ESCUELA INTERNACIONAL DE DOCTORADO
Programa de Doctorado en Ciencias del Deporte

Heart rate variability in acute resistance training: monitoring recovery.

Author:
Sajith Udayanga Marasingha Arachchige

Directors:
Dr. Linda Haiwon Chung
Dr. Pedro Emilio Alcaraz Ramón

Murcia, October of 2021

ESCUELA INTERNACIONAL DE DOCTORADO
Programa de Doctorado en Ciencias del Deporte

Heart rate variability in acute resistance training: monitoring recovery.

Author:
Sajith Udayanga Marasingha Arachchige

Directors:
Dr. Linda Haiwon Chung
Dr. Pedro Emilio Alcaraz Ramón

Murcia, October of 2021

UCAM

UNIVERSIDAD CATÓLICA DE MURCIA

AUTORIZATION OF THE DIRECTORS OF THE THESIS FOR SUBMISSION

Dr. Linda Haiwon Chung and Dr. Pedro Emilio Alcaraz Ramón as Directors of the Doctoral Thesis titled "Heart rate variability in acute resistance training: monitoring recovery" carried out by Sajith Udayanga Marasingha Arachchige in the Doctoral Program in Sports Sciences, **authorize for submission** since it has the conditions necessary for its defence.

Sign to comply with the Royal Decrees 99/2011, 1393 / 2007, 56 / 2005 and 778/98 in Murcia, July 28th, 2021.

ALCARAZ RAMON PEDRO EMILIO - 23014777B

Firmado digitalmente por ALCARAZ RAMON PEDRO EMILIO - 23014777B
Fecha: 2021.07.30 10:10:26 +02'00'

LINDA HAIWON| CHUNG

Firmado digitalmente por LINDA HAIWON|CHUNG
Nombre de reconocimiento (DN): cn=LINDA HAIWON|CHUNG, serialNumber=Y1463302X, givenName=LINDA HAIWON, sn=CHUNG, ou=CIUDADANOS, o=ACCV, c=ES
Fecha: 2021.07.29 10:01:40 +02'00'

DEDICATION

මා

වෙනුවෙන්

සියලුම දේ කැප කල

මාගේ

ආදරණීය

අම්මා සහ තාත්තා

වෙනුවෙන්

නමස්කාර පුර්වකව

පුදමී...!!!

ACKNOWLEDGEMENTS

In life, the journey towards success is a challenging one. Even more so when one embarks it alone. However, I was privileged during my personal journey to be a part of a brilliant team of collective minds. I highly doubt my doctoral thesis would have seen the light of day if not for the support, guidance and encouragement I received from them throughout my journey. I want to sincerely acknowledge each and every one of them.

First and foremost, I am extremely grateful to my supervisors, Dr. Linda H. Chung and Dr. Pedro E. Alcaraz, for their invaluable advice, continuous support, and patience during my PhD study. Thank you for all the lessons and for always pushing me to achieve the highest standards possible. Furthermore, encouraging me every single day with a smile, specifically when things got hard and I was struggling, and being my compass in the middle of the sea. I'm eternally grateful for everything you've done. Thank you so much.

Secondly, I would like to thank Dr. Jacobo A. Rubio-Arias, for your guidance, help and collaboration in Study 1's published article, included in the present thesis. I learnt a lot from you. I truly appreciate everything you have done.

To my colleagues and friends in the Research Centre for High Performance - UCAM. To Dr. Tomás T. Freitas, Dr. Cristian Marín Pagán, Dr. Elena Marín Cascales, Dr. Jorge Carlos Vivas, Antonio Martínez Serrano, Florencia Crystel, Javier Martínez Noguera and Laura Campoy de Haro, thank you for your guidance and unconditional help during these last few years. You all have made my life easier and kept a smile on my face during this journey. Thank you for all the great moments spent in the lab and in Madrid.

Thank you, Ms. Ana Baella Negrillo and all the members of Vice-rector's Office for Research, the UCAM International Doctoral School and all of the participants who humbly offered their valuable time and effort to help my research studies.

I wish to express my sincere gratitude to Isabel María and her mother. I do not have enough words to express my gratitude for making my life easier in Spain and for all the support during this challenging process. Moreover, to Pepe, Amalia, Virginia, Ivan, Encarni and María José for all the great moments.

To all my friends back home in Sri Lanka. Thank you for proving that no distance can weaken our true friendship. Specifically, Mevan Gunathilake and Devmith Athulathmudali for all the support and encouragement in this ride.

To my dear family, my parents, Asilin and Gunarathne. Thank you so much for your unconditional support, encouragement and guidance in all of the stages in my life. For the sacrifice you make every day to give me the opportunity to fulfil my life goals and for never holding me back in the pursuit of my dreams. No words will ever be enough to describe how fortunate I feel to have you in my life. බොහොම පින් මාගේ ආදරණීය අම්මේ සහ තාත්තේ.....මම ගොඩක් පින් කරලා තියෙනවා ඔබේ පුතෙකු ලෙස ඉපදෙන්නට.....!!! To my brother, Sandun, and sister, Hashima. Thank you for all of your help and support throughout my life. I love you all…!!!

Last, but by no means least, thank you so much for my body and my laptop. I know I gave you a lot of hard times and that I did not take care of you well. But, you too are stronger than I thought. You never gave up on me and kept it together when things did not go in the right direction. Fighting with me to the end….

To all. Thank you…!!!

" If I have seen further than others, it is by standing upon the shoulders of giants."

Isaac Newton

From this thesis, Study 1 has been published in a peer-reviewed journal and several abstracts have been submitted to national or international conferences, where oral communications and posters were presented. Below lists the references of the dissemination of the work:

- Marasingha-Arachchige SU, Rubio-Arias JÁ, Alcaraz PE, Chung LH. Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis. Journal of Sport and Health Science. 2020. DOI: 10.1016/j.jshs.2020.11.008
- Marasingha-Arachchige SU, Alcaraz PE, Chung LH. Comparing the root mean square of successive differences (RMSSD) recovery between strength and power training following a modified BEAST fatigue protocol. VI Jornadas de Investigación y Doctorado: ODS con Ciencia (2020). Universidad Católica San Antonio de Murcia, Spain.
- Marasingha-Arachchige SU, Rubio-Arias JÁ, Alcaraz PE, Chung LH. Acute Effect of Resistance Exercises on Heart Rate Variability: A Meta-Analysis. International Sport Forum on Strength, Training and Nutrition (2019), International Sport Forum of the Strength & Conditioning Society (SCS) and the European Sport Nutrition Society (ESNS), Spain.
- Marasingha-Arachchige SU, Alcaraz PE, Chung LH. Effect of Acute Resistance Exercise on Heart Rate Variability: A Systematic Review. IV Jornadas de Investigación y Doctorado: Women in Science (2018). Universidad Católica San Antonio de Murcia, Spain.

INDEX

ABSTRACT

A sufficient level of training stimulus and adequate recovery from a training stress facilitates the improvement of sports performance and reduction of injuries. However, proper training load manipulation and adequate optimal recovery from such training stress are essential components to achieve an individual's potential, as well as to evade negative consequences of excessive fatigue. Among the many monitoring tools, heart rate variability is gaining attention in the scientific community and coaches to evaluate the athlete's training load, post-exercise fatigue or recovery level in the field. This doctoral thesis aimed to: 1) study the acute effect of resistance exercise on the heart rate variability parameters and the possible moderating factors contributing to cardiac autonomic activity during post-exercise recovery, 2) examine the changes and recovery of heart rate variability parameters induced by different resistance training modalities (strength and power) and training loads (100%, 75%, 50%), following intensive fatigue conditions within the micro training cycle and 3) identify the optimal training loads, based on heart rate variability parameters, that are needed to maintain adequate recovery within the micro-training cycle in strength and power training modalities.

To achieve these aims, the doctoral thesis was divided into two primary studies: 1) a systematic review and meta-analysis and 2) an experimental study that was based on some of the findings of the systematic review with meta-analysis and designed to answer aims 2 and 3. The experimental study was randomised, cross-over design study that lasted for eight weeks and consisted of six trials (Block 1 strength training modality and Block 2 power training modality; each block containing 3 different training loads). Forty-eight hours prior to all trials, participants performed a 45-minute modified BEAST fatigue protocol. In the subsequent visit (post-48H modified BEAST), participants performed one of 3 training loads (100% (4 sets), 75% (3 sets) or 50% (2 sets)), which consisted of half

squat, bench press and hip thrust exercises (Strength: 5-reps, 90% one-repetition maximum, 4-minutes rest between sets; Power: 5-reps, Optimal load, 3-minutes rest between sets). Heart rate variability parameters were recorded and analysed before and after the fatigue protocol and the resistance training sessions, as well as at post-6 hours, -24 hours, and -48 hours.

The main findings from Study 1 showed a decrease in overall autonomic modulation, withdrawal of cardiac parasympathetic modulation and activation of cardiac sympathetic modulation following an acute resistance exercise session (after around 30 minutes). Moreover, training volume demonstrated a greater effect on the withdrawal of cardiac parasympathetic modulation and the activation of cardiac sympathetic modulation in healthy individuals. Additionally, the number of sets, the intensity of exercise, and the rest between sets can be considered a moderating factor on heart rate variability parameters. The main findings from Study 2 demonstrated that strength training modality and higher training loads disturbed the cardiac autonomic modulation more so than the power training modality and lower training loads. In addition, cardiac autonomic modulation recovered sooner following the power training modality and lower training loads compared to the strength training modality and higher training loads. Lastly, based on the natural log of root mean square of the successive differences parameter (Ln RMSSD), 75% of strength training load and 100% of power training load may be considered the optimal training load to achieve adequate recovery within the microcycle when athletes are under the influence of fatigue from previous training sessions.

Keywords: fatigue, sympathetic, parasympathetic, cardiac autonomic modulation, training load manipulation, optimal recovery, strength training, power training, training stress, HRV, RMSSD, microcycle stress management

RESUMEN

Un nivel suficiente de estímulo de entrenamiento y una adecuada recuperación tras el estrés producido por este facilita la mejora del rendimiento deportivo y la reducción de las lesiones. Sin embargo, la manipulación eficiente de la carga de entrenamiento y de los procesos de recuperación son componentes esenciales para alcanzar el potencial de un individuo, así como para evitar las consecuencias negativas de la fatiga excesiva. Entre las muchas herramientas de monitorización, la variabilidad de la frecuencia cardíaca está ganando atención en la comunidad científica y en los entrenadores para evaluar la carga de entrenamiento del deportista, la fatiga post-ejercicio o el nivel de recuperación en el campo. Esta tesis doctoral tenía como objetivo: 1) estudiar el efecto agudo del ejercicio de fuerza sobre los parámetros de variabilidad de la frecuencia cardíaca y los posibles factores moderadores que contribuyen a la actividad autonómica cardíaca durante la recuperación post-ejercicio, 2) examinar los cambios y la recuperación de los parámetros de variabilidad de la frecuencia cardíaca inducidos por diferentes modalidades de entrenamiento de fuerza (fuerza y potencia) y cargas de entrenamiento (100%, 75%, 50%), tras condiciones de fatiga intensa dentro del microciclo de entrenamiento e 3) identificar las cargas de entrenamiento óptimas, en función de los parámetros de variabilidad de la frecuencia cardíaca que permitan mantener una recuperación adecuada dentro del microciclo de entrenamiento en las modalidades de entrenamiento de fuerza y potencia.

Para alcanzar estos objetivos, la tesis doctoral se dividió en dos estudios principales: 1) una revisión sistemática y meta-análisis y 2) un estudio experimental basado en los hallazgos de la revisión sistemática con meta-análisis, diseñado para responder a los objetivos 2 y 3. El estudio experimental fue un estudio aleatorizado con diseño cruzado que duró ocho semanas y consistió en seis ensayos (bloque 1 de la modalidad de entrenamiento de fuerza y bloque 2 de la modalidad de entrenamiento de potencia; cada bloque contenía 3 cargas de entrenamiento

diferentes). Cuarenta y ocho horas antes de todos los ensayos, los participantes realizaron un protocolo de fatiga BEAST modificado de 45 minutos. En la visita posterior (post-48h), participantes realizaron uno de los 3 cargas de entrenamiento (100% (4 series), 75% (3 series) o 50% (2 series)), que consistía en ejercicios de media sentadilla, press de banca y empuje de cadera (Fuerza: 5 repeticiones, 90% de una repetición máxima, 4 minutos de descanso entre series; Potencia: 5 repeticiones, carga de entrenamiento óptima, 3 minutos de descanso entre series). Se registraron y analizaron los parámetros de variabilidad de la frecuencia cardíaca antes y después del protocolo de fatiga y de las sesiones de entrenamiento de fuerza, así como después de 6, 24 y 48 horas.

Los principales resultados del Estudio 1 mostraron una disminución de la modulación autonómica general, la retirada de la modulación parasimpática cardíaca y la activación de la modulación simpática cardíaca tras una sesión de ejercicio de fuerza agudo (después de unos 30 minutos). Además, el volumen de entrenamiento demostró tener un mayor efecto sobre la retirada de la modulación parasimpática cardíaca y la activación de la modulación simpática cardíaca en individuos sanos. Además, el número de series, la intensidad del ejercicio y el descanso entre series pueden considerarse un factor moderador de los parámetros de variabilidad de la frecuencia cardíaca. Los principales resultados del Estudio 2 demostraron que la modalidad de entrenamiento de fuerza y las cargas de entrenamiento más elevadas alteraron la modulación autonómica cardíaca en mayor medida que la modalidad de entrenamiento de potencia y las cargas de entrenamiento más bajas. Además, la modulación autonómica cardíaca se recuperó antes tras la modalidad de entrenamiento de potencia y las cargas de entrenamiento más bajas en comparación con la modalidad de entrenamiento de fuerza y las cargas de entrenamiento más altas. Por último, basándose en el logaritmo natural del cuadrado medio del parámetro de diferencias sucesivas (RMSSD), el 75% de la carga de entrenamiento de fuerza y el 100% de la carga de entrenamiento de potencia podrían considerarse como la carga de entrenamiento

óptima para lograr una recuperación adecuada dentro del microciclo cuando los atletas se encuentran bajo los efectos de la fatiga producida por las anteriores sesiones de entrenamiento.

Palabras clave: fatiga, simpático, parasimpático, modulación autonómica cardíaca, manipulación de la carga de entrenamiento, recuperación óptima, entrenamiento de fuerza, entrenamiento de potencia, estrés del entrenamiento, VFC, RMSSD, gestión del estrés del microciclo

ABBREVIATIONS

The abbreviations of the units from the International System Units are not included in the following list as there are internationally accepted standards for their use.

ANS	Autonomic Nervous System
ApEN	Approximate Entropy
ARE	Acute Resistance Exercise
BMI	Body Mass Index
BP	Bench press
CAR	Central activation ratio
CK	Creatine kinase
CMJ	Countermovement jump
CRP	C-reactive protein
DOMS	Delayed onset of muscle soreness
EMG	Electromyography
Gln	Glutamine
Glu	Glutamate
HF	High-frequency power
HR	Heart rate
HRV	Heart rate variability
LF	Low-frequency power
Ln	Natural logarithm
MTDS	Multicomponent Training Distress Scale
MVC	Maximal Voluntary Isometric Contraction
NN	Normal-to-Normal
NSCA	National Strength and Conditioning Association

nu	Normalized values
pNN50	Proportion of interval differences of successive NN intervals lasting more than 50 ms
PNS	Parasympathetic Nervous System
POMS	Profile of Mood States
RBP	Resting blood pressure
RESTQ-Sport	Recovery-stress questionnaire for athletes
RFD	Rate of force development
RFR	Rate of force relaxation
RMSSD	Root Mean Square of Successive Differences
RP	Repetition maximum
RPE	Rating of Perceived Exertion
RPP	Relative peak power
RT	Resistance training
SampEn	Sample entropy
SD1	Poincaré plot standard deviation perpendicular the line of identity
SD2	Poincaré plot standard deviation along the line of identity
SDNN	Standard deviation of all NN time intervals
SNS	Sympathetic Nervous System
SS	Stress Score Index
TP	Total power
TQR	Total Quality Recovery
VLF	Very low frequency

LIST OF FIGURES

LIST OF TABLES

LIST OF APPENDIXES

I - INTRODUCTION

I. INTRODUCTION

Resistance training (RT) plays an essential role in professional player's training programmes and physically active individuals targeting to improve variety of their physical qualities (muscle strength, power, hypertrophy, local muscular endurance, balance and coordination) and body composition (bone mass, body fat and muscle mass) to better their performance and overall health (1-5). According to the National Strength and Conditioning Association (NSCA) explanation, RT entails a wide range of resistive loads and a variety of training modalities to optimise the effects of training and improve sports performance and overall health (6). Among these physical qualities, muscle strength and power play a crucial part in most sports performances. Muscle strength could be the basis of many other physical qualities (4, 7, 8), which used to sports performance and physically active life. Maximal power production is one of the most important physical quality, frequently used during sports performance. It is important to mention that most sports-related skills must be performed quickly and generate greater force with a shorter time period (9).

Sufficient level of training stimulus required for physiological adaptation and challenge the physiological capacity. If not, insufficient training might reduce the athlete's physiological adaptation and reduce performance or detraining (10). Another vital factor in improving the performance and reducing the injuries was proper recovery from the training stress. Recovery is an umbrella term used to describe the restorative process of physiological and psychological tiredness (fatigue) due to physical and mental effort (11, 12). Concerning the physiological point of view of sports and exercise context, recovery is mainly referred to as regeneration follows physical fatigue induced by training (11, 13). If players cannot get adequate recovery from the training stimulus, the athletes' body may

continuously expose to training-induced fatigue and lead to non-functional overreaching or overtraining, which might end up with fatigue syndrome (1, 11, 14).

Even though appropriate training stimulus and recovery levels, athletes can improve their performance and reduce the possibility of injuries, To achieve their real potential, proper training load manipulation and adequate optimal recovery of such training stress are essential (15). Morden commercial-based setting of the sports requires athletes to perform in several competitions quickly and participate in an intense and regular training session. Similarly, in a highly competitive environment, most efficient training programs might help keep an athlete ahead of other players. Identifying the optimal training load and optimal recovery time are few of the significant objectives of sports coaches and fitness professionals. It allows more time for improving an athlete's performance (i.e., more training sessions, better training adaptations and less risk of injuries). These circumstances demanded to closely monitor the athletes' fatigue level and adjust the training stimulus according to the recovery status to achieve optimal training adaptation.

There are many metabolic, performance and hormonal monitoring tools used to evaluate the athletes training load, post-exercise fatigue or recovery level by the trainers. Among them, objective tests like percentage of maximal oxygen uptake (VO_2max) test (12), jump tests (16-18), creatine kinase (CK) (19, 20), C-reactive protein (CRP) (19), cortisol (13, 21), free-testosterone (13, 19), blood lactate concentration (12), glutamine (Gln) (19), glutamate (Glu) (19) and subjective tests like recovery-stress questionnaire for athletes test (RESTQ-Sport) scores (13, 22, 23), total quality recovery (TQR) (24), rating of perceived exertion (RPE) (13, 25), delayed onset of muscle soreness (DOMS) (10) and profile of mood states questionnaire (POMS) scores (13, 26) can see in the lab environment and sports filed.

Most of the objective post-exercise recovery status or fatigue monitoring methods or tests are invasive, time-consuming and need specialised knowledge to

collect data and analyse. Most importantly, unable to use easily in the field and expensive for everyday use. On the other hand, subjective tests accuracy depends on the particular subject's psychological situation. So, it is important to use a physiological marker with the minimum interference to the training and recovery process. With the importance of a non-invasive, comfortable, affordable and field use friendly testing method, with the rapidly growing of technology, heart rate variability (HRV) is being increasingly attracting the scientific communities attention to used as a monitoring tool to evaluate the training load, training adaptation, status of fatigue or level of recovery (13, 27-31).

HRV is the physiological variation in the time interval between heart beats (32), which provides essential information of the autonomic nervous system's (ANS) cardiac sympathetic and cardiac parasympathetic nervous systems (1, 13, 28, 33). These cardiac sympathetic and parasympathetic modulation changes can be monitored by examining HRV parameters (34-38). Previous studies have shown that acute bout of resistance exercise (ARE) affects cardiac sympathetic modulation and cardiac parasympathetic modulation (34, 39, 40). However, some inconsistencies in the literature show the opposite effect on HRV parameters following an ARE session (40-43). Moreover, it is unclear what the magnitude of the ARE has on HRV parameters. Additionally, to our knowledge, no study has examined (i.e., meta-analyses) the possible moderating factors of ARE that affect HRV parameters. Therefore, the first stage of this doctoral study consisted of a systematic review and meta-analysis to understand how an ARE session affects the HRV characteristics and identify the possible moderating factors contributing to cardiac autonomic activity during post-exercise recovery.

After identifying the effect of ARE session on the HRV parameters and possible moderating factors, the second stage was to examine RT manipulation after a high-fatiguing session and determine the recovery within the microcycle. Large individual variation can see in training adaptation after utilised standardised training program (44). Some individuals show greater adaptation and

improvement in performance following a standardised training program, while others do not show a high degree of improvement, decrement in performance or no changes in performance (44, 45). For proper training manipulation, training load should be prescribed individually, specifically paying attention to the responses of daily variations in the training load after an intense training-induced fatigue level of the athlete, in order to facilitate proper recovery (14, 46, 47) and adaptation. The individualised training program would help achieve more significant improvements and a smaller variation in training adaptation. However, identifying optimal training load under the fatigued condition based on HRV parameters was not widely examined. Therefore, this doctoral study's second phase was conducted as an experimental study to evaluate and compare the changes and recovery of HRV parameters and other objective and subjective responses induced by strength training and power training modalities and different training loads, following a prior intensive fatigue session within the micro training cycle. Furthermore, identify the optimal training loads related to strength and power training modalities following a high-fatiguing session to maintain the proper recovery status within the microcycle.

II – LITERATURE REVIEW

II. LITERATURE REVIEW

The purpose of this chapter is to provide a comprehensive background in the areas that support the studies presented in this doctoral thesis. The chapter is split into ten sections, which will cover RT, importance of periodization, monitoring training load, manipulation of training load, fatigue and recovery, monitoring fatigue and recovery status, the ANS, physiology behind the effects of exercise training to ANS, HRV and acute effect of resistance exercise on HRV parameters.

2.1. RESISTANCE TRAINING

RT began its popularity in the 1970s and has long been a well-recognized form of exercise to improve physical fitness, health and athletic performance (48-51). It is also considered an important part of a complete exercise program (52). The skeletal muscles can be trained dynamically and or isometrically (53). Concentric and/or eccentric contractions changes muscle length (shortening and lengthening) and the tension produced by the muscles is considered dynamic RT, while isometric RT is described as a continuous contraction against a fixed load or resistance with no change in muscle length (50, 53). The primary goal of RT is to develop muscle fitness using a muscle or group of muscles against external resistance like bodyweight, free weight, weight training machines etc. (50, 53, 54). It is important to mention that in this context, muscular fitness is an umbrella term for muscular strength, muscular power, and local muscular endurance (55).

Previous studies demonstrate that RT has the ability to increase physical muscle qualities, like strength, power, hypertrophy, local muscle endurance, balance, and coordination (4, 56). Among these physical qualities, muscle strength and muscle power are considered the most essential qualities to improve sports performance, since most competition requires greater force application within a

limited time frame (9). When comparing these two qualities, muscle strength plays a central role in the eyes of fitness trainers and sports science researchers because strength is considered the basis for other physical qualities and several sports-related skills (4, 8). It is also notably associated to decrease the risk of injuries (4). Furthermore, there is a fundamental relationship between strength and power where individuals with higher strength levels produce higher power output compared to lower strength level of individuals (8, 57). Moreover, several studies have revealed that strength training improves both maximal strength and maximal power production (57, 58). This makes sense since power is defined by the product of force and velocity (59).

However, it's important to keep in mind that strength and conditioning practitioners use considerably different strength and power training programs that vary in number of sets, reps, percentage of 1RM, rest between sets, etc (1). One of the main differences between these two training programs is the lifting velocity of the exercise. The force-velocity curve (60) illustrates that greater generation of concentric muscular force corresponds with slower muscle shortening and corresponding movement velocity. On the other hand, higher the muscle shortening and corresponding movement velocity coincides with lower concentric muscular force generation.

Muscle strength is defined by the ability to exert force on an external object or resistance (4, 7, 61, 62). Thus, maximum strength is produced under low velocities, and maximum speed is produced under low loads. Power output is defined as the time rate of doing work (1) and is calculated as the product of force (strength) and velocity (speed) (59). Among athletes and trainers, power is loosely considered as "explosive strength" (1). However, according to the power equation (8, 63), where force is multiplied by velocity, the maximum power output is generated using the optimal load (i.e., ideal balance between force and velocity) (8, 64). The optimal load is defined as the load that generates peak power output in a

given exercise (60). Figure 1 summarizes the force-velocity, force-power and velocity-power curves, as well as the optimal load relationship.

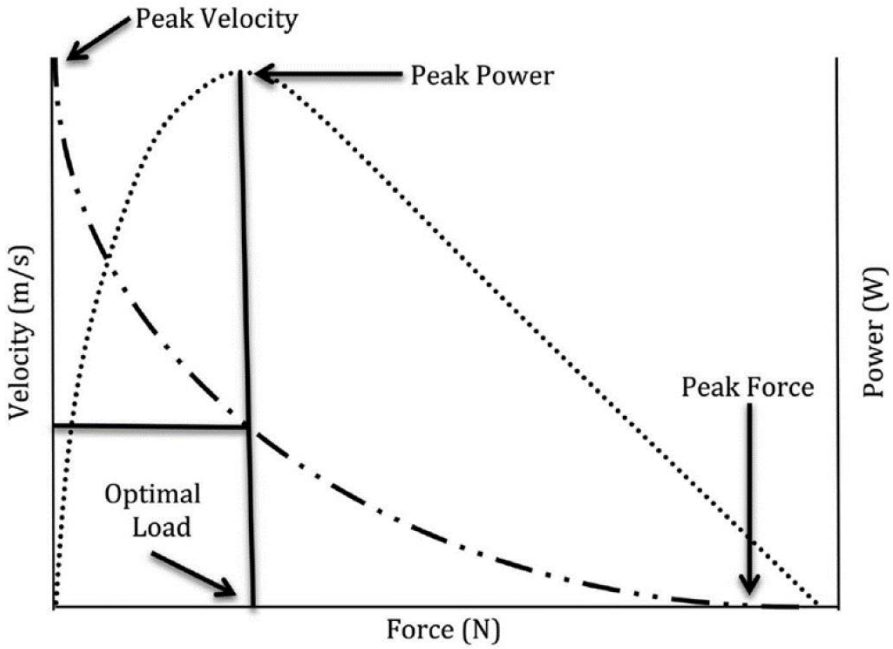


Figure 1. Force-velocity, force-power, velocity-power, and optimal load relationship. Source: Haff *et al.* (60)

The NSCA (1) and previous studies (1, 50) have provided recommendations for strength training protocols, where ≥85% of 1RM, ≤6 repetitions, 2-6 sets, 2-5 minutes rest is performed in a single training session. For maximal power training, power exercises use moderate resistances compared to strength exercises, and the former requires acceleration during the full range of movement with high lifting velocities. The NSCA (1) and previous studies (1, 64-66) have also provided power training protocol recommendations, which are 75-85 % of 1RM or optimal load, 3-5 repetitions, 3-5 sets, 2-5 minutes rest in a single training session. When using optimal load for power exercise, the load must be individualized and calculated separately for each exercise used in power training (8, 64, 66).

The development of muscle strength and maximal power depends on a combination of several morphological and neurological adaptations (67-69) apart

from training status, training age, initial strength level and genetics of the athlete (68, 70). These adaptations are: (i) increase in the muscle's anatomical cross-sectional area (CSA; muscle hypertrophy) and change in muscle architecture, which increase the number of cross-bridge interactions between actin and myosin within the previously and newly generated sarcomeres (67, 71-73), (ii) increase in musculotendinous stiffness (ability to spring back), which enhances force transmission because of the increased tissue stiffness (67, 74), (iii) increase in motor unit discharge rate (firing frequency) and decrease in the recruitment-threshold force of motor units (67, 73, 75), (iv) increase in motor unit synchronization (67, 76) and iv) decrease in inhibition on neuromuscular activation (73, 76). These morphological and neurological changes interplay and together contribute in the improvement of muscle strength and maximal power production.

It is important to note that a successful training program gives the personal trainer and coach an opportunity to manage the training adaptations and recovery responses in a structured and controlled manner. In order to achieve the main training objective, strength and conditioning coaches design training programs with different time durations to target short-term and long-term goals. This planning process is referred to as periodization (1).

2.2. IMPORTANCE OF PERIODIZATION

Previous studies showed that periodized RT increases maximal strength and power than non-periodized RT (48, 77). Periodization is the logical integration and sequencing of specific training factors by manipulating training variables over interdependent training periods to optimize specific physical, psychological, and performance outcomes at predetermined time points (78, 79). Depending on the athlete's needs, the training plan can range from a year to several years. For example, a school athlete's training plan may be designed for the National School Games that occur at the end of the year, and an Olympic athlete's training plan spans over four years. Although the athlete pursues a key goal over a number of

years, the training plan is usually divided into more detailed, one-year training plans. The annual training plan consists of single or multiple macrocycles, which depends on the number of competitive seasons in a year. It is divided into different training periods, such as preparation, competitive and transition periods (1, 73). Macrocycles can last from a few months to a year, and some authors refer to the annual training plan as the macrocycle. One macrocycle is made up of a collection of several mesocycles, where a mesocycle can last from a few weeks to several months and is usually limited to 2 weeks to 6 weeks. Each mesocycle can be divided into microcycles that can range from a few days to weeks. At the bottom of periodization hierarchy, a microcycle is divided into several training days and is commonly consider as seven days (1). Each training session can last up to several hours (1, 78, 80).

Regarding periodization, the microcycle is considered the most important period because daily training interventions are the foundation of the whole training plan (73). These periods are primarily used to modulate training stress in line with training objectives by manipulating training volume-load across each microcycle to stimulate adaptation and give recovery time (78). The training that takes place in a microcycle is decided solely by the objectives set out in the mesocycle, macrocycle and, ultimately, the annual training plan (1, 78, 80, 81).

Integrating a comprehensive monitoring and testing program in the periodization program allows coaches to track the development of athlete performance and health safety (73). It is important to guide the periodization process using the data collected from the athlete throughout the training program. Specifically, data should be gathered using various monitoring tools to evaluate training stress, training-induced fatigue level, recovery status and the adaptation to the training stimulus to understand the response to the training program day-to-day. Information provided by constant monitoring can be used to modify the original training program via manipulation of the training loads within the training session based on the fatigue/recovery status of the athlete. Thus, fatigue

management and long-term training goal can be achieved with adequate and appropriate periodization.

2.3. MONITORING TRAINING LOAD

Monitoring the athlete's training load gives coaches an understanding of the effectiveness of the training program and the individual adaptation/responses of the athletes. Training load can be divided into two parts: internal and external training load (82-84). The internal training load is defined as the relative physiological and psychological stress imposed on the athlete during the training, and the external training load is described as the work performed by the athlete (12, 82).

Specifically, internal training load refers to the impact of the work performed by the athlete (82). It is measured using objective tools (e.g., heart rate (HR) recovery, HRV, oxygen consumption, blood lactate concentration, training impulse (TRIMP)) and subjective tools (e.g., Borg's RPE, psychological inventories (POMS, RESTQ- Sport)) (12, 82). External training load implies the combination of work performed by the athlete (82). It was commonly calculated using the training volume and training intensity of the RT program. The total amount of work performed during a training session is considered the training volume (1, 50). The volume is calculated by the number of sets multiplied by the number of repetitions per set (1, 85). Repetition is defined as the complete execution of a specific movement technique, and a set is considered as a group of repetitions (1). Alternatively, the training load is referred to the amount of weight assigned to an exercise (1). The load is commonly prescribed as either a percentage of the 1-repetition maximum (1RM), percentage of body mass (1, 64) or based on barbell velocity (86). Trainers commonly use volume-load, which is defined as the density of volume performed at the prescribed intensities, and it can be calculated as the total number of sets × the number of repetitions per set × the weight lifted per repetition (1).

It is important to monitor both internal and external training loads, as they provide valuable information regarding the athlete's training load and fatigue level. Because external load quantifies how much work an athlete performs, and internal load presents information on how that training load affects the athlete. However, it is important to understand that the effect of the training load (internal load) is more vital than how much work has been done by the athlete. This is because when an athlete repeats the same training protocol over a few days, the effect on the internal load may be quite different, although the external training load is unchanged. The impact on the training load may depend on several physiological and psychological factors, like fatigue/recovery status, emotional status, training age, nutrition level, disease, etc. Among these factors, the athlete's current fatigue/recovery status plays a vital role in training adaptation, particularly during the microcycle.

However, in recent years, competitions occur more frequently and thus poses a challenge to sports coaches and fitness trainers to find the right balance between optimal recovery time and efficient training protocols to promote continual improvements, without incurring negative sequelae of overtraining, in the athlete's performance during the competitive season. Therefore, optimizing training sessions by manipulating the variables of a training program based on fatigue and recovery status may be the solution to manage the training- and competition-induced fatigue and recovery of the athletes in order to attain optimal performance.

2.4. MANIPULATION OF TRAINING LOAD

It is well established that physiological adaptation requires an adequate exercise stimulus and proper recovery duration. With greater training stress, there is an elevated accumulation of fatigue in the body, and therefore, there is the need for longer recovery to complete training adaptations. Although recovery is an essential part of training adaptation, completing the recovery process is not required to engage in a new training bout (87). Manipulation of the variables of

training load within the session and microcycle can be used to manage the fatigue level, thus enabling the recovery process and avoiding overtraining (88, 89).

In an acute RT session, choice of exercises, exercise intensity, loading form, training to failure, speed of contraction, psychological factors, number of sets per exercise, number of repetitions per set, rest between sets and exercise order are considered as the training variables (90). Choice of the exercises depends on the sport or target muscle group. Intensity represents the amount of resistance (load) used during the exercise. Scientific literature and recognized professional organizations, like NSCA, have divided training intensity into three levels: high (>85 % 1RM), moderate (65–85 % 1RM), and low (<65 % 1RM), and also introduced specific weight ranges aimed at improving certain muscle performance characteristics (strength: ≥85 % 1RM, power: 75-85 % 1RM or optimal load, hypertrophy: 67-85 % 1RM, muscular endurance: ≤67 % 1RM) (1, 50) based on literature findings. It is important to remember that there is an inverse relationship between training intensity and volume, where increases in training intensity requires lower number of repetitions to be performed (91). Volume can be manipulated by changing the number of repetitions performed in a set or the number of sets performed during an exercise. Similar to training intensity, the scientific literature and recognized professional organizations, like NSCA, provided sets and repetitions guidance based on training goals:

- Strength: repetitions are ≤6 and sets 2-6.
- Power: repetitions are 3-5 and sets 3-5.
- Hypertrophy: repetitions are 6-12 and sets 3-6.
- Muscular endurance: repetitions – ≥12 sets – 2-3) (1, 64-66).

Rest duration between sets and exercises highly depend on the training goal, intensity, and the physical condition of the athlete. The recommended resting periods based on training goals between sets and exercise were: strength 2-5 min, power 2-5 min, hypertrophy 30 sec to 1.5 min and muscular endurance ≤30 sec.

However, depending on the intensity and the condition of the athlete, the athlete might need a longer period of rest between sets to complete the training program safely and with proper technique (1, 50, 92). Even though there are many ways to organize the exercise order in a training session, one must consider how one exercise affects the technique or the quality of effort of the next exercise (1), especially when performing two exercises that require similar muscles (i.e., the former exercise could decrease in the quality of the latter exercise (90)). Therefore, all of the aforementioned variables can be manipulated to adjust the training load based on the fatigue and recovery status of the athlete and the training goals.

2.5. FATIGUE AND RECOVERY

Intense training often leads to a disruption in the physiological systems of the body (93). After the body experiences new or more intense training stress, the body's initial response is a decrease in performance capacity due to fatigue, muscle soreness, stiffness and or a decrease in energetic stores (1, 79). The magnitude of the stress that the athlete is experiencing can last for hours (acute), days (residual) or weeks (chronic) (1) and can depend on several factors, such as the type of muscle contraction (isometric, isotonic, concentric, eccentric and intermittent or continual), duration or volume, velocity, frequency, intensity of exercise and type of muscles used for the exercise (12, 94-96).

There are numerous definitions of fatigue, but the most common is "an exercise-induced reduction in the ability to exert muscle force or power" (97). In other words, the "failure to maintain the required or expected force or power output" (98) and "an inability to complete a task that was once achievable within a recent time frame" (99). Fatigue is a complex and multifaceted phenomenon that has a variety of mechanisms that affect the central nervous system (central fatigue) and the skeletal muscle (peripheral fatigue) (12, 97). Central fatigue is related to the reduction of muscle force production during volitional contractions as a result of a decrease in neural drive that originates from the motor cortex of the brain (i.e.,

decreased motoneuron firing frequency and/or a number of functioning motor units) (100). Peripheral fatigue is associated with impaired muscle contractile activity, leading to loss of muscle fibre force (100). This phenomenon is thought to be caused by impaired neuromuscular transmission, impaired excitation-contraction coupling or failure of muscle action potentials (101). Peripheral fatigue is also associated with alterations in calcium (Ca^{2+}) concentrations. The extracellular Ca^{2+} concentration is fundamental in the forming of cross-bridges. The binding of Ca^{2+} to troponin displaces tropomyosin away from the myosin-binding site on actin, thus allowing cross-bridge formation. Therefore, a decrease in Ca^{2+} release from the sarcoplasmic reticulum and lack of reabsorption to the sarcoplasmic reticulum can contribute to peripheral fatigue (102-104).

Post-exercise recovery is one of the essential factors in the training adaptation cycle. Recovery is defined as the "ability to meet or exceed performance in a particular activity" (105). During the recovery period, the body restores homeostasis to maintain internal conditions stable and relatively constant to perform normal functional capacity (1, 93). Meanwhile, if the training stress/stimulus is appropriate, the body is also performing biochemical, structural, and mechanical adjustments to increase resistance to the training stress (adaptation) (1, 93). Thus, proper recovery from such training stress is necessary because the body may be exposed to continuous training-induced fatigue, which could lead to non-functional overreaching or overtraining and, ultimately, to overtraining syndrome (1, 14).

A prolonged maladaptation can be referred to as overtraining syndrome, also known as burnout and can last for six months or more. Decreased performance, high fatigue, severe loss of energy, reduced appetite, disturbed sleep patterns and hormonal disturbances are some of the signs and symptoms of overtraining syndrome (1, 106, 107). Two types of overtraining syndromes are proposed by the scientific literature and are sympathetic and parasympathetic overtraining syndrome (1, 106, 108). The sympathetic overtraining syndrome refers to an

increase in the cardiac sympathetic modulation at rest, and the parasympathetic overtraining syndrome involves an increase in cardiac parasympathetic modulation at rest and during exercise (1, 108). Sympathetic overtraining syndrome develops before parasympathetic overtraining syndrome, and in this stage, chronic suppression of physiological systems in the body (1, 106). Physiological markers associated with sympathetic overtraining syndromes include an increase in resting and exercise HR, hypertension, insomnia (sleep disorder), restlessness, elevated basal metabolic rate, negative nitrogen balance and Electrocardiogram (ECG) abnormalities (21, 106, 109). Physiological markers of parasympathetic overtraining syndrome includes low resting (bradycardia) and relatively low exercise HR, progressive anaemia, low blood pressure and digestive disturbances (21, 106, 109). Unfortunately, an athlete's career may have grave consequences if these are not treated. Therefore, athletes need the opportunity for adequate recovery from the training stress and constant monitoring of fatigue and recovery status can help avoid the negative outcomes of non-functional overreaching and overtraining.

2.6. MONITORING FATIGUE AND RECOVERY STATUS

Among the factors that contribute to the success and failure of a sport, fatigue and recovery status play a major role. Proper monitoring of these factors and making training decisions based on the athlete's status may increase the effectiveness of training adaptation and reduce the risk of injuries. When considering fatigue levels and recovery status, it is important to consider both physiological and psychological fatigue and recovery (23). The physiological and psychological impact of a training stimulus, as well as the time it takes to recover from that effect, varies from athlete to athlete because those processes depend on several internal (age, training age, genetics) and external (nutrition, training stress, hydration) factors related to the individual (13). Therefore, monitoring fatigue

levels and recovery status is extremely important at every stage of the training process.

Physiological fatigue and recovery status monitoring consist of biomarkers (biochemical, biological or muscle-status marker) and performance-based tests. These tests are objective indicators of fatigue and recovery in the sports context. The biochemical markers identify the inter-individual variabilities of the metabolic process and residues of muscle damage (110). Blood lactate concentration, cortisol, ammonia level, CK, myoglobin (Mb), lactate dehydrogenase (LDH), blood leukocyte count, interleukin-6 (IL-6), testosterone, testosterone/ cortisol ratio and C-reactive protein (CRP) are used to identify the fatigue and recovery status (111-114). Performance markers that are commonly used by researchers and coaches are: jump tests (CMJ, vertical, squat jumps) (115, 116), maximal isometric voluntary strength (117), sprint (118) and repeated sprint ability (119).

A study conducted by González Badillo *et al.* (120) with 9 males reported a decrease in CMJ height and an increase in cortisol and CK following 2 resistance exercise protocols: (i) 2 exercises (Bench Press (BP) and squat) of 80% 1RM, 3 sets of 4 repetitions; and ii) 2 exercises (BP and squat) 80% 1RM, 3 sets of 8 repetitions) and gradually returned to the baseline values in different time frames within 48 hours from the training session. Another study performed by Morán-Navarro *et al.* (121) observed a decrease in CMJ height and an increase in cortisol and CK following 3 resistance exercise protocols: (i) 2 exercises (BP and full squat) of 75% 1RM, 3 sets of 5 repetitions; ii) 2 exercises (BP and full squat) 75% 1RM, 6 sets of 5 repetitions; and iii) 2 exercises (BP and full squat) 75% 1RM, 3 sets of 10 repetitions) and gradually returned to the baseline values in different time frames within 48 hours from the training session in 10 resistance-trained men. Bartolomei *et al.* (122) conducted a study with 12 experienced resistance-trained men and showed a decrease in CMJ peak power, maximal isometric strength, testosterone concentration and an increase in cortisol, IL-6, CRP concentrations, CK, Mb , LDH following 2 resistance exercise protocols: (i) 90% 1RM of squat exercise, 8 sets of 3

repetitions with 3 min rest between sets; and ii) 70% 1RM of squat exercise, 8 sets of 10 repetitions with 3 min rest between sets) and gradually returned to baseline in different time frames within post-72 hours. Although there a number of biomarkers, the majority require specialized equipment and human resources to perform the measurements. Also, the results of some biomarkers cannot be derived in real-time due to the length of time it takes for process the results. Thus, the best option for coaches is performance-based testing, where the athlete performs at his or her maximum capacity, preferably in the rested or recovered state.

Psychological monitoring tools are commonly referred to as subjective tools (113, 114) and primarily assess changes in the psychological state (stress and mood) caused by training. This information is obtained using a self-reported questionnaire, and several appropriate and sensitive psychometric questionnaires are currently used in the field of sports. Among them, the Multicomponent Training Distress Scale (MTDS) (123), the POMS (124), TQR (125) and the RESTQ-Sport (23) are widely used.

A study conducted by Halson *et al.* (126) with 8 endurance cyclists observed an increase in POMS total mood disturbance following a period of 2-week high intensity training protocol and a decrease during the recovery period (post 2 weeks). Moreover, O'Connor *et al.* (127) showed a significant increase in the POMS total mood disturbance score after a 3-day high volume training cycle in swimmers. Furthermore, Coutts *et al.* (128) demonstrated that a 4-week high volume physical training program significantly increased total stress and decreased total recovery scores of RESTQ-Sport questionnaire in 16 well-trained male triathletes. Another study conducted with 12 national level male rowers reported a significant decrease in RESTQ-Index (increased stress and decreased recovery scores) during a high volume training period of 3 weeks and gradually recovered (decreased stress and increase recovery scores) during the following 2-week recovery period (129).

Although the questionnaire is a cost effective and non-invasive method of monitoring fatigue and recovery status, it’s uncertain whether subjective tools

accurately reflect changes in the athlete's fatigue and recovery status. Because the accuracy of the results depends on the athlete's mentality and motivation level, and provided data may under- or over-estimate fatigue or recovery level. Furthermore, there is lack of evidence regarding the association between subjective and objective tools (125). In summary, the above-mentioned objective and subjective tools appear to be problematic in their daily use in measuring fatigue levels and recovery status. Therefore, there is a need for a non-invasive, comfortable, affordable, and field user-friendly testing method. HRV may be the optimal tool to measure the fatigue and recovery status. But, in order to understand its potential, one must first understand the physiology of the ANS.

2.7. THE AUTONOMIC NERVOUS SYSTEM

The human body has two motor systems: the voluntary motor system and the ANS. While the voluntary motor system activates the skeletal muscles, the ANS is responsible for involuntary muscle contractions of the rest of the body's organs and is affected by internal and external situations. This self-regulating system influences the functions of organs (cardiovascular and respiratory control, thermal regulation, gastrointestinal mobility, urinary and bowel excretory functions, reproduction, metabolic and endocrine functions) to make sure that they adapt to the external stress and disturbance to maintain the stability of the body's systems (39).

ANS can be further divided into two opposing branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Primarily, these two systems have reciprocal effects on most of the organs. The PNS can be thought of as the "rest and digestion" mechanism that slows the HR, lowers blood pressure and increases the activity of the digestive system, thus preserving energy for maintenance and conservation of the body's functions during the normal resting situation (i.e., when physiological activity or psychological stress is not present (39, 130). On the other hand, the SNS is rapidly activated during periods of

physiological or psychological stress, thereby increasing the HR and contractility, blood pressure, blood flow to the skeletal muscles, release of glucose from the liver and elevate blood glucose and free fatty acid, as well as decreasing the activity of the digestive system to meet the physiological demands. Hence, it is called the "fight or flight" response (39, 131).

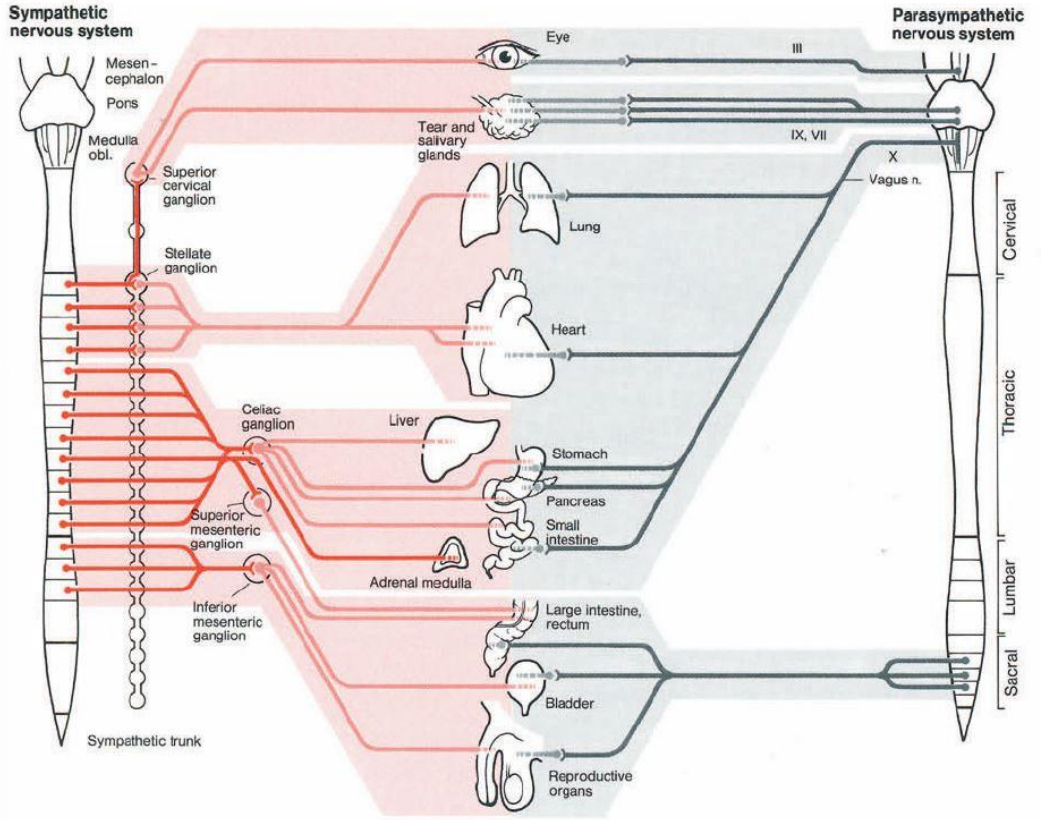


Figure 2. Structure of the ANS. The SNS (left) is activated as a coping response under stressful situations by increasing HR, cardiac output, blood flow to the muscles and decreasing digestive activity. The PNS (right) is activated when physiological activity or psychological stress is no longer present. Source: Jänig (132)

Moreover, these two branches (SNS and PNS) work together to regulate the HR to ensure optimal functions as demanded by the body without unnecessary waste of energy. The balance between SNS and PNS activity is constantly changing in an effort to achieve optimal efficiency in the body, taking into account all internal

and external stimuli. However, one branch always dominates the ANS, depending on the stress or recovery state that the body experiences at the moment (39). Figure 2 depicts the structure of the ANS.

2.8. PHYSIOLOGY BEHIND THE EFFECTS OF EXERCISE TRAINING TO ANS

When the human body is engaged in exercise or sports training, adjustments in the cardiovascular system is required in order to meet the metabolic demands of the active skeletal muscles and the thermoregulatory demands to control core temperature, as well as maintaining the vital functions, such as blood pressure and adequate perfusion to other organs. The ANS plays a key role in responding to and meeting these metabolic and thermoregulatory demands (133, 134).

According to the model established by many authors (135-142), when exercise begins, the "feed-forward" signals that descend from the higher brain centres (“central command”) enter the medullary cardiovascular centre and bring the arterial baroreflex to a higher operating level. As a result, there is an increase in HR, which is primarily mediated by a withdrawal of the cardiac parasympathetic modulation of the ANS. Quick feedback from muscle mechanoreceptors help to initiate the withdrawal of cardiac parasympathetic modulation, while activation of the cardiopulmonary baroreceptors (as a result of increased secondary venous benefits to muscle pump action) elicits withdrawal of cardiac parasympathetic modulation, as well as an initiate the reduction of cardiac sympathetic modulation. During the entire training session or exercise period, both the cardiac sympathetic and parasympathetic modulation control the HR according to the level of exercise intensity. During this time, the cardiac sympathetic modulation acts as a "tone-setter" and the cardiac parasympathetic modulation acts as a "rapid responder or modulator". Cardiac parasympathetic modulation dominates resting and low-intensity exercise conditions, and as the intensity of exercise gradually increases, it triggers further parasympathetic withdrawal and cardiac sympathetic activation.

Thus, the cardiac sympathetic modulation dominates the ANS as the athlete engages in high-intensity exercise (133, 136, 143, 144).

When the athlete returns to post-exercise rest, the higher brain centre lowers the arterial baroreflex, causing a decrease in HR, thereby increasing cardiac parasympathetic modulation activity (133, 145, 146). The rest that ensues immediately after exercise triggers a rapid decrease in HR (HR recovery), which is explained by the reactivation of cardiac parasympathetic modulation of ANS (145-149), but some studies suggest that the cardiac sympathetic modulation is also involved in this process (134, 150). As recovery continues, very slow reduction of HR and recovery of other vital organs can be observed due to gradual domination of cardiac parasympathetic modulation and withdrawal of cardiac sympathetic modulation (133). Although there is a rapid recovery within minutes after cessation of training, it can take up to 48 hours to return to pre-training or complete recovery levels. In some cases, even before or after 48 hours, the athlete may be able to fully recover and reach higher status of recovery than pre-training level (151-153). Most importantly, these changes in SNS and PNS can be monitored and quantified by examining HRV (154, 155). With the recent technological advancements, HRV is a tool that is increasingly used to assess fatigue status and recovery.

2.9. HEART RATE VARIABILITY

The time between successive heartbeats is never constant and there is an oscillation around the mean value between consecutive heartbeats. This oscillation or variation in the time interval between consecutive heartbeats (RR interval) is known as HRV (32, 156, 157). In a typical Electrocardiograph trace, we can identify the: (i) P wave that is produced by electrical potentials generated depolarization of the atria, (ii) QRS complex that represents the depolarization of the ventricles and (iii) T wave that signifies the repolarization of the ventricles (158). The time interval variation between consecutive RR intervals is used to measure HRV (see Figure 3).

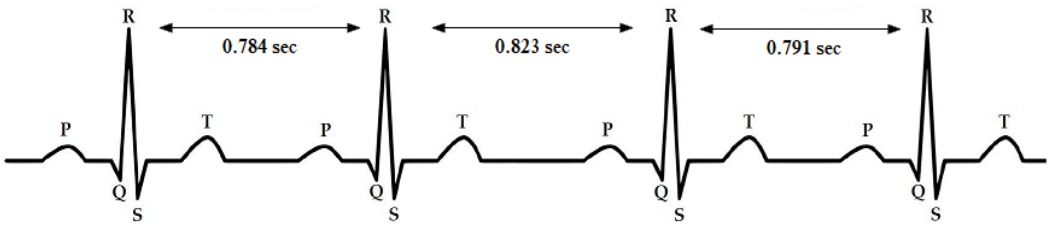


Figure 3: Time interval variation between consecutive RR intervals

Briefly, about the history of HRV, the changes in the RR intervals of the human heart was first identified in the early 1600s (159). However, its physiological importance was not realized until 1965 when Hon (160) observed that fatal distress was preceded by changes in the variation of the RR intervals before a significant change in HR. Subsequently, research into the field of HRV was carried out extensively, and by the 1980s methodological issues related to this field were solved, the physiology behind HRV, and the relationship with ANS were explained and many clinical applications were found (39). The rapid development of the field of HRV and the realization of the clinical value of HRV led to the formation of the joint Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology in 1996 (161). The joint task force established the minimal technical requirements, definitions, standardized the areas of Powerbands in frequency domain and offered recommendations for conducting clinical research and patient examinations using HRV (39, 161, 162). With the current advancements of the technology, non-invasive, reliable and practical on-field HRV monitoring devices are available with the capability of continuous recording long period (more than 24 hours) (114).

2.9.1. Heart rate variability parameters

HRV can be analysed in several ways, and each method has its strengths and weaknesses. There are three main methods used to evaluate HRV, which are "time

domain", “frequency domain” and "non-linear" analysis. Each method has a number of different HRV indicators that can be calculated:

1. Time-domain analysis (30, 161, 163, 164)
    - Statistical methods
    - Geometric methods
2. Frequency domain analysis (30, 163-165)
    - Autoregression
    - Fast Fourier transforms
3. Non-linear analysis (155, 164, 166, 167)
    - Poincare plots (168)
    - Spectral slope in the log-log scale
    - Kolmogorov entropy
    - Correlation dimension
    - Approximate entropy
    - Spectral Coarse Graining
    - Lyapunov exponents
    - Complex demodulation / Homomorphic filtering
    - QIS-A (Quartile deviation of integrated and subtracted fluctuation) (167)
    - Alpha-stable distributions
    - Higher-order spectral methods
    - Detrended fluctuation analysis (166)

In the scientific literature, many research papers have performed the below mentioned HRV analysis methods, and these methods were used in the present dissertation thesis to allow for comparisons with the literature.

- Time domain analysis (Statistical method)
- Frequency domain analysis (Auto regression and Fast Fourier transforms)
- Non-linear Analysis (Poincare plot analysis)

2.9.1.1. Time domain analysis

Among the HRV analysis methods, the simplest method of analysing RR intervals is time-domain analysis, and in this method, time-domain indicators calculate the amount of variability in measurements of the time period between successive normal-to-normal (NN) heartbeats during the monitoring period (161, 169).

In the statistical method, the standard deviation of all NN time intervals over the selected time period (SDNN) is the simplest and most frequently used HRV parameter. SDNN mirrors all cyclical components responsible for variability during data collection periods (161). It is not recommended to compare SDNN measurements obtained by different recording time duration. Because SDNN estimates cycle length depends on the monitoring time. It should also be noted that the total variance of the HRV depends on the length of the analysed recording (161). In short-term rest recordings, the SDNN parameter can be used as an indicator of overall autonomic modulation (161). In practice, short-term 5-minute recordings and 24-hour recordings are more ideal (161). Another time-domain HRV parameter commonly used is the proportion of interval differences of successive NN intervals lasting more than 50 ms (pNN50) and requires a 2-min epoch. (161, 169). The pNN50 parameter can also be used as an indicator of cardiac parasympathetic activity (170), and pNN50 parameter has been shown to be correlated with RMSSD and HF power parameters (169). It's also important to mention that pNN50 is considered a more reliable parameter than short-term SDNN parameter (169).

Among the time domain parameters, the most widely used parameter is the Root Mean Square of Successive Differences (RMSSD). To calculate RMSSD, first one must compute each successive time difference between NN intervals in ms. After that, each of the values is squared and the result is averaged before the square root of the total is obtained (169). The recommended minimum data recording time is 5 minutes (short-term), but some researchers have suggested an ultra-short-term duration of 10s, 30s and 60s (169, 171-174). RMSSD is the primary time domain

parameter used to evaluate the cardiac parasympathetic activity (169) and is a promising method for monitoring individual adaptation to training when measured during resting or post-exercise recovery conditions (173). Most importantly, RMSSD parameter is less affected by fluctuations in respiration and more stable parameter, therefore it is a more robust indicator of cardiac parasympathetic effect (175, 176). Table 1. summarizes the above-mentioned time-domain parameters.

Table 1. Selected Time Domain parameters

Variables	Units	Description
SDNN	ms	Standard deviation of all NN intervals
pNN50	%	NN50 count divided by the total number of all NN intervals
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals

Abbreviations: % = Percentage; ms = milliseconds; NN = Normal to normal; pNN50 = proportion of interval differences of successive NN intervals lasting more than 50 ms; RMSSD = Root Mean Square of Successive Differences; SDNN = standard deviation of all NN time intervals over the selected time period;

2.9.1.2. Frequency domain analysis

The power spectral density method describes the periodic oscillation of the HR signal, converts it into different frequency bands (high and low) and gives numerical values of their relative intensity, which is termed variance or power (155, 176). This provides an idea of the activities of the different branches of ANS (163). Power spectral analysis can be performed using parametric and non-parametric methods. Due to the simplicity of the algorithm and the high processing speed, the non-parametric Fast Fourier Transformation method is widely used among frequency domain analysis methods (155) because the parametric methods of discrete Fourier transformation are more complex and depend on the model used.

Therefore, it cannot be applied to subjects whose HR changes rapidly during the measurement period due to the fact that the time series under investigation must be static (162). Among the usual frequency domain parameters, total power (TP), low-frequency power (LF), high-frequency power (HF) and ratio of LF to HF (LF/HF ratio) are commonly used to measure the changes of ANS (162).

The TP (0.003–0.4 Hz) represents the overall autonomic activity (177, 178). The HF band (0.15–0.40 Hz) is often considered a proxy of cardiac parasympathetic modulation and is affected by the respiratory rate, although it is considered reliable with normal respiration (163, 169, 179). Lower HF value is related to stressful situations (169). A minimal, one-minute data recording is recommended to analyze HF (169). On the other hand, it is recommended to record at least two minutes of data for analysis of LF bands 0.04–0.15 Hz. High LF value reflects increased cardiac sympathetic activity due to mental and physical stress (155, 162). However, some researchers consider the LF parameter as a cardiac sympathetic modulation marker, while others believe it reflects the cardiac sympathetic and parasympathetic modulation (161, 176). When LF is expressed in normalized units, it is a quantitative marker for sympathetic modulations (161, 176, 180). LF and HF power can be expressed as absolute values (ms^2) or normalized values (nu) (30, 161). Normalization reduces the effect of TP changes on the values of the LF and HF components and reduces the effects of noise due to artifacts (176). It is important to use normalization when investigating the impact of different interventions on the same subject or when comparing subjects with major differences in TP (181).

LF/HF ratio parameter estimates the balance between cardiac sympathetic and parasympathetic modulation (Sympathovagal balance) (155, 162, 182). A decreased LF/HF ratio reflects higher cardiac parasympathetic activity compared to cardiac sympathetic activity, but this ratio might shift due to decreased LF. Conversely, an increased LF/HF ratio indicates higher cardiac sympathetic activity compared to cardiac parasympathetic activity. Therefore, the LF/HF ratio should be cautiously interpreted when taking into account the mean values of the HF and

LF, especially during short recordings (182). Table 2. summarizes the aforementioned frequency domain parameters.

Table 2. Selected Frequency Domain parameters

Variables	Units	Description
TP	ms^2	The variance of all NN intervals
LF	ms^2	Low frequency power
LF(nu)	nu	Low frequency power in normalized units
HF	ms^2	High frequency power
HF(nu)	nu	High frequency power in normalized units
LF/HF	---	Ratio of LF power to HF power

Abbreviations: HF = High frequency; LF= Low frequency; ms^2 = milliseconds squared; NN = normal-to-normal; nu = normalized units; TP = Total power

2.9.1.3. Non-linear analysis

There are several non-linear methods (fractal analysis) used to evaluate HRV. These methods are based on the "chaos theory" and fractals. Chaos refers to a study of multivariable, nonlinear and nonperiodic systems (183, 184). Among the non-linear methods, Poincaré Plot (SD1, SD2 and SD1/SD2), Approximate Entropy (ApEN) and Sample Entropy are the most commonly used methods.

The standard descriptors of the Poincaré plot are SD1 (width of the ellipse calculated as standard deviation of the distance of each point from the y = x-axis) and SD2 (length of the ellipse calculated as standard deviation of each point from the y = x + average R–R interval). The line of identity is the 45º fictional diagonal line on the Poincaré plot (169, 185, 186). SD1 describes the fast beat-to-beat variability in the R-R intervals, while SD2 represents the longer-term variability (163, 169). Moreover, the SD1 parameter is associated with cardiac parasympathetic modulation, while SD2 parameter is inversely proportional to the sympathetic activity. However, some researchers consider SD2 parameter to reflect sympathetic and parasympathetic activity (163, 187, 188). Unpredictability of the NN time series

is measured using the SD1/SD2 ratio and provides information about the autonomic balance when the monitoring period is long enough and there is domination of cardiac sympathetic modulation. However, this interpretation is quite controversial because SD1 (numerator) is associated to cardiac parasympathetic modulation, while SD2 (denominator) is inversely related to cardiac sympathetic modulation (187). On the other hand, SD2/SD1 is considered as the sympathovagal balance and is correlated with LF/HF ratio (189-191). Moreover, Orellana *et al.* (187) introduced the Stress Score Index (SS) parameter using SD2 value (SS = 1,000 × 1/SD2) to get a better understanding of the sympathetic activity. The SS value has been used in several studies and its usefulness has been validated (187, 188, 192, 193).

The ApEN parameter was introduced by Pincus (194), which measures the regularity and complexity of the time series (169). ApEN presents a number between 0 and 1, and a small value of ApEN indicates that the signal is constant and predictable, while higher values indicate a lower prediction of fluctuations in NN intervals (162, 195, 196). A major drawback of the ApEN parameter is the lack of internal consistency (162). Therefore, Richman *et al.* (197) has introduced a different algorithm called the "sample entropy" (SampEn) as an alternative. Most importantly, SampEn is not affected by record duration and displays relative consistency compared to ApEN (197). Therefore, SampEn provides a less biased and reliable measure of signal formality and complexity compared to ApEN (162, 198).

Table 3. Selected Non-linear parameters

Variables	Units	Description
SD1	ms	Poincaré plot standard deviation perpendicular the line of identity
SD2	ms	Poincaré plot standard deviation along the line of identity

SD2/SD1	---	Ratio of SD2-to-SD1
SS	s^{-1}	Stress Score index
ApEN	---	Approximate entropy, which measures the regularity and complexity of a time series
SampEn	---	Sample entropy, which measures the regularity and complexity of a time series

Abbreviations: ApEN = Approximate entropy; ms = milliseconds; s^{-1} = per second; SampEn; SD1 = Poincaré plot standard deviation perpendicular the line of identity; SD2 = Poincaré plot standard deviation along the line of identity; SS = Stress Score index

2.9.2. Measuring heart rate variability

According to the Task Force recommendations, HRV data should be recorded for 24 hrs for long-term and about 5 min for short-term data analysis (161). However, 5 min of data recording is methodologically suitable and offers more advantages in practical application compared to long-term data recording. With the technological advancement and new discoveries, some studies have recommended: "ultra-short-term analysis" (< 5 minutes) for some HRV parameters (172).

Another important factor to consider when recording HRV data is the body posture of the athlete. Posture may influence HRV reliability. As some studies suggest, the dominance of cardiac sympathetic modulation is detected when standing or when in the passive head-up-tilt position, while the dominance of cardiac parasympathetic modulation is detected in the supine body position (199-202). A systematic review conducted by da Silva *et al.* (203) reported no difference between supine or standing position. However, to ensure consistency, the use of a single posture to record HRV data throughout the study is most appropriate.

The athlete's respiratory rate during HRV recording can also affect the data. It has been reported that there is a negative correlation between respiratory rate

and spectral measurements of cardiac parasympathetic modulation (204-207). Therefore, one must control the respiration rate or pattern during the recording period in order to have interpretable results. Investigators have accepted different respiration rates (i.e., 6-15 beats/minute) (163) and some studies have used 12 breaths per minute during HRV data collection (37, 43, 208-212). Kingsley *et al.* (38) reported that 66% of the included ARE used a controlled breathing rate of 12 breaths per minute in their review study.

Once data is collected, pre-processing the HRV raw data is necessary before applying any HRV analysis methods. In many cases, it is difficult to obtain a good HRV measurement that is completely devoid of artifacts, and this may be due to technical (missing, extra or misaligned beat detections) or physiological (ectopic beats and arrhythmic events) artifacts (213, 214). Technical artifacts may be due to measurement noise or inaccuracy in the identification algorithm and ectopic beats, which is normal and relatively common among healthy subjects (161). HRV is sensitive to data quality, and untreated artifacts can significantly alter the values of HRV parameters (213). There are several methods and algorithms for editing or correcting dubious R–R intervals, and the most common artifact correction and editing techniques are: deletion, interpolation of degree zero, interpolation of degree one (linear interpolation) and cubic spline interpolation (213). According to the Task Force, human visual inspection of RR interval raw data to remove artifact is considered the gold standard (161). However, it has been identified that human assessment is prone to error and depends on skill level, which can be problematic for reliability and reproducibility. Now, advanced software is capable of identifying and treating these technological and physical artifacts and is recommended over manual visual inspection to verify the correctness of the algorithms (215).

2.10. ACUTE EFFECT OF RESISTANCE EXERCISE ON HRV PARAMETERS

The effects of exercise on ANS can be measured using HRV parameters and there are several studies that have examined the acute effect of RT on HRV parameters in healthy adults and trained athletes. Heffernan *et al.* (208) studied 14 young men and found a significant decrease in HF power and LF power, and an increase in the LF/HF ratio 30 minutes following RT (10-RM for eight resistance exercises - three sets each with 90 sec of rest between sets) session. In a recent study, Thamm *et al.* (152) reported a significant decrease in RMSSD parameter following hypertrophic and maximum strength training sessions, which recovered back to baseline within 30 minutes from the training sessions. Interestingly, LF, HF and LF/HF ratio parameters remained statistically unaltered during the recovery period up to one hour from the training session. These results suggest that resistance exercises acutely increase cardiac sympathetic modulation and decrease in cardiac parasympathetic modulation.

Chen *et al.* (153) conducted a study with 7 weightlifters using a 2-hour weight training session (back squat, seated shoulder press, deadlift, and front squat - intensity for each training started from 60% 1RM 3 times, 70% 1RM 3 times, 80% 1RM 3 times, 90% 1RM 2 times, 95% 1RM 1 time with 90 sec rest on each repetition) and HRV parameters were measured before training and at 3, 24, 48 and 72 hours after training. The results revealed that, within 3 hours from the training session, HF power, very low frequency (VLF) and median variability decreased significantly and gradually returned to baseline after 48 hours. LF(nu) increased significantly 3 hours post-resistance exercise and returned to baseline after 48 hours, indicating that resistance exercises acutely increased cardiac sympathetic modulation and decreased cardiac parasympathetic modulation. Most importantly, it took around 48 hours from the training session to recovered. Another study performed with 17 resistance-trained and 17 untrained participants performing various (whole, lower or upper body - three sets at 10RM with 90 sec of rest between sets) acute RT exercise bouts showed that there was a significant

increase in LF(nu) and LF/HF ratio, while natural logarithm (Ln) HF and HF(nu) significantly decrease after 25 minutes after the intervention (210). In another study, 17 healthy participants participated in low-intensity (40% 1RM) and high-intensity (80% 1RM) resistance exercise sessions, showed that LF(nu) and LF/HF ratio increased while HF(nu) decreased compared to pre-training levels in both exercise intensity levels following 15 to 75 minutes post-exercise recovery stage (216) suggesting an increase in cardiac sympathetic modulation and a decrease in cardiac parasympathetic modulation following the ARE.

In addition, eight recreationally-trained women performed 10 RM load test in 4 resistance exercises (smith back squat, leg press incline, leg extension, and leg flexion), resulting in a significant decrease in RMSSD and pNN50 parameters compared to baseline following 15 minutes from the exercises (217). Another study conducted by Lima *et al.* (218) with 12 normotensive men revealed that 1 RM knee extension test decreased RMSSD, pNN50, HF(nu) parameters and increased LF(nu), LF/HF ratio and VLF(nu) parameters, 40 minutes after the training session. Interestingly, SDNN parameter did not changed as a result of 1 RM knee extension test. Kingsley *et al.* (212) conducted a study with 14 men and 13 women using ARE session, consisting of 3 exercises (squat, BP and deadlift), 3 sets of 10 repetitions at 75% 1RM, showed that Ln RMSSD, Ln TP and Ln HF parameters decreased and Ln LF/HF ratio increased in both genders. Interestingly, after the RT sessions in this study, the Ln LF parameter increased for men and decreased for women. However, the study concluded that there were no significant differences between sex in the alterations in HRV parameters following RT session.

Among the aforementioned studies, most of the studies revealed that RMSSD, pNN50, HF, HF(nu) and TP parameters decreased and increased LF, LF(nu) and LF/HF ratio parameters as a result of ARE session (36, 42, 43, 152, 153, 209, 210, 218-220). The review by Kingsley *et al.* (38) that examined 10 studies published before 2014 also showed similar results (decrease in HF(nu) parameter and increase in LF(nu) and LF/HF ratio) following a RT session in healthy young

men and women. Since then, several studies have examined the effect of ARE on HRV parameters (36, 37, 41, 42, 152, 217, 218, 220-231), and there are some discrepancies in the findings as some studies show the opposite effect on HRV parameters following an ARE session (41-43, 217). Therefore, it may be important to systematically review and conduct a meta-analysis of the studies that have investigated ARE on HRV parameters to understand how an ARE session affects the HRV parameters and identify the possible moderating factors that contribute to the cardiac autonomic activity during post-exercise recovery.

III – HYPOTHESIS

III. HYPOTHESIS

3.1. GENERAL HYPOTHESIS

Study 1

- HRV parameters would show that ARE session negatively affects the cardiac autonomic activity during post-exercise recovery (around 30 minutes) and subject's characteristics and resistance exercise training session variables act as possible moderating factors on the cardiac autonomic activity during post-exercise recovery.

Study 2

- Both strength and power RT modalities negatively affect the cardiac autonomic activity (HRV parameters), performance, neuromuscular fatigue, central fatigue, peripheral fatigue and perceptual responses and these changes would recover sooner in power training compared to strength training. Furthermore, lower training loads would be better at maintaining the recovery level in the subsequent training microcycle (at 48H following an intensive fatigue session) than higher training loads in strength and power training modalities.

3.2. SPECIFIC HYPOTHESIS

Study 1

1. The systematic review would show that cardiac parasympathetic modulation and overall autonomic modulation decreases and cardiac sympathetic modulation increases following an ARE session.
2. The meta-analysis would show that subject characteristics (gender, body mass index (BMI), and training status) and training characteristics (training

intensity, number of repetitions, sets, rest between sets, amount of exercise per workout and training volume) are moderating factors to the cardiac autonomic activity during post-exercise recovery.

Study 2

1. 100% training load of strength training modality would show greater effect on HRV parameters (pNN50 ↓, SDNN ↓, Ln RMSSD ↓, HF(nu) ↓, TP ↓, SampEn ↓, SD1 ↓, LF(nu) ↑, LF/HF ratio ↑, SD2 ↑, SD2/SD1 ratio ↑, SS ↑, RHR ↑), performance (BP relative peak power (RPP) ↓, CMJ height ↓, CMJ RPP ↓), neuromuscular fatigue (MVC peak force ↓, Rate of force development $(RFD)^{200MVC}$ ↓), central fatigue (Central activation ratio (CAR) ↓, MVC/tetanic force ↓), peripheral fatigue (tetanic force ↓, RFD^{tet} ↓, Rate of force relaxation $(RFR)^{tet}$ ↑, twitch force ↓, $T_{1/2}$ ↑, twitch-to-tetanus ratio ↓), and perceptual responses (DOMS ↑, POMS ↓) compared to 100% training load of power training modality following the intensive fatigue session.
2. 100% training load of power training modality would return to the Pre-B value (recover) sooner on HRV parameters (pNN50, SDNN, Ln RMSSD, HF(nu), TP, SampEn, SD1, LF(nu), LF/HF ratio, SD2, SD2/SD1 ratio, SS, RHR), performance (BP RPP, CMJ height, CMJ RPP), neuromuscular fatigue (MVC peak force, RFD^{200MVC}), central fatigue (CAR, MVC/tetanic force), peripheral fatigue (tetanic force, RFD^{tet}, RFR^{tet} , twitch force, $T_{1/2}$, twitch-to-tetanus ratio), and perceptual responses (DOMS, POMS) compared to 100% training load of strength training modality following the intensive fatigue session.
3. Some HRV parameters (pNN50, SDNN, Ln RMSSD, HF(nu), TP, SampEn, SD1), performance (BP RPP, CMJ height, CMJ RPP), neuromuscular fatigue (MVC peak force, RFD^{200MVC}), central fatigue (CAR, MVC/tetanic force), peripheral fatigue (tetanic force, RFD^{tet}, twitch force, twitch-to-tetanus ratio), and perceptual responses (POMS) would decrease while some HRV parameters (LF(nu), LF/HF ratio, SD2, SD2/SD1 ratio, SS, RHR), peripheral

fatigue (RFR^{tet}, $T_{1/2}$), and perceptual responses (DOMS) would increase following the intensive fatigue session and subsequent ARE strength training sessions (100, 75 or 50%) and gradually return to the respective Pre-B values within the microcycle.

4. Some HRV parameters (pNN50, SDNN, Ln RMSSD, HF(nu), TP, SampEn, SD1), performance (BP RPP, CMJ height, CMJ RPP), neuromuscular fatigue (MVC peak force, RFD^{200MVC}), central fatigue (CAR, MVC/tetanic force), peripheral fatigue (tetanic force, RFD^{tet}, twitch force, twitch-to-tetanus ratio), and perceptual responses (POMS) decrease while some HRV parameters (LF(nu), LF/HF ratio, SD2, SD2/SD1 ratio, SS, RHR), peripheral fatigue (RFR^{tet}, $T_{1/2}$), and perceptual responses (DOMS) increase following the intensive fatigue session and subsequent ARE power training session (100, 75 or 50%) and gradually return to the respective Pre-B values within the microcycle.
5. 100% training load of strength training modality trial would show the greatest effect on HRV parameters (pNN50 ↓, SDNN ↓, Ln RMSSD ↓, HF(nu) ↓, TP ↓, SampEn ↓, SD1 ↓, LF(nu) ↑, LF/HF ratio ↑, SD2 ↑, SD2/SD1 ratio ↑, SS ↑, RHR ↑), performance (BP RPP ↓, CMJ height ↓, CMJ RPP ↓), neuromuscular fatigue (MVC peak force ↓, RFD^{200MVC} ↓), central fatigue (CAR ↓, MVC/tetanic force ↓), peripheral fatigue (tetanic force ↓, RFD^{tet} ↓, RFR^{tet} ↑, twitch force ↓, $T_{1/2}$ ↑, twitch-to-tetanus ratio ↓), and perceptual responses (DOMS ↑, POMS ↓) compared to 75% and 50% training load of strength training modality trial following the intensive fatigue session.
6. 100% training load of power training modality trial would show the greatest effect on HRV parameters (pNN50 ↓, SDNN ↓, Ln RMSSD ↓, HF(nu) ↓, TP ↓, SampEn ↓, SD1 ↓, LF(nu) ↑, LF/HF ratio ↑, SD2 ↑, SD2/SD1 ratio ↑, SS ↑, RHR ↑), performance (BP RPP ↓, CMJ height ↓, CMJ RPP ↓), neuromuscular fatigue (MVC peak force ↓, RFD^{200MVC} ↓), central fatigue (CAR ↓, MVC/tetanic force ↓), peripheral fatigue (tetanic force ↓, RFD^{tet} ↓,

RFR^{tet} ↑, twitch force ↓, $T_{1/2}$ ↑, twitch-to-tetanus ratio ↓), and perceptual responses (DOMS ↑, POMS ↓) compared to 75% and 50% training load of power training modality trial following the intensive fatigue session.

7. 50% training load of strength training modality would return HRV parameters (pNN50, SDNN, Ln RMSSD, HF(nu), TP, SampEn, SD1, LF(nu), LF/HF ratio, SD2, SD2/SD1 ratio, SS, RHR), performance (BP RPP, CMJ height, CMJ RPP), neuromuscular fatigue (MVC peak force, RFD^{200MVC}), central fatigue (CAR, MVC/tetanic force), peripheral fatigue (tetanic force, RFD^{tet}, RFR^{tet} , twitch force, $T_{1/2}$, twitch-to-tetanus ratio), and perceptual responses (DOMS, POMS) to Pre-B value (recover) sooner than 100% and 75% training loads of strength training modality following the intensive fatigue session.
8. 50% training load of power training modality would return HRV parameters (pNN50, SDNN, Ln RMSSD, HF(nu), TP, SampEn, SD1, LF(nu), LF/HF ratio, SD2, SD2/SD1 ratio, SS, RHR), performance (BP RPP, CMJ height, CMJ RPP), neuromuscular fatigue (MVC peak force, RFD^{200MVC}), central fatigue (CAR, MVC/tetanic force), peripheral fatigue (tetanic force, RFD^{tet}, RFR^{tet} , twitch force, $T_{1/2}$, twitch-to-tetanus ratio), and perceptual responses (DOMS, POMS) to Pre-B value (recover) sooner than 100% and 75% training load of power training modality following the intensive fatigue session.
9. Among the three (100%, 75%, 50%) strength training loads, 75% strength training load would be the optimal training load for the subsequent training session following an intensive fatigue session to achieve adequate recovery within microcycle, based on HRV parameters.
10. Among the three (100%, 75%, 50%) power training loads, 100% power training load would be the optimal training load for the subsequent training session following the intensive fatigue session to achieve adequate recovery within microcycle, based on HRV parameters.

IV - OBJECTIVES

IV. OBJECTIVES

4.1. GENERAL OBJECTIVES

Study 1

- To evaluate the effects of ARE session on the HRV characteristics and identify the possible moderating factors contributing to cardiac autonomic activity during post-exercise recovery.

Study 2

- To evaluate and compare the changes and recovery of HRV parameters and other objective and subjective responses induced by strength training and power training modalities and different training loads of strength and power training modalities, following an intensive fatigue condition within the micro training cycle. Furthermore, identify the optimal training loads related to strength and power training modalities following a high-fatiguing session to maintain the adequate recovery within the micro-training cycle

4.2. SPECIFIC OBJECTIVES

Study 1

1. Conduct a systematic review with meta-analysis regarding the effect of ARE on HRV parameters.
2. Determine the moderating factors of acute RT session that affect cardiac autonomic modulation during recovery.

Study 2

1. Compare the effect and recovery of 100% training load session between ARE strength and power training modalities following the intensive fatigue session on HRV parameters, performance, neuromuscular fatigue, central fatigue, peripheral fatigue, and perceptual responses.
2. Analyze the change in HRV parameters, performance, neuromuscular fatigue, central fatigue, peripheral fatigue, and perceptual responses following the intensive fatigue session and subsequent ARE strength training session using a given training load (100, 75 or 50%) within the microcycle.
3. Analyze the change in HRV parameters, performance, neuromuscular fatigue, central fatigue, peripheral fatigue, and perceptual responses following the intensive fatigue session and subsequent ARE power training session using a given training load (100, 75 or 50%) within the microcycle.
4. Compare the effect and recovery of different load (100 vs. 75 vs. 50%) sessions used within each ARE training modality (strength or power) following the intensive fatigue session on HRV parameters, performance, neuromuscular fatigue, central fatigue, peripheral fatigue, and perceptual responses.
5. To identify the optimal training load for strength and power training within the microcycle to achieve adequate recovery following the intensive session based on HRV parameters.

.

V – METHODS

V. METHODS

This chapter provides information regarding the methodology of the studies conducted during this doctoral thesis. This doctoral thesis consists of a systematic review with meta-analysis and an experimental study to respond to the objectives mentioned in Chapter 3. The systematic review with meta-analysis examines the state of the literature regarding the ARE on HRV parameters to understand how an ARE session affects the HRV parameters and identify the possible moderating factors that contribute to the cardiac autonomic activity during postexercise recovery. The experimental study was designed based on the findings from the systematic review and meta-analysis to examine the effect and recovery of different training loads of strength or power RT sessions following an intensive fatigue session. Another aim of the experimental study was to identify the optimal training load for strength and power training for the subsequent training session following an intensive fatigue session based on HRV parameters. This would provide evidence that HRV parameters can be used as a tool to establish the optimal volume for the subsequent training session after a high fatiguing session (earlier in the microcycle). The following sections describe more in detail the methodology used for the systematic review with meta-analysis (Section 5.1) and the experimental study (Section 5.2).

5.1. STUDY 1

In recent years, there has been great interest in investigating the acute effects of RT on HRV parameters. However, it is unclear what the magnitude of the effects of RT, as well as the possible moderating factors, are on HRV parameters. Therefore, this work aimed to systematically review the literature and conduct a meta-analysis on the studies that have investigated the acute effect of RT on HRV

parameters and determine which variables of RT moderate the cardiac autonomic recovery status following a RT session. The recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) declaration (232) were followed during this methodological process.

5.1.1. Data sources

A comprehensive literature search was performed in the PubMed-Medline, Web of Science, SPORTDiscus and Cochrane Library electronic databases with the inception established through November 30, 2019. The keywords and categorical searches were: (i) "heart rate variability" OR "HRV" OR "vagal" OR "autonomic function" and (ii) "resistance training" OR "strength training" OR "weight training" OR "power training" OR "weightlifting" OR "full body" OR "circuit*" OR "neuromuscular training" OR "bodyweight training". Second, the Boolean operator AND was used to combine categories (i) and (ii). Additional records were identified while reviewing the reference lists of the books written in the relevant area.

5.1.2. Selection criteria

The eligibility criteria were pre-established. Articles were included if they: (1) examined the ARE on HRV after one training session; (2) conducted the study on healthy individuals (males or females); (3) contained a detailed explanation of the RT protocol; (4) provided information of outcomes both at baseline and following intervention; (5) reported that post-data was recorded between 8 and 30 minutes after the intervention; and (6) included at least one ARE training intervention group. Research studies were excluded if they: (1) had a sample population with pathologies; (2) were not an original investigation published in peer-reviewed journals; (3) did not specify the test battery to be evaluated; (4) did not make available the data or did not provide those data a posteriori with the corresponding

author; and (5) had methodological issues that may have had potential risk of carryover effects due to inadequate recovery period (≤24 hours).

5.1.3. Study selection and data extraction

The electronic database search and selection of the studies for inclusion were conducted by two authors (SUMA and JARA), according to the criteria previously established. Any disagreements regarding the inclusion/exclusion of articles were discussed and resolved by consensus. The following data were extracted from the selected articles: authors, number of participants, subject characteristics, exercise protocol and outcomes of selected HRV parameters. SDNN, RMSSD, HF(nu), LF(nu) and LF/HF ratio were considered the most examined HRV parameters (155, 161, 162, 169). RMSSD and HF(nu) indicate the level of cardiac parasympathetic modulation (161, 169), while LF(nu) provides the degree of cardiac sympathetic modulation (161, 162). LF/HF ratio presents the extent of sympathovagal balance, and SDNN represents overall autonomic modulation (161). Thus, an increase in cardiac sympathetic modulation corresponds to an increase in LF(nu) and LF/HF ratio, while domination of cardiac parasympathetic modulation is shown by an increase in RMSSD and HF(nu) parameters.

5.1.4. Data synthesis

Mean ($\bar{x}$), standard deviation (SD) and sample size (n) data were recorded from the included articles (SUMA) and were confirmed by another investigator (JARA). Corresponding authors of the included articles were contacted if necessary data were not available in print. When studies reported two or more subgroups, those subgroups were combined into a single group, in accordance with the Cochrane Handbook for Systematic Reviews of Interventions (233). For pre-post intervention studies, $\bar{x}$, SD and n values and post-intervention $\bar{x}$, SD and n values of Experimental-Control studies were uploaded to the Review Manager software (RevMan 5.3; Cochrane Collaboration, Oxford, UK). For each study, mean

difference (MD), change in SD, 95% confidence intervals (CI) were calculated between pre- and post-intervention (i.e., differences within groups) and between experimental and control groups.

5.1.5. Meta-analysis

Meta-analyses were conducted on the changes in each outcome using Review Manager software (RevMan 5.3; Cochrane Collaboration, Oxford, UK). Since SDNN, RMSSD, LF(nu), HF(nu) and LF/HF ratio data were measured with different time durations (i.e., time period of collected data) or were presented with different units (e.g., natural logarithm or milliseconds squared), the MD's were standardized by dividing the values with their corresponding SD and weighted according to the inverse variance method. The standardized mean difference (SMD) in SDNN, RMSSD, LF(nu), HF(nu) and LF/HF ratio data of each study was pooled with a random-effects model (233). The data analysis was focused on the magnitude of the effects obtained.

5.1.6. Heterogeneity and risk of bias

The statistical heterogeneity between studies was evaluated using the Cochrane χ^2 test(I^2). The I^2 values of <30%, 30% - 60%, and >60% were considered as low, moderate and high levels of heterogeneity, respectively. A P value <0.05 from the χ^2 test suggested the presence of heterogeneity (234), which was likely due to the methodological diversity of the studies. Methodological quality was also evaluated using the "Study quality assessment tools" provided by the National Heart, Lung, and Blood Institute (235). The "Quality assessment of controlled intervention studies" tool was used for studies that included control groups, and the tool for "quality assessment tool for before-after (Pre-Post) studies with no control group" was used for studies that included only an experimental group. Publication bias was evaluated by analysing the funnel plot asymmetry test.

5.1.7. Subgroup analysis

In this study, the authors decided to perform subgroup analyses using categorical variables and continuous variables without conducting meta-regression analysis for continuous variables. The reason for representing continuous variables as categorical variables for the subgroup analyses was to match how these variables are presented by organizations like the NSCA (1). The recommendations of general training protocols are: high intensity > 85% 1RM and low intensity < 65% 1RM. It is important to analyze the data with these recommendations taken into consideration to help reduce the gap between the scientific evidence and practical application in RT sessions in the field or gym. Therefore, subgroup analyses were defined considering the real practice of RT sessions in the field, as well as the NSCA guidelines (1, 236, 237).

Subjects characteristics (Gender, BMI, and training status) and training characteristics (training intensity (% 1RM), number of repetitions, sets, rest between sets, amount of exercises per workout and training volume (number of repetitions × sets × exercises)) were assessed by subgroup analysis to examine its effect on selected HRV parameters. With regards to BMI, ≤24.9 kg/m^2 (healthy weight) or > 24.9 kg/m^2 (overweight) were considered as cut-off values based on the guidelines by the Centers for Disease Control and Prevention (238). Regarding gender, male and female were used for grouping trials. For RT variables, cut-off values for grouping trials were determined by considering the practical approach of RT sessions in the field and the NSCA guidelines (1, 236, 237). High (> 85% 1RM), moderate (>65% to 85% 1RM) and low (≤65% 1RM) values were used as cut-off points for training intensity (1, 236, 237). For the number of repetitions, < 6, 6 to 10 and >10 repetitions were considered as cut-off values. With respect to the number of sets, cut-off values were set as < 3, exactly 3, and > 3 sets, and for the number of exercises, < 6, exactly 6, and > 6 exercises per workout cut-off values were established. For resting time between sets, <2 min, exactly 2 min, and >2 min were used as cut-off points. Regarding training volume (calculated as the number of

repetitions × sets × exercises), cut-off points were set at <108 (low), 108 to <180 (medium) and ≥180 (high). Changes in possible moderating factors were expressed and analysed as the difference between post- and pre-intervention values. Subgroup analyses were also performed using Review Manager software (RevMan 5.3; Cochrane Collaboration, Oxford, UK).

5.2. STUDY 2

This experimental study was based on some of the findings of the systematic review with meta-analysis. The main purpose of the experimental study was to evaluate and compare the changes and recovery of HRV parameters and other (objective and subjective) responses induced by strength training and power training under the fatigue conditions within the micro training cycle and identify the optimal training loads based on HRV parameters, need to maintain the adequate recovery within the micro training cycle in strength and power training modalities. Which could be use HRV parameters as a tool to determine the appropriate optimal training load for the subsequent training session to maintain the adequate recovery

5.2.1. Participants selection criteria

Participants were included if they: (1) were aged between 18 to 35 years old; (2) were non-smokers; (3) were absence of cardiovascular events or metabolic disease as determined via the Physical Activity Readiness Questionnaire (PAR-Q); (4) had a resting blood pressure (RBP) less than 140 (systolic)/90 (diastolic) mmHg; (5) were physically active (muscle-strengthening activities involving major muscle groups on 2 or more days a week for more than 3 months); (6) were not taking any medications including anti-inflammatories and any supplements (7) did not have any orthopaedic injuries during the past 3 months.

5.2.2. Study design

This randomized, cross-over design study lasted for 8 weeks, where participants underwent six experimental trials. Figure 4. shows the study timeline. Participants in this study visited the research centre on two occasions during the first week (familiarization). The first visit was to provide a basic understanding of the study, as well as to get information mentioned in the selection criteria (5.2.1) for the study and to obtain anthropometric measures (weight and height). In the second visit, the resting blood lactate level was measured and then estimated maximal dynamic strength and power-load profiling tests in BP, half squat and hip thrust exercises were determined. Finally, the familiarization week was completed after a practical session on the use of the HR sensor and an explanation of the study protocol.

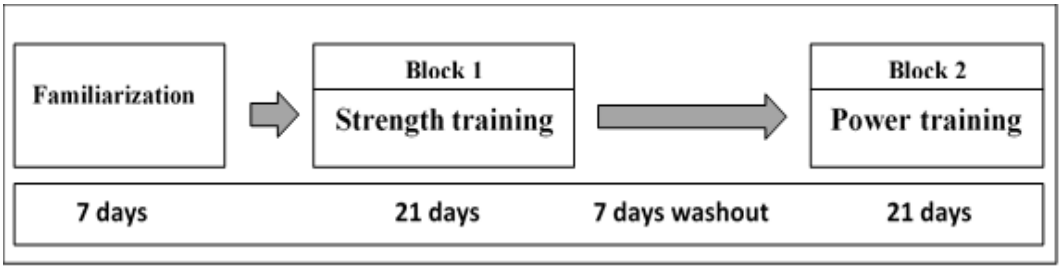


Figure 4. Research study timeline.

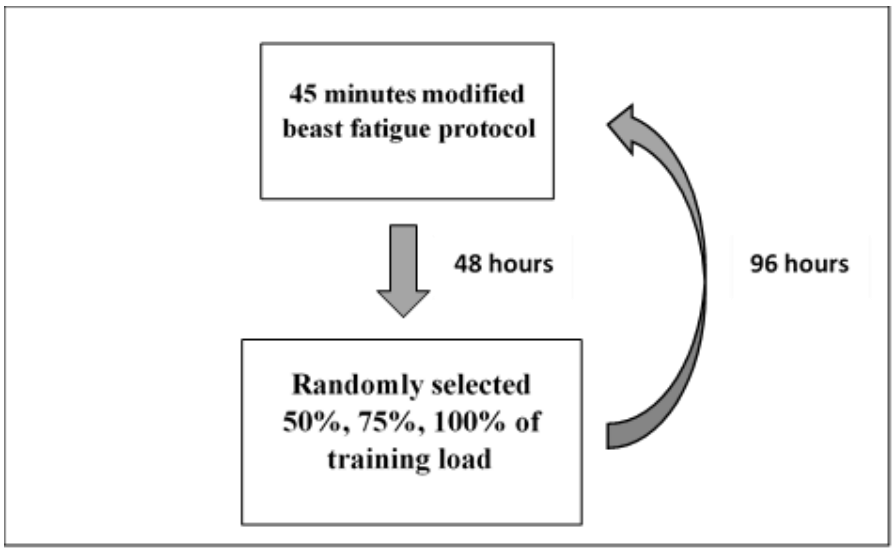


Figure 5. Each week within the block (Strength or power) for a given treatment scenario

Each block lasted for 3 weeks. For Block 1, all participants underwent strength training, and for Block 2, power training experimental trials. There was a 7 day washout period between blocks. In each block, there was a fatigue protocol visit, followed by a specific training load (100%, 75% or 50% of training load; experimental trials) in the subsequent visit, where the training load was assigned using manual randomization method (Figure 5). At the end of the study, all participants performed six experimental trials (i.e., 3 trials per training modality (strength and power). The same testing procedures were performed every week.

5.2.3. Testing procedures

This study was conducted in the UCAM Research Centre for High-Performance Sports (Murcia, Spain). Each experimental trial lasted for 5 days (Figure 6). On the morning of the first day of each experimental week, the participants rested for 10 minutes after arriving to the research centre. During this time, the POMS and Delayed onset muscle soreness (DOMS) scale were answered. Participants were then taken to a quiet, private room to measure RBP, resting HR, and RR intervals data for 10 minutes. Afterwards, they performed a standard 5-minute warm-up of light cycling on an upright exercise bike followed by 5 minutes of joint mobility exercises and dynamic stretching. Then, CMJ, maximal voluntary isometric contraction (MVC), electromyography activity (EMG) and BP power output tests.

Next, participants performed the 45 minute beast fatigue protocol under the supervision of the investigator. Blood lactate concentration (BLC) was measured immediately following the Beast protocol. Participants also repeated the CMJ, MVC, EMG and the BP power output tests soon afterwards. Once the post-measurements were conducted, participants rested for 10 minutes, and during this time, they answered the Borg rating of perceived exertion score (RPE), POMS questionnaires and DOMS scale. To finish the visit, participants returned to the quiet, private room to measure again the RBP, RHR and RR intervals data for 10

minutes. The order of the tests was kept the same for all the first visit morning sessions (Figures 7 and 8). Hereafter, from the moment the participant arrives at the research centre in the morning to the BP power output test, the process described above will be called as "pre-test" (Figure 7), and from the BLC measure to 10 minutes RR intervals data recording will be referred as "post-test" (Figure 8) for ease of reading.

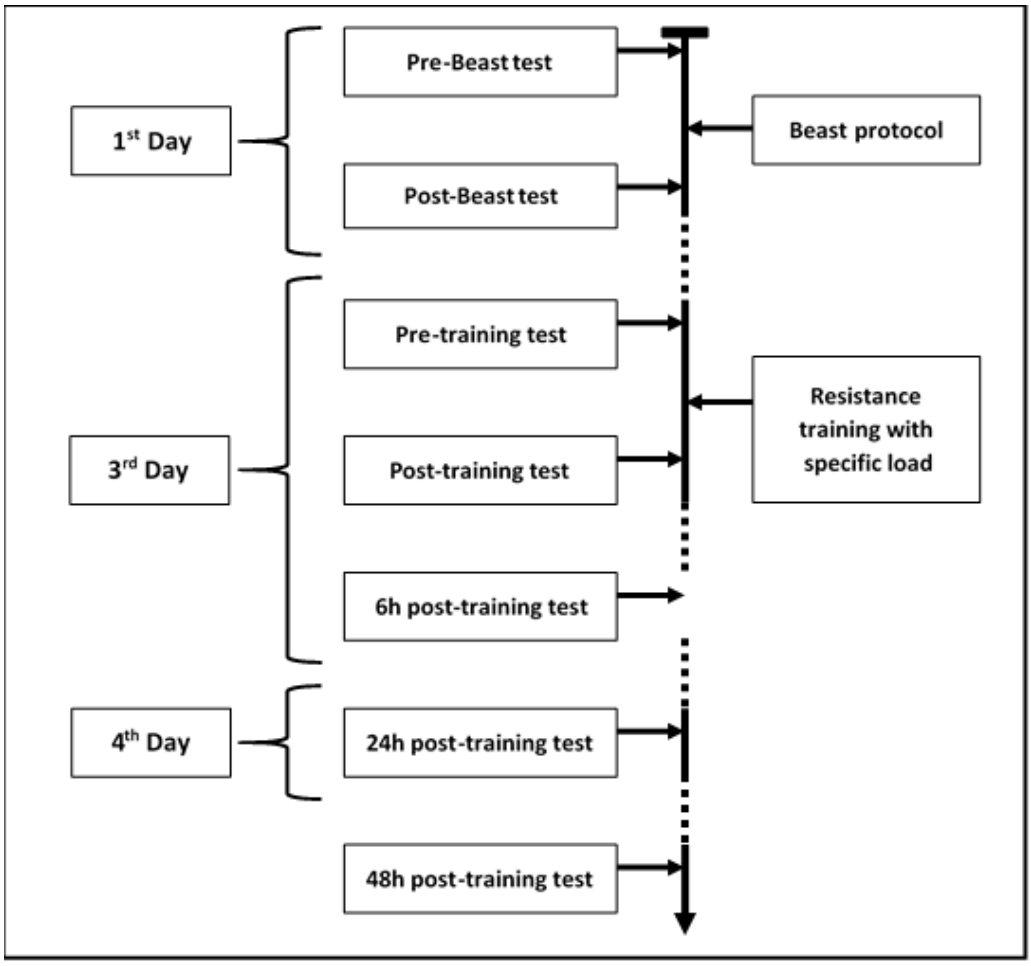


Figure 6. Scenario of one experimental trial (within a week)

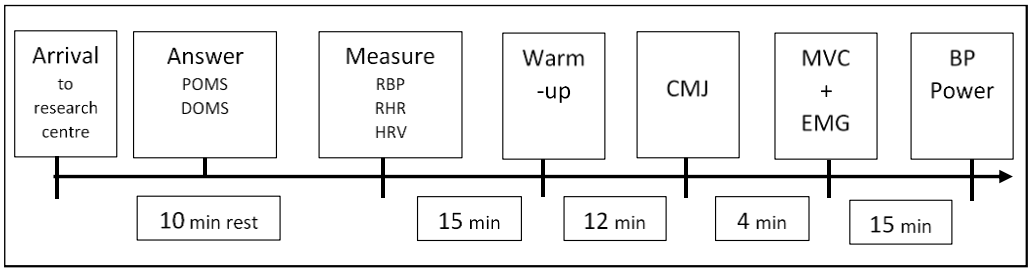


Figure 7. Pre-test measurements and timeline

Abbreviations: BP = Bench press; CMJ = Countermovement jump; DOMS = Delayed onset muscle soreness; EMG = Electromyography; HRV = Heart rate variability; min = minutes; MVC = Maximal voluntary isometric contraction; POMS = Profile of mood states; RBP = Resting blood pressure; RHR = Resting heart rate.

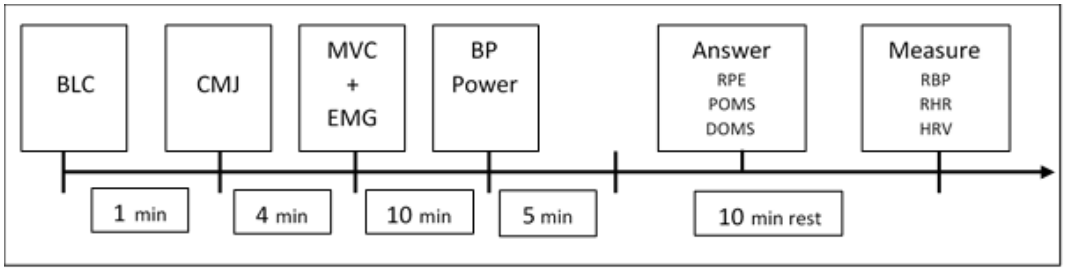


Figure 8. Post-test measurements and timeline

Abbreviations: BLC = Blood lactate concentration; BP = Bench press; CMJ = Countermovement jump; DOMS = Delayed onset muscle soreness; EMG = Electromyography; HRV = Heart rate variability; min = minutes; MVC = Maximal voluntary isometric contraction; POMS = Profile of mood states; RBP = Resting blood pressure; RHR = Resting heart rate.

After 48 hours (3rd day of the week), participants returned to the research centre early in the morning and completed the "pre-test" procedure (like on the first day). Afterwards, participants underwent the experimental trial (i.e., RT session), where the training load was randomly assigned by the investigator. Participants were only informed about the amount of weight, the number of repetitions, sets and resting time between sets used during the training session. The anticipated changes in "training volume" between weeks were not given to the

participants to minimize it's effect on the results. At the end of the RT session, participants completed the "post-test" measurements. Six hours after the RT session, the POMS Questionnaire, DOMS scale were administered and 10 minutes of RR intervals data were measured.

At post-24 hours (4th day of the week), participants repeated the "pre-test" measurements. At post-48 hours (5th day of the week), participants answered the POMS questionnaires and DOMS scale and 10 minutes RR intervals data were measured. In addition, participants were asked to record RR intervals data for 10 minutes every morning after they woke up from the start to finish of the study.

5.2.3.1. Maximal dynamic strength

The maximal dynamic strength of the lower and upper body was evaluated by estimating the 1RM value of BP, half-squat and hip thrust exercises. These exercises were performed using a modified Smith machine, where the linear encoder (Chronojump BoscoSystem, Spain) was connected to the barbell, and all data were recorded using Chronojump-BoscoSystem software. After the standardized warm-up session, participants performed 1 set of 10 repetitions with the minimum weight allowed by the machine (14 kg) and another 1 set of 10 repetitions with a submaximal weight based on their previous experience. Then, to estimate the 1RM of the BP and half-squat exercises, participants performed 3 repetitions with the weight of their predicted 5RM based on previous training experience. The participant was instructed to move the barbell as fast as possible in the concentric phase of the exercise. Two to 3 minutes of rest was given between sets and exercises, respectively. Manual randomization method was used to determine the order of the exercises for each participant, with the condition of that the BP was always the second exercise, to avoid performing two lower-body exercises consecutively. A spotter was present during the tests to ensure safety and proper technique.

Upper-body maximal dynamic strength was assessed using a 1RM BP. During the BP exercise, participants were instructed to lower the barbell in a

controlled manner (3 seconds count by the spotter) to the lowest position possible but not touching the chest during the eccentric phase and perform the concentric phase (upward movement) at maximum velocity (explosive manner). However, if the barbell touched the chest, the results of that effort was not considered as a valid attempt. The formula proposed by Jidovtseff *et al.* (239) was used to predict the 1RM value of the BP exercise.

$$\%\ 1RM\ of\ Bench\ Press = \frac{AV^{1} - 1.7035}{-0.0146}$$

Lower-body maximal dynamic strength was assessed using a 1RM half squat. During the half-squat exercise, participants were instructed to go downwards in a controlled manner (3 seconds count by the spotter; eccentric phase) until thighs were parallel to the floor (knees were at 90 degrees) and perform the concentric phase (upward movement) at maximum velocity (explosive manner). The formula proposed by Loturco *et al.* (240) was used to predict the 1RM value of the half squat exercise.

$$\%\ 1RM\ of\ Half\ squat = \left(-105.05\ \times MPV^{2}\right) + 131.75$$

Concerning the hip thrust, after performing the warm-up set with a submaximal weight, as mentioned above, the participant performed predicted 5RM weight based on previous training experience. After the initial set, the weight was adjusted for the next set, based on the number of repetitions the subject was performed. If the subject performed 6 repetitions, the weight was increased by

[1] AV = Average velocity

[2] MPV = Mean propulsive velocity

around 2%, and if it is ≥7 repetitions, the weight was increased by around 5%. Similarly, if participants performed only 4 repetitions, the weight was reduced by around 2%, and if participants only perform ≤3 repetitions, the weight was reduced by around 5%. The 5RM was assessed in 3 attempts for all participants with 2 min of rest between each attempt (241). The 1RM load was calculated using the Brzycki equation (242)

$$1RM = \frac{100 \times Weight^{3}}{102.78 - (2.78 \times Reps^{4})}$$

5.2.3.2. Power load curve

The power-load curve was conducted for BP, half squat and hip thrust exercises to identify the load that the participant could generate maximum power output. The relative weights of 30%, 45%, 60%, 75% and 90% of the previously estimated 1RM values of the respective exercises were used. Participants were instructed to perform the eccentric phase of the exercise in a controlled manner (3 seconds count by the spotter) and perform the concentric phase (upward movement) at maximum velocity (explosive manner) in each exercise. However, during the BP exercise, if the barbell touched the chest area, the results of that effort was not considered as a valid attempt. Participants performed 3 repetitions with each relative weight and 3 minutes of rest was given between each relative weight. Peak power output was recorded for each repetition, and the weight corresponding to its highest power output was considered for the power training. The order in which the exercises were performed was randomly selected for each player, with the condition of that the BP was always the second exercise, to avoid performing two lower-body exercises consecutively. A spotter was present during the tests to ensure safety and proper technique.

---

[3] Weight = Weight lifted by the participant in Kg

[4] Reps = Total number of repetitions completed (between 1 to 10)

5.2.3.3. RR intervals data collecting and analysis of HRV parameters

A Polar H10 HR sensor (Polar Electro Oy, Professorintie 5, FI-90440 Kempele, Finland) was used to measure RR intervals data. This technique has been validated against the gold standard ECG Holter device (243). The electrodes of the HR sensor was moistened with room temperature water to ensure good conductivity and strapped just below the chest muscles (under the garment and below the nipple line). The strap was adjusted accordingly for proper fit around the chest. In the supine position, resting RR interval data were recorded for 10 minutes and stored using the Elite HRV mobile application (Elite HRV Version 4.3, Asheville, North Carolina, USA) via Bluetooth 4.0 technology. Participants were asked to relax and not talk or move the body during the recording period. Additionally, participants were instructed to follow the visual guide for breathing using the "open readings" function of the mobile application. The controlled breathing rate (12 breaths per minute) and depth was applied because previous studies have reported that breathing rate and depth significantly affect the length of RR intervals (244). The room temperature was maintained at 26°C for all sessions.

In addition to the laboratory recordings, the RR intervals were assessed after waking up in the morning in the participant's home. Participants were instructed to empty the urinary bladder after waking up and before recording the RR intervals data, moisten the electrode of the HR sensor. Participants were previously educated and practised on how to wear the HR sensor and familiarized on how to use the Elite HRV mobile application. For each recording session, raw RR interval data were exported as a text file to a computer using Elite HRV's email exporting function.

Kubios HRV version 3.3.1 software (Biosignal Analysis and Medical Imaging Group, Department of Physics, University of Kuopio, Kuopio, Finland) was used to analysis the HRV parameters. The last 5 minutes of each of the 10 minutes RR interval data recordings were used for analysis. Prior to this, an artifact was corrected using the medium threshold artifact correction function. If the number of

sample artifacts of the relevant RR interval sample was more than 5%, the sample was not included for the study (artifacts acceptance threshold was 5%) and the next best 5 minutes RR interval data sample (from the end of the recording) were selected for analysis. Time and frequency domain data were applied to the linear analysis method. From the time domain parameters, SDNN, pNN50, RMSSD, the natural logarithm of RMSSD (Ln RMSSD) were determined. Normalized units of low frequency (LF(nu); 0.04–0.15 Hz), high frequency (HF(nu); 0,15–0,4 Hz), the ratio between LF/HF and the TP were acquired from the frequency domain. For non-linear measures, Poincaré plot standard deviation perpendicular the line of identity (SD1), Poincaré plot standard deviation along the line of identity (SD2), Ratio of SD2-to-SD1 (SD2/SD1) and Sample Entropy (SampEn) were calculated. These determination of these parameters are standard for HRV analysis by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (161). Moreover, Orellana *et al.* (187) proposed SS was calculated using the following equation.

$$SS = \left(\frac{1}{SD2}\right) * 1000$$

5.2.3.4. Countermovement jump test

CMJ test was conducted using Kistler 9286BA portable force platform (Kistler Group, Winterthur, Switzerland) with a sample rate of 1000Hz, and all data were recorded using ForceDecks software version 1.2.6464 (Neuromuscular performance technologies). Participant started each trial by standing in an upright position with feet placed shoulder width apart on the centre of the force platform. The participant was asked to perform a fast-downward movement to about 90 knee flexion (visually monitored knee angle) and instantly follow it with an explosive-upward vertical jump (with a rapid countermovement) as high as possible, all in one sequence. Also, they were asked to try and land on both feet with their balance centred on the force platform. Arm-swing was prohibited, as they were instructed to keep their hands on the hips throughout the trial.

Participants performed one trail as a warm-up jump and then executed two maximal CMJ on the force platform with one-minute rest between trails. The best jump height trial was considered. Jump height and RPP (245) were analysed because previous studies have demonstrated that they are accurate indicators of neuromuscular and metabolic fatigue.

5.2.3.5. Bench press relative power output test

The BP power output test was used to assess the upper body power production. The test was performed using a modified Smith machine, where the barbell was connected to the linear encoder (Chronojump BoscoSystem, Spain), and all data were recorded using Chronojump-BoscoSystem software. Participants performed 10 repetitions, with the minimum weight allowed by the modified Smith machine (only barbell – 14 kg), to warm-up before starting the testing session. After two minutes of rest, participants were instructed to perform the eccentric phase of the exercise in a controlled manner (3 seconds count by the spotter) and execute the concentric phase (upward movement) of the BP exercise at maximum velocity (explosive manner). However, if the barbell touched the chest area, the results of that effort was not considered as a valid attempt. Three trials of BP power output test were performed with half of the bodyweight of the participant for resistance and peak power output, where the best repetition was considered for the study. RPP was calculated by dividing peak power output by body mass. A spotter was present during the tests to ensure safety and proper technique.

5.2.3.6. Resting blood pressure

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using an automated oscillometric blood pressure device (OMRON HEM-7203-AP, OMRON Healthcare Co. Ltd, Japan.) and performed prior to the recordings of the RR intervals. The data were recorded in a relaxed, calm environment free of any disturbances. Room temperature was maintained at 26°C.

The equipment was auto calibrated before each use. Participants were asked to remain relaxed and remained in the seated position and not to talk during the measurement. Measurements were performed on the left arm, following the American Heart Association's recommendations (246).

5.2.3.7. Neuromuscular function / fatigue test

Neuromuscular function / fatigue test setup

EMG activity during an MVC was assessed to evaluate central and peripheral fatigue. Each participant practiced the MVC and electrical stimulation protocol during the familiarization session. Two pre-gelled Ag-AgCl single-use ECG electrodes (Ambu® BlueSensor N – Ambu A/S, Denmark) were attached on the surface of Vastus Lateralis (VL) muscle of the right leg with an inter-electrode distance of 20 mm. Electrode placement was marked with a permanent marker to ensure consistent placement during the study. Before attaching the electrodes, the skin surface was shaved and cleaned with alcohol. The electrode placement and skin preparation were performed as recommended by the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project (247) by the European concerted action in the Biomedical Health and Research Program (BIOMED II-Program). Then, participants were asked to sit on the Biodex System 3VR dynamometer (Biodex medical, Shirley, New York). Conductive gel (Electro-Gel, Telic, S.A, Barcelona, Spain) where cathode and anode pads (11 * 8 cm) were placed over the upper and lower quadriceps femoris muscle group, respectively. Velcro bands were used to fix them to the skin. The quadriceps muscle was stimulated using Signal 6.02 software and the constant current stimulator (Digitimer DS7H, Digitimer, Welwyn Garden City, UK). The participant was securely strapped to the dynamometer chair in the seated, upright position. The hip and knee were fixed at 90 degrees of flexion, and the ankle was secured to the arm of a custom-built apparatus, which was connected with the force transducer (Model SML-500, Interface, Scottsdale, AZ, USA). Wireless DTS force sensor

(Noraxon USA INC, Scottsdale, AZ, USA) received the signal from the force transducer to transmit force production of MVC

Wireless DTS EMG sensor electrode (Noraxon USA INC, Scottsdale, AZ, USA) was placed over the belly of the vastus lateralis muscle. Another velcro band was used to fix the EMG sensor over the skin, which minimized displacement of the EMG sensor and reduce movement artifacts from the cables (95, 247). Noraxon EMG TeleMyo DTS Desk receiver (Noraxon USA INC, Scottsdale, AZ, USA) was used to receive the data sent by the EMG sensor and force sensor. On the first day of every trial (each week), maximal stimulus intensity was determined for each subject by applying a single pulse at progressively higher intensity amplitudes until 50% of the highest peak force from the previously measured MVC (Baseline MVC) was achieved. This amplitude value was used as for the stimulated supramaximal train for the CAR protocol.

Force measurements protocol

Participants performed two MVCs that lasted around 5s with 2-minutes of rest between each attempt. Participants were asked to perform the MVC as rapidly and forcefully as possible, and verbal encouragement was given for every attempt. All the force and EMG data were synchronously acquired using the Noraxon MR 3.6.20 software (Noraxon, Scottsdale, AZ, USA) and stored in the laptop. EMG activity signals were processed using Filtering (Filter: FIR, Window: 79 points, Type: Bandpass, Low frequency: 20Hz, High frequency: 500Hz, Window: Lancosh), Rectification and Smoothing (Algorithm: RMS, Window: 100ms) methods. All the raw data were exported, and peak force and rate of RFD during the first 200ms were calculated with Microsoft Excel (Microsoft Corporation, Redmond, WA, USA). The data of the highest peak force attempt was used for this study.

Central and peripheral activation protocol

To determine central and peripheral activation, participants performed the following protocol (Figure 9) 2 times with 2-minute rest in between. The quadriceps were stimulated by applying first a single 50-Hz 1s train (Twitch), which was followed by a supramaximal train of stimuli (50 Hz, 250ms) during the plateau of a 5s MVC (superimposed tetanus). Then a tetanic train (potentiated train; 50 Hz, 250ms) was applied while the subject remained relaxed, followed by another single supramaximal stimulus (potentiated twitch). Three seconds of rest was given between each stimulated measurement. Raw data were exported and analysed using AcqKnowledge 3.9.1 (BIOPAC Systems Inc, CA, USA) and Microsoft Excel (Microsoft Corporation, Redmond, WA, USA).

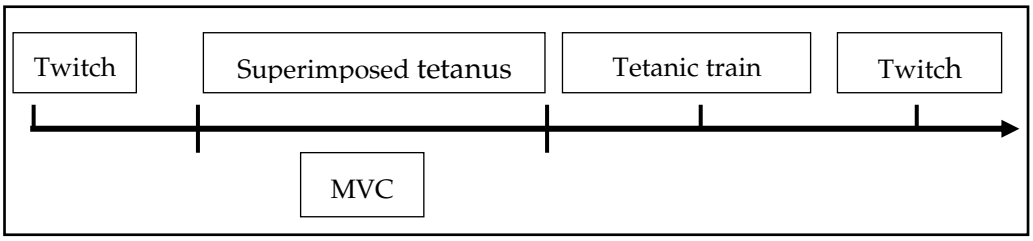


Figure 9. Central and peripheral activation protocol.

Assessing the neuromuscular, central and peripheral fatigue

Changes in MVC peak force related to the baseline was determined as an indicator of neuromuscular fatigue level (248). CAR (249) and MVC-to-tetanic ratio (MVC force/ tetanic force) (250) compared to the baseline were considered as an indication of central fatigue. Peripheral fatigue was assessed by changes in tetanic force and twitch force compared to baseline (251). CAR was calculated using the peak force production before the superimposed tetanus divided by the peak force production during the superimposed tetanus (Total force) (249).

$$CAR = \frac{\text{peak force production before the superimposed tetanus}}{\text{peak force production during the superimposed tetanus (Total force)}}$$

Contractile function

Peak force production, the maximum RFD, the maximum RFR, and the half-time of force relaxation ($T_{1/2}$) were calculated from the tetanic train. The $T_{1/2}$ was measured as the time needed for force to decline to 50% of the potentiated peak force during the relaxation phase. Furthermore, twitch peak force production and twitch-to-tetanus ratio (Tw/Tet) was calculated as an indicator of low-frequency fatigue in the contraction protocols (252).

5.2.3.8. Delayed on set muscle soreness

Visual Analogue Scale (VAS) was used to quantify the level of muscle soreness of the participants. VAS method is most often used during the previous studies to assess DOMS (253-256) and have reported as highly reliable and validated (257-259). Using a 100-mm horizontal line, participants were instructed that 0 indicated "*No pain*" and 100 represented "*Extreme pain*". Each participant was asked to mark a vertical line to indicate their level of muscle pain. The level of pain was calculated using the distance from the "0" to the vertical mark.

5.2.3.9. Borg CR-10 scale of rating of perceived exertion

The Borg Category Ratio-10 rating of perceived exertion (RPE) scale was utilized to quantify the feeling of exertion of the physical activity performed by the subject.

The scale consists of 11 points on a Likert scale from 0 to 10 ("Nothing at all" to "Very, very hard"). RPE scale was explained to the participants prior to the start of the study. Around 30 minutes after the 45 min beast fatigue protocol or RT session, RPE test was conducted, as recommended by Foster *et al.* (25) and Day *et al.* (260). Participants answered based on the following question: "*How was your workout?*" to gauge their sensation of physical stress and fatigue level.

5.2.3.10. Blood lactate concentration

A capillary blood sample was collected from a fingertip. Before collecting the blood, the selected area was cleaned and disinfected with alcohol. After that, it was dried with cotton and sterile gauze so that there were no remains of any substance that could contaminate the blood sample. The puncture was performed with a sterile and single-use disposable lancet (MenaLancetPro, Leczyca, Poland). The first drop of blood was discarded, and the second drop was considered as the sample for each measurement. The blood was introduced into a test strip, which was analysed by a portable lactate analyser, Lactate Pro 2 LY-1730 model (Arkray, Kyoto, Japan), which had previously been calibrated using a calibration strip, following the manufacturer's instructions. Blood samples were collected on the 1st day before the warm-up, just after 45 minutes of the beast fatigue protocol and on the 2nd day just after the RT session in each trial. During the whole process, recommended hygiene practices were followed, and all the biological samples and sharp elements were disposed of in a special container.

5.2.3.11. Profile of mood states questionnaire (POMS)

The revised POMS questionnaire (261) was used to evaluate the mood states of the participants during the study. POMS is reliable and valid for use in a sport setting (261). The questionnaire consists of 40 adjectives with 5-point Likert scale ("Not at all" to "Extremely"), assessing 7 factors (tension, depression, anger, vigour, fatigue, confusion and esteem) related to the mood states.

5.2.4. Training protocol

5.2.4.1. 45 minutes Beast fatigue protocol (M-BEAST)

The modified 45-minute Beast fatigue protocol (M-BEAST) (262) is a modified version of the Ball-sport Endurance And Sprint Test (BEAST90) (263) and was used to induce fatigue on the 1st day of each trial. During the familiarization session, M-

BEAST protocol was explained, and a practical session was conducted. A summary of the M-BEAST session is presented in Figure 10.

This procedure was conducted in the Gymnasium of the UCAM Sports Centre, Murcia, Spain. It involves continuous walking, jogging, 80% of speed running, 100% of speed sprinting, maximum speed side shuffle between corns and maximum jump to head the imaginary ball (Figure 11). Two locations of the court were used for this purpose.

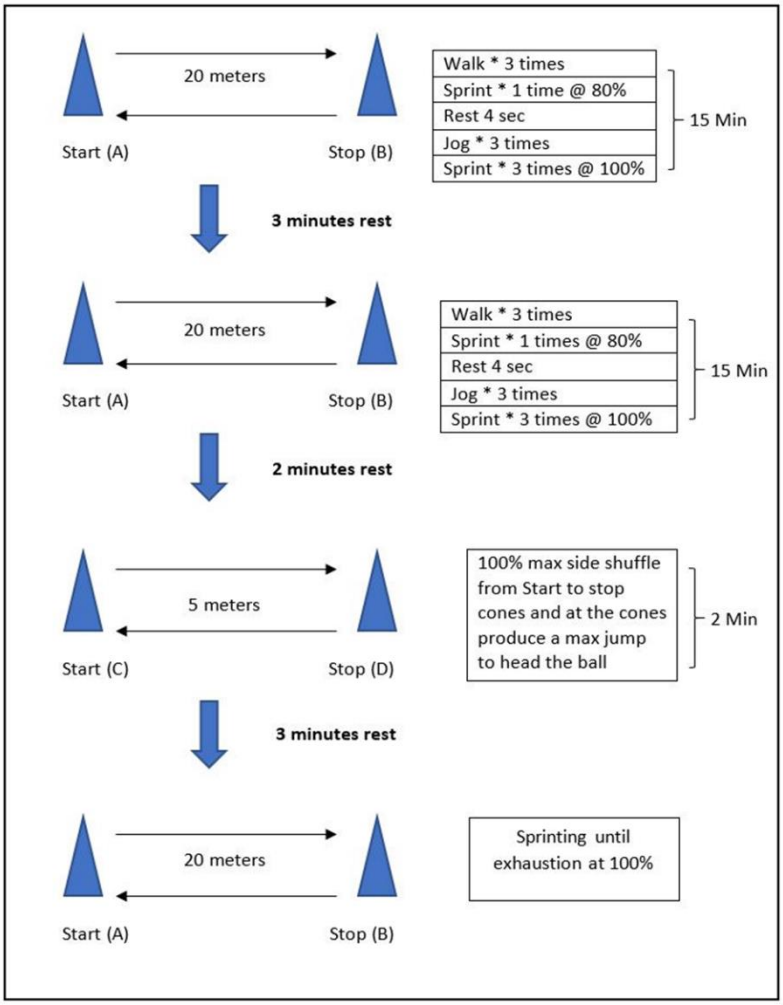


Figure 10. 45 min modified Beast fatigue protocol (Source: Martyn Matthews (262))
Abbreviations: Min = minutes; sec = seconds

In Location 1, two cones (A and B) were placed 20m apart, while Location 2 had two cones (C and D) placed 5m apart. The procedure began at Location 1 where participants walked the 20m length 3 times continuously (A to B, B to A and A to B), and then participants sprinted one time at 80% maximum speed from B to A cone. Thus, the participants performed the activities illustrated in Figure 11 for 15 minutes continuously. After a 3-minute break, the same activities were repeated for another 15 minutes.

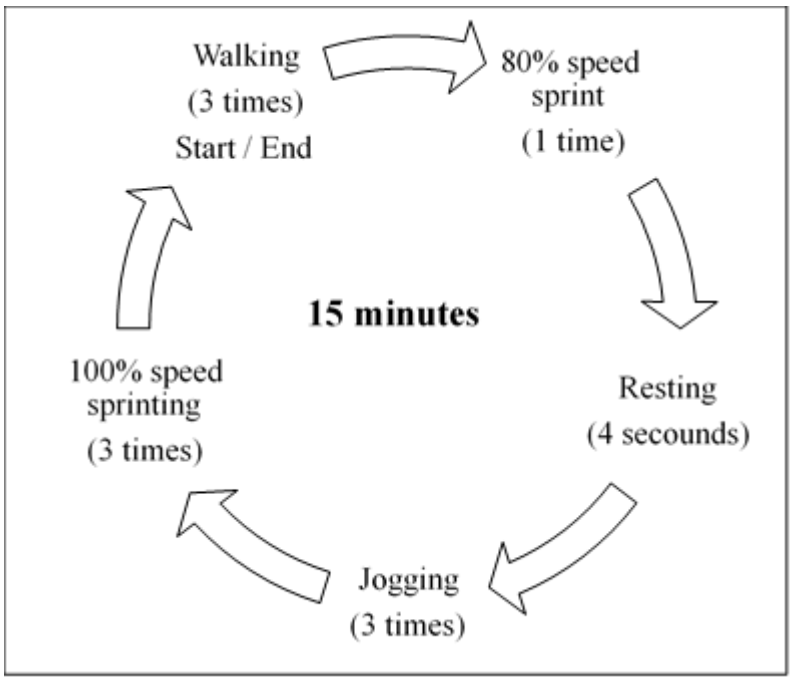


Figure 11. Process of Location 1 in the M-BEAST protocol

Participants then proceeded to the second location after resting for 2 minutes. They performed 100% maximum side shuffle between the 5m distanced cones, where afterwards they had to perform maximum jumps to head an imaginary ball for another 2 minutes. After that, they went back to Location 1 with another 3 minutes of rest and started sprinting 100% maximum speed between the 20m distanced cones for 5 minutes or until they were exhausted. All M-BEAST sessions

were supervised by the investigator, and verbal encouragements were given to the participants throughout the protocol.

5.2.4.2. Resistance training protocol

BP, hip thrust, and half squat exercises were performed during the RT program in every trial during the study. During the 1st block, all the participants performed strength training, and, in the 2nd block, they performed power training with different training loads.

5.2.4.3. Strength training

During the familiarization session, participants performed 5 repetitions with 90% of estimated 1RM for each exercise. For each trial, they were instructed to perform a different number of sets that was pre-randomized (4, 3 sets or 2 sets) with 4 minutes of rest between sets. Furthermore, for each repetition, the eccentric phase was executed over 1 second and the concentric phase was conducted in a slow, controlled manner over 3 seconds (1:3).

5.2.4.4. Power training

Similar to strength training, participants performed 5 repetitions with a pre-randomized number of sets for each exercise for each trial. Optimal load was determined during the familiarization session for each exercise and was used as the resistance load for each exercise with 3 minutes of rest between sets. The investigator was instructed to move the barbell as fast as possible in the concentric phase of the exercise. Table 4. summarizes the characteristics of the RT protocol.

Table 4. Characteristics of the resistance training protocol

	Sets	Repetitions	Resistance	Rest
Strength training				
100 % of the training load	4	5	90% 1RM	4 min
75 % of the training load	3	5	90% 1RM	4 min

50 % of the training load	2	5	90% 1RM	4 min
Power Training				
100 % of the training load	4	5	OL	3 min
75 % of the training load	3	5	OL	3 min
50 % of the training load	2	5	OL	3 min

Abbreviations: OL = Optimal load; 1RM = 1 Repetition maximum

5.2.5. Statistical analysis

Statistical analysis was conducted using IBM SPSS Statistics 23.0 for Windows (IBM SPSS Inc., Chigaco, IL, USA). All the data were analyzed using absolute values and expressed as mean ± standard deviation (SD), unless otherwise stated. The statistical significance level was set at $p \leq 0.05$, while $p \leq 0.06$ was accepted as a significant trend. Normality was assessed using the Shapiro-Wilk test of normality on the studentized residuals, and outliers were assessed by no studentized residuals greater than ± 3 standard deviations. If data were not normally distributed, transformation methods were used. The assumption of sphericity was assessed by using Mauchly's test of sphericity ($p > 0.05$). A two-way repeated-measures ANOVA test was performed to determine whether there was a significant two-way interaction (group*time) of the dependent variable.

If the results indicated that there was a significant two-way interaction, the simple main effect for treatment (between treatments at each time points) and time points (between time points at each treatment) were tested using one-way repeated measure ANOVA. In the case of statistically significant difference, post-hoc analyses were performed with the Bonferroni adjustments to determined the differences between treatments and over time points. If the results indicated that there was no significant two-way interaction, but there was statistically significant main effects of treatment and/or time, a one way repeated measure ANOVA test

was performed. Spearman's correlations were used to quantify relationships between changes (Post-B / Pre-T / Post-T / Post-24H – Pre-B) in Ln RMSSD, performance, neuromuscular, central, peripheral, and perceptual markers. Cohen's *d* effect size (ES) was calculated to determine the magnitude of differences between the time points ($d = 0.20$ – small, $d = 0.50$ – moderate, $d \geq 0.8$ – large as magnitude thresholds).

VI - RESULTS

VI. RESULTS

In this chapter, the results of the Systematic review, meta-analysis and experimental study are presented. Accordingly, this chapter is divided into two main sections. The first section presents systematic reviews and meta-analyses results (Study 1), while the other section presents the results of the experiment study (Study 2). Furthermore, the results of the systematic reviews and meta-analyses study presented under several subsections, namely, study selection, characteristics of the interventions, heterogeneity and risk of bias assessment, main effects analysis and subgroup analysis results. Results of the experimental study also reported under several subsections: those are Participants, HRV parameters, performance variables and physical functions, neuromuscular fatigue, central fatigue, peripheral fatigue, perceptual responses, other and time-course of recovery monitoring using different monitoring tools.

6.1. STUDY 1[5]

Under the subsection of study selection, reported the number of articles found from initial electronic database search and other sources, and the final number of articles included after performing the inclusion and exclusion criteria. On the characteristics of the interventions, reported the descriptive characteristics of the participants and methodological characteristics of the selected studies. Variation in study outcomes between selected studies (heterogeneity) and quality

[5] Marasingha-Arachchige SU, Rubio-Arias JÁ, Alcaraz PE, Chung LH. Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis. Journal of Sport and Health Science. 2020.

of the selected studies reported under the heterogeneity and risk of bias assessment subsection.

To achieve one of the primary objectives of this doctoral thesis, firstly reported the main effects analysis results, which was to determine whether and if so, how ARE effect on HRV parameters (SDNN, RMSSD, HF(nu), LF(nu) and LF/HF ratio) in the previously published studies. Secondly, subgroups analysis results were reported to determine whether and how ARE effects vary among the different subject's characteristics and training characteristics on the HRV parameters.

6.1.1. Study selection

From the initial electronic database search and other sources, 1449 records were identified. After removal of duplicates, 1076 titles and abstracts were evaluated, and 1003 were excluded. Thus, the full text of 73 articles was assessed to determine eligibility for the inclusion of studies, and 2 additional studies were screened as a result of reviewing the reference lists. From these studies, 49 articles were excluded because they did not meet the inclusion criteria. After review, a total of 26 studies were included in the systematic review and meta-analysis (36, 37, 40, 42, 43, 152, 208-212, 216, 218-221, 223-226, 228-231, 264, 265). All included articles were published between 2006 and 2019 (Figure 12).

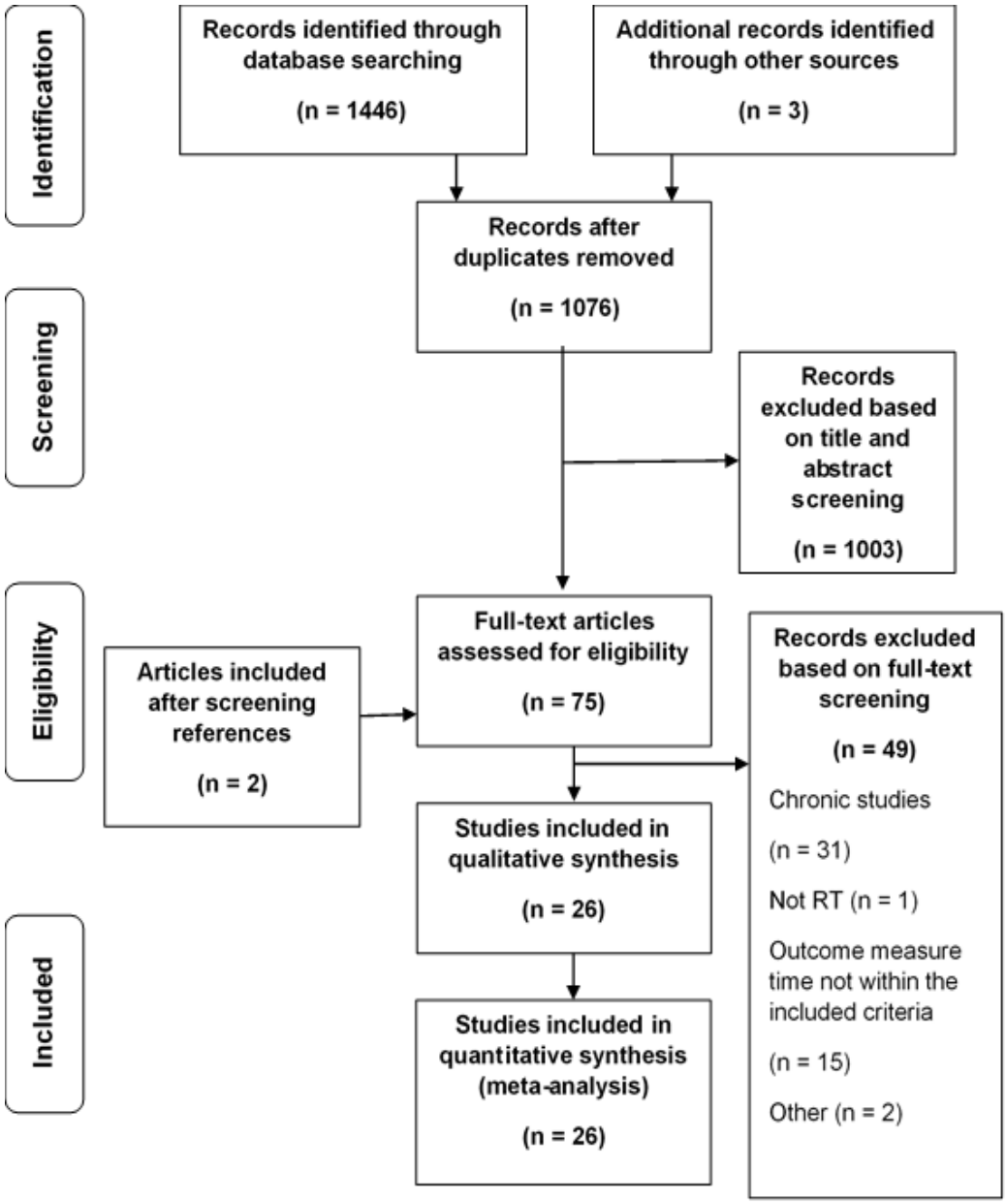


Figure 12. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram regarding article selection for each stage of the systemic eligibility process. RT = resistance training.

6.1.2. Characteristics of the intervention

Participants were healthy and physically active, and the majority were resistance-exercise-trained individuals. Their age ranged (SD) between 15 ± 1 and 48 ± 2 years. The samples included both males and females. BMI (SD) ranged from 20.0 ± 1.0 kg/m^2 to 27.5 ± 2.1 kg/m^2, although some studies did not report BMI values. The sample sizes in the included studies ranged from 8 to 34 participants. Among the included studies, there were a total of 412 participants for this systematic review and meta-analysis.

The amount of exercise performed during the RT sessions ranged from 1 to 8 exercises. The intensity of the resistance exercises performed ranged from bodyweight to 100% 1RM. Among these studies, 13 study groups performed at low intensity (≤65% 1RM), 25 performed at moderate intensity (>65% to 85% 1RM), and 3 performed at high intensity (>85%) (Table 5). With regards to measuring HRV parameters, most of the studies used Polar HR monitors and ECG monitors, with participants in a supine or seated position for 5–15 min. Additionally, most of the studies identified and corrected for or excluded the abnormalities (ectopic/artefacts) of beat-to-beat interval data before analyzing the HRV parameters. HRV measurement and data analyzing methods used in the included studies are presented in Table 6.

Table 5. Characteristics of the ARE sessions

Study	Participants	Control group	Training status	Age (years)	Protocol				
					N of exercises	N of sets	N of reps	Intensity (% 1RM)	Rest between sets
Macêdo et al., 2019 (42)	34M: Healthy weight 19, Overweight 15	No	Adolescent	Healthy weight: 15 ± 1 Overweight: 16 ± 1	5	3	12	60	-
Thamm et al., 2019 (152)	10M	No	RT	24 ± 3.8	1	5	10	70	120 s
					1	15	1	100	180 s
Lima et al., 2019 (218)	12M	Yes (not healthy)	normotensive	25.5 ± 5.7	1			1RM test	
Kingsley et al., 2018 (212)	27: 14M, 13F	Yes	RT	Men: 22 ± 3 Women: 23 ± 3	3	3	10	75	120 s
Monteiro et al., 2018 (40)	8F	No	Recreationally-RT	21.8 ± 2.2	4			1RM test	
De Freitas et al., 2018 (221)	16M	No	Recreationally-RT	24.9 ± 5.3	6	3	10	65	90 s
					3	6	10	65	90 s
					3	6	10	65	90 s
Paz et al., 2017 (211)	13M	No	RT	26.2 ± 3.9	6	3	10	75	90 s
					6	3	10	75	90 s
					6	3	10	75	180 s
Corrêa et al., 2017 (223)	24M	Yes	RT	20.5 ± 0.6	1	4	UF	67	180 s
				25 ± 4.1	1	4	8	67	180 s
Xavier et al., 2017 (264)	29M	No	Physically active	21.62 ± 2.63	3	3	12	70	-
Figueiredo et al., 2016 (225)	11M (Pre-hypertensive)	No	RT	26.1 ± 3.6	8	3	8 - 10	70	120 s
					8	3	8 - 10	70	60 s

Table 5. (Continued)

Kingsley et al., 2016 (37)	16: 11M, 5F	No	RT	23 ± 3	3	3	12	70	120 s
Kliszczewicz et al., 2016 (224)	10M	No	Physically fit	26.4 ± 2.7	3	-	-	-	-
Mayo et al., 2016b (36)	13M	Yes	RT	23 ± 3	1	5	34	75	720 s
					1	5	23.6	75	720 s
					1	5	32	75	720 s
					1	5	22.9	75	720 s
Mayo et al., 2016a (220)	17: 12M, 5F	Yes	RT	23 ± 3	1	5	8	80	180 s
					1	10	4	40	30 s
					1	40	1	10	18.5 s
Iglesias et al., 2015 (226)	10M	No	RT	23 ± 4	1	3	UF	90	180 s
					1	3	-	90	-
Kingsley et al., 2014 (210)	34: Trained (9F, 8M), Untrained (7F, 10M)	Yes	Trained: whole-body RT for 6 ± 2 yrs, > 3 days a week. Untrained: not participated in RT for at least 1 year	Trained: 22 ± 1 Untrained: 22 ± 2	4	3	10	75	120 s
Okuno et al., 2014 (229)	9M	No	RT	24 ± 2.9	1	5	8 and last set UF	80	60 s
					1	5	16 and last set UF	40	60 s
Saccomani et al., 2014 (228)	10M	Yes	RT	24.5 ± 1.1	8	3	12	60	120 s
Tibana et al., 2013 (230)	9F	Yes (not healthy)	Sedentary	35.0 ± 6.7	6	3	10	60	120 s

Table 5. (Continued)

Fabiana et al., 2013 (231)	10M	No	Physically-active	23 ± 2	4	3	UF	75	120 s
					4	3	UF	75	120 s
					4	3	UF	75	Self-suggested
Teixeira et al., 2011 (219)	20: 10M, 10F	Yes	Healthy, normotensive	26 ± 1	6	3	20	50	45 s; 90 s btwn exer
Lima et al., 2011 (265)	15M	Yes	Healthy	22.2 ± 3.2	5	3	12,9,6	50	120 s
					5	3	12,9,6	70	120 s
Kingsley et al., 2010 (43)	15M	Yes (not healthy)	Healthy	45 ± 5	1	5	10	75	90 s
Kingsley et al., 2009 (209)	9M	Yes (not healthy)	Healthy	48 ± 2	10	1	12	60	-
Rezk et al., 2006 (216)	17: 8M, 9F	Yes	Healthy	23 ± 1	6	3	20	40	45 s; 90 s btwn exer
					6	3	10	80	45 s; 90 s btwn exer
Heffernan et al., 2006 (208)	14M		Moderately active		8	3	10	75	90 s

Abbreviations: 1RM = 1 repetition maximum; Btwn = between; exer = exercise; F = females; M = males; RT = resistance-trained; s = seconds; UF = until failure; yrs = year.

Table 6. HRV measurements and data analysing methods

Study	HRV measurement					Data analysing		
	Tool used to measure	Position	Duration of measurement	Respiration control	Analysed time duration	Abnormal beat-to-beat interval identification method	Ectopic/artefacts beats correction method	Software
Macêdo et al. 2019 (42)	Polar RS800 HR monitor	Seated	10 min	N/A	10 min	Visual inspection	Manual correction	Kubios HRV analysis
Thamm et al. 2019 (152)	Firstbeat Bodyguard 2 (ECG)	Supine	10 min	N/A	2 min	Manual inspection with support of internal algorithms	>10% artifacts were excluded	ARTiiFact version 2
Lima et al. 2019 (218)	N/A	Supine	10 min	N/A	1,000 consecutive R-R intervals	N/A	N/A	Kubios HRV analysis version 2.2
Kingsley et al. 2018 (212)	Modified CM5 configuration	Supine	5 min	12 breaths/min	5 min	Visual inspection	Interpolated	WinCPRS
Monteiro et al. 2018 (40)	Polar RS800cx HR monitor	Supine	15 min	N/A	5 min	N/A	Period of greater signal stability	Kubios HRV analysis version 2
de-Freitas et al. 2018 (221)	Polar RS800 HR monitor	Supine	5 min	breathing spontaneously	5 min	Digital filtering using Polar software + manual filtering	>95% sinoatrial node beats were included	Kubios HRV

Table 6. (Continued)

Paz et al. 2017 (211)	Polar RS800cx HR monitor	Seated	5 min	12 breaths/min	5 min	N/A	N/A	Kubios HRV analysis version 2
Corrêa et al. 2017 (223)	Polar RS800 HR monitor	Seated	10 min	N/A	10 min	N/A	N/A	Matlab
Xavier et al. 2017 (264)	Polar RS800cx HR monitor	N/A	10 min	N/A	10 min	Manual filtering	sinus rhythm >95% were included	Kubios HRV analysis
Figueiredo et al. 2016 (225)	Polar RS800cx HR monitor	N/A	10 min	N/A	10 min	N/A	N/A	Matlab
Kingsley et al. 2016 (37)	Modified CM5 configuration	Supine	5 min	12 breaths/min	5 min	N/A	N/A	WinCPRS
Kliszczewicz et al. 2016 (224)	Modified lead II configuration with Biopac MP100 data acquisition system	Supine	10 min	N/A	5 min	Visual inspection	Ectopic/nonsinus beats replaced by the adjacent R-R interval	Specialized HRV software
Mayo et al. 2016b (36)	Polar RS800cx HR monitor	Seated	10 min	breathing spontaneously	5 min	Automatic	Medium-level automatic artifact correction	Kubios HRV analysis version 2.1
Mayo et al. 2016a (220)	Task Force monitor	Seated	10 min	breathing spontaneously	5 min	Automatic	Medium-level automatic artifact correction	Kubios HRV analysis version 2.1
Iglesias et al. 2015 (226)	Suunto memory belt	Seated	10 min	breathing spontaneously	8 min	Automatic	Strong-level automatic artifact correction	Kubios HRV analysis

Table 6. (Continued)

Kingsley et al. 2014 (210)	Modified CM5 configuration	Supine	5 min	12 breaths/min	5 min	Visual inspection	N/A	AcqKnowledge 4.2
Okuno et al. 2014 (229)	Polar RS800cx HR monitor	Seated	5 min	Not controlled	5 min	Visual inspection	Manual correction	Kubios HRV analysis version 2
Saccomani et al. 2014 (228)	Polar S810i HR monitor	Seated	10 min	N/A	10 min	N/A	N/A	Kubi HRV analysis version 2
Tibana et al. 2013 (230)	Polar S810i HR monitor	N/A	5 min	N/A	5 min	Manual inspection	Excluded artifacts	Kubios HRV analysis version 2
Fabiana et al. 2013 (231)	Polar RS800cx HR monitor	Seated	5 min	N/A	5 min	N/A	N/A	N/A
Teixeira et al. 2011 (219)	ECG (TEB, D10)	N/A	10 min	N/A	10 min	N/A	N/A	PRE software and LA software
Lima et al. 2011 (265)	Polar RS800cx HR monitor	Seated	10 min	Not controlled	10 min	N/A	N/A	Kubios HRV analysis
Kingsley et al. 2010 (43)	Modified CM5 configuration with a Biopacd data acquisition system	Seated	5 min	12 breaths/min	5 min	Visual inspection	N/A	WinCPRS

Table 6. (Continued)

Kingsley et al. 2009 (209)	Modified CM5 configuration with a Biopacd data acquisition system	Seated	5 min	12 breaths/min	5 min	Visual inspection	Linearly interpolated	WinCPRS
Rezk et al. 2006 (216)	ECG (TEB, D10)	N/A	10 min	N/A	10 min	N/A	N/A	Levinson–Durbin recursion
Heffernan et al. 2006 (208)	Modified CM5 configuration	Supine	10 min	12 breaths/min	10 min	Visual inspection	Linearly interpolated	WinCPRS

Abbreviations: ECG = Electrocardiogram; HR = Heart rate; N/A = Not available; R-R interval = interbeat intervals between all successive heartbeats.

6.1.3. Heterogeneity and risk of bias assessment

Except for SDNN ($I^2 = 47\%$, $p = 0.06$), heterogeneity was present for changes in RMSSD ($I^2 = 71\%$, $p < 0.001$), LF(nu) ($I^2 = 83\%$, $p < 0.001$), HF(nu) ($I^2 = 85\%$, $p < 0.001$), and LF/HF ratio ($I^2 = 40\%$, $p = 0.03$) parameters among the pre-post intervention studies. Regarding control group interventions, heterogeneity was detected in LF(nu) ($I^2 = 86\%$, $p < 0.001$), HF(nu) ($I^2 = 80\%$, $p < 0.001$), and LF/HF ratio ($I^2 = 78\%$ $p < 0.001$), but not in RMSSD ($I^2 = 26\%$, $p = 0.24$).

The quality of the studies, according to the National Heart, Lung, and Blood Institute Study Quality Assessment Tools (235), was high for the pre-post interventions (8.18 ± 0.53, out of a possible 12 points) and experimental-control interventions (9.56 ± 0.53, out of a possible 14 points) (see Appendix 13.1.2 Table 11-12 which illustrates the results of study quality). A funnel plot asymmetry test was used to determine publication bias. Visual interpretation of the funnel plot asymmetry tests (SMD values between pre-post tests and control-experimental tests) showed that SDNN, RMSSD, LF(nu), HF(nu) and LF/HF ratio variables were asymmetrical, suggesting the presence of publication bias) (see Appendix 13.1.1 Figure. 160–168, which illustrate the results of the funnel plot asymmetry tests).

6.1.4. Main effects analysis

6.1.4.1. RMSSD

There were 18 ES calculations from 15 studies (mean age = 23.5 years; 199 males, 42 females) that showed a decrease in RMSSD ($p < 0.001$; SMD = –1.01; 95%CI: –1.29 to –0.74) of ~30 min (8–30 min) after the ARE session compared to pre-test values. There were 6 ES calculations from 4 studies (mean age = 22.3 years; 64 males, 58 females) that demonstrated a decrease in RMSSD ($p < 0.001$; SMD = –0.75; 95%CI: –1.01 to –0.49) post ~30 min (8–30 min) for ARE session compared to control groups (Figure. 13).

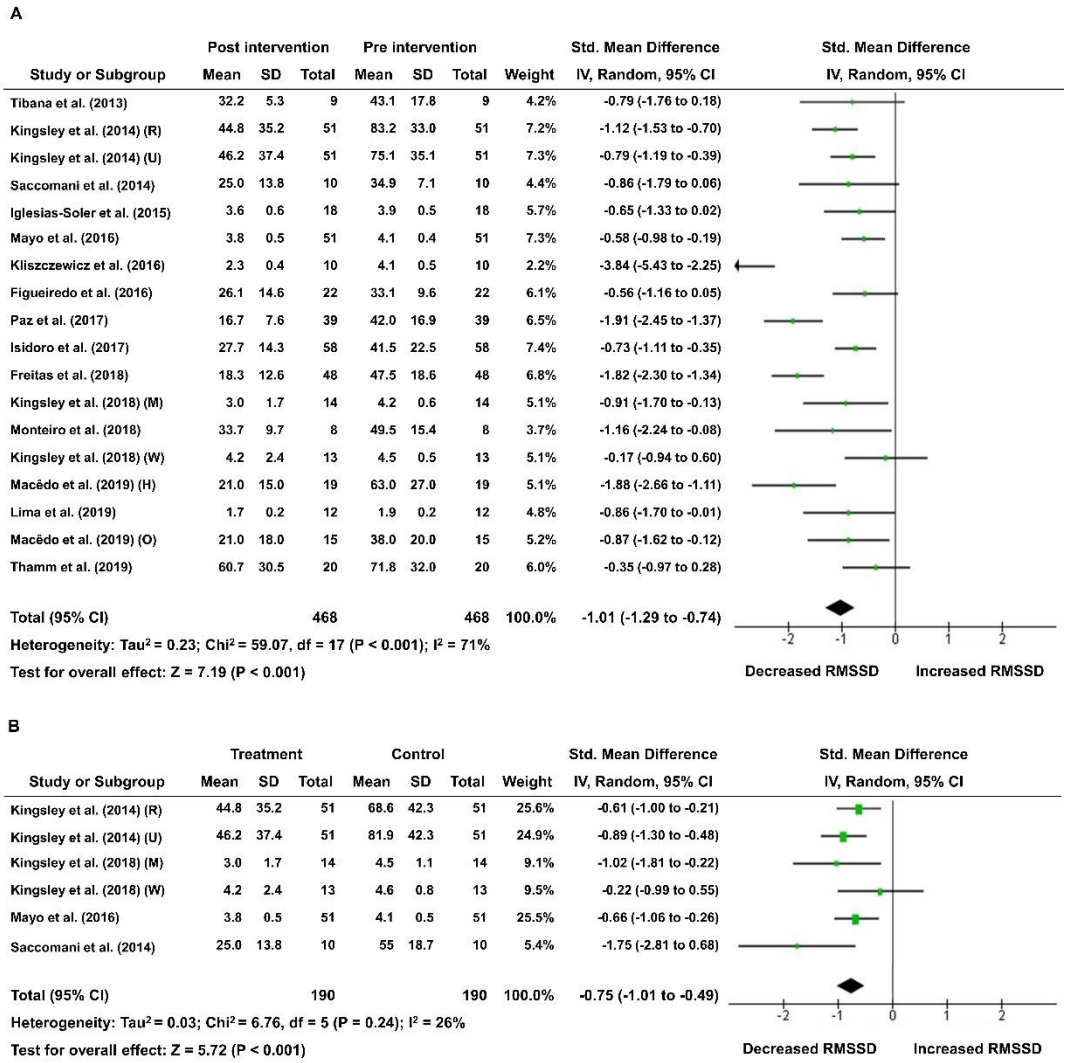


Figure 13. Forest plots for the acute effects of RT on RMSSD. (A) Acute effects of RT sessions on RMSSD pre- vs. post-intervention. (B) Acute effects of RT sessions on RMSSD control group vs. treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; df = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained; RMSSD = root mean square of the successive differences; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

6.1.4.2. HF(nu)

There was a decrease in HF(nu) ($p < 0.001$; SMD = –1.08; 95%CI: –1.43 to –0.73) in 23 ES calculations from 20 studies (mean age = 24.6 years; 251 males, 52 females) following ARE compared to baseline. When compared to a control group, the ARE group also decreased HF(nu) ($p < 0.001$; SMD = –1.06; 95%CI: –1.52 to –0.60) ~30 min (8–30 min) after the ARE session (Figure. 14) in 8 ES calculations from 6 studies (mean age = 23.2 years; 74 males, 35 females).

6.1.4.3. LF(nu)

A total of 20 studies (mean age = 24.6 years; 250 males; 57 females), with 22 ES calculations, showed an increase in LF(nu) ($p < 0.001$; SMD = 0.78; 95%CI: 0.46–1.11) after an ARE session compared to pre-intervention. Similarly, 6 studies (mean age = 23.2 years; 73 males, 40 females), with 7 ES calculations, showed an increase in LF(nu) ($p < 0.001$; SMD = 1.00; 95%CI: 0.43–1.56) in the ARE group compared to the control group (Figure. 15).

6.1.4.4. LF/HF ratio

In the 21 ES calculations in 19 studies (mean age = 25.4 years; 235 males, 66 females), there was an increase in LF/HF ratio ($p < 0.001$; SMD = 0.82; 95%CI: 0.64–0.99) ~30 min (8–30 min) after ARE compared to baseline. A total of 10 ES calculations from 8 studies (mean age = 22.9 years; 93 males, 53 females) also showed an increase in LF/HF ratio ($p < 0.001$; SMD = 1.02; 95%CI: 0.62–1.43) in the ARE group compared to the control group (Figure. 16).

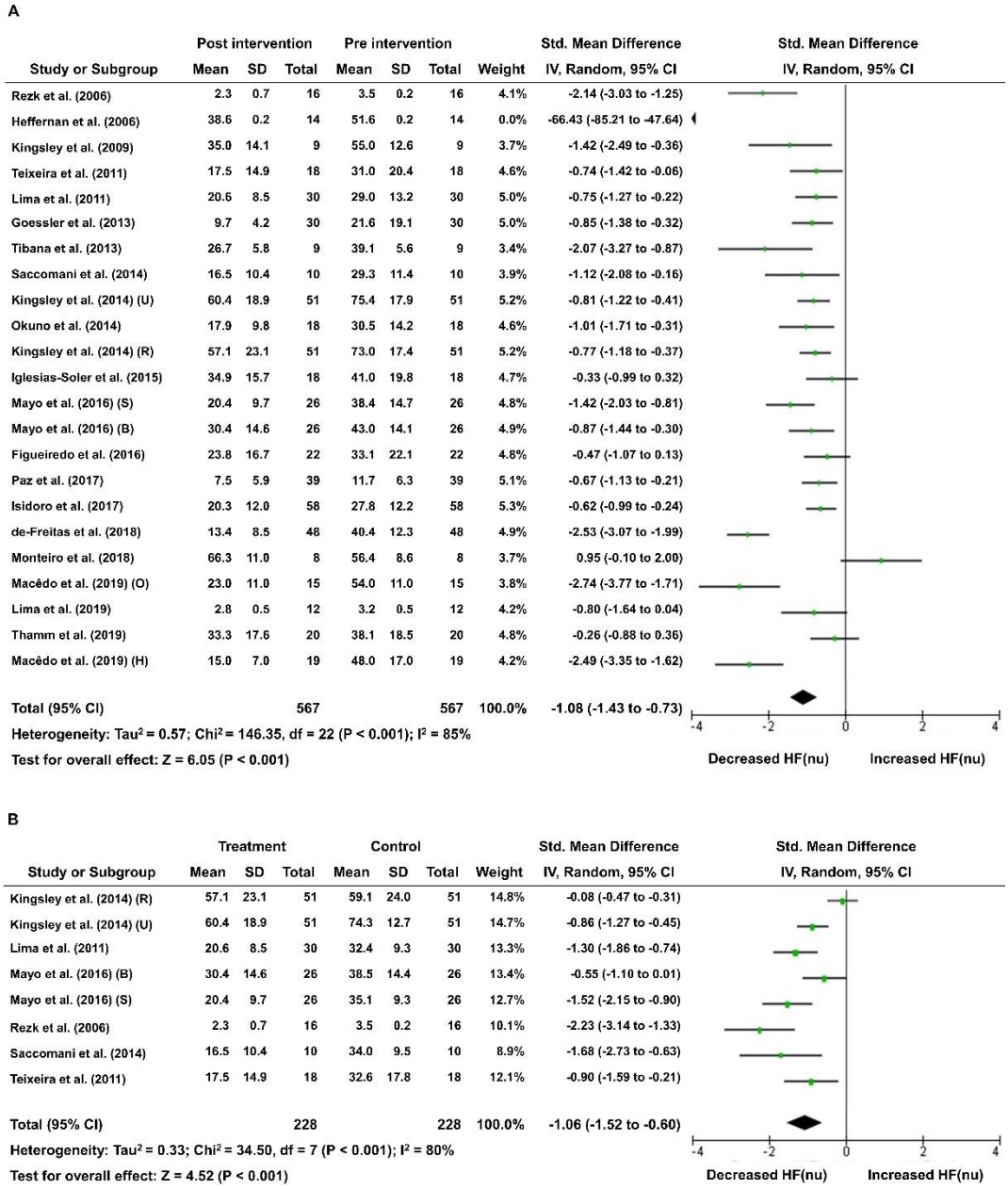


Figure 14. Forest plots for the acute effects of RT on HF(nu). (A) Acute effects of RT sessions on HF(nu) pre- vs. post-intervention. (B) Acute effects of RT sessions on HF(nu) control group vs. treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. B = bench press; CI = confidence interval; df = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained; S = parallel squat; HF(nu) = normalized units high frequency;

RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

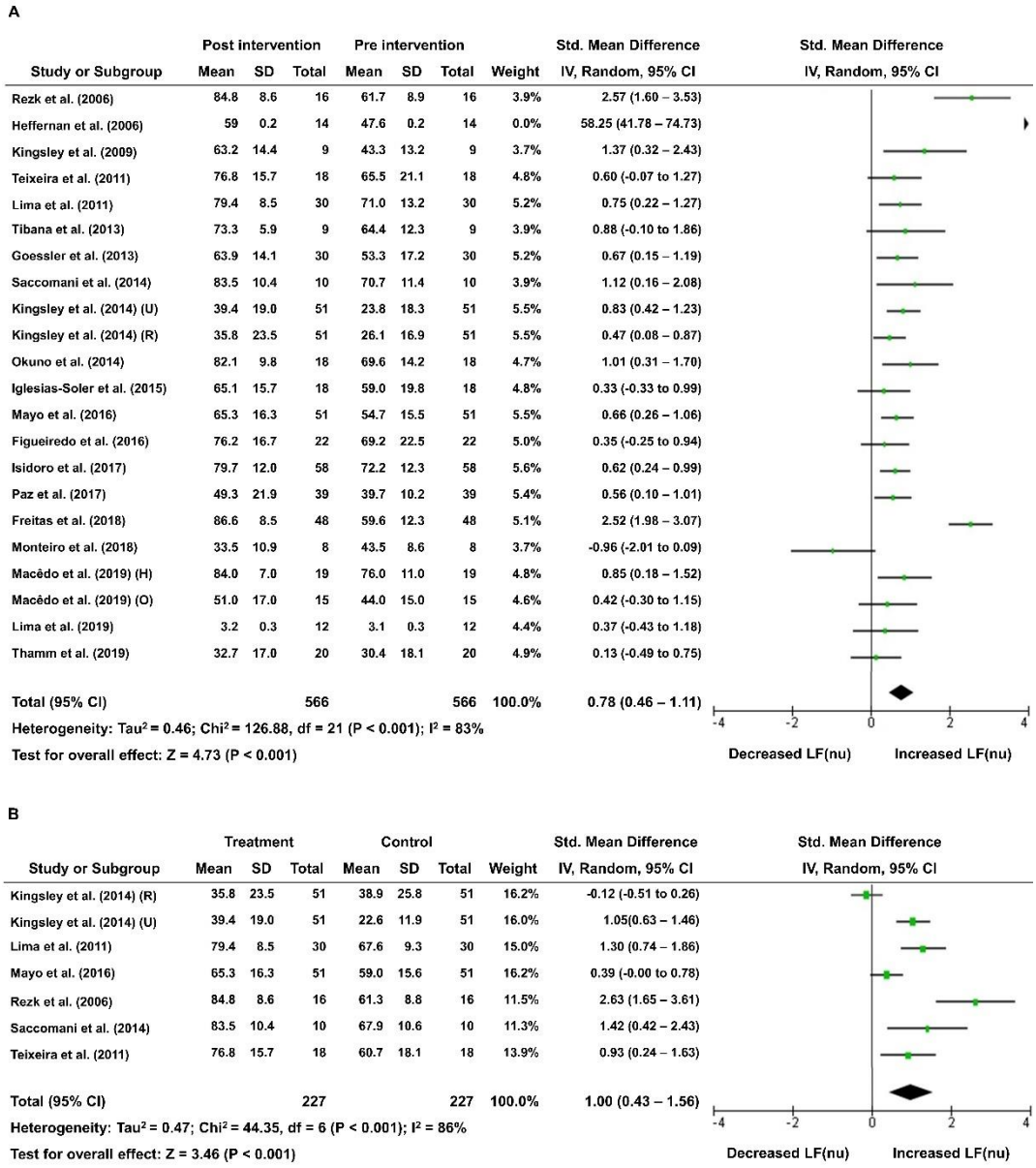


Figure 15. Forest plots for the acute effects of RT on LF(nu). (A) Acute effects of RT sessions on LF(nu) pre- vs. post-intervention. (B) Acute effects of RT sessions on LF(nu) control group vs. treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; df = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained;

LF(nu) = normalized units low frequency; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women

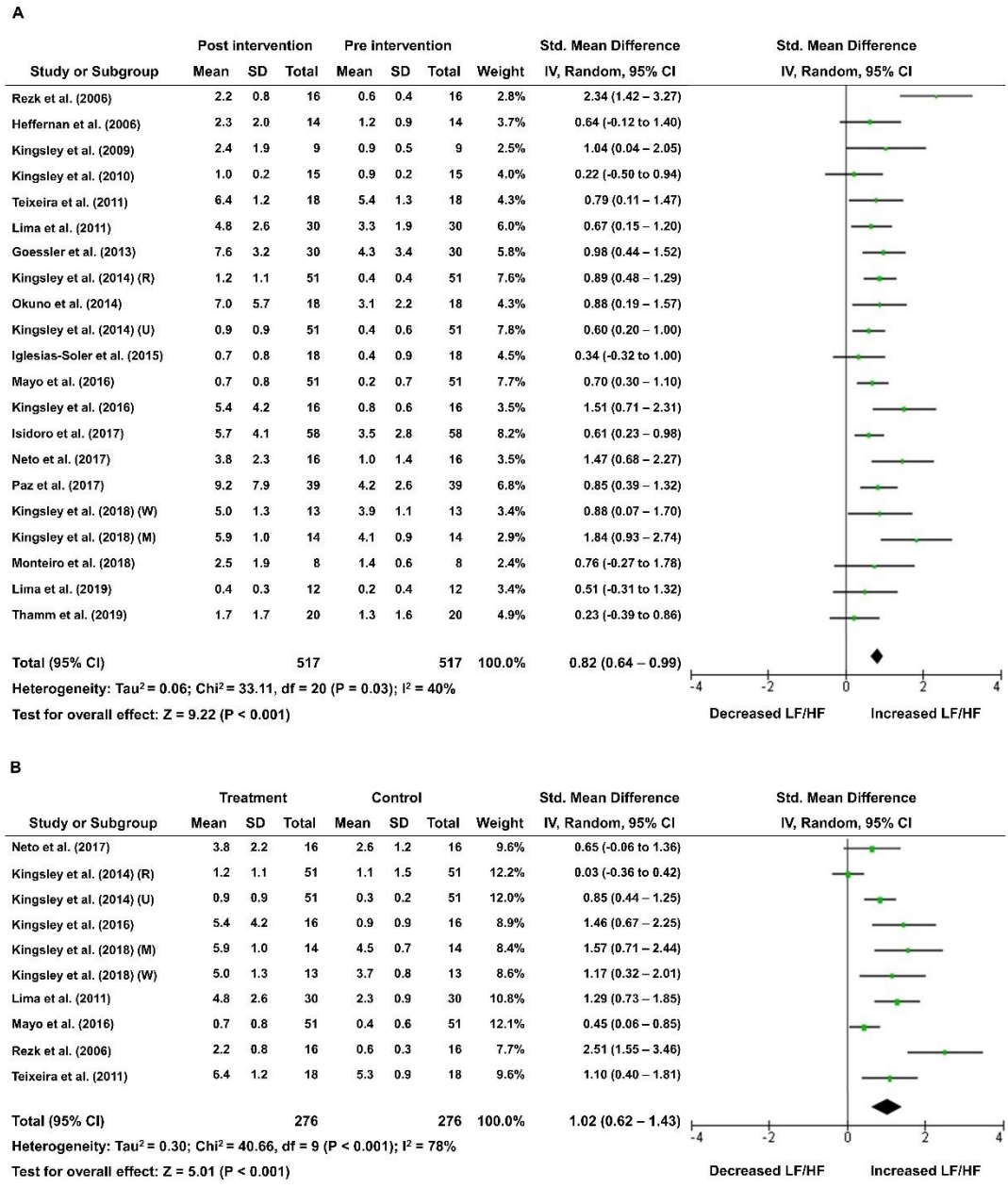


Figure 16. Forest plots for the acute effects of RT on LF/HF ratio. (A) Acute effects of RT sessions on LF/HF ratio pre vs. post-intervention. (B) Acute effects of RT sessions on LF/HF ratio control group vs. treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled across trials. CI = confidence interval; df = degrees of

freedom; H = healthy weight; IV = inverse variance; M = men; R = resistance trained; LF/HF = low frequency/high frequency; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

6.1.4.5. SDNN

A total of 7 studies (mean age = 22.4 years; 103 males, 33 females), with 9 ES calculations, showed a decrease in SDNN ($p < 0.001$; SMD = –0.58; 95%CI: –0.85 to –0.30) after an ARE session compared to pre-intervention (Figure. 17). However, the main effect analysis was not conducted for the ARE group compared with the control group due to the limited number of studies (only 1 study).

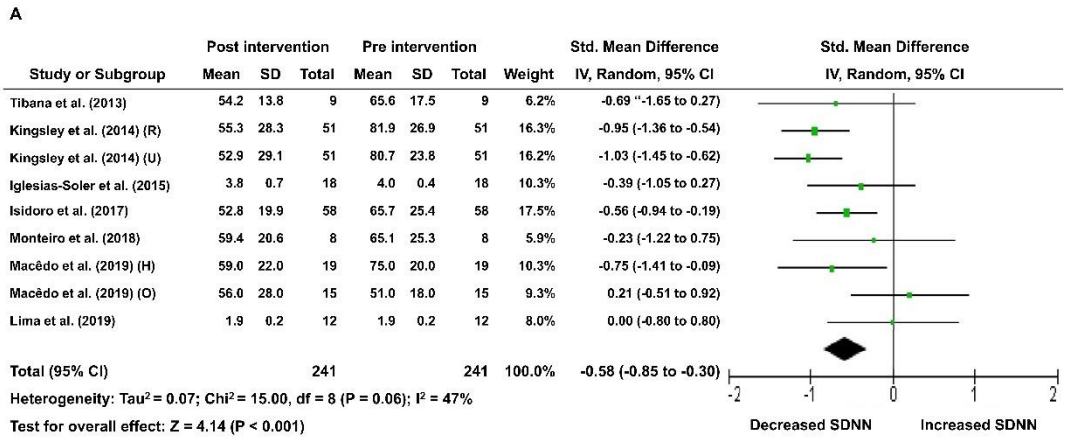


Figure 17. Forest plots for the acute effects of RT on SDNN. Acute effects of RT sessions on SDNN pre vs. post-intervention. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; df = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; R = resistance trained; RT = resistance training; SDNN = standard deviation of the NN interval; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

6.1.5. Subgroup analysis

6.1.5.1. RMSSD

For the subject characteristics, there was no difference in effect between subgroups based on gender ($p = 0.12$), BMI ($p = 0.44$), or training status ($p = 0.48$). With respect to RT variables, the number of sets ($p = 0.05$) and training volume

($p = 0.01$) showed a difference in effect between subgroups. Moreover, the SMD data showed that 3 sets and higher training volume had the greatest effect on RMSSD, whereas <3 sets and lower training volume had the least effect when comparing subgroups following resistance exercises. However, no other variables (exercises ($p = 0.07$), intensity ($p = 0.41$), repetitions ($p = 0.39$), and rest ($p = 0.31$)) indicated a difference in effect between subgroups (Table 7).

6.1.5.2. HF(nu)

For the subject characteristics, there was no difference in effect between subgroups for gender ($p = 0.75$), BMI ($p = 0.74$), or training status ($p = 0.15$). Regarding RT variables, intensity ($p = 0.01$), rest between sets ($p = 0.05$), and training volume ($p = 0.003$) showed a difference in effect between subgroups. Furthermore, SMD data revealed that low intensity, <2 min of rest and higher training volume had the greatest effect on HF(nu), whereas high intensity, 2 min of rest and lower training volume had the least effect compared to subgroups following ARE. However, there was no difference in effect between subgroups for all the other variables (repetitions ($p = 0.10$), sets ($p = 0.93$), and exercises ($p = 0.37$)) (Table 7).

6.1.5.3. LF(nu)

Regarding the subject characteristics, there was no difference in effect between subgroups for gender ($p = 0.63$), BMI ($p = 0.37$), and training status ($p = 0.45$). Except for training volume ($p = 0.02$), all the other RT variables (intensity ($p = 0.15$), sets ($p = 0.90$), exercises ($p = 0.17$), repetitions ($p = 0.46$), and rest ($p = 0.41$)) show no difference in effect between subgroups following resistance exercises. SMD data for training volume showed that a higher training volume had a greater effect and that a lower training volume had a lesser effect on LF(nu) compared to other subgroups following resistance exercises (Table 7).

6.1.5.4. LF/HF ratio

Concerning the subject characteristics (gender ($p = 0.65$), BMI ($p = 0.77$), and training status ($p = 0.55$)) and RT variables (intensity ($p = 0.24$), repetitions ($p = 0.82$), sets ($p = 0.56$), exercises ($p = 0.51$), rest ($p = 0.99$), and volume ($p = 0.62$)), there was no difference in effect between subgroups (Table 7).

Table 7. Subgroup analyses assessing potential moderating factors for heart rate variability parameters in studies included in the meta-analysis

Methodological factors	Studies			Acute resistance exercise			
	N[a]	References	SMD (95%CI)	I^2	I_p	P	P_{diff}
RMSSD							
Gender							
Male	12	(42, 152, 211, 212, 218, 221, 224-226, 228, 264)	-1.16 (-1.56 to -0.76)	77	<0.001	<0.001	0.12
Female	3	(40, 212, 230)	-0.61 (-1.19 to -0.03)	16	0.30	0.04	
BMI (kg/m²)							
≤24.9	8	(42, 211, 212, 218, 220, 228, 230, 264)	-0.98 (-1.41 to -0.54)	73	<0.001	<0.001	0.44
>24.9	3	(42, 212, 225)	-0.74 (-1.14 to -0.34)	0	0.72	<0.001	
Training status							
Resistance trained	11	(40, 152, 210-212, 220, 221, 225, 226, 228)	-0.94 (-1.30 to -0.57)	74	<0.001	<0.001	0.48
Not trained	7	(42, 210, 218, 224, 230, 264)	-1.15 (-1.62 to -0.67)	70	0.002	<0.001	
Exercise intensity (%RM)							
High (>85)	2	(152, 226)	-0.52 (-1.06 to 0.01)	0	0.53	0.06	0.41
Moderate (>65–85)	9	(152, 210-212, 225, 264)	-0.89 (-1.20 to -0.58)	63	0.006	<0.001	
Low (≤65)	7	(42, 220, 221, 228, 230)	-1.01 (-1.56 to -0.46)	75	<0.001	<0.001	
Number of repetitions							
<6	2	(152, 220)	-0.49 (-1.03 to -0.06)	0	0.59	0.08	0.39
6–10	8	(152, 210, 212, 220, 221, 230)	-0.94 (-1.31 to -0.58)	64	0.008	<0.001	
>10	5	(42, 220, 228, 264)	-0.86 (-1.37 to -0.35)	66	0.02	0.001	
Number of sets							
<3	1	(220)	-0.10 (-0.78 to -0.57)	-	-	0.76	0.05
3	13	(42, 210-212, 221, 225, 226, 228, 230, 264)	-1.02 (-1.31 to -0.73)	65	<0.001	<0.001	
>3	5	(152, 220, 221)	-0.99 (-1.50 to -0.49)	58	0.05	<0.001	

Table 7. (Continued)

Number of exercises							
<6	14	(40, 42, 152, 210, 212, 218, 220, 221, 226, 264)	-0.89 (-1.11 to -0.67)	44	0.04	<0.001	0.07
6	3	(211, 221, 230)	-1.69 (-2.42 to -0.96)	61	0.08	<0.001	
>6	2	(225, 228)	-0.65 (-1.15 to -0.14)	0	0.59	0.01	
Rest between sets (minutes)							
<2	9	(42, 211, 220, 221, 225, 230)	-1.16 (-1.63 to -0.70)	70	<0.001	<0.001	0.31
2	7	(152, 210, 212, 225, 228)	-0.77 (-1.02 to -0.51)	13	0.33	<0.001	
>2	4	(152, 211, 220, 226)	-1.01 (-1.68 to -0.33)	58	0.07	0.003	
Training volume							
Low (<108)	6	(152, 210, 212, 220)	-0.63 (-0.85 to -0.41)	0	0.44	<0.001	0.01
Moderate (108–<180)	4	(210, 211, 264)	-1.29 (-1.88 to -0.70)	76	0.006	<0.001	
High (≥180)	5	(42, 221, 228, 230)	-1.32 (-1.83 to -0.81)	56	0.06	<0.001	
HFnu							
Gender							
Male	16	(36, 42, 152, 208, 211, 218, 221, 225, 226, 228, 229, 231, 264, 265)	-1.14 (-1.59 to -0.68)	88	<0.001	<0.001	0.75
Female	3	(40, 209, 230)	-0.84 (-2.65 to 0.98)	88	<0.001	0.37	
BMI (kg/m²)							
≤24.9	12	(36, 42, 208, 211, 216, 218, 219, 228, 230, 264, 265)	-1.25 (-1.78 to -0.71)	86	<0.001	<0.001	0.74
>24.9	3	(42, 209, 225)	-1.50 (-2.87 to -0.13)	86	<0.001	0.03	
Training status							
Resistance trained	10	(36, 40, 152, 210, 211, 221, 226, 228, 229)	-0.85 (-1.33 to -0.36)	84	<0.001	<0.001	0.15
Not trained	12	(42, 208-210, 216, 218, 219, 230, 231, 264, 265) 264, 265)	-1.40 (-1.97 to -0.83)	87	<0.001	<0.001	

Table 7. (Continued)

Exercise intensity (%RM)							
High (>85)	2	(152, 226)	-0.34 (-0.87 to 0.19)	0	0.97	0.20	0.01
Moderate (>65–85)	13	(36, 152, 208, 210, 211, 216, 225, 229, 231, 264, 265)	-0.93 (-1.32 to -0.53)	81	<0.001	<0.001	
Low (≤65)	10	(42, 209, 216, 219, 221, 228-230, 265)	-1.58 (-2.19 to -0.96)	80	<0.001	<0.001	
Number of repetitions							
<6	1	(152)	-0.36 (-1.24 to -0.53)	-	-	0.43	0.10
6–10	7	(152, 208, 210, 216, 221, 230)	-1.58 (-2.64 to -0.53)	93	<0.001	0.003	
>10	9	(36, 42, 209, 216, 219, 228, 264)	-1.39 (-1.87 to -0.91)	74	<0.001	<0.001	
Number of sets							
<3	1	(209)	-1.42 (-2.49 to -0.36)	-	-	0.009	0.93
3	16	(42, 208, 210, 211, 216, 219, 221, 225, 226, 228, 230, 231, 264, 265)	-1.21 (-1.63 to -0.79)	85	<0.001	<0.001	
>3	5	(36, 152, 221, 229)	-1.22 (-1.94 to -0.49)	85	<0.001	0.001	
Number of exercises							
<6	15	(36, 40, 42, 152, 210, 218, 221, 226, 229, 231, 264, 265)	-1.00 (-1.35 to -0.65)	81	<0.001	<0.001	0.37
6	5	(211, 216, 219, 221, 230)	-1.51 (-2.26 to -0.75)	79	<0.001	<0.001	
>6	4	(208, 209, 225, 228)	-2.04 (-4.48 to 0.39)	94	<0.001	0.10	
Rest between sets (minutes)							
<2	11	(42, 208, 211, 216, 219, 221, 225, 229-231)	-1.72 (-2.49 to -0.95)	89	<0.001	<0.001	0.05
2	7	(152, 210, 225, 228, 231, 265)	-0.72 (-0.94 to -0.51)	0	0.57	<0.001	
>2	5	(36, 152, 211, 226)	-0.85 (-1.23 to -0.47)	25	0.25	<0.001	
Training volume							
Low (<108)	4	(152, 210, 231)	-0.56 (-0.82 to -0.29)	0	0.52	<0.001	0.003
Moderate (108–<180)	8	(36, 209-211, 264, 265)	-1.02 (-1.33 to -0.70)	55	0.03	<0.001	
High (≥180)	8	(42, 208, 216, 219, 221, 228, 230)	-2.17 (-3.22 to -1.12)	90	<0.001	<0.001	

Table 7. (Continued)

LFnu							
Gender							
Male	14	(36, 42, 152, 208, 211, 218, 221, 225, 226, 228, 229, 231, 264, 265)	0.79 (0.33–1.26)	87	<0.001	<0.001	0.63
Female	3	(40, 209, 230)	0.43 (-0.93 to 1.80)	81	0.005	0.53	
BMI (kg/m²)							
≤24.9	18	(42, 208, 211, 216, 218-220, 228, 230, 264, 265)	0.91 (0.41–1.41)	84	<0.001	<0.001	0.37
>24.9	4	(42, 209, 225)	0.58 (0.06–1.11)	31	0.24	0.03	
Training status							
Resistance trained	10	(40, 152, 210, 211, 220, 221, 225, 226, 228, 229)	0.65 (0.18–1.13)	86	<0.001	0.007	0.45
Not trained	12	(42, 208-210, 216, 218, 219, 230, 231, 264, 265)	0.91 (0.44–1.38)	83	<0.001	<0.001	
Exercise intensity (% RM)							
High (>85)	2	(152, 226)	0.32 (-0.21 to 0.85)	0	0.94	0.24	0.15
Moderate (>65–85)	12	(152, 208, 210, 211, 216, 220, 225, 229, 231, 264, 265)	0.81 (0.38–1.24)	82	<0.001	<0.001	
Low (≤65)	11	(42, 209, 216, 219-221, 228-230, 265)	1.02 (0.53–1.52)	78	<0.001	<0.001	
Number of repetitions							
<6	2	(152, 220)	0.59 (0.04–1.14)	0	0.40	0.04	0.46
6–10	8	(152, 208, 210, 216, 220, 221, 230)	1.27 (0.35–2.19)	93	<0.001	0.007	
>10	8	(42, 209, 216, 219, 220, 228, 264)	0.77 (0.45–1.08)	34	0.16	<0.001	
Number of sets							
<3	2	(209, 220)	0.78 (-0.21 to 1.76)	61	0.11	0.12	0.90
3	16	(42, 208, 210, 211, 216, 219, 221, 225, 226, 228, 230, 231, 264, 265)	0.86 (0.50–1.22)	81	<0.001	<0.001	
>3	5	(152, 220, 221, 229)	1.05 (0.24–1.86)	86	<0.001	0.01	

Table 7. (Continued)

Number of exercises							
<6	14	(40, 42, 152, 210, 218, 220, 221, 226, 229, 231, 264, 265)	0.66 (0.38–0.95)	73	<0.001	<0.001	0.17
6	5	(211, 216, 219, 221, 230)	1.33 (0.52–2.15)	83	<0.001	0.001	
>6	4	(208, 209, 225, 228)	2.11 (-0.32 to 4.54)	94	<0.001	0.09	
Rest between sets (minutes)							
<2	12	(42, 208, 211, 216, 219-221, 225, 229-231)	1.08 (0.43–1.74)	89	<0.001	0.001	0.41
2	7	(152, 210, 225, 228, 231, 265)	0.62 (0.39–0.85)	6	0.38	<0.001	
>2	4	(152, 211, 220, 226)	0.63 (0.22–1.04)	0	0.85	0.002	
Training volume							
Low (<108)	5	(152, 210, 220, 231)	0.46 (0.25–0.68)	0	0.41	<0.001	0.02
Moderate (108–<180)	6	(209-211, 264, 265)	0.97 (0.57–1.37)	62	0.02	<0.001	
High (≥180)	8	(42, 208, 216, 219, 221, 228, 230)	1.51 (0.45–2.56)	92	<0.001	0.005	
LF/HF ratio							
Gender							
Male	11	(152, 208, 211, 212, 218, 223, 226, 229, 231, 264, 265)	0.77 (0.54 – 0.99)	33	0.13	<0.001	0.65
Female	4	(40, 43, 209, 212)	0.65 (0.22 – 1.08)	0	0.51	0.003	
BMI (kg/m²)							
≤24.9	10	(208, 211, 212, 216, 218-220, 223, 264, 265)	0.85 (0.60 – 1.10)	43	0.07	<0.001	0.77
>24.9	3	(43, 209, 212)	1.00 (0.02 – 1.98)	74	0.02	0.05	
Training status							
Resistance trained	11	(37, 40, 152, 210-212, 220, 223, 226, 229)	0.87 (0.63–1.12)	38	0.09	<0.001	0.55
Not trained	10	(43, 208-210, 216, 218, 219, 231, 264, 265)	0.76 (0.51–1.02)	44	0.07	<0.001	

Table 7. (Continued)

Exercise intensity (% RM)							
High (>85)	2	(152, 226)	0.42 (-0.11 to 0.95)	0	0.70	0.12	0.24
Moderate (>65–85)	16	(37, 43, 152, 208, 210-212, 216, 220, 223, 229, 231, 264, 265)	0.89 (0.69–1.09)	37	0.07	<0.001	
Low (≤65)	6	(209, 216, 219, 220, 229, 265)	0.73 (0.35–1.11)	30	0.21	<0.001	
Number of repetitions							
<6	2	(152, 220)	0.71 (0.16–1.27)	0	0.67	0.01	0.82
6–10	11	(37, 43, 208, 210, 212, 216, 220, 223)	0.89 (0.59–1.19)	48	0.04	<0.001	
>10	5	(209, 216, 219, 220, 264)	0.78 (0.39–1.17)	35	0.19	<0.001	
Number of sets							
<3	2	(209, 220)	0.62 (0.04–1.19)	3	0.31	0.03	0.56
3	13	(37, 208, 210-212, 216, 219, 226, 231, 264, 265)	0.90 (0.65–1.15)	53	0.01	<0.001	
>3	5	(43, 152, 220, 223, 229)	0.71 (0.30–1.12)	50	0.09	<0.001	
Number of exercises							
<6	16	(37, 40, 43, 152, 210, 212, 218, 220, 223, 226, 229, 231, 264, 265)	0.76 (0.58–0.93)	30	0.12	<0.001	0.51
6	3	(211, 216, 219)	1.25 (0.44–2.06)	77	0.01	0.003	
>6	2	(208, 209)	0.79 (0.18–1.40)	0	0.53	0.01	
Rest between sets (minutes)							
<2	8	(43, 208, 211, 216, 219, 220, 229, 231)	0.87 (0.52–1.23)	51	0.04	<0.001	0.99
2	8	(37, 152, 210, 212, 231, 265)	0.91 (0.60–1.22)	45	0.08	<0.001	
>2	5	(152, 211, 220, 223, 226)	0.90 (0.54–1.27)	0	0.60	<0.001	
Training volume							
Low (<108)	10	(37, 43, 152, 210, 212, 220, 223, 231)	0.79 (0.51–1.07)	49	0.04	<0.001	0.62
Moderate (108–<180)	6	(209-211, 264, 265)	0.93 (0.61–1.26)	44	0.11	<0.001	
High (≥180)	3	(208, 216, 219)	1.22 (0.25–2.18)	78	0.01	0.01	

Note: I^2 = heterogeneity; *Ip* = p values for heterogeneity; N[a] = number of acute resistance exercise-trained groups within the selected studies; *P* = test for overall effect; *Pdiff* = test for subgroup differences.

Abbreviations: ARE = acute resistance exercise; BMI = body mass index; HF(nu) = normalized units high frequency; MD = mean difference; %RM = Percentage of 1 repetition maximum; SMD = standardized mean difference.

6.2. STUDY 2

For the experimental study, the results are presented in the following manner. First, I will present the results regarding the impact of a fatigue-intensive session on the dependent variables during the subsequent strength or power training session and how the effects differed by ARE modalities. Additionally, I will present how these effects changed over time compared to baseline and whether the training modality played a role in those changes. Furthermore, I will show the impact of a fatigue-intensive session on the dependent variables during the subsequent session of different ARE training loads within a given ARE modality and how these effects changed over time compared to baseline and whether ARE training load had an influence on those changes.

6.2.1. Participants

Overall, seventeen participants (12 males and 5 females) volunteered for the study. Four (3 males and 1 female) of the 17 were excluded for not meeting the inclusion criteria (see below). Thus, thirteen (9 males and 4 females) healthy, physically-active adults participated in this study and two (1 male and 1 female) discontinued after completing the first block of the study because of relocation due to Erasmus student program. Therefore, 11 participants participated in the comparison of strength versus power modality as well as the comparison of different power training loads, and 13 participants participated in the comparison of different strength training loads.

The participants were informed about the study procedures, possible risks and benefits and instructed not to consume caffeine- and alcohol- containing products during the study period. They were also asked to maintain their daily activities and eating habits but avoid vigorous physical activities or training during the study. All of the participants signed an informed consent form. The study was conducted according to the Declaration of Helsinki, and the study was approved by the Ethics committee at the Universidad Católica San Antonio de Murcia Spain (No. CE111806). Participants' characteristics are shown in Table 8.

Table 8. General characteristics of the different comparisons

Comparison	Age (years)	Height (cm)	Body Mass (kg)	BMI (kg/m^2)
S100 Vs P100 (n = 11)	21.36 ± 3.29	170.18 ± 12.06	67.07 ± 12.57	23.06 ± 3.13
S100 Vs S75 Vs S50 (n = 13)	22.15 ± 3.72	172 ± 12.85	67.38 ± 12.07	22.71 ± 2.99
P100 Vs P75 Vs P50 (n = 11)	21.36 ± 3.29	170.18 ± 12.06	67.07 ± 12.57	23.06 ± 3.13

Data are expressed as mean ± standard deviation.

Abbreviations: BMI = Body mass index (kg/m^2); S = Strength training modality; Vs = Versus; P = Power training modality

Table 9. External weight used during the training sessions.

Exercise	Strength (kg)	Power - OL (kg)	OL % of 1RM
Bench press (n = 11)	48.41 ± 25.42	27.74 ± 14.54	51.09 ± 13.94
Half squat (n = 11)	77.27 ± 44.84	46.82 ± 32.91	52.49 ± 16.28
Hip thrust (n = 11)	71.36 ± 32.47	54 ± 25.88	66.63 ± 16.44
Bench press (n = 13)	49.27 ± 23.66	-	-
Half squat (n = 13)	80.38 ± 41.64	-	-
Hip thrust (n = 13)	75.65 ± 32.78	-	-

Data are expressed as mean ± standard deviation

Abbreviations: kg = Kilograms; OL = optimal load; % of 1RM = Percentage of one-repetition maximum

Table 10. External resistance training loads used during the sessions

Comparison	S100 – TL (kg)		P100 – TL (kg)
S100 Vs P100 (n = 11)	3940.91 ± 1878.33		2571.09 ± 1309.10
	S100 – TL (kg)	**S75 – TL (kg)**	**S50 – TL (kg)**
S100 Vs S75 Vs S50 (n = 13)	4106.15 ± 1763.87	3079.62 ± 1322.9	2053.08 ± 881.93
	P100 – TL (kg)	**P75 – TL (kg)**	**P50 – TL (kg)**
P100 Vs P75 Vs P50 (n = 11)	2571.09 ± 1309.10	1928.32 ± 981.82	1285.55 ± 654.55

Data are expressed as mean ± standard deviation

Abbreviations: kg = Kilograms; P = Power training modality; S = Strength training modality; TL = Training load (Training load = weight repetitions sets)

6.2.2. Heart rate variability parameters

6.2.2.1. Strength 100 versus Power 100 training

HRV parameters for the comparison between 100% training load strength (S100) modality and 100% training load power (P100) modality are reported below.

1.2.1.1.1. pNN50

There was an overall treatment effect ($p = 0.012$) and an overall time effect ($p < 0.001$) on pNN50. There was a significant treatment x time interaction for pNN50 ($p = 0.011$), where simple main effects for treatment showed that pNN50 was significantly lower in the strength modality at Post-T ($p = 0.051$), Post-6H ($p = 0.032$), Post-24H ($p = 0.012$) and Post-48H ($p < 0.001$; Figure 18) compared to the power modality. This indicates that S100 modality for AREs decreased more in pNN50 than P100 modality.

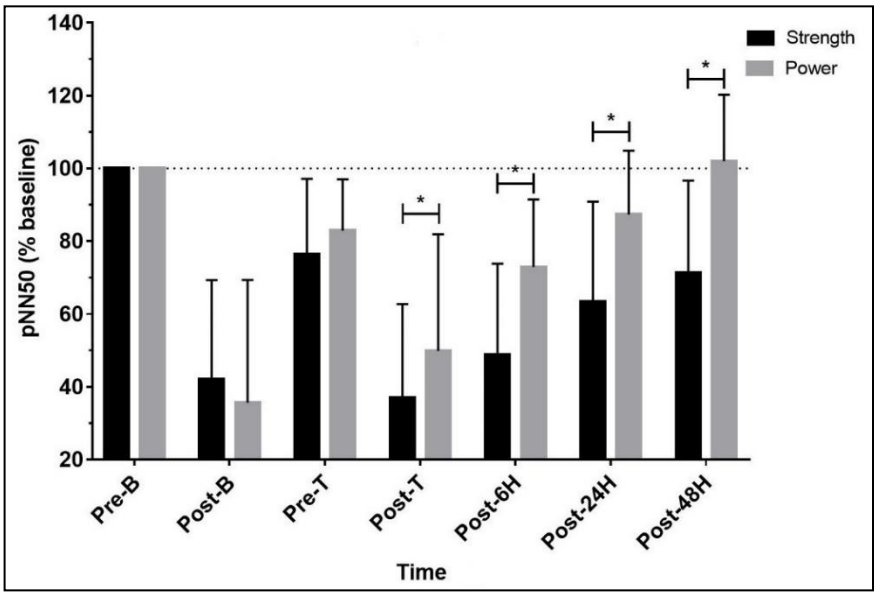


Figure 18. Comparison between S100 and P100 on pNN50 values (n = 10). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that pNN50 differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Post-B ($P = 0.001$, ES = -1.76), Post-T ($P < 0.001$, ES = -1.98), Post-6H ($P = 0.001$, ES = -1.47) and Post-24H ($P = 0.029$, ES = -0.92), except at Pre-T ($P = 0.121$, ES = -0.63) and Post-48H ($P = 0.103$, ES = -0.80), compared to Pre-B value. In P100, significant time differences were shown at Post-B ($p = 0.005$, ES = -1.55), Pre-T ($p = 0.026$, ES = -0.43), Post-T ($p = 0.001$, ES = -1.14),

Post-6H (p = 0.052, ES = -0.72) except at Post-24H (p = 0.604, ES = -0.37) and Post-48H (p = 1.000, ES = 0.03), compared to Pre-B value.

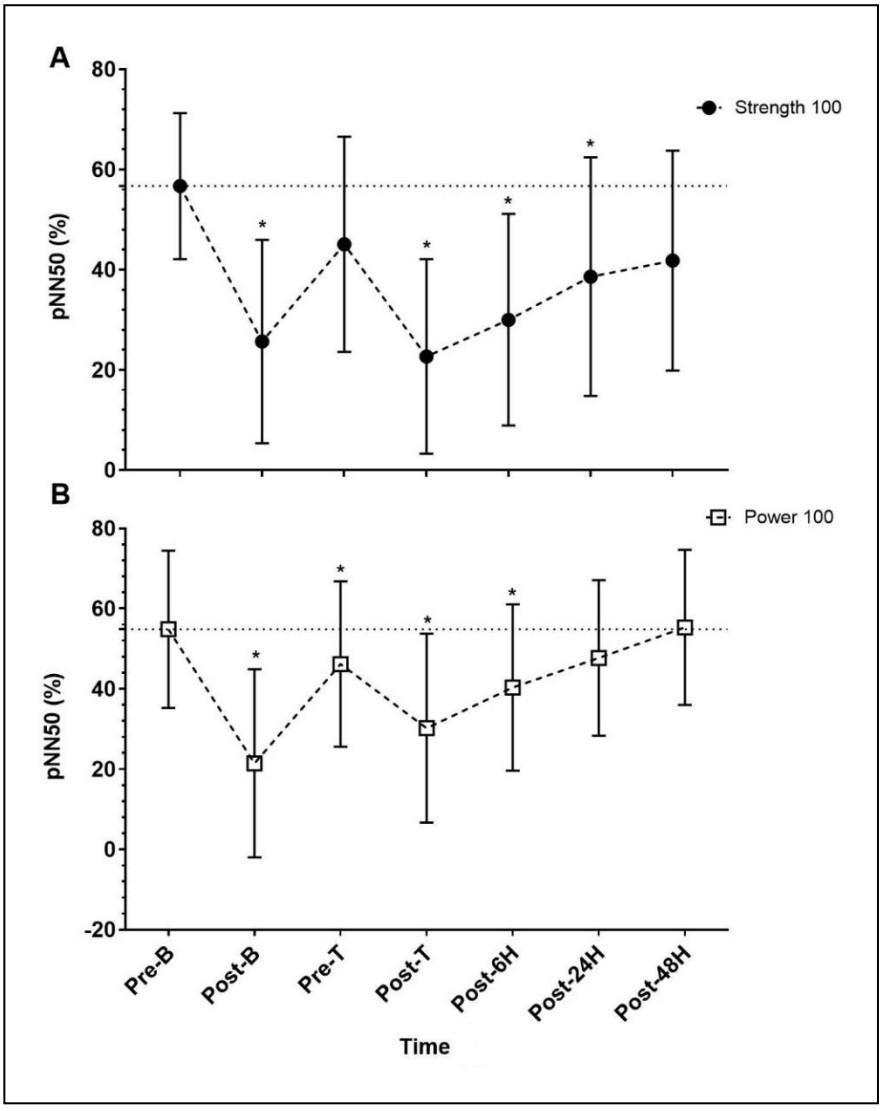


Figure 19. Changes in pNN50 value parameter in (A) S100 and (B) P100 protocols (n = 10). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-48H for P100, whereas S100 did

not yet recover at Post-48H. Similarly, ES results also showed that, cardiac parasympathetic modulation of P100 recovered at post-48H, whereas S100's level did not yet recover at Post-48H (Figure 19).

6.2.2.1.1. SDNN

There was an overall treatment effect ($p = 0.010$) and an overall time effect ($p < 0.001$) on SDNN. There was a significant treatment x time interaction for SDNN ($p = 0.002$), where simple main effects for treatment showed that SDNN was significantly lower in the strength modality at Post-T ($p = 0.005$), Post-6H ($p = 0.014$) and post-24H ($p = 0.007$; Figure 20) compared to the power modality. This indicates that S100 modality for AREs decreased more in SDNN than P100 modality.

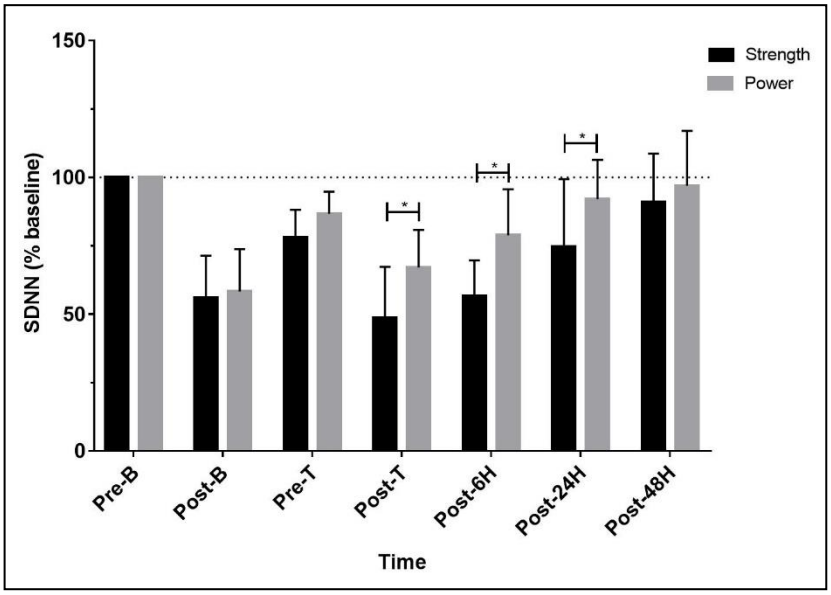


Figure 20. Comparison between S100 and P100 on SDNN values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that SDNN differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Post-B ($P = 0.001$, ES = -1.27), Pre-T ($P = 0.002$, ES = -0.57), Post-T ($P = 0.004$, ES = -1.38) and Post-6H ($P < 0.001$, ES = -1.09)

except at Post-24H (P = 0.066, ES = -0.50) and Post-48H (P = 1.000, ES = -0.21), compared to Pre-B value. In P100, significant time differences were shown at Post-B (p = 0.002, ES = -1.16), Pre-T (p = 0.009, ES = -0.36) and Post-T (p = 0.001, ES = -0.83) except at Post-6H (p = 0.169, ES = -0.59), Post-24H (p = 1.000, ES = -0.23) and Post-48H (p = 1.000, ES = -0.22), compared to Pre-B value.

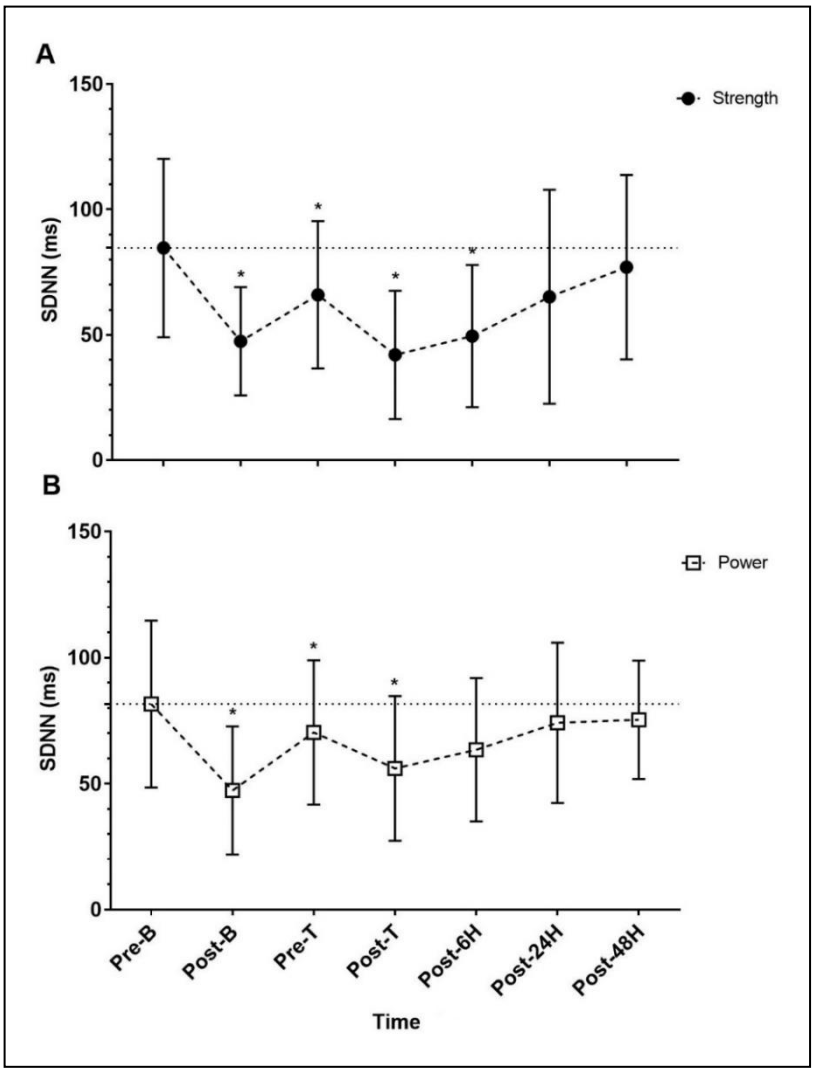


Figure 21. Changes in SDNN parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that overall autonomic modulation decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, overall autonomic modulation recovered to baseline (Pre-B) at Post-6H for P100, whereas S100 needed longer time (Post-24H) to recover. According to the ES results, overall autonomic modulation of both training modalities did not yet recover at Post-48H (Figure 21).

6.2.2.1.2. Ln RMSSD

There was an overall treatment effect ($p = 0.007$) and an overall time effect ($p < 0.001$) on Ln RMSSD. There was a significant treatment x time interaction for Ln RMSSD ($p = 0.019$), where simple main effects for treatment showed that Ln RMSSD was significantly lower in the strength modality at Post-T ($p = 0.015$), Post-6H ($p = 0.003$), Post-24H ($p < 0.001$) and Post-48H ($p = 0.006$; Figure 22) compared to the power modality. This indicates that S100 for AREs decreased more in Ln RMSSD than P100.

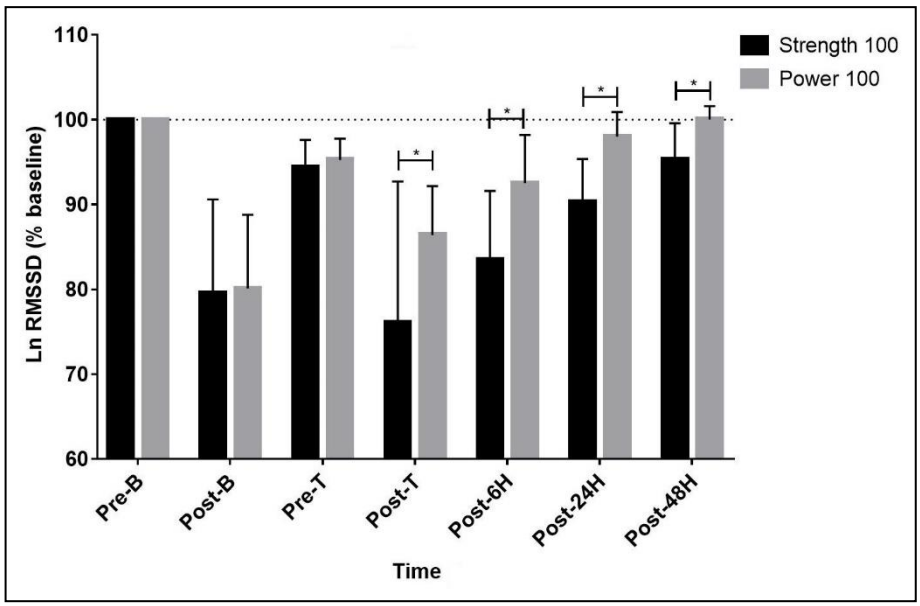


Figure 22. Comparison between S100 and P100 on Ln RMSSD values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that Ln RMSSD differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Post-B ($P < 0.001$, ES = -1.41), Pre-T ($P = 0.002$, ES = -0.50), Post-T ($P = 0.005$, ES = -1.42), Post-6H ($P < 0.001$, ES = -1.27) and Post-24H ($P = 0.001$, ES = -0.82), except at Post-48H ($P = 0.077$, ES = -0.41), compared to Pre-B value. In P100, significant time differences were shown at Post-B ($p < 0.001$, ES = -1.60), Pre-T ($p = 0.001$, ES = -0.42), Post-T ($p < 0.001$, ES = -1.06), Post-6H ($p = 0.040$, ES = -0.65), except at Post-24H ($p = 0.968$, ES = -0.20) and Post-48H ($p = 1.000$, ES = -0.01), compared to Pre-B value. These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-24H for P100, whereas S100 needed longer time (Post-48H) to recover. According to the ES results, cardiac parasympathetic modulation of P100 recovered at post-24H, whereas S100's level did not yet recover at Post-48H (Figure 23).

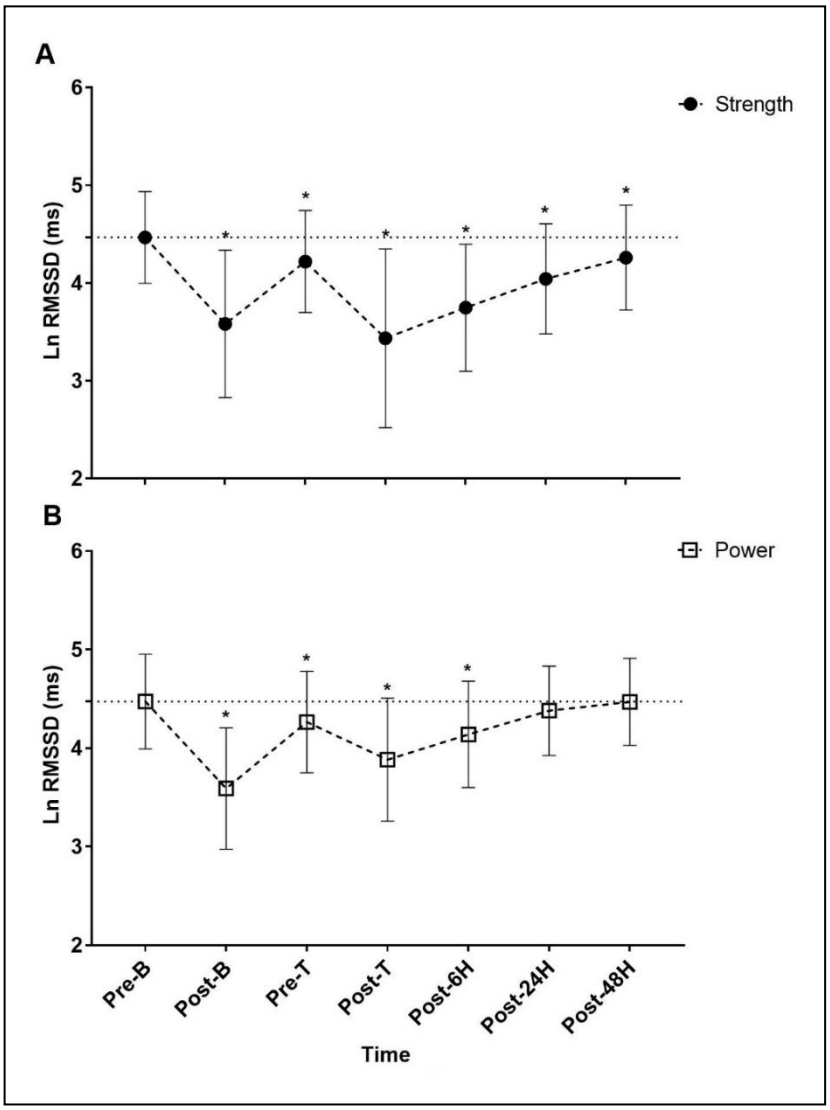


Figure 23. Changes in Ln RMSSD value parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.1.3. HF(nu)

There was no overall treatment effect on HF(nu) ($p = 0.736$). However, there was an overall time effect on HF(nu) ($p = 0.053$). No significant group x time interaction for HF(nu) was observed ($p = 0.279$; Figure 24).

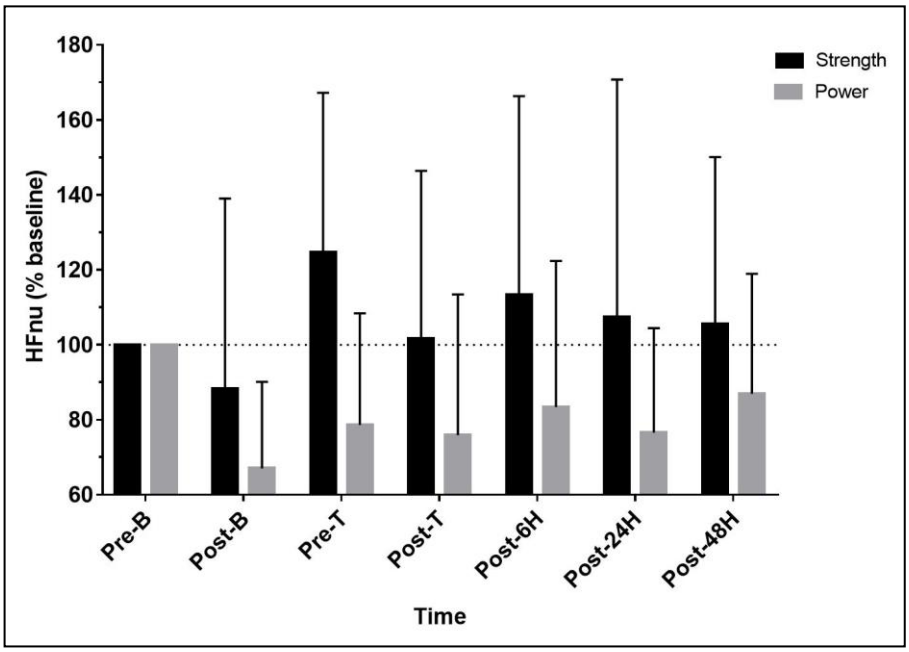


Figure 24 Comparison between S100 and P100 on HF(nu) values (n = 11).

Simple main effects over time revealed that there was no significant difference between time points in S100 (P = 0.291), except for P100 (P = 0.049) trial. Compared to Pre-B, significant difference was shown only at Post-B (p = 0.031, ES = -1.17) in P100 trial. ES results (S100: Post-B (ES = -0.37), Pre-T (ES = 0.53), Post-T (ES = -0.16), Post-6H (ES = 0.27), Post-24H (ES = -0.04) and Post-48H (ES = 0.02), P100: Pre-T (ES = -0.75), Post-T (ES = -0.97), Post-6H (ES = -0.84), Post-24H (ES = -1.01) and Post-48H (ES = -0.59)) showed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline at Post-6H for S100, whereas P100's level did not yet recover at Post-48H (Figure 25). According to the ES results, cardiac parasympathetic modulation of S100 recovered at post-48H, whereas P100's level did not yet recover at Post-48H.

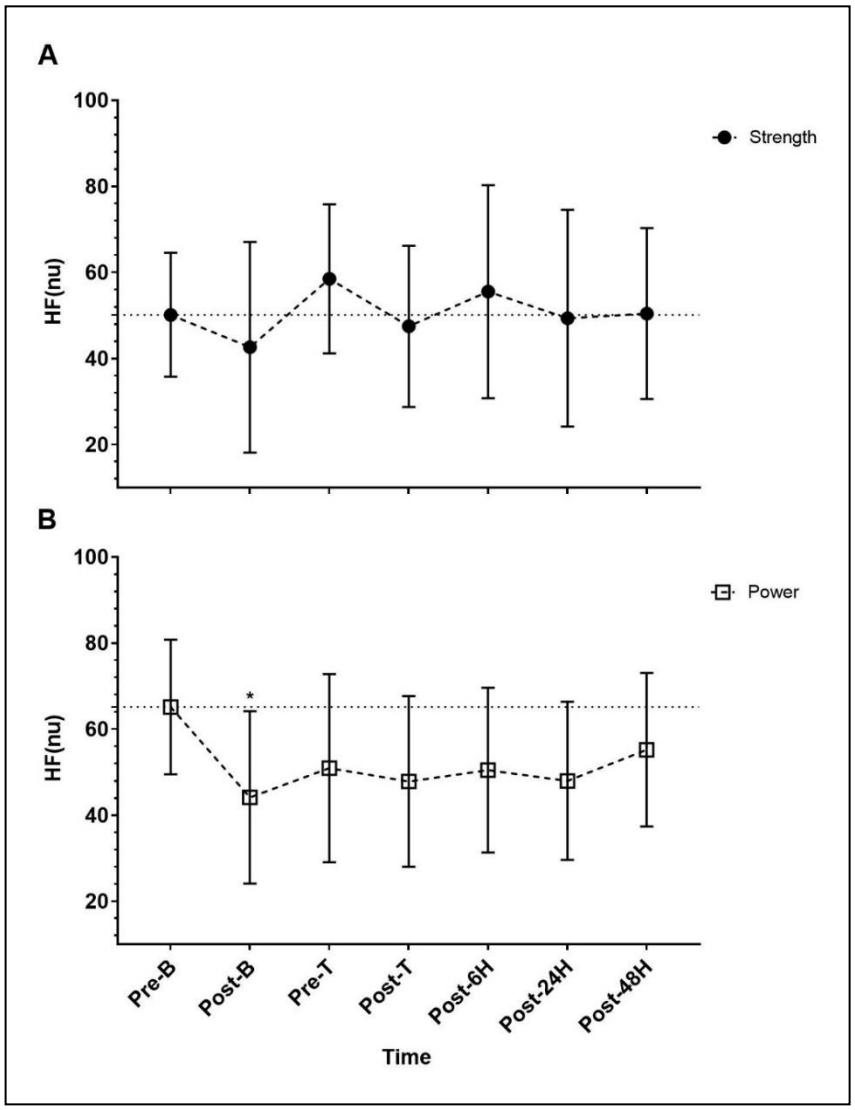


Figure 25. Changes in HF(nu) value parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.1.4. LF(nu)

There was no overall treatment effect on LF(nu) ($p = 0.741$). However, there was an overall time effect on LF(nu) ($p = 0.054$). No significant group x time interaction for LF(nu) was observed ($p = 0.276$; Figure 26).

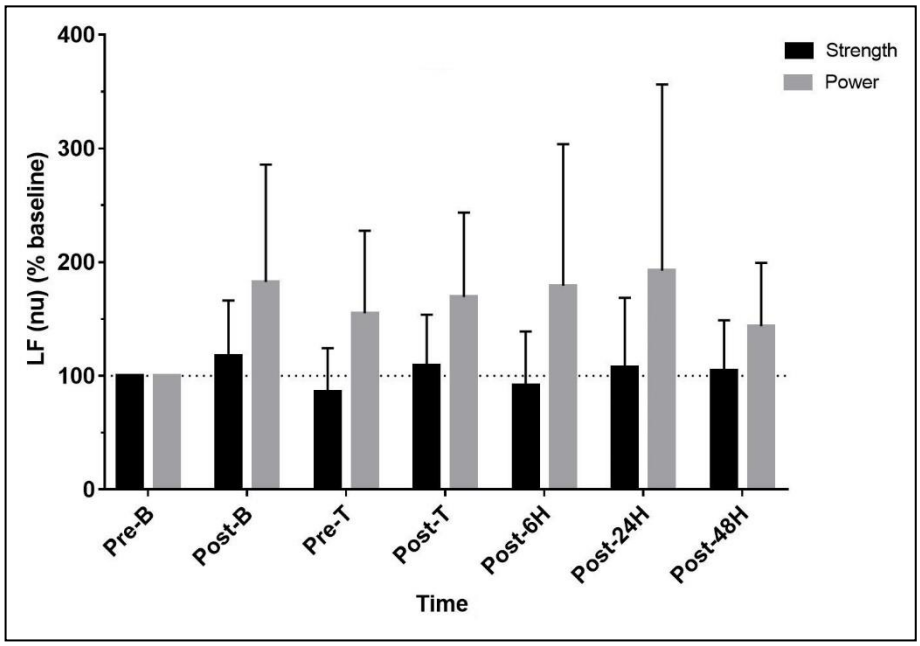


Figure 26. Comparison between S100 and P100 on LF(nu) values (n = 11)

Simple main effects over time revealed that there was no significant difference between time points in S100 (P = 0.291), except for P100 (P = 0.049) trial. Compared to Pre-B, significant difference was showed only at Post-B (p = 0.031, ES = -1.17) in P100 trial. ES results (S100: Post-B (ES = 0.37), Pre-T (ES = -0.53), Post-T (ES = 0.15), Post-6H (ES = -0.27), Post-24H (ES = 0.04) and Post-48H (ES = -0.02), P100: Pre-T (ES = 0.75), Post-T (ES = 0.97), Post-6H (ES = 0.84), Post-24H (ES = 1.01) and Post-48H (ES = 0.59)) showed that cardiac sympathetic modulation increased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, cardiac sympathetic modulation recovered to baseline at Post-6H for S100, whereas P100's level did not yet recover at Post-48H (Figure 26). According to the ES results, cardiac sympathetic modulation of both training modalities did not recover at Post-48H (Figure 27). Even though not fully recovered, S100 showed better recovery level than P100 at Post-48H.

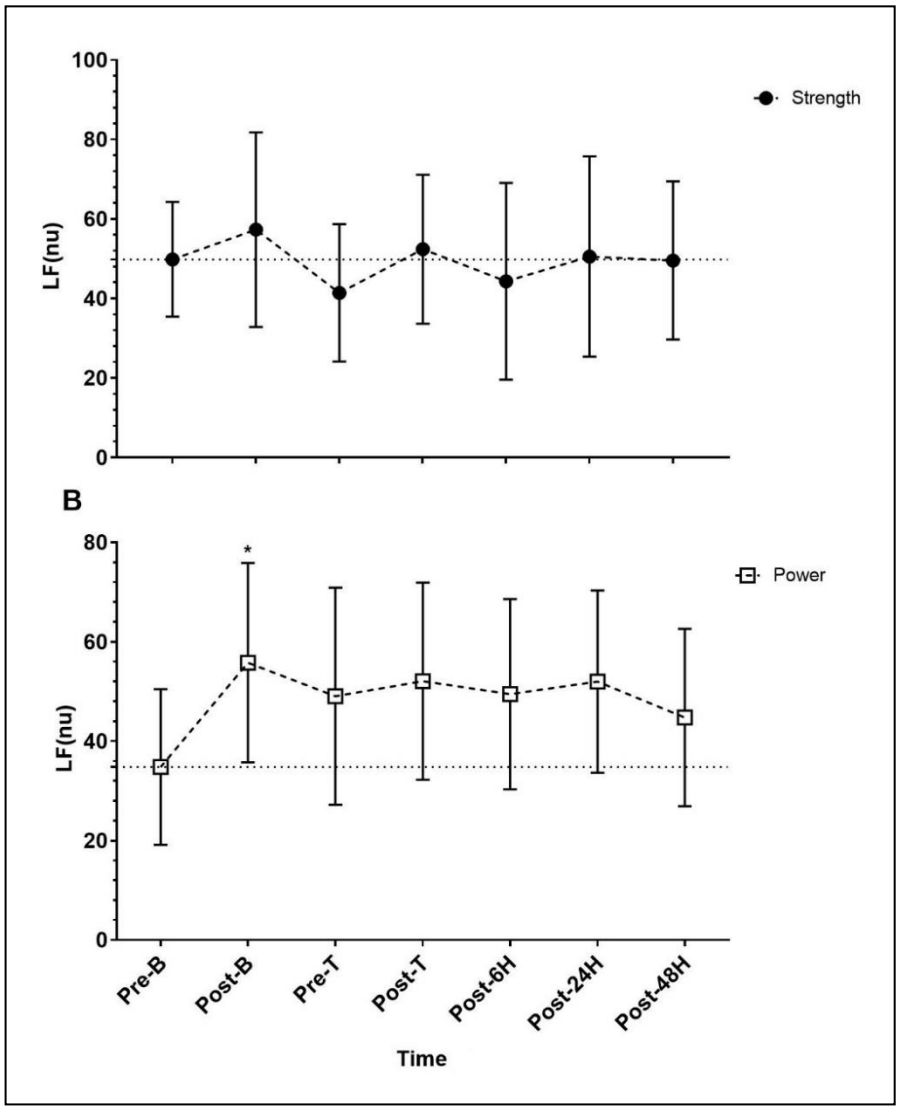


Figure 27. Changes in LF(nu) value parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.1.5. LF/HF ratio

There was neither an overall treatment effect ($p = 0.762$) nor an overall time effect ($p = 0.097$) on LF/HF ratio. No significant group x time interaction for LF/HF ratio was observed ($p = 0.269$; Figure 28). These results showed that S100 and P100 trials did not significantly effect the cardiac sympathovagal balance.

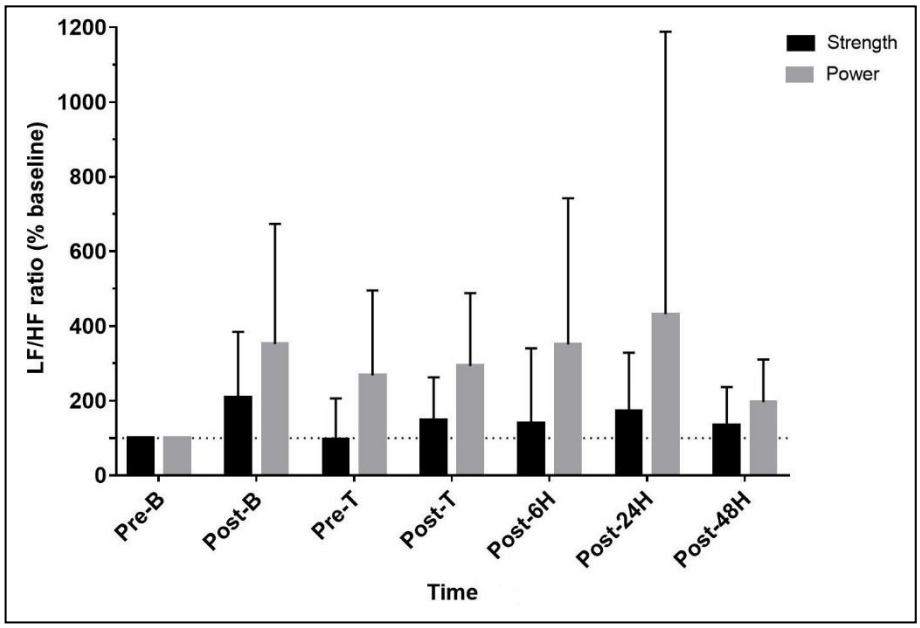


Figure 28. Comparison between S100 and P100 on LF/HF ratio values (n = 11).

6.2.2.1.6. Total power

There was an overall treatment effect ($p = 0.024$) and an overall time effect ($p < 0.001$) on TP. There was a significant treatment x time interaction for TP ($p < 0.001$), where simple main effects for treatment showed that TP was significantly lower in the strength modality at Post-T ($p = 0.001$), Post-6H ($p = 0.036$) and Post-24H ($p = 0.022$; Figure 29)) compared to the power modality. This indicates that S100 modality for AREs decreased more in TP than P100 modality.

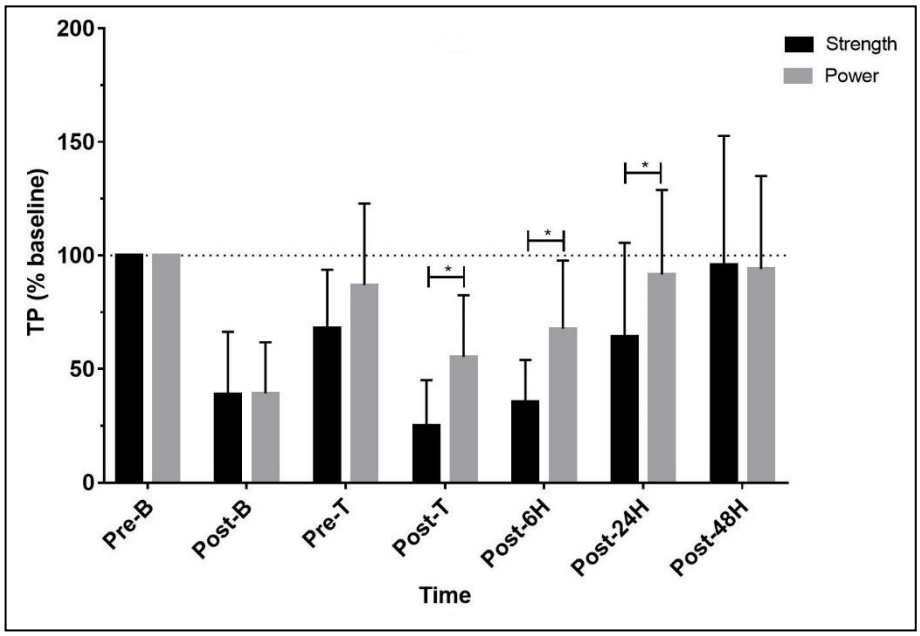


Figure 29. Comparison between S100 and P100 on TP values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that TP differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Post-B ($p = 0.010$, ES = -1.00), Post-T ($p = 0.004$, ES = -1.20) and Post-6H ($P = 0.002$, ES = -0.86), except at Pre-T ($p = 0.090$, ES = -0.47), Post-24H ($p = 0.099$, ES = -0.35) and Post-48H ($p = 1.000$, ES = -0.15), compared to Pre-B value. In P100, significant time differences were shown at Post-B ($p = 0.013$, ES = -0.90) and Post-T ($p = 0.054$, ES = -0.65), except at Pre-T ($p = 1.000$, ES = -0.35), Post-6H ($P = 0.403$, ES = -0.61), Post-24H (P100: $p = 1.000$, ES = -0.10) and Post-48H ($p = 1.000$, ES = -0.38), compared to Pre-B value. These results revealed that total autonomic activity decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, total autonomic activity recovered to baseline (Pre-B) at Post-6H for P100, whereas S100 needed longer time (Post-24H) to recover. According to the ES results, total autonomic activity of both training modalities did not recover at Post-

48H (Figure 30). Although not fully recovered, S100 showed better recovery level than P100 at Post-48H.

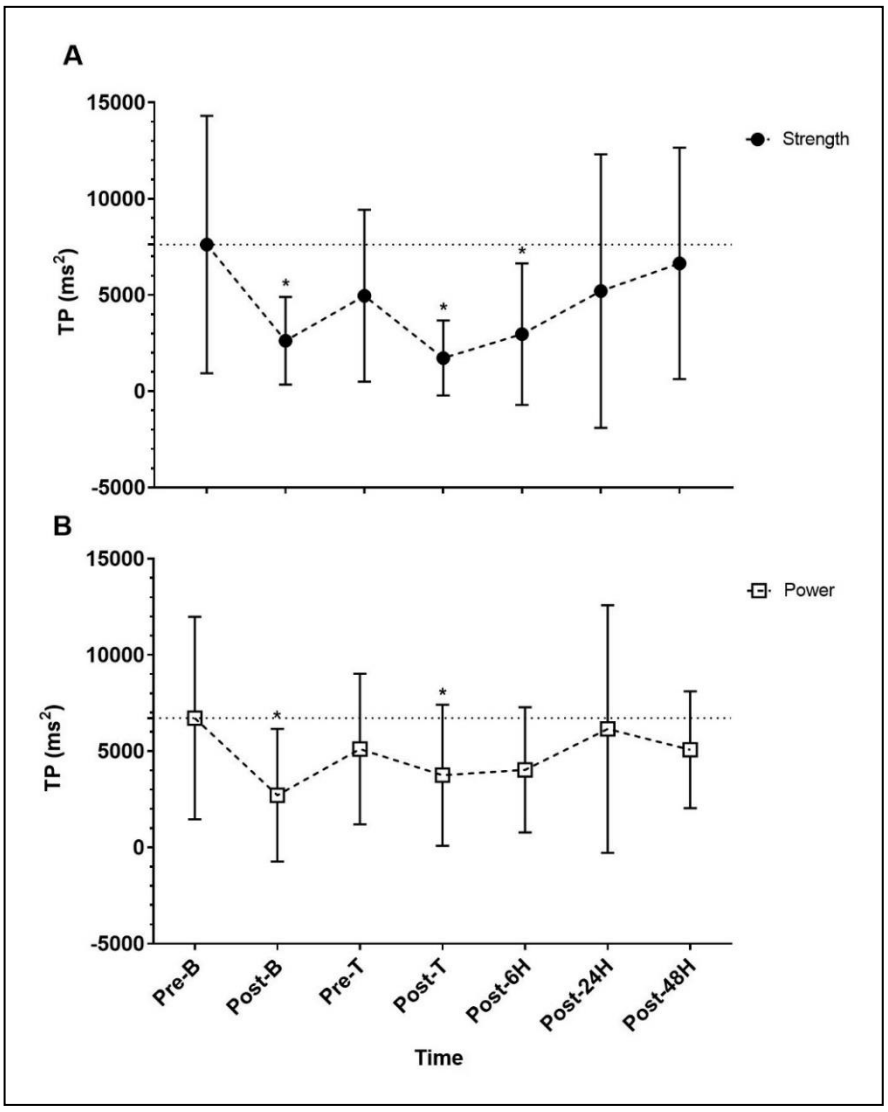


Figure 30. Changes in TP parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.1.7. SD1

There was an overall treatment effect ($p = 0.007$) and an overall time effect on SD1 ($p < 0.001$). There was significant treatment x time interaction for SD1 ($p =$

0.019), where simple main effects for treatment showed that SD1 was significantly lower in the strength modality at Post-T (p = 0.015), Post-6H (p = 0.003), post-24H (p < 0.001) and post-48H (p = 0.006; Figure 31)) compared to the power modality. This indicates that S100 modality for AREs decreased more in SD1 than P100 modality.

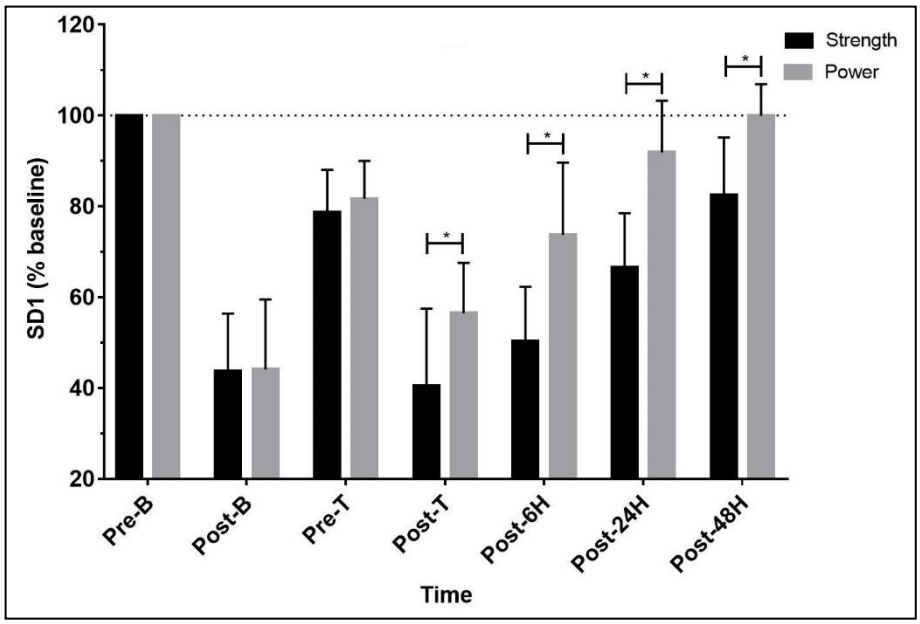


Figure 31. Comparison between S100 and P100 on SD1 values (n = 11). * Significant pairwise comparison differences between strength and power modalities (p ≤ 0.05).

Simple main effects over time revealeded that SD1 differed significantly between time points in S100 (P < 0.001) and P100 (P < 0.001) trials. In S100, significant time differences were observed at Post-B (P < 0.001, ES = -1.30), Pre-T (P = 0.002, ES = -0.42), Post-T (p = 0.005, ES = -1.42), Post-6H (P < 0.001, ES = -1.14) and Post-24H (P = 0.001, ES = -0.72), except at Post-48H (P = 0.076, ES = -0.33), compared to Pre-B value. In P100, significant time differences were shown at Post-B (P < 0.001, ES = -1.35), Pre-T (P = 0.001, ES = -0.38), Post-T (P < 0.001, ES = -0.94), Post-6H (P = 0.040, ES = -0.57), except at Post-24H (P = 0.967, ES = -0.21) and Post-48H (P = 1.000, ES = -0.04), compared to Pre-B value.

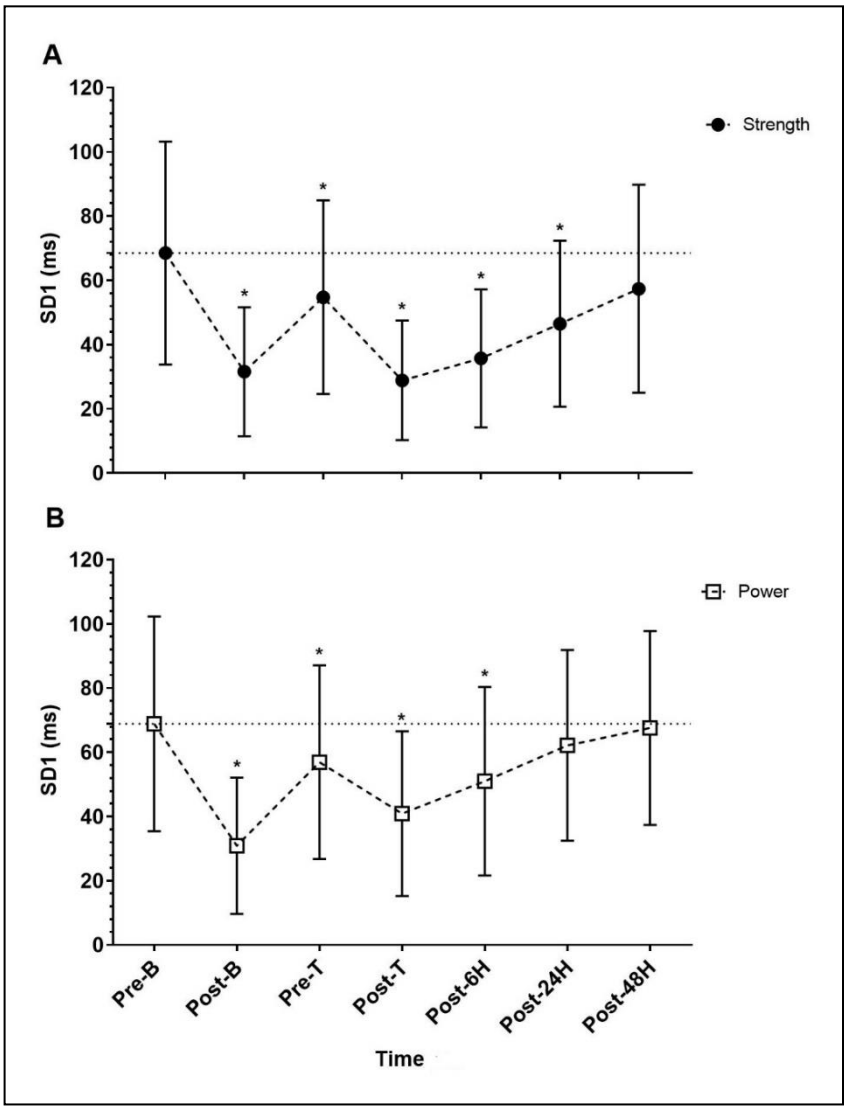


Figure 32. Changes in SD1 parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-24H for P100, whereas S100 needed longer time (Post-48H) to recover. According to the ES results, cardiac parasympathetic modulation of both training modalities did not recover at Post-

48H (Figure 32). Although not fully recovered, P100 showed better recovery level than S100 at Post-48H.

6.2.2.1.8. SD2

There was an overall treatment effect ($p = 0.040$) and an overall time effect on SD2 ($p < 0.001$). There was a significant treatment x time interaction for SD2 ($p = 0.037$), where simple main effects for treatment showed that SD2 was significantly lower in the strength modality at Post-T ($p = 0.007$), Post-6H ($p = 0.047$) and post-24H ($p = 0.053$; Figure 33)) compared to the power modality. This indicates that S100 modality for AREs decreased more in SD2 than P100 modality.

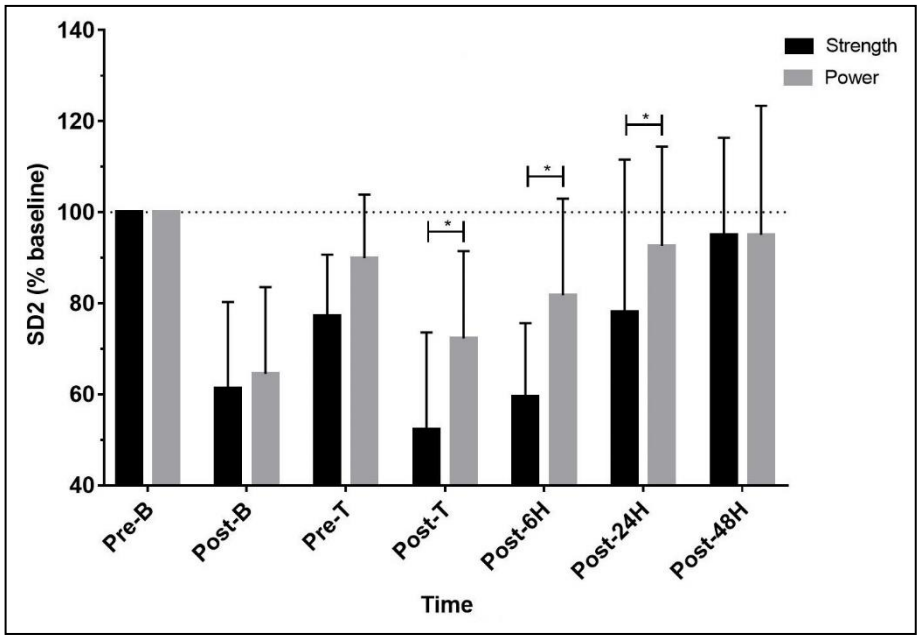


Figure 33. Comparison between strength 100 and power 100 on SD2 values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that SD2 differed significantly between time points in S100 ($P = 0.033$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Pre-T ($P = 0.012$, ES = -0.66), Post-6H ($P = 0.013$, ES = -1.04), except at Post-B ($P = 0.407$, ES = -1.21), Post-T ($P = p = 0.640$,

ES = -1.30), Post-24H (P = 0.126, ES = -0.40) and Post-48H (P = 1.000, ES = -0.14), compared to Pre-B value.

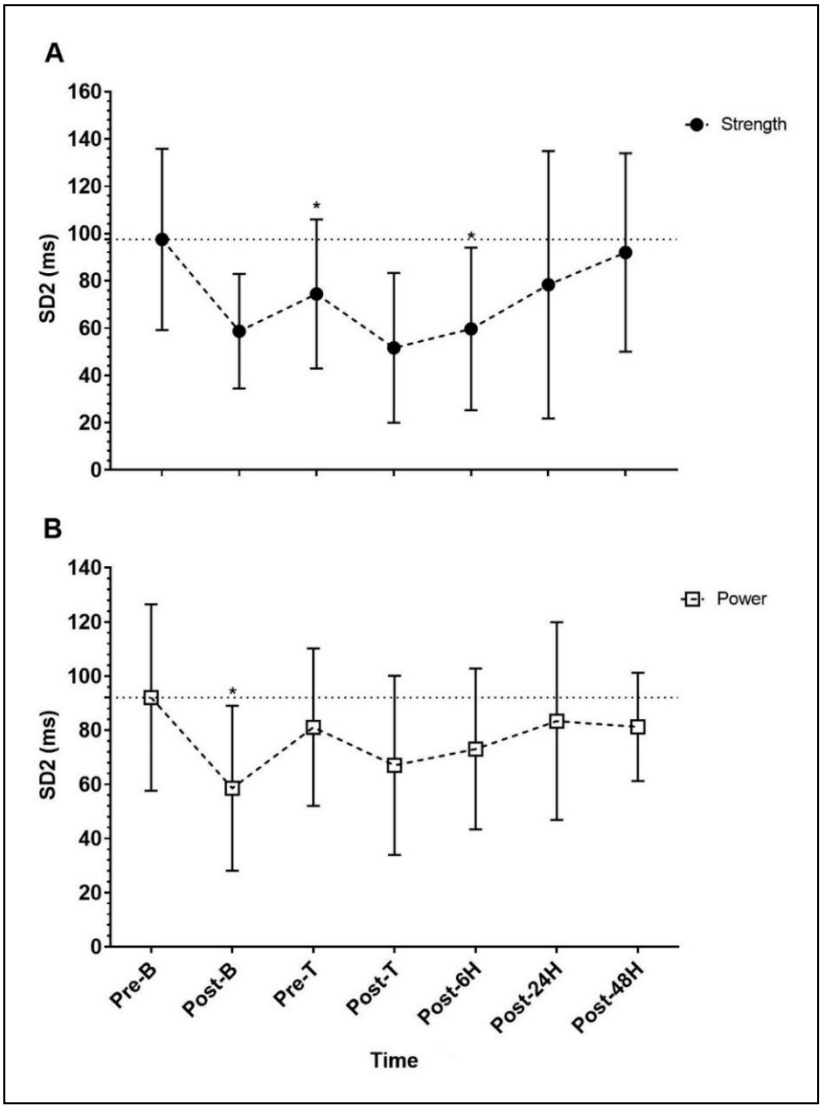


Figure 34. Changes in SD2 parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In P100, no significant time differences were shown at Pre-T (P = 1.000, ES = -0.35), Post-T (P = 0.093, ES = -0.74), Post-6H (P = 0.923, ES = -0.59), Post-24H (P = 1.000, ES = -0.25) and Post-48H (P = 1.000, ES = -0.39), except at Post-B (P = 0.020, ES = -1.03) compared to Pre-B value. These results revealed that SD2 decreased

following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, SD2 recovered to baseline at Post-24H for S100, whereas cardiac sympathetic modulation was not significantly affected by P100 training modality following ARE protocol (Figure 34).

6.2.2.1.9. SD2/SD1 ratio

There was an overall treatment effect (p=0.052) and an overall time effect on SD2/SD1 ($p < 0.001$). There was no significant treatment x time interaction for SD2/SD1 ($p = 0.258$). Simple main effects for treatment showed that SD2/SD1 ratio was significantly lower in the strength modality at Post-24H ($p = 0.027$) and Post-48H ($p = 0.007$; Figure 35) compared to the power modality. This indicates that S100 modality for AREs increased more in SD2/SD1 ratio than P100 modality.

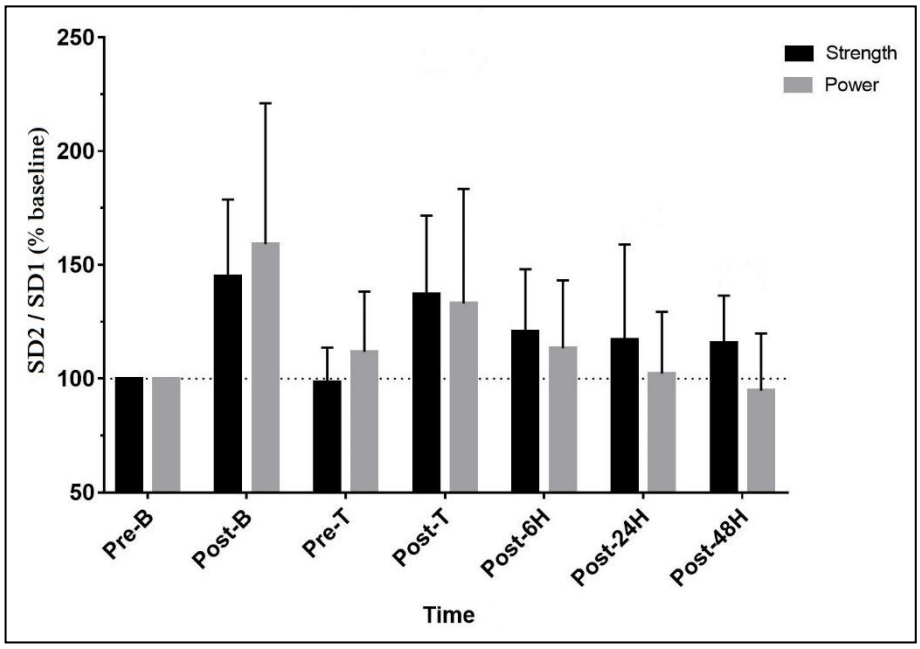


Figure 35. Comparison between S100 and P100 on SD2/SD1 ratio values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that SD2/SD1 ratio differed significantly between time points in S100 ($P = 0.006$) and P100 ($P < 0.001$) trials. In

S100, no significant time differences were observed at Pre-T (P = 1.000, ES = -0.05), Post-T (P = 0.309, ES = 0.89), Post-6H (P = 1.000, ES = 0.59), Post-24H (P = 1.000, ES = 0.50) and Post-48H (p = 1.000, ES = 0.58), except at Post-B (P = 0.054, ES = 1.20), compared to Pre-B value. In P100, no significant time differences were shown at Post-B (P = 0.102, ES = 1.50), Pre-T (P = 1.000, ES = 0.48), Post-T (P = 0.700, ES = 0.98), Post-6H (P = 1.000, ES = 0.48), Post-24H (P = 1.000, ES = 0.01) and Post-48H (P = 1.000, ES = -0.24), compared to Pre-B value. These findings indicate that the balance of cardiac sympathetic and parasympathetic modulation shifted to the cardiac sympathetic modulation after M-Beast protocol and ARE training, and it gradually recovered to baseline values in both S100 and P100. However, it's also showed that these trials did not significantly effect the cardiac sympathovagal balance except for M-Beast protocol of S100. According to the ES results, cardiac sympathovagal balance of P100 recovered at Post-24H, whereas S100's level did not yet recover at Post-48H (Figure 36).

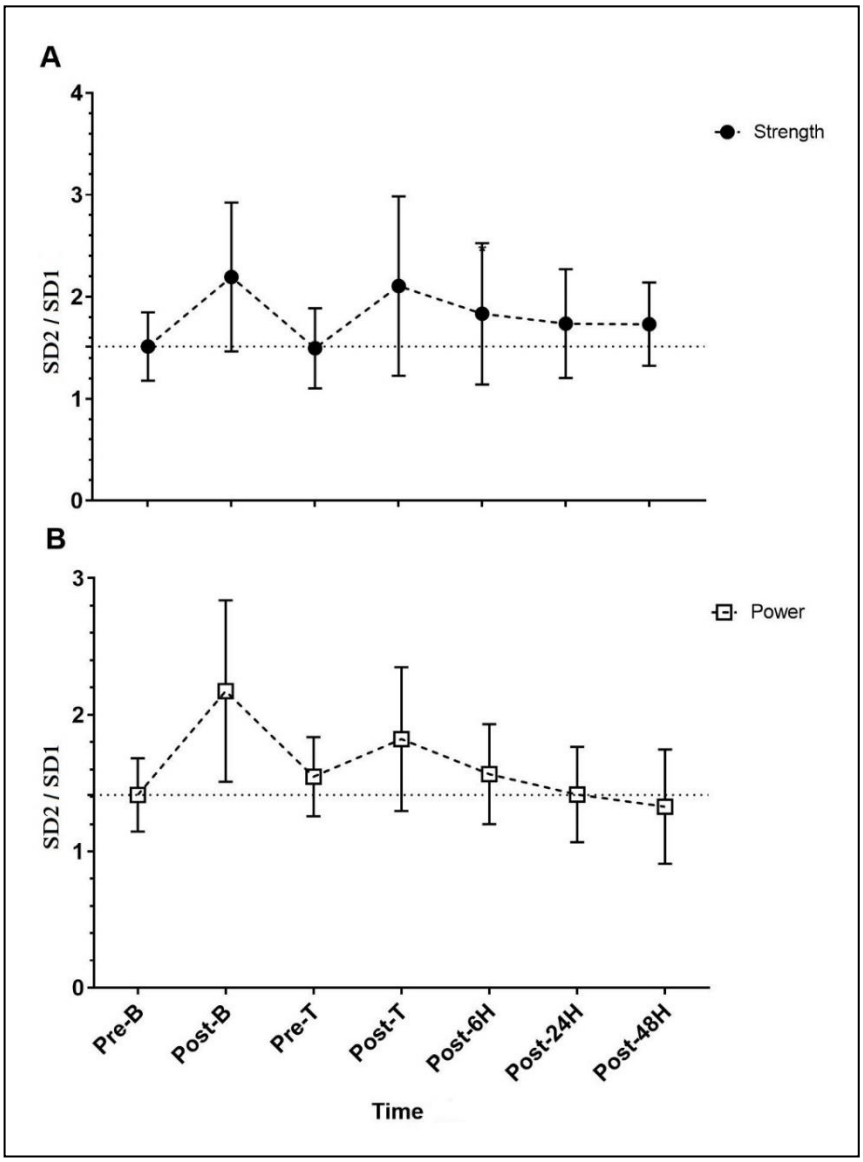


Figure 36. Changes in SD2/SD1 parameter in (A) S100 and (B) P100 protocols (n = 11).

6.2.2.1.10. Stress Score Index (SS)

There was an overall treatment effect ($p = 0.040$) and an overall time effect on SS ($p < 0.001$). There was a significant treatment x time interaction for SS ($p = 0.037$), where simple main effects for treatment showed that SS was significantly lower in the strength modality at Post-T ($p = 0.007$), Post-6H ($p = 0.047$) and Post-24H ($p =$

0.053; Figure 37)) compared to the power modality. This indicates that S100 modality for AREs decreased more in SS than P100 modality.

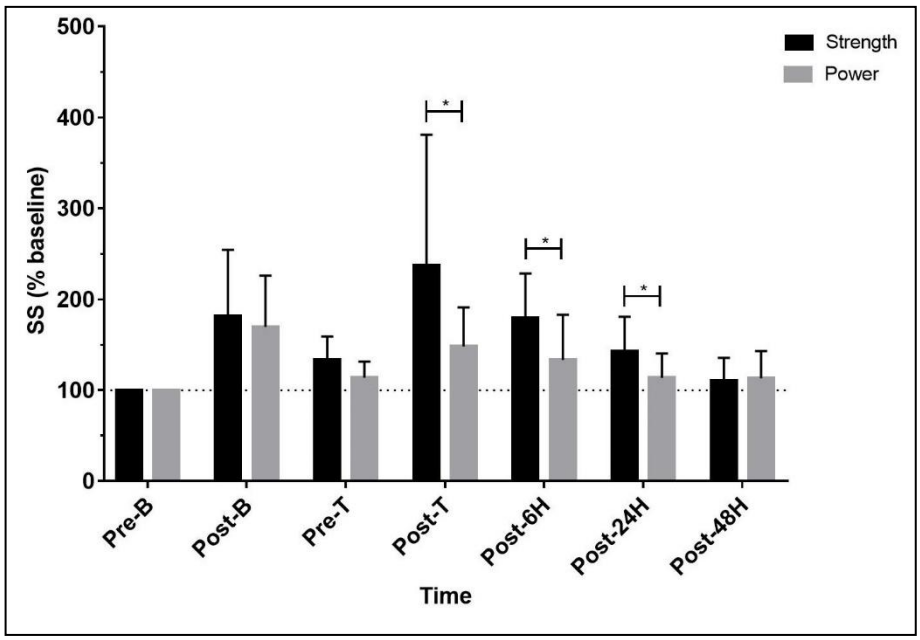


Figure 37. Comparison between S100 and P100 on SS values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that SS differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time differences were observed at Post-B ($P = 0.008$, ES = 0.99), Pre-T ($p = 0.012$, ES = 0.83), Post-T ($P = 0.010$, ES = 0.98), and Post-6H ($P = 0.001$, ES = 1.34), except at Post-24H (P p = 0.284, ES = 0.90), at Post-48H ($P = 1.000$, ES = 0.25), compared to Pre-B value. In P100, significant time differences were shown at Post-B ($P = 0.010$, ES = 1.26), Post-T ($P = 0.034$, ES = 0.85), except at Pre-T: ($P = 0.661$, ES = 0.27), Post-6H

(P = 0.566, ES = 0.60), Post-24H (P = 1.000, ES = 0.24) and Post-48H (P = 1.000, ES = 0.14), compared to Pre-B value.

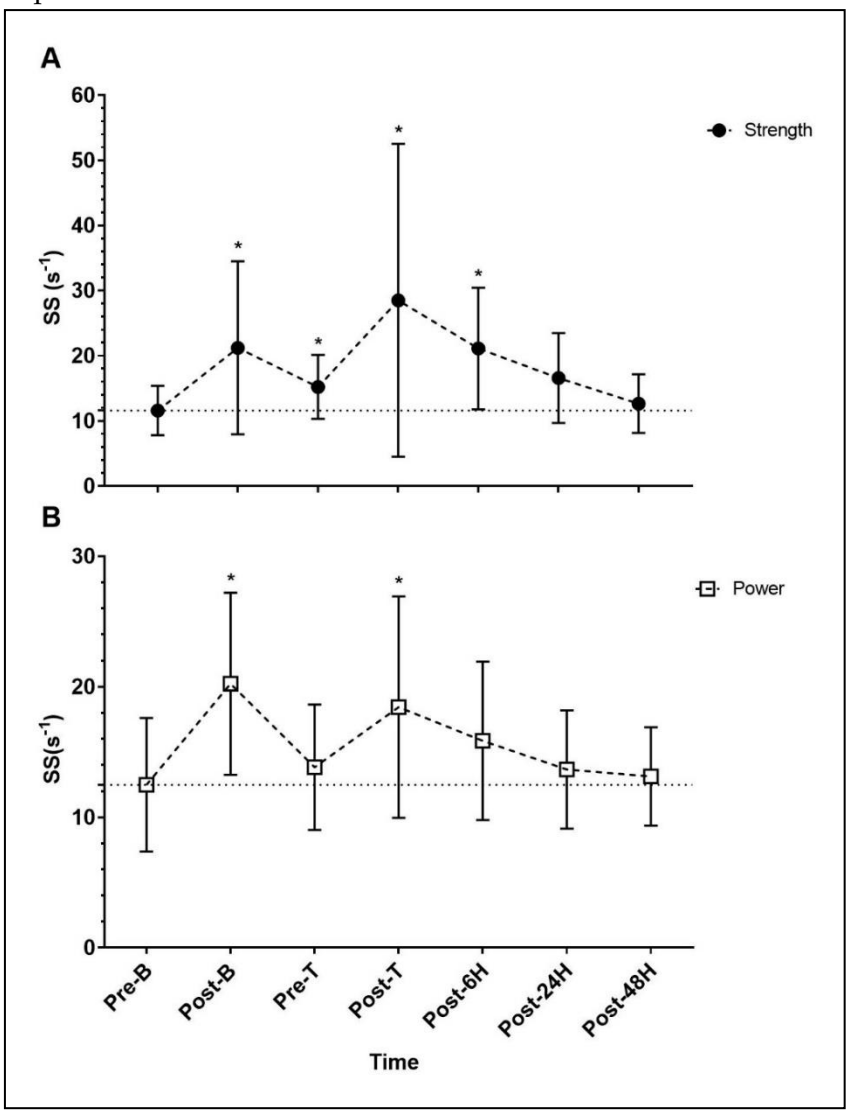


Figure 38. Changes in SS parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results suggest revealed that SS increased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, SS recovered to baseline (Pre-B) at Post-6H for P100, whereas S100 needed longer time (Post-24H) to recover. According to the ES results, SS of both training modalities were not recovered at Post-48H (Figure 38).

Even though not fully recovered, P100 showed better recovery level than S100 at Post-48H.

6.2.2.2. Strength 100 versus Strength 75 versus Strength 50 training

The results of the HRV parameters for comparison between 100% of training load of strength training modality (S100), 75% of training load of strength training modality (S75) and 50% of training load of strength training modality (S50) are reported below.

6.2.2.2.1. pNN50

There was an overall treatment (i.e., Training load) effect ($p = 0.013$) and an overall time effect ($p < 0.001$) on pNN50. There was a significant treatment x time interaction for pNN50 ($p < 0.001$), where simple main effects for treatment showed that pNN50 was significantly different between treatments (S100 vs S75 vs S50) at Pre-B ($p = 0.007$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.029$)), Post-T ($p = 0.002$, (S100 vs S75: $p = 0.018$; S100 vs S50: $p = 0.008$)), Post-6H ($p < 0.001$, (S100 vs S75: $p = 0.003$; S100 vs S50: $p = 0.006$)), Post-24H ($p = 0.015$, (S100 vs S75: $p = 0.029$; S100 vs S50: $p = 0.059$)) and Post-48H ($p < 0.001$, (S100 vs S75: $p = 0.001$; S100 vs S50: $p = 0.041$)) (Figure 39).

Simple main effects over time revealed that pNN50 was significantly different between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P < 0.001$) trials. Compared to Pre-B, significant time differences were observed at Post-B (S100: $p = 0.001$, ES = -1.98; S75: $p < 0.001$, ES = -1.91 and S50: $p = 0.005$, ES = -1.26) and Post-T (S100: $p < 0.001$, ES = -1.79; S75: $p < 0.001$, ES = -1.15 and S50: $p = 0.002$, ES = -0.40) in all training loads. At Pre-T, significant differences were shown in S100 ($p = 0.021$, ES = -0.63) and S75 ($p = 0.003$, ES = -0.51), except in S50 ($p = 1.000$, ES = -0.12) compared to Pre-B value. Similar to Pre-T, Post-6H also showed significant differences in S100 ($p < 0.001$, ES = -1.67) and S75 ($p = 0.006$, ES = -0.97), except in S50 ($p = 1.000$, ES = -0.25) compared to Pre-B value. At Post-24H (S75: $p = 0.114$, ES = -0.50; S50: $p = 1.000$, ES = 0.08) and Post-48H (S75: $p = 1.000$, ES = -0.02; S50: p =

0.867, ES = 0.28) no significant differences were shown compared to Pre-B values in S75 and S50, except in S100 (Post-24H: P = 0.006, ES = -0.86; Post-48H: P = 0.041, ES = -0.69).

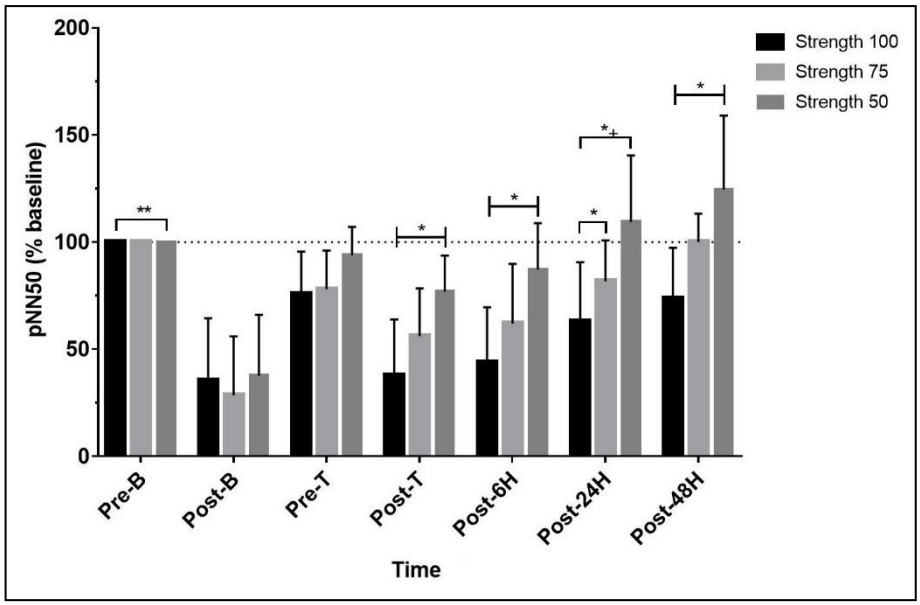


Figure 39. Comparison between S100, S75 and S50 on pNN50 values (n = 12). * Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$). * Significant pairwise comparison differences in S75 compared to S100 ($p \leq 0.05$). ** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$). *+ Significant trend pairwise comparison differences in S50 compared to S100 ($p \leq 0.06$).

These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-6H for S50, whereas S75 needed longer time (Post-24H) to recover. But, S100 did not recover at Post-48H (Figure 40).

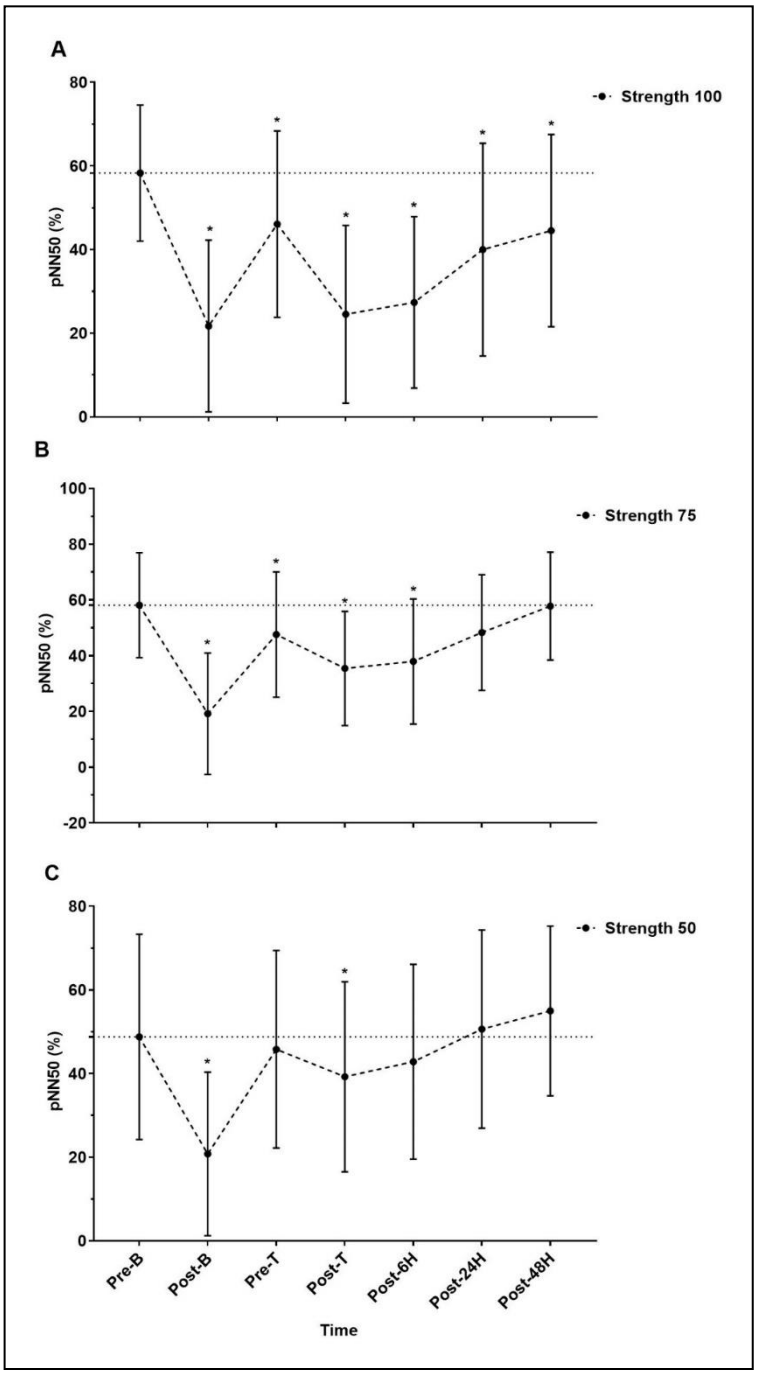


Figure 40. Changes in pNN50 parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 12).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

According to the ES results, cardiac parasympathetic modulation of S50 did recover at post-24H, whereas S75's level almost recovered and S100's level did not yet recover at Post-48H.

6.2.2.2.2. SDNN

There was an overall treatment effect ($p = 0.032$) and an overall time effect ($p < 0.001$) on SDNN. There was a significant treatment x time interaction for SDNN ($p = 0.004$), where simple main effects for treatment showed that SDNN was significantly different between treatments (S100 vs S75 vs S50) at Post-T ($p < 0.001$, (S100 vs S75: $p = 0.002$; S100 vs S50: $p = 0.002$)), Post-6H ($p = 0.006$, (S100 vs S75: $p = 0.044$; S100 vs S50: $p = 0.025$)) and Post-24H ($p = 0.004$, (S100 vs S75: $p = 0.094$; S100 vs S50: $p = 0.017$)) (Figure 41).

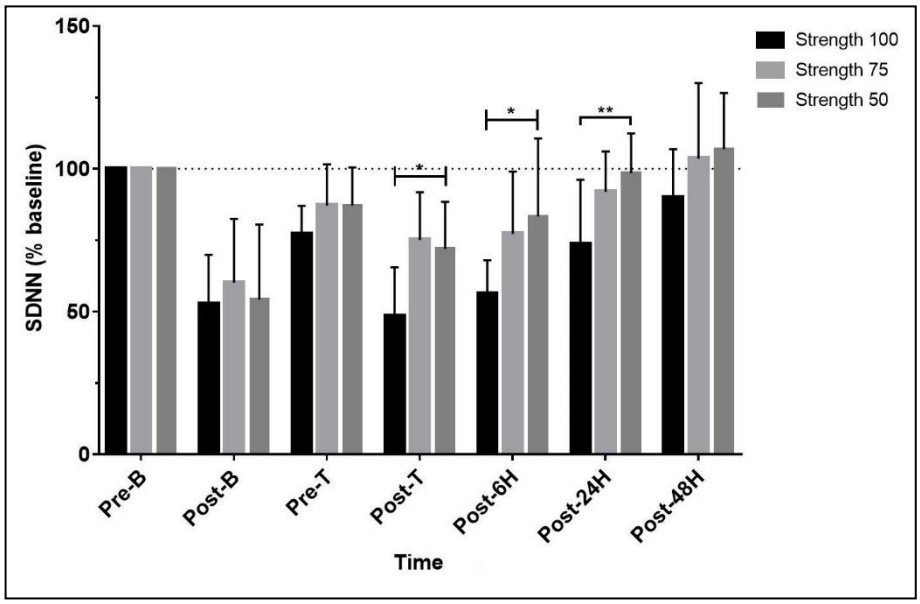


Figure 41. Comparison of S100, S75 and S50 on SDNN values (n = 13). $\vdash^{*}\dashv$ Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$). $\vdash^{**}\dashv$ Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that SDNN different significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P = 0.001$).

Compared to Pre-B, a significant time differences were observed at Post-B (S100: p = 0.001, ES = -1.40; S75: p = 0.004, ES = -1.30 and S50: p = 0.036, ES = -1.17) and Post-T (S100: p = 0.001, ES = -1.41; S75: p = 0.006, ES = -0.72 and S50: p = 0.002, ES = -0.68) in all training modalities. At Pre-T (S75: p = 0.185, ES = -0.40; S50: p = 0.078, ES = -0.32), Post-6H (S75: p = 0.065, ES = -0.66; S50: p = 0.331, ES = -0.54) and Post-24H (S75: p = 1.000, ES = -0.31; S50: p = 1.000, ES = -0.09), there were no significant differences compared to Pre-B in S75 and S50, except in S100 (Pre-T: P < 0.001, ES = -0.62; Post-6H: p < 0.001, ES = -1.13 and Post-24H: p = 0.010, ES = -0.56). However, no significant difference was shown at Post-48H (S100: p = 0.812, ES = -0.27; S75: p = 1.000, ES = -0.03 and S50: p = 1.000, ES = 0.09) compared to Pre-B in all training loads. These results indicate that overall autonomic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, overall autonomic modulation recovered to baseline at Post-6H in S75 and S50, whereas S100 needed longer time (Post-48H) to recover (Figure 42). According to the ES results, overall autonomic modulation of S50 recovered at post-24H, whereas S100 and S75 did not recover at Post-48H. Even though not fully recovered, S75 showed better recovery level than S100 at Post-48H.

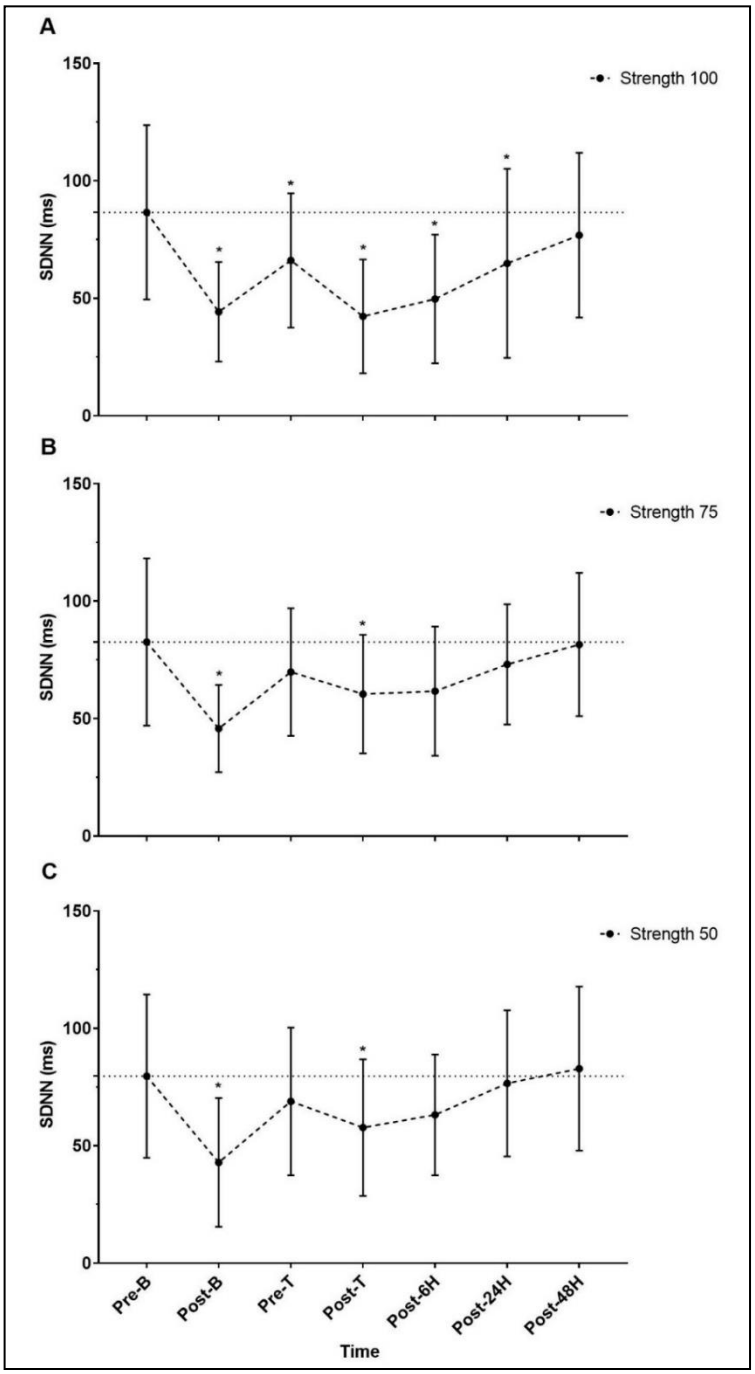


Figure 42. Changes in SDNN parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.2.3. Ln RMSSD

There was an overall treatment effect (p = 0.008) and an overall time effect (p < 0.001) on Ln RMSSD. There was a significant treatment x time interaction for Ln RMSSD (p = 0.001), where simple main effects for treatment showed that Ln RMSSD was significantly different between treatments (S100 vs S75 vs S50) at Pre-B (p = 0.026, (S100 vs S75: P = 1.000 ; S100 vs S50: P = 0.084)), Post-T (P = 0.001, (S100 vs S75: p = 0.014 ; S100 vs S50: p = 0.002)), Post-6H (p < 0.001, (S100 vs S75: p = 0.009 ; S100 vs S50: p < 0.001)), Post-24H (p < 0.001, (S100 vs S75: p = 0.002 ; S100 vs S50: p = 0.002)) and Post-48H (p = 0.013, (S100 vs S75: p = 0.015 ; S100 vs S50: p = 0.167)) (Figure 43).

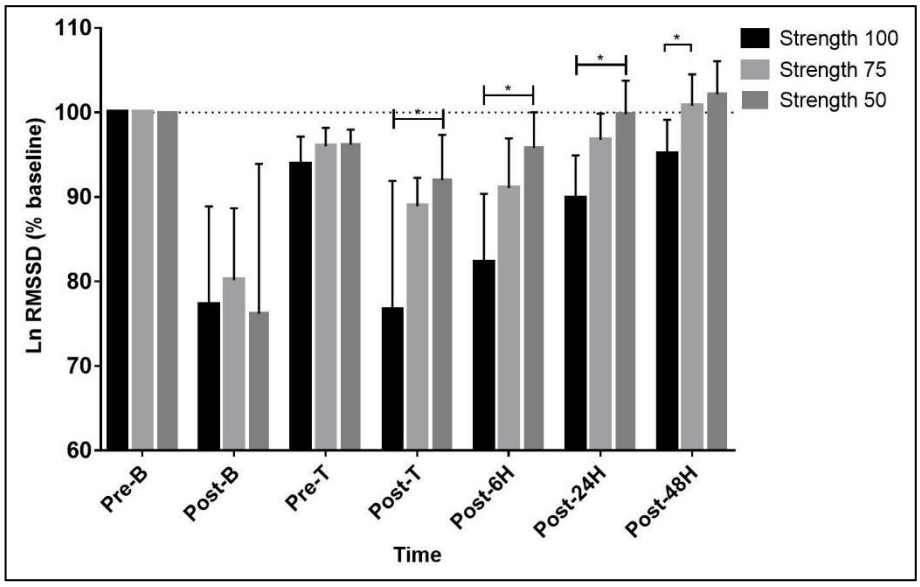


Figure 43. Comparison between S100, S75 and S50 on Ln RMSSD values (n = 13). * Significant pairwise comparison differences in S75 and S50 compared to S100 (p ≤ 0.05). * Significant pairwise comparison differences in S75 compared to S100 (p ≤ 0.05).

Simple main effects over time revealed that Ln RMSSD different significantly between time points in S100 (P < 0.001), S75 (P < 0.001) and S50 (P < 0.001). Compared to Pre-B, significant time differences were observed at Post-B (S100: p < 0.001, ES = -1.65; S75: p < 0.001, ES = -1.62 and S50: p = 0.004, ES = -1.27), Pre-T

(S100: $p < 0.001$, ES = -0.54; S75: $p < 0.001$, ES = -0.35 and S50: $p < 0.001$, ES = -0.28) and Post-T (S100: $p = 0.001$, ES = -1.45; S75: $p < 0.001$, ES = -0.99 and S50: $p = 0.001$, ES = -0.57) in all training loads. At Post-6H (S100: $p < 0.001$, ES = -1.39; S75: $p = 0.002$, ES = -0.78) and Post-24H (S100: $p < 0.001$, ES = -0.84; S75: $p = 0.039$, ES = -0.30), there were significant differences compared to Pre-B in S100 and S75, except in S50 (Post-6H: $p = 0.067$, ES = -0.33; Post-24H: $p = 1.000$, ES = -0.02). However, except in S100 ($p = 0.013$, ES = -0.42), there was no significant differences were shown in S75 ($p = 1.000$, ES = 0.06) and S50 ($p = 1.000$, ES = 0.15) at Post-48H compared to Pre-B values. These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-6H for S50, whereas S75 needed longer time (Post-48H) to recover. Although, S100 was not recovered at Post-48H (Figure 44). According to the ES results, cardiac parasympathetic modulation of S75 and S50 recovered at post-48H, whereas S100's level did not yet recover at Post-48H. Interestingly, S50 showed better recovery level than S75 at Post-48H.

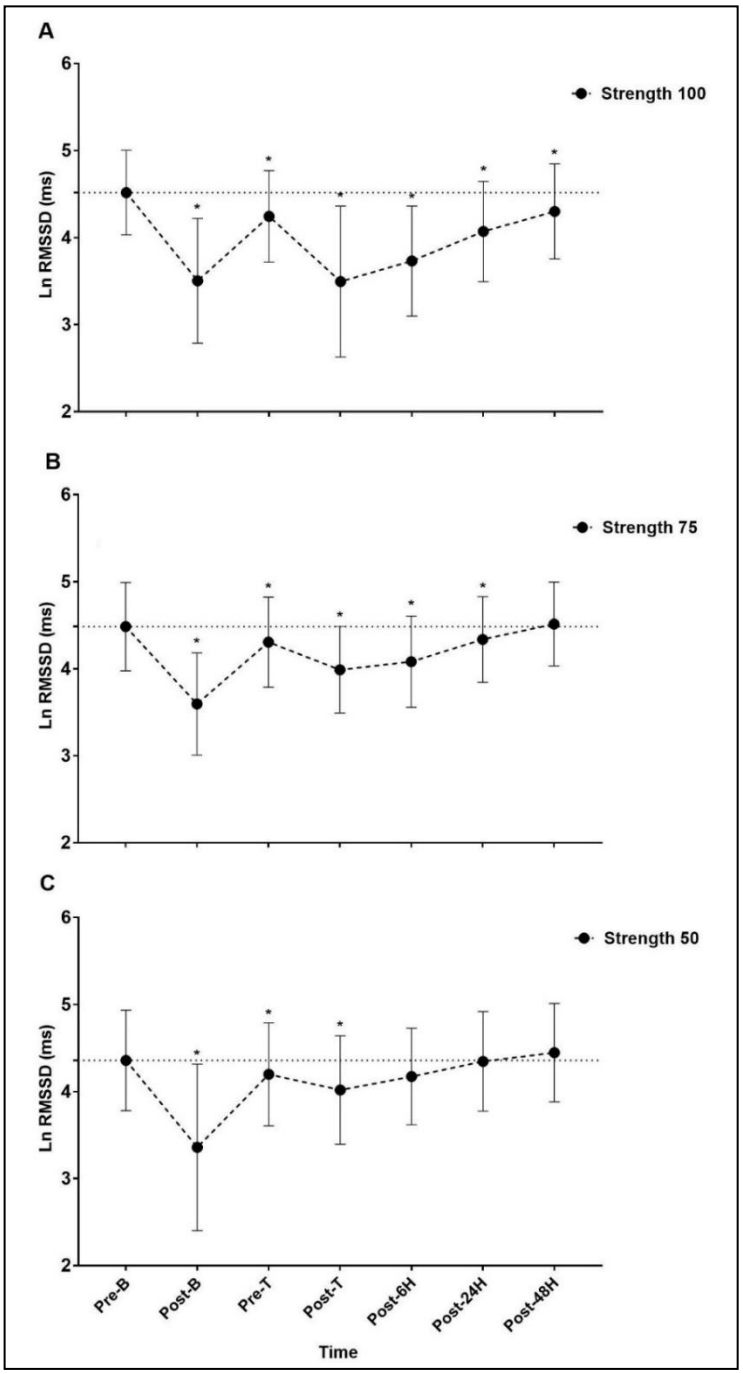


Figure 44. Changes in Ln RMSSD parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.2.4. HF(nu)

There was no overall treatment effect on HF(nu) (p = 0.618). However, there was an overall time effect on HF(nu) (p = 0.001). No significant group x time interaction for HF(nu) was observed (p = 0.172; Figure 45).

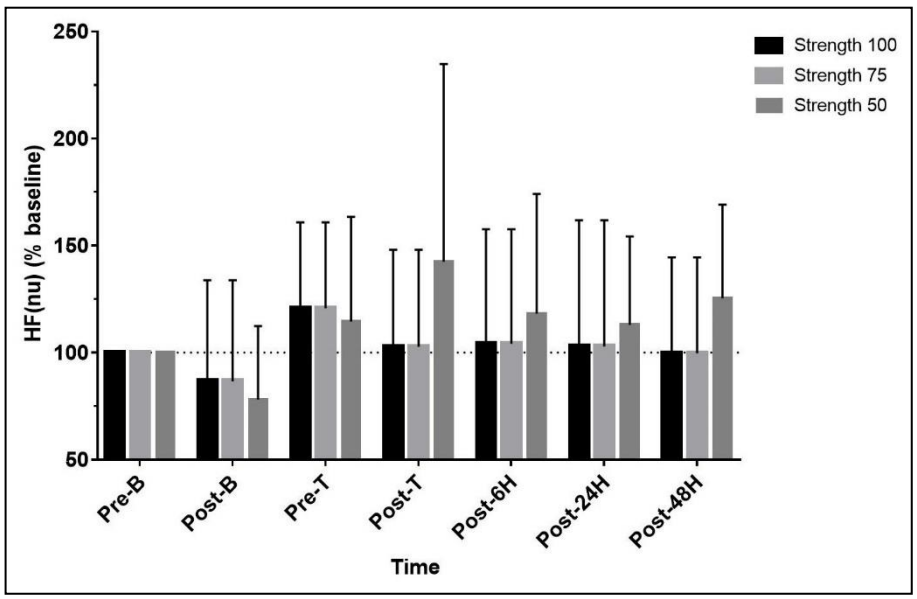


Figure 45. Comparison of S100, S75 and S50 on HF(nu) values (n = 13).

Simple main effects over time revealed that HF(nu) different significantly between time points in S75 (P = 0.004) and S50 (P = 0.007), except S100 (P = 0.354) trial. Compared to Pre-B, no significant time differences were observed at Post-B (S100: p = 1.000, ES = -0.41; S75: p = 0.159, ES = -0.85 and S50: p = 0.591, ES = -0.63), Pre-T (S100: p = 1.000, ES = 0.41; S75: p = 1.000, ES = -0.16 and S50: p = 1.000, ES = 0.16), Post-T (S100: p = 1.000, ES = -0.13; S75: p = 0.075, ES = -1.29 and S50: p = 1.000, ES = 0.38), Post-6H (S100: p = 1.000, ES = -0.05; S75: p = 0.604, ES = -0.95 and S50: p = 1.000, ES = 0.10), Post-24H (S100: p = 1.000, ES = -0.16; S75: p = 0.355, ES = -0.61 and S50: p = 1.000, ES = 0.02) and Post-48H (S100: p = 1.000, ES = -0.16; S75: p = 1.000, ES = -0.18 and S50: p = 1.000, ES = 0.35) in all training loads.

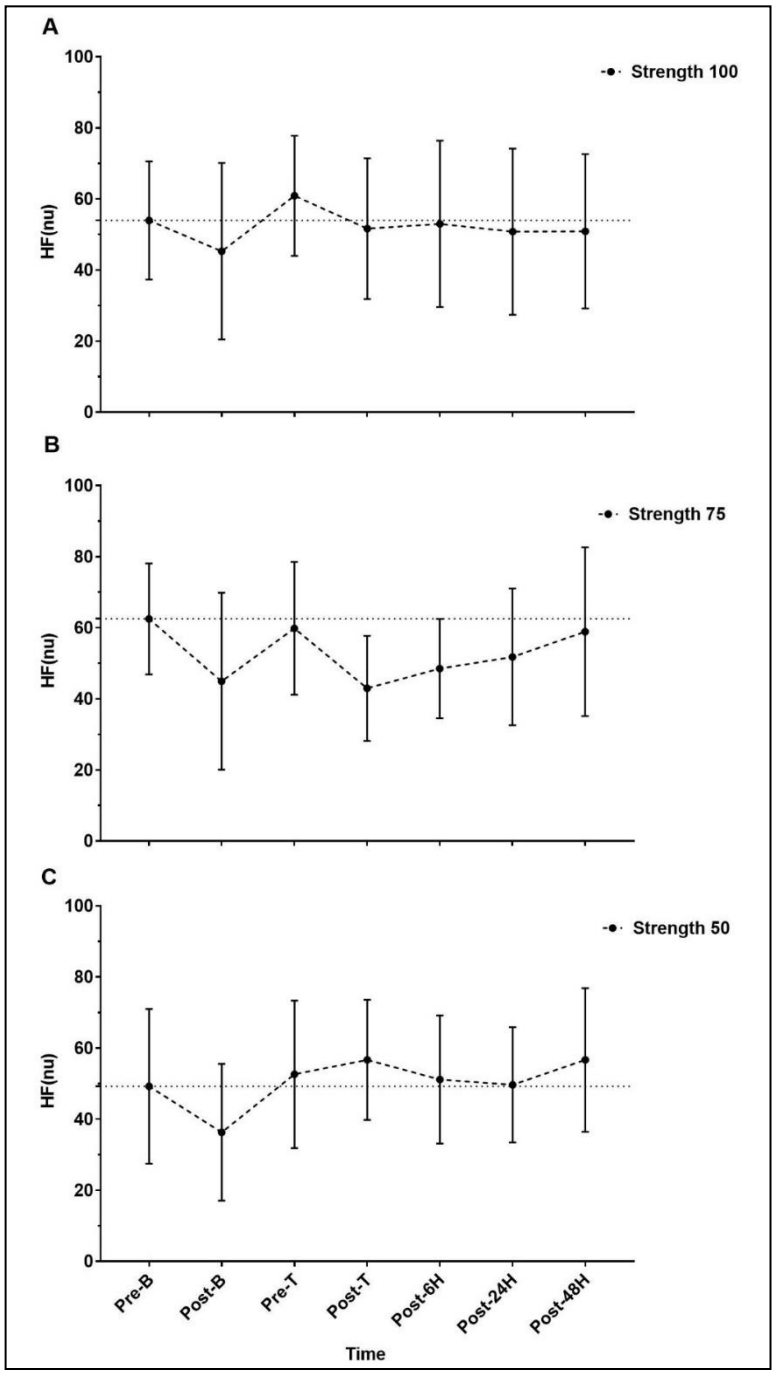


Figure 46. Changes in HF(nu) parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results show that cardiac parasympathetic modulation decreased following the M-Beast protocol in all 3 training loads and ARE protocols of S100 and S75 gradually returned to Pre-B values. According to the ES results, cardiac parasympathetic modulation of S50 was remained recovered from Pre-T, whereas S75's and S100's level did not yet recover at Post-48H. (Figure 46).

6.2.2.2.5. LF(nu)

There was no overall treatment effect on LF(nu) ($p = 0.630$). However, there was an overall time effect on LF(nu) ($p = 0.001$). No significant group x time interaction for LF(nu) was observed ($p = 0.165$; Figure 47).

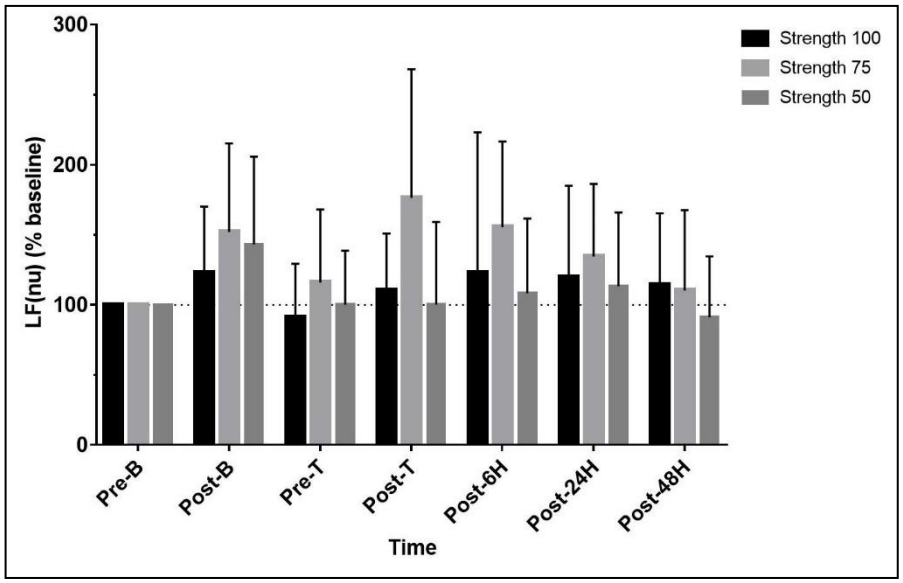


Figure 47. Comparison of S100, S75 and S50 on LF(nu) values (n = 13).

Simple main effects over time revealed that LF(nu) different significantly between time points in S75 ($P = 0.004$) and S50 ($P = 0.008$), except S100 ($P = 0.341$) training loads.

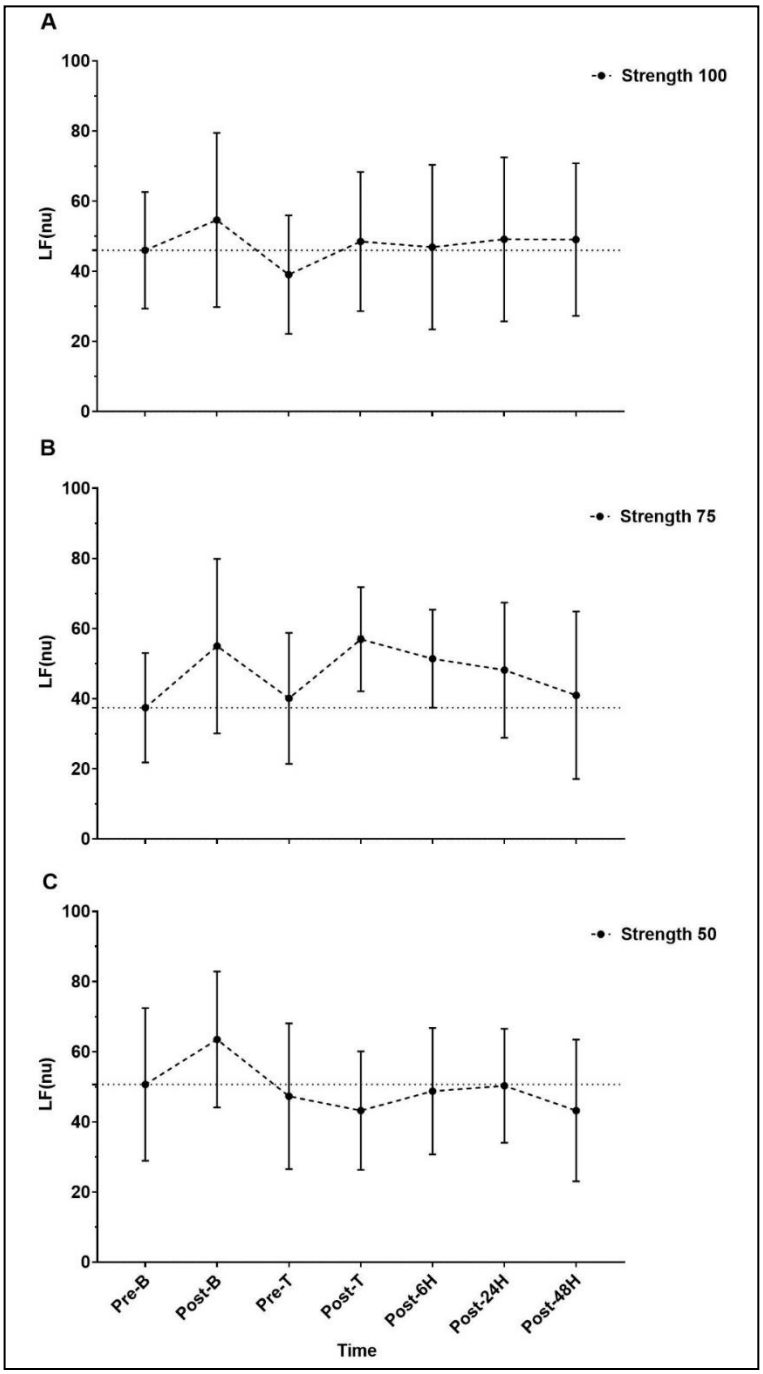


Figure 48. Changes in LF(nu) parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13).

Compared to Pre-B, no significant time differences were observed at Post-B (S100: p = 1.000, ES = 0.41; S75: p = 0.159, ES = 0.85 and S50: p = 0.609, ES = 0.62),

Pre-T (S100: p = 1.000, ES = -0.41; S75: p = 1.000, ES = 0.16 and S50: p = 1.000, ES = -0.16), Post-T (S100: p = 1.000, ES = 0.14; S75: p = 0.076, ES = 1.28 and S50: p = 1.000, ES = -0.38), Post-6H (S100: p = 1.000, ES = 0.04; S75: p = 0.610, ES = 0.94 and S50: p = 1.000, ES = -0.10), Post-24H (S100: p = 1.000, ES = 0.16; S75: p = 0.355, ES = 0.61 and S50: p = 1.000, ES = -0.02) and Post-48H (S100: p = 1.000, ES = 0.16; S75: p = 1.000, ES = 0.18 and S50: p = 1.000, ES = -0.35) in all training loads. These results revealed that cardiac sympathetic modulation increased following the M-Beast protocol in all 3 trials and ARE protocols in S100 and S75 and it gradually returned to Pre-B values. According to the ES results, cardiac sympathetic modulation of S50 returned to Pre-T, whereas S75's and S100's level did not yet recover at Post-48H (Figure 48).

6.2.2.2.6. LF/HF ratio

There was no overall treatment effect on LF/HF ratio (p = 0.619). However, there was an overall time effect on LF/HF ratio (p = 0.001). No significant group x time interaction for LF/HF ratio was observed (p = 0.190; Figure 49).

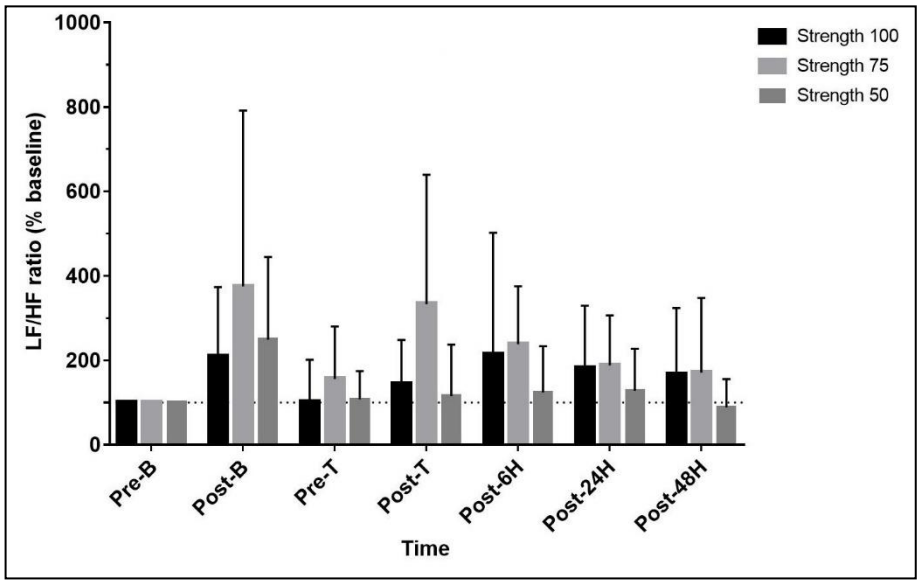


Figure 49. Comparison of S100, S75 and S50 on LF/HF ratio values (n = 13).

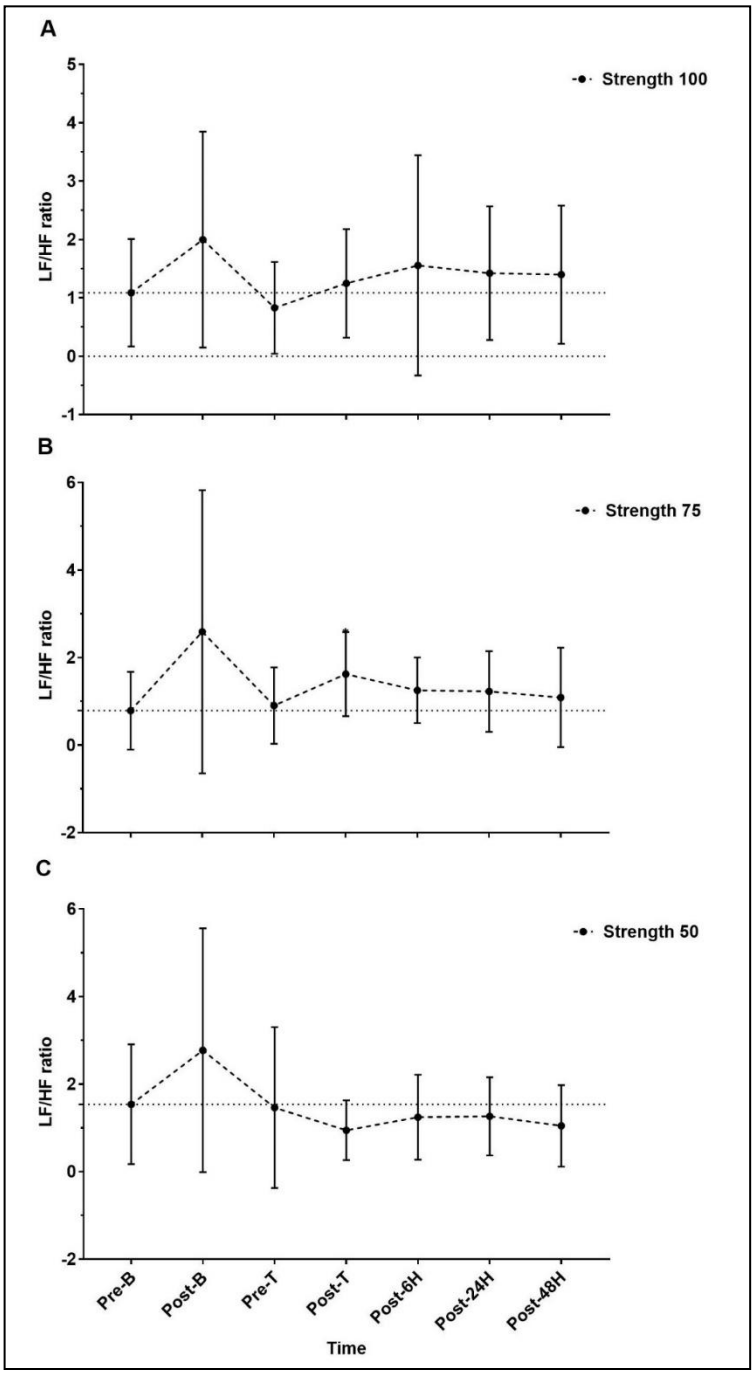


Figure 50. Changes in LF/HF ratio parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13).

Simple main effects over time revealed that LF/HF ratio different significantly between time points in S75 (P = 0.004) and S50 (P = 0.005) trials, except S100 (P = 0.444). In S100, there were no significant time differences observed at all the time points (Post-B (p = 1.000, ES = 0.62), Pre-T (p = 1.000, ES = -0.30), Post-T (p = 1.000, ES = 0.17), Post-6H (p = 1.000, ES = 0.32), Post-24H (p = 1.000, ES = 0.32) and Post-48H (p = 1.000, ES = 0.29)) compared to Pre-B. Similar results were reported in S75 (Post-B (p = 0.264, ES = 0.76), Pre-T (p = 1.000, ES = 0.13), Post-T (p = 0.086, ES = 0.90), Post-6H (p = 0.637, ES = 0.57), Post-24H (p = 0.584, ES = 0.49) and Post-48H (p = 1.000, ES = 0.30)) and S50 (Post-B (p = 0.421, ES = 0.56), Pre-T (p = 1.000, ES = -0.05), Post-T (p = 1.000, ES = -0.55), Post-6H (p = 1.000, ES = -0.25), Post-24H (p = 1.000, ES = -0.24) and Post-48H (p = 1.000, ES = -0.42)) trials compared to Pre-B values. These results revealed that cardiac sympathovagal balance shifted to cardiac sympathetic modulation following the M-Beast protocol in all three trials and S100 and S75 and it gradually returned to Pre-B values. According to the ES results, cardiac sympathovagal balance of S50 returned to Pre-T, whereas S75's and S100's level did not yet recover at Post-48H (Figure 50).

6.2.2.2.7. Total power

There was an overall treatment effect (p = 0.013) and an overall time effect (p < 0.001) on TP. There was a significant treatment x time interaction for TP (p < 0.001), where simple main effects for treatment showed that TP was significantly different between treatments (S100 vs S75 vs S50) at Post-T (p < 0.001, (S100 vs S75: p = 0.002; S100 vs S50: p < 0.001)), Post-6H (p = 0.014, (S100 vs S75: p = 0.065 ; S100 vs S50: p = 0.038)) and Post-24H (p = 0.038, (S100 vs S75: p = 0.095 ; S100 vs S50: p = 0.122)) (Figure 51).

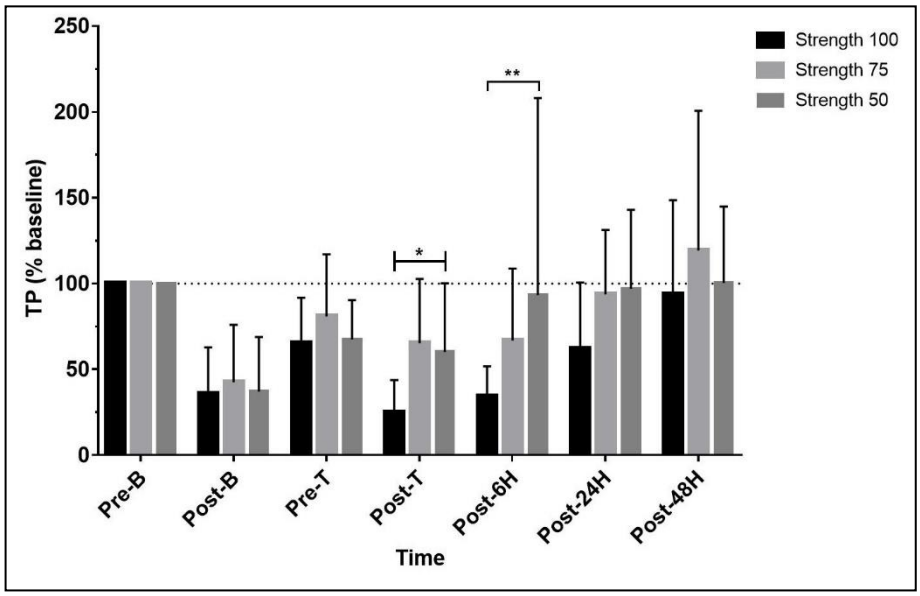


Figure 51. Comparison between S100, S75 and S50 on pNN50 values (n = 13). *—* Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$). **—** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that TP different significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P = 0.007$) trials. Compared to Pre-B, significant time differences were observed at Post-B in S100 ($p = 0.04$, ES = -1.10) and S75 ($p = 0.005$, ES = -1.05), except for S50 ($p = 0.084$, ES = -1.02). Pre-T (S100: $p = 0.032$, ES = -0.58; S50: $p = 0.021$, ES = -0.53) and Post-T (S100: $p = 0.015$, ES = -1.23; S50: $p = 0.029$, ES = -0.57) showed significant differences compared to Pre-B in S100 and S50, except for S75 (Pre-T: $p = 0.667$, ES = -0.42; Post-T: $p = 0.089$, ES = -0.68). Post-6H (S75: $p = 0.155$, ES = -0.65; S50: $p = 1.000$, ES = -0.60) and Post-24H (S75: $p = 1.000$, ES = -0.30; S50: $p = 1.000$, ES = -0.22) showed no significant differences compared to Pre-B in S75 and S50, except for S100 (Pre-T: $p < 0.001$, ES = -0.42; Post-T: $p = 0.020$, ES = -0.68). Interestingly, no significant differences were shown at Post-48H (S100: $p = 1.000$, ES = -0.26; S75: $p = 1.000$, ES = -0.27 and S50: $p = 1.000$, ES = -0.07) in all training loads.

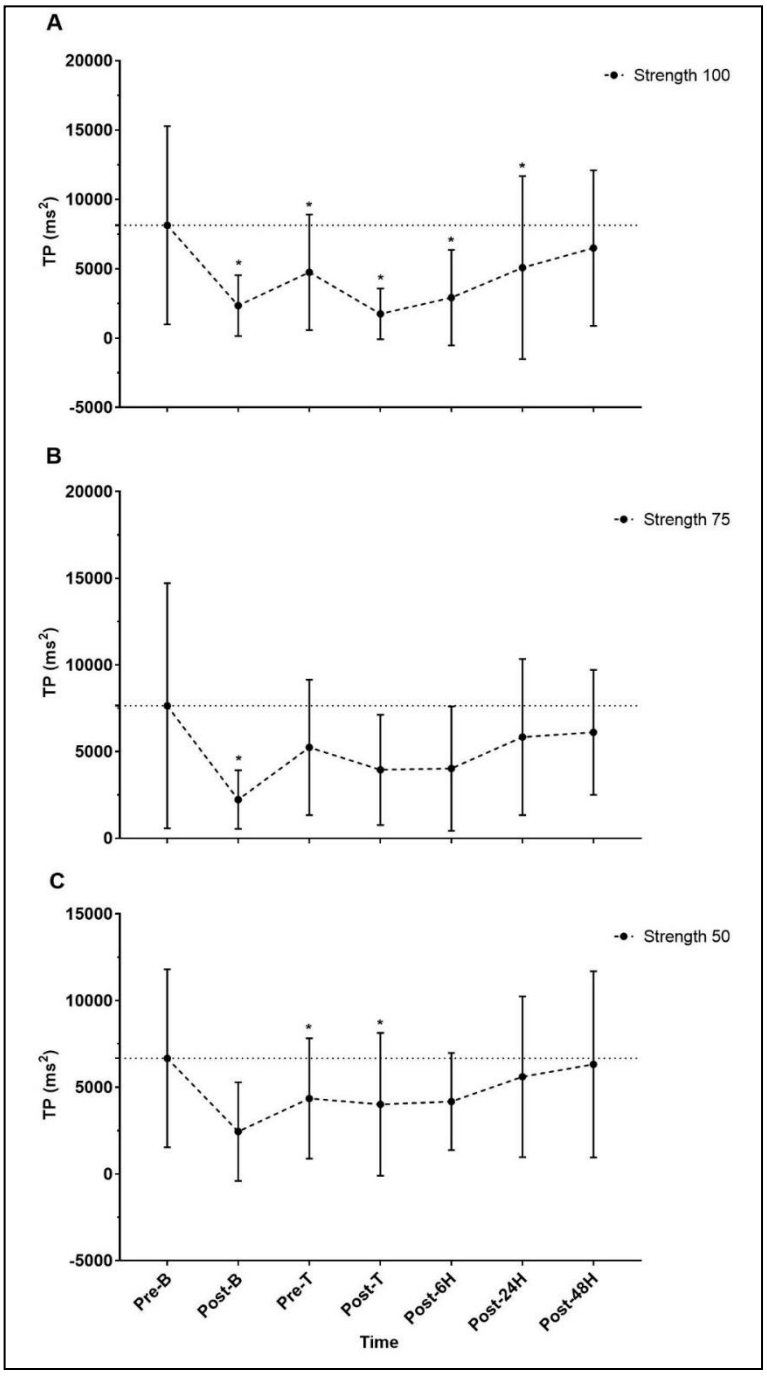


Figure 52. Changes in TP parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that total autonomic activity decreased following the M-Beast protocol and ARE protocols for all training loads and gradually returned to Pre-B values. Interestingly, total autonomic activity recovered to baseline (Pre-B) at Post-6H for S50, whereas S100 needed longer time (Post-24H) to recover. Moreover, compared to Pre-B, S75's total autonomic activity remained decreased without significant difference after M-Beast protocol. According to the ES results, total autonomic activity of all three training loads did not yet recover at Post-48H. Even though not fully recovered, S50 showed better recovery level than S100 and S75 at Post-48H (Figure 52).

6.2.2.2.8. SD1

There was an overall treatment effect ($p = 0.008$) and an overall time effect ($p < 0.001$) on SD1. There was a significant treatment x time interaction for SD1 ($p = 0.001$), where simple main effects for treatment showed that SD1 was significantly different between treatments (S100 vs S75 vs S50) at Pre-B ($p = 0.026$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.084$)), Post-T ($p = 0.001$, (S100 vs S75: $p = 0.014$; S100 vs S50: $p = 0.002$)), Post-6H ($p < 0.001$, (S100 vs S75: $p = 0.009$; S100 vs S50: $p < 0.001$)), Post-24H ($p < 0.001$, (S100 vs S75: $p = 0.002$; S100 vs S50: $p = 0.002$)) and Post-48H ($p = 0.013$, (S100 vs S75: $p = 0.015$; S100 vs S50: $p = 0.166$)) (Figure 53).

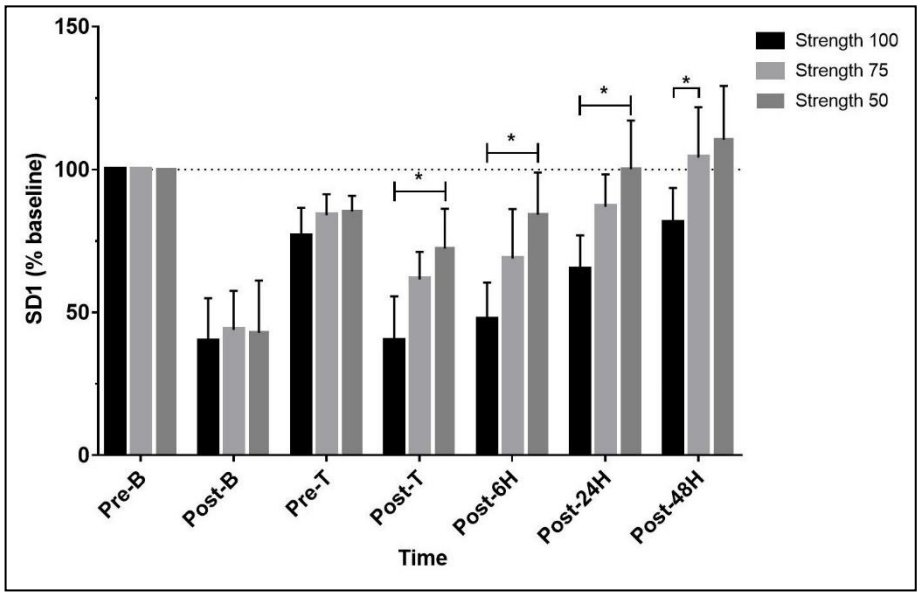


Figure 53. Comparison between S100, S75 and S50 on SD1 values (n = 13). * Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$). * Significant pairwise comparison differences in S75 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that SD1 was significantly different between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P < 0.001$) trial. Compared to Pre-B, significant time differences were observed at Post-B (S100: $p < 0.001$, ES = -1.46; S75: $p < 0.001$, ES = -1.41 and S50: $p = 0.004$, ES = -1.25), Pre-T (S100: $p < 0.001$, ES = -0.48; S75: $p < 0.001$, ES = -0.35 and S50: $p < 0.001$, ES = -0.27) and Post-T (S100: $p = 0.001$, ES = -1.45; S75: $p < 0.001$, ES = -0.99 and S50: $p = 0.001$, ES = -0.58) in all training modalities. Post-6H (S100: $p < 0.001$, ES = -1.25; S75: $p = 0.002$, ES = -0.78) and Post-24H (S100: $p < 0.001$, ES = -0.75; S75: $p = 0.039$, ES = -0.32) showed significant differences compared to Pre-B in S100 and S75, except for S50 (Post-6H: $p = 0.067$, ES = -0.38; Post-24H: $p = 1.000$, ES = -0.03). However, except in S100 ($p = 0.013$, ES = -0.35), there was no significant differences shown in S75 ($p = 1.000$, ES = 0.03) and S50 ($p = 1.000$, ES = 0.15) at Post-48H compared to Pre-B.

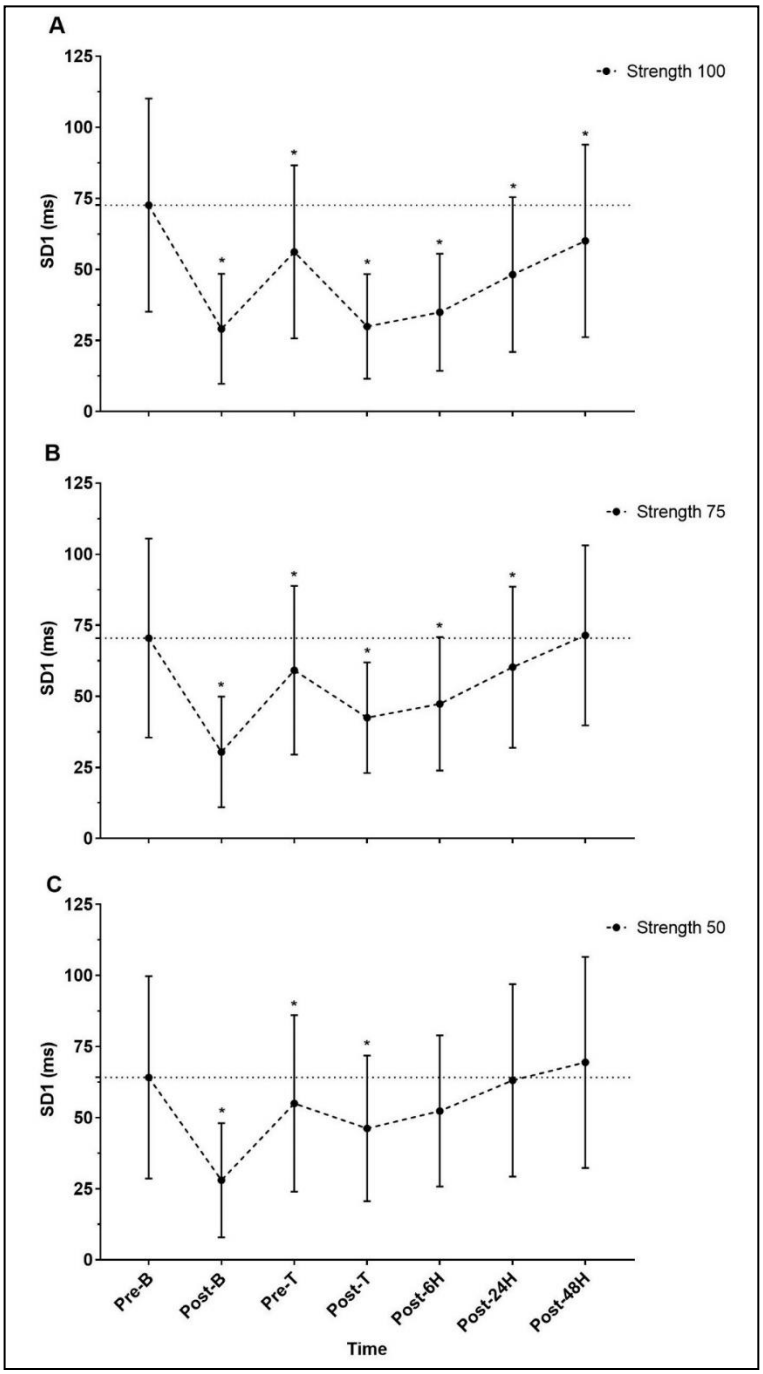


Figure 54. Changes in SD1 parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-6H for S50, whereas S75 needed longer time (Post-48H) to recover. S100 did not recover at Post-48H (Figure 54). According to the ES results, cardiac parasympathetic modulation of S50 and S75 recovered at Post-48H, whereas S100's level did not recover at Post-48H. Where there was full recovery, S50 showed better recovery level compared to S75 at Post-48H.

6.2.2.2.9. SD2

There was no overall treatment effect on SD2 ($p = 0.087$). However, there was an overall time effect on SD2 ($p < 0.001$). There was a significant treatment x time interaction for SD2 ($p = 0.027$), where simple main effects for treatment showed that SD2 was significantly different between treatments (S100 vs S75 vs S50) at Post-T ($p < 0.001$, (S100 vs S75: $p = 0.002$; S100 vs S50: $p = 0.005$)) and Post-24H ($p = 0.028$, (S100 vs S75: $p = 0.248$; S100 vs S50: $p = 0.084$)) (Figure 55).

Simple main effects over time revealed that SD2 differed significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P = 0.004$) trial. In S100, significant time differences were observed at Post-B ($p = 0.003$, ES = -1.32), Pre-T ($p = 0.003$, ES = -0.69), Post-T ($p = 0.002$, ES = -1.33) and Post-6H ($p < 0.001$, ES = -1.02) except at Post-24H ($p = 0.086$, ES = -0.46) and Post-48H ($p = 1.000$, ES = -0.21) compared to Pre-B. In S75, no significant time differences were shown at Pre-T ($p = 1.000$, ES = -0.43), Post-T ($p = 0.326$, ES = -0.54), Post-6H ($p = 0.485$, ES = -0.56), Post-24H ($p = 1.000$, ES = -0.29) and Post-48H ($p = 1.000$, ES = -0.10) except at Post-B ($P = 0.029$, ES = -1.19) compared to Pre-B.

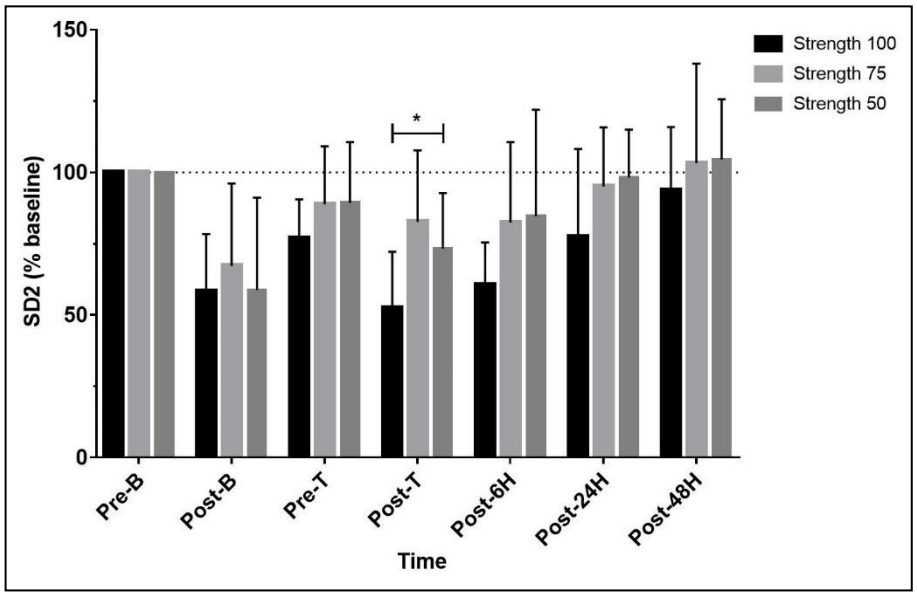


Figure 55. Comparison of S100, S75 and S50 on SD2 values (n = 13). ⊢*⊣ Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$).

In S50, no significant time differences were shown at Post-B ($p = 0.092$, ES = -1.07), Pre-T ($p = 0.989$, ES = -0.33), Post-6H ($p = 0.853$, ES = -0.60), Post-24H ($p = 1.000$, ES = -0.12) and Post-48H ($p = 1.000$, ES = 0.04) except at Post-T ($p = 0.011$, ES = -0.69) compared to Pre-B. These results revealed that SD2 decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, SD2 recovered to baseline (Pre-B) at Post-6H for S50, whereas S100 needed longer time (Post-24H) to recover. Moreover, compared to Pre-B, S75's SD2 remained decreased without significant difference after M-Beast protocol. According to the ES results, SD2 of S50 recovered at post-48H, whereas S100's and S75's level did not recover at Post-48H (Figure XX). Even though not fully recovered, S75 showed better recovery level than S100 at Post-48H (Figure 56).

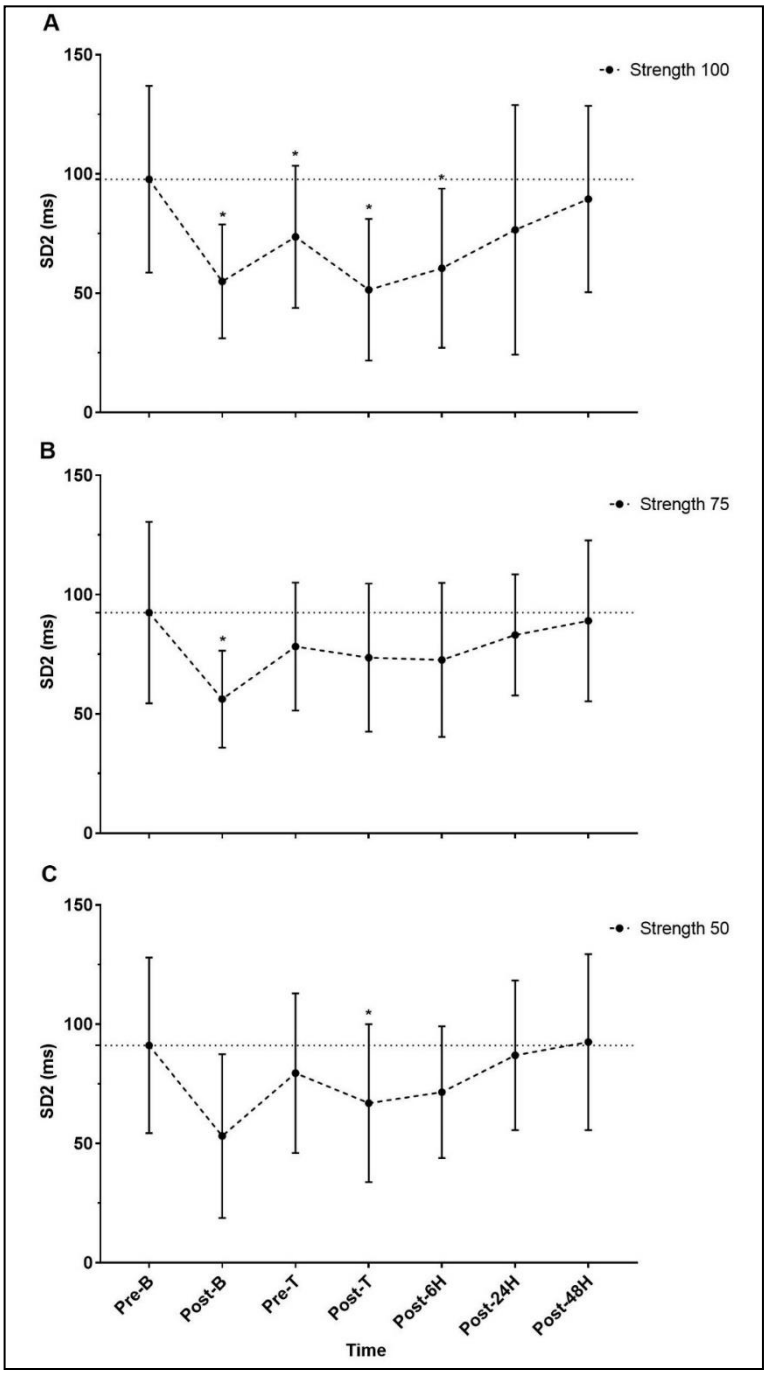


Figure 56. Changes in SD2 parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.2.10. SD2/SD1 Ratio

There was no overall treatment effect on SD2/SD1 ratio ($p = 0.102$). However, there was an overall time effect on SD2/SD1 ratio ($p < 0.001$) and a significant group x time interaction for SD2/SD1 ratio was observed ($p = 0.046$). Simple main effects for treatment showed that SD2/SD1 ratio was significantly different between treatments at the Post-T ($p = 0.004$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.013$)), and Post-6H ($p = 0.040$, (S100 vs S75: $p = 0.388$; S100 vs S50: $p = 0.098$)) (Figure 57).

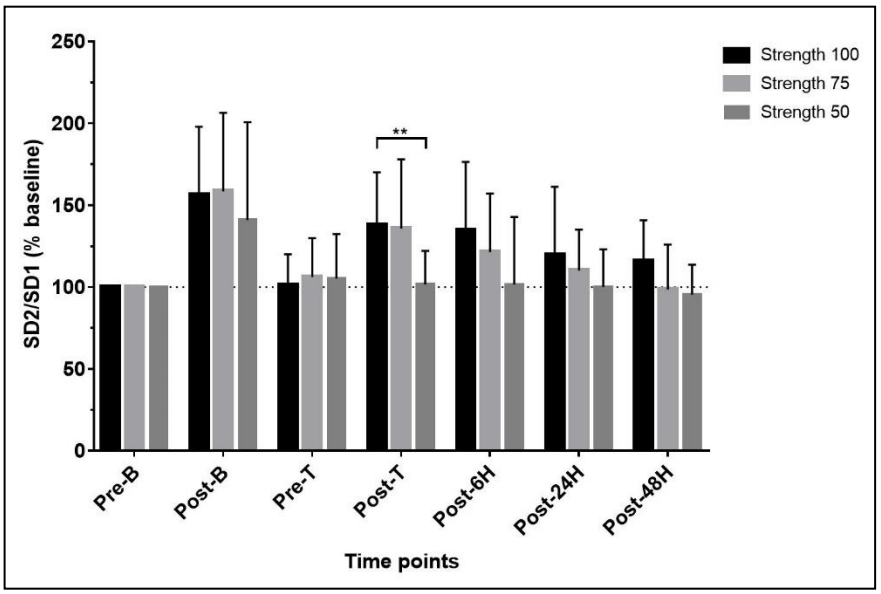


Figure 57. Comparison of S100, S75 and S50 on SD2/SD1 ratio values (n = 13). ** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that SD2/SD1 ratio differed significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P = 0.040$) trial. Compared to Pre-B, significant time differences were observed at Post-B in S100 ($p = 0.002$, ES = 1.42) and S75 ($p = 0.015$, ES = 1.46) except in S50 ($p = 0.650$, ES = 0.71). At Post-T, no significant differences were shown in S75 ($p = 0.144$, ES = 1.22) and S50 ($p = 1.000$, ES = -0.10), except in S100 ($p = 0.007$, ES = 0.88).

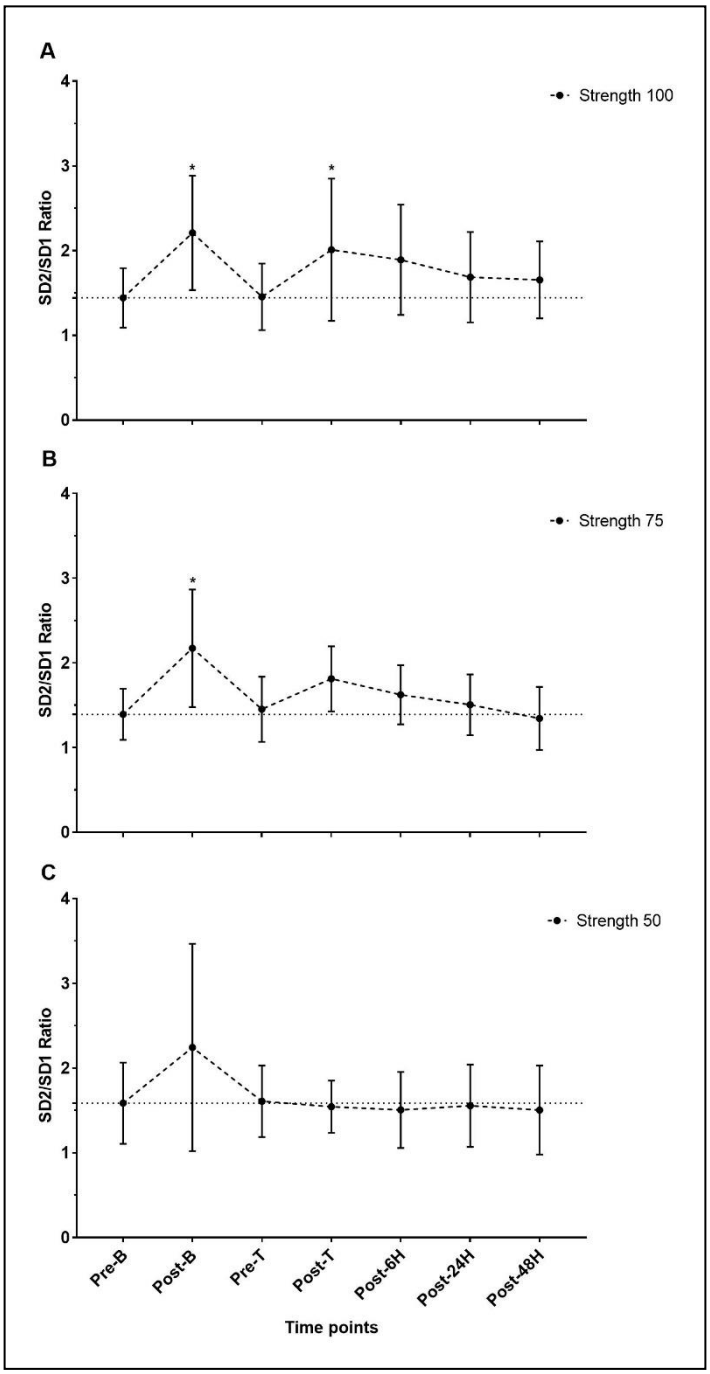


Figure 58. Changes in SD2/SD1 parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

There was no significant difference observed at Pre-T (S100: p = 1.000, ES = 0.03; S75: p = 1.000, ES = 0.17; S50: p = 1.000, ES = 0.05), Post-6H (S100: p = 0.188, ES = 0.86; S75: p = 1.000, ES = 0.71; S50: p = 1.000, ES = -0.17), Post-24H (S100: = 1.000, ES = 0.54; S75: p = 1.000, ES = 0.34; S50: p = 1.000, ES = -0.06) and Post-48H (S100: = 1.000, ES = 0.52; S75: p = 1.000, ES = -0.14; S50: p = 1.000, ES = -0.16) in all training loads compared to Pre-B. According to ES results, cardiac sympathovagal balance shifted to cardiac sympathetic modulation following the M-Beast protocol in all three trials and S100 and S75 and it gradually returned to Pre-B values. According to the ES results, cardiac sympathovagal balance of S50 recovered at Pre-T, whereas S75 needed longer time (Post-48H) to recover. Interestingly, S100's level did not recover at Post-48H (Figure 58).

6.2.2.2.11. Stress Score Index (SS)

There was no overall treatment effect on SS (p = 0.102). However, there was an overall time effect on SS (p < 0.001) and a significant group x time interaction for SS was observed (p = 0.027). Simple main effects for treatment showed that SS was significantly different between treatments (S100 vs S75 vs S50) at Post-T (p < 0.001, (S100 vs S75: p = 0.002; S100 vs S50: p = 0.005)) and Post-24H (p = 0.028, (S100 vs S75: p = 0.248; S100 vs S50: p = 0.084)) (Figure 59).

Simple main effects over time revealed that SS differed significantly between time points in S100 (P < 0.001), S75 (P < 0.001) and S50 (P = 0.004) trial. In S100, significant time differences were shown at Post-B (p = 0.003, ES = 1.15), Pre-T (p = 0.003, ES = 0.80), Post-T (p = 0.002, ES = 0.99) and Post-6H (p < 0.001, ES = 1.27) except at Post-24H (p = 0.086, ES = 0.88) and Post-48H (p = 1.000, ES = 0.26) compared to Pre-B. In S75, no significant time differences were shown at Pre-T (p = 1.000, ES = 0.29), Post-T (p = 0.326, ES = 0.52), Post-6H (p = 0.485, ES = 0.56), Post-24H (p = 1.000, ES = 0.10) and Post-48H (p = 1.000, ES = 0.01) except at Post-B (p = 0.029, ES = 1.15) compared to Pre-B. In S50, no significant time differences were shown at Post-B (p = 0.092, ES = 0.88), Pre-T (p = 0.989, ES = 0.30), Post-6H (p =

0.853, ES = 0.51), Post-24H (p = 1.000, ES = 0.02) and Post-48H (p = 1.000, ES = -0.11) except at Post-T (p = 0.011, ES = 0.71) compared to Pre-B. These results revealed that SS increased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, stress level recovered to baseline (Pre-B) at Post-6H for S50, whereas S100 needed longer time (Post-24H) to recover. However, S75 ARE protocol did not significantly affect the SS of the participants (Figure 60). According to the ES results, the SS of S50 and S75 recovered at Post-48H, whereas S100's level did not recover at Post-48H. Interestingly, the SS of S50 showed better recovery level compared to S75 at Post-48H.

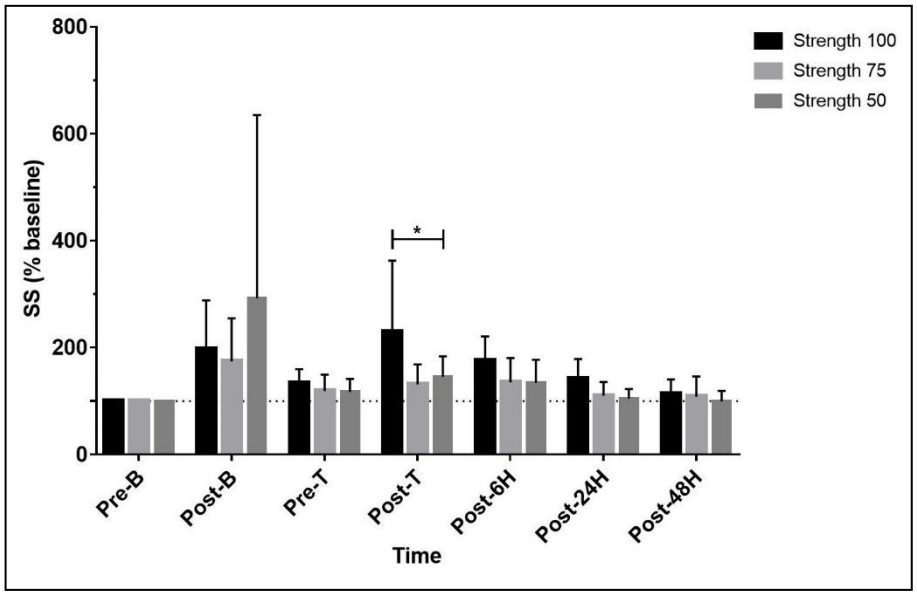


Figure 59. Comparison of S100, S75 and S50 on SS values (n = 13). *—| Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$).

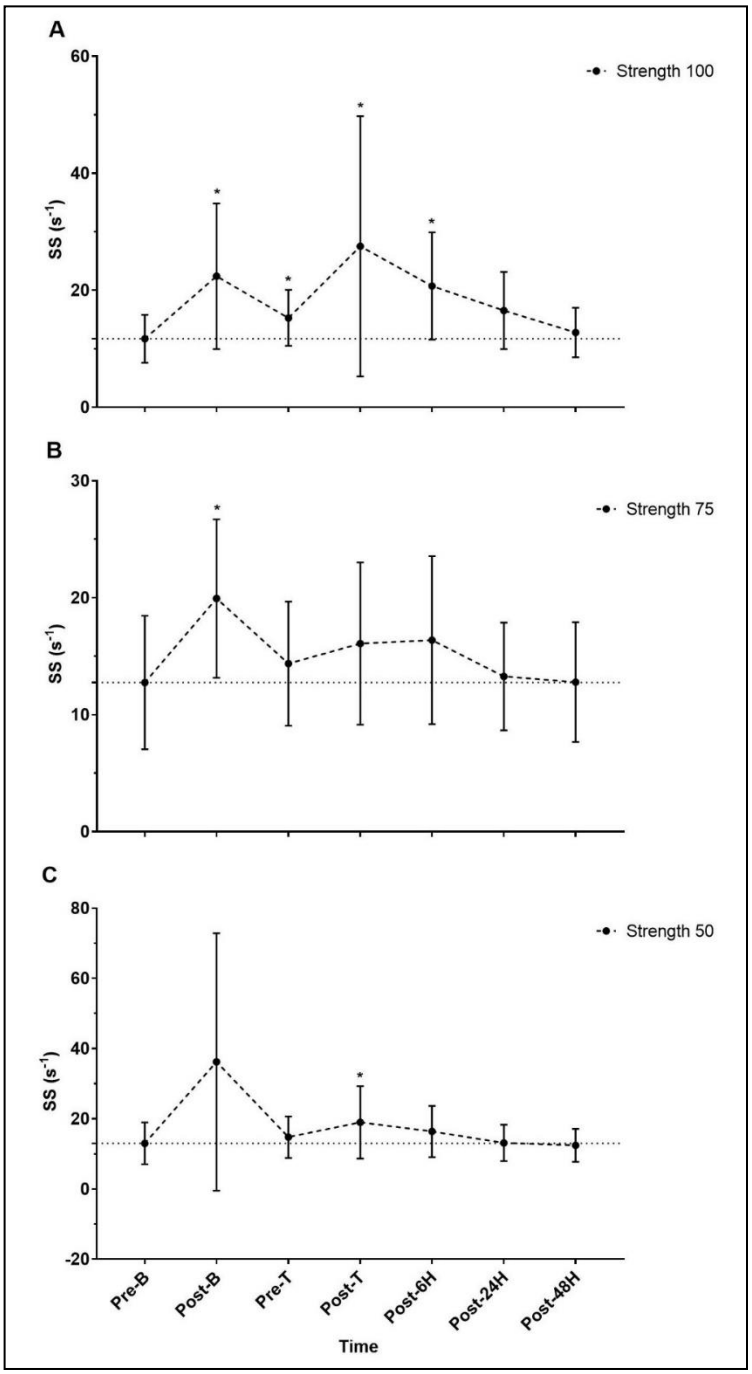


Figure 60. Changes in SS parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.3. Power 100 versus Power 75 versus Power 50 training

The results from the HRV parameters for the comparison between 100% of training load of power training modality (P100), 75% of training load of power training modality (P75) and 50% of training load of power training modality (P50) are reported below.

6.2.2.3.1. pNN50

There was no overall treatment effect on pNN50 ($p = 0.333$). However, there was an overall time effect on pNN50 ($p < 0.001$). No significant group x time interaction for pNN50 was observed ($p = 0.453$; Figure 61).

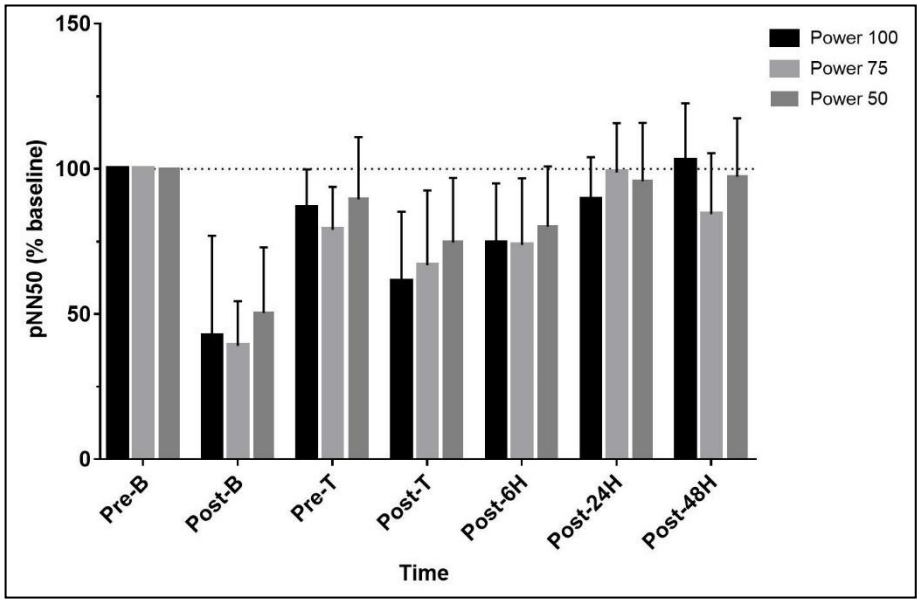


Figure 61. Comparison of P100, P75 and P50 on pNN50 values (n = 08).

Simple main effects over time revealed that pNN50 differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial.

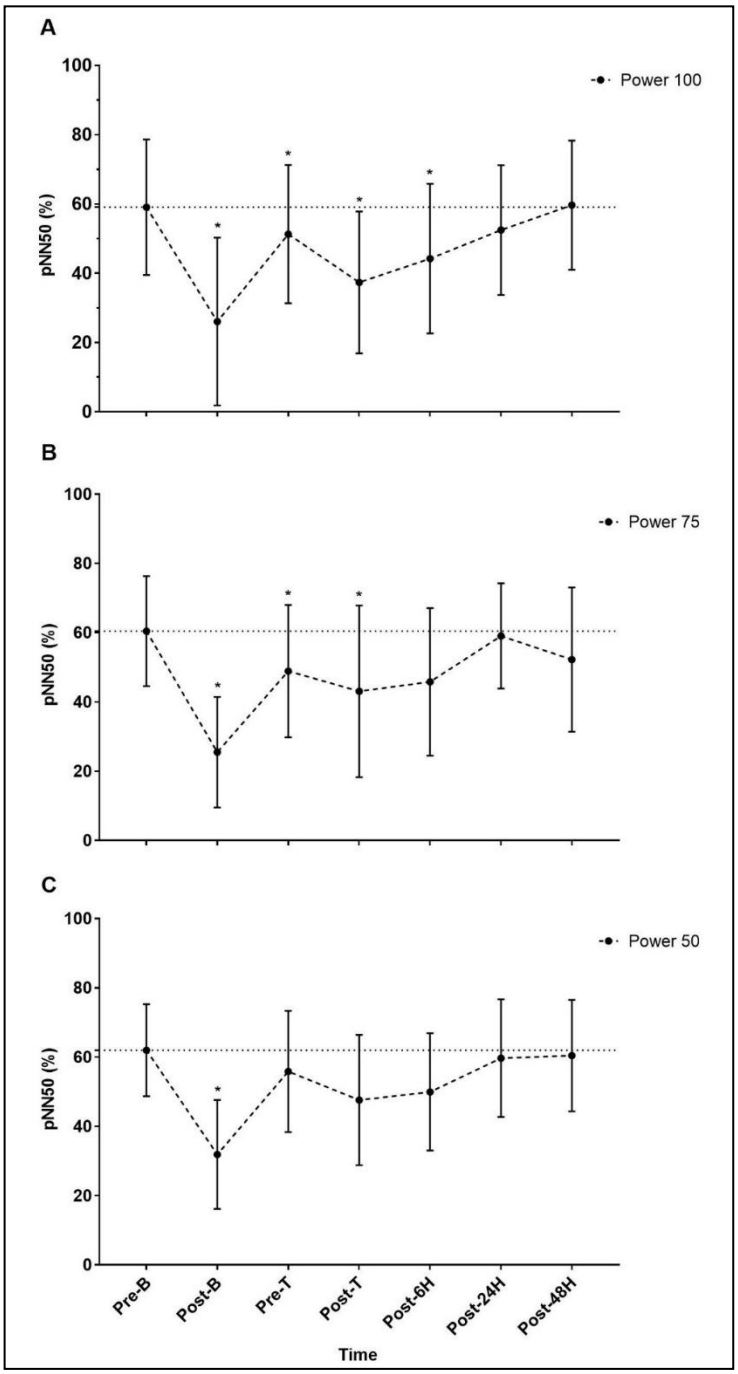


Figure 62. Changes in pNN50 parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 08).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In P100, significant time differences were observed at Post-B ($p = 0.005$, ES = -1.50), Pre-T ($p = 0.026$, ES = -0.39), Post-T ($p = 0.001$, ES = -1.08), Post-6H ($p = 0.052$, ES = -0.72) except at Post-24H ($p = 0.604$, ES = -0.34) and Post-48H ($p = 1.000$, ES = 0.03) compared to Pre-B. In P75, significant time differences were observed at Post-B ($p < 0.001$, ES = -2.20), Pre-T ($p = 0.024$, ES = -0.66), Post-T ($p = 0.032$, ES = -0.83) except at Post-6H ($p = 0.484$, ES = -0.78), Post-24H ($p = 1.000$, ES = -0.09) and Post-48H ($p = 1.000$, ES = -0.44) compared to Pre-B. In P50, no significant time differences were observed at Pre-T ($p = 1.000$, ES = -0.39), Post-T ($p = 0.066$, ES = -0.89), Post-6H ($p = 0.291$, ES = -0.79), Post-24H ($p = 1.000$, ES = -0.15) and Post-48H ($p = 1.000$, ES = -0.11) except at Post-B ($p = 0.002$, ES = -2.07) compared to Pre-B. These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-6H for P75, whereas P100 needed longer time (Post-24H) to recover. However, P50 ARE protocol did not significantly affect the cardiac parasympathetic modulation of the participants (Figure 62). Moreover, ES results showed that cardiac parasympathetic modulation recovered to Pre-B at Post-48H for P100. Surprisingly, P75 and P50 did not recover at Post-48H.

6.2.2.3.2. SDNN

There was no overall treatment effect on SDNN ($p = 0.558$). However, there was an overall time effect on SDNN ($p < 0.001$). No significant group x time interaction for SDNN was observed ($p = 0.193$; Figure 63).

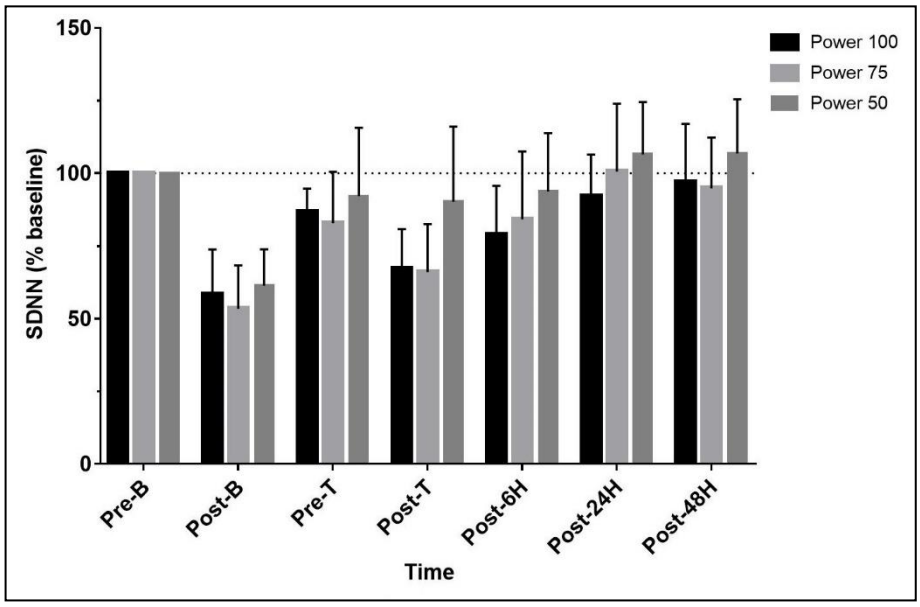


Figure 63. Comparison of P100, P75 and P50 on SDNN values (n = 11).

Simple main effects over time revealed that SDNN differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100, significant time differences were shown at Post-B ($p = 0.002$, ES = -1.16), Pre-T ($p = 0.009$, ES = -0.36), Post-T ($p = 0.001$, ES = -0.83) except at Post-6H ($p = 0.169$, ES = -0.59), Post-24H ($p = 1.000$, ES = -0.23) and Post-48H ($p = 1.000$, ES = -0.22) compared to Pre-B. In P75, significant time differences were shown at Post-B ($p = 0.001$, ES = -1.33) and Post-T ($p = 0.004$, ES = -0.82) except at Pre-T ($p = 0.245$, ES = -0.45), Post-6H ($p = 0.682$, ES = -0.37), Post-24H ($p = 1.000$, ES = 0.01) and Post-48H ($p = 1.000$, ES = -0.08) compared to Pre-B. In P50, no significant time differences were shown at Pre-T ($p = 1.000$, ES = -0.32), Post-T ($p = 1.000$, ES = -0.18), Post-6H ($p = 1.000$, ES = -0.34), Post-24H ($p = 1.000$, ES = 0.09) and Post-48H ($p = 1.000$, ES = 0.09) except at Post-B ($p < 0.001$, ES = -1.04) compared to Pre-B. These results showed that overall autonomic modulation decreased following the M-Beast protocol in all 3 training loads and ARE protocols of P100, and P75 gradually returned to Pre-B values.

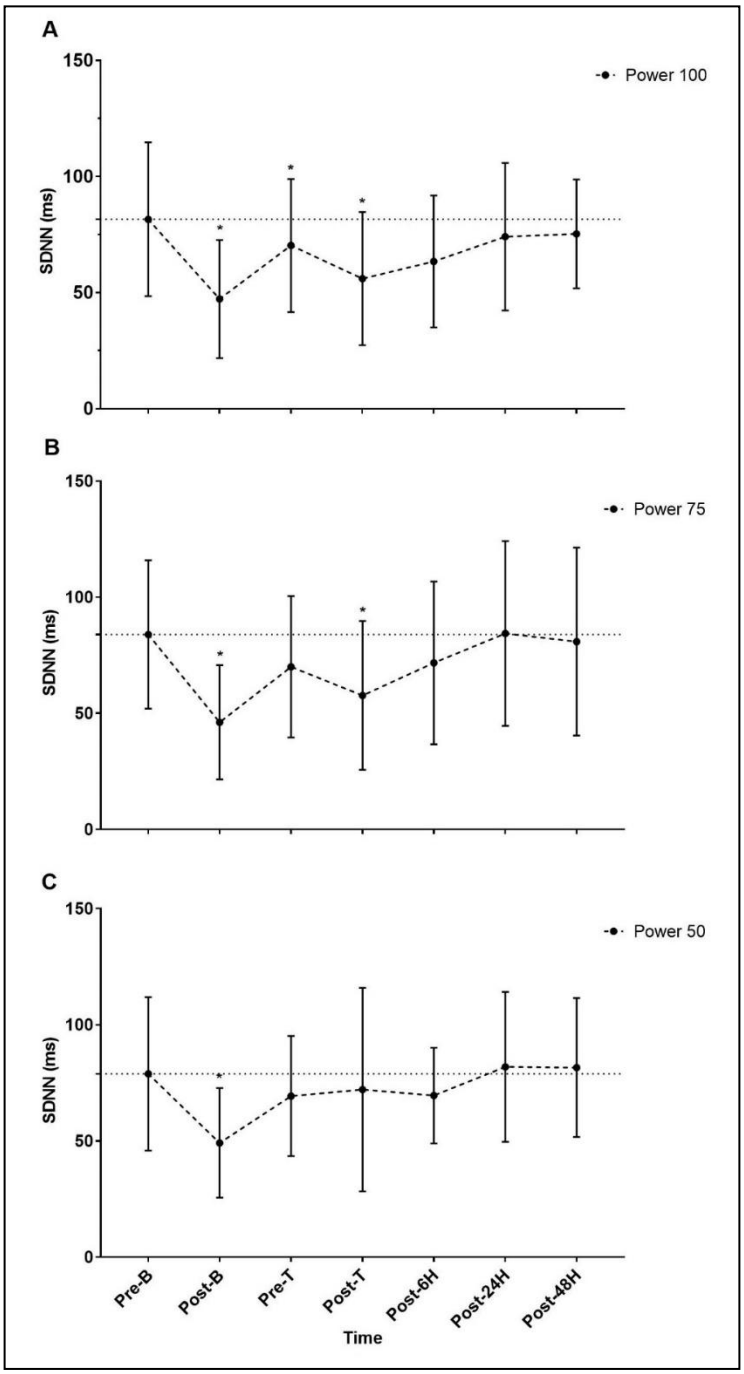


Figure 64. Changes in SDNN parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Interestingly, ARE protocol of P50 did not decrease the overall autonomic modulation. Moreover, overall autonomic modulation recovered to baseline (Pre-B) at Post-24H for P100 and P75, whereas P50 did not change from Pre-T. According to the ES results, overall autonomic modulation of P50 and P75 recovered at Post-24H (P50 showed better recovery level than P75), whereas P100's level did not recover at Post-48H (Figure 64).

6.2.2.3.3. Ln RMSSD

There was no overall treatment effect on Ln RMSSD ($p = 0.645$). However, there was an overall time effect on Ln RMSSD ($p < 0.001$). No significant group x time interaction for Ln RMSSD was observed ($p = 0.377$; Figure 65).

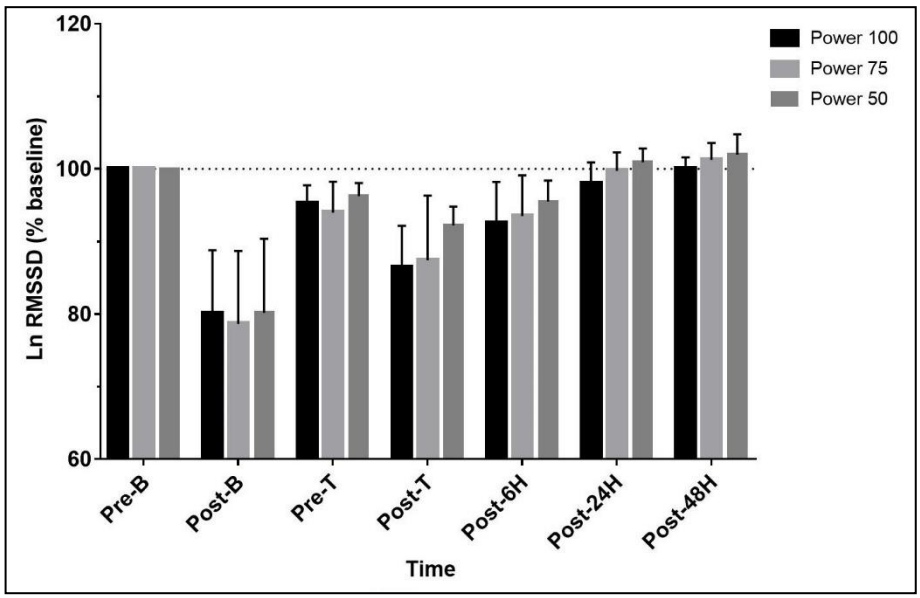


Figure 65. Comparison of P100, P75 and P50 on Ln RMSSD values (n = 11).

Simple main effects over time revealed that Ln RMSSD differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100, significant time difference were observed at Post-B ($p < 0.001$, ES = -1.60), Pre-T ($p = 0.001$, ES = -0.42), Post-T ($p < 0.001$, ES = -1.06), Post-6H ($p = 0.040$, ES = -0.65) except at Post-24H ($p = 0.968$, ES = -0.20) and Post-48H ($p = 1.000$, ES = -0.01)

compared to Pre-B. In P75, significant time difference were observed at Post-B ($p < 0.001$, ES = -1.48), Pre-T ($p = 0.012$, ES = -0.48), Post-T ($p = 0.003$, ES = -0.85), Post-6H ($p = 0.049$, ES = -0.50) except at Post-24H ($p = 1.000$, ES = -0.03) and Post-48H ($p = 1.000$, ES = 0.11) compared to Pre-B. In P50, significant time differences were observed at Post-B ($p < 0.001$, ES = -1.21), Pre-T ($p = 0.001$, ES = -0.30), Post-T ($p < 0.001$, ES = -0.60), Post-6H ($p = 0.011$, ES = -0.38) except at Post-24H ($p = 1.000$, ES = 0.07) and Post-48H ($p = 0.591$, ES = 0.15) compared to Pre-B. These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-24H for all three training loads (Figure 66). According to the ES results, cardiac parasympathetic modulation of P50 recovered at Post-24H, whereas P75's and P100's level recovered at Post-48H. P75 showed better recovery level than P100 at Post-48H.

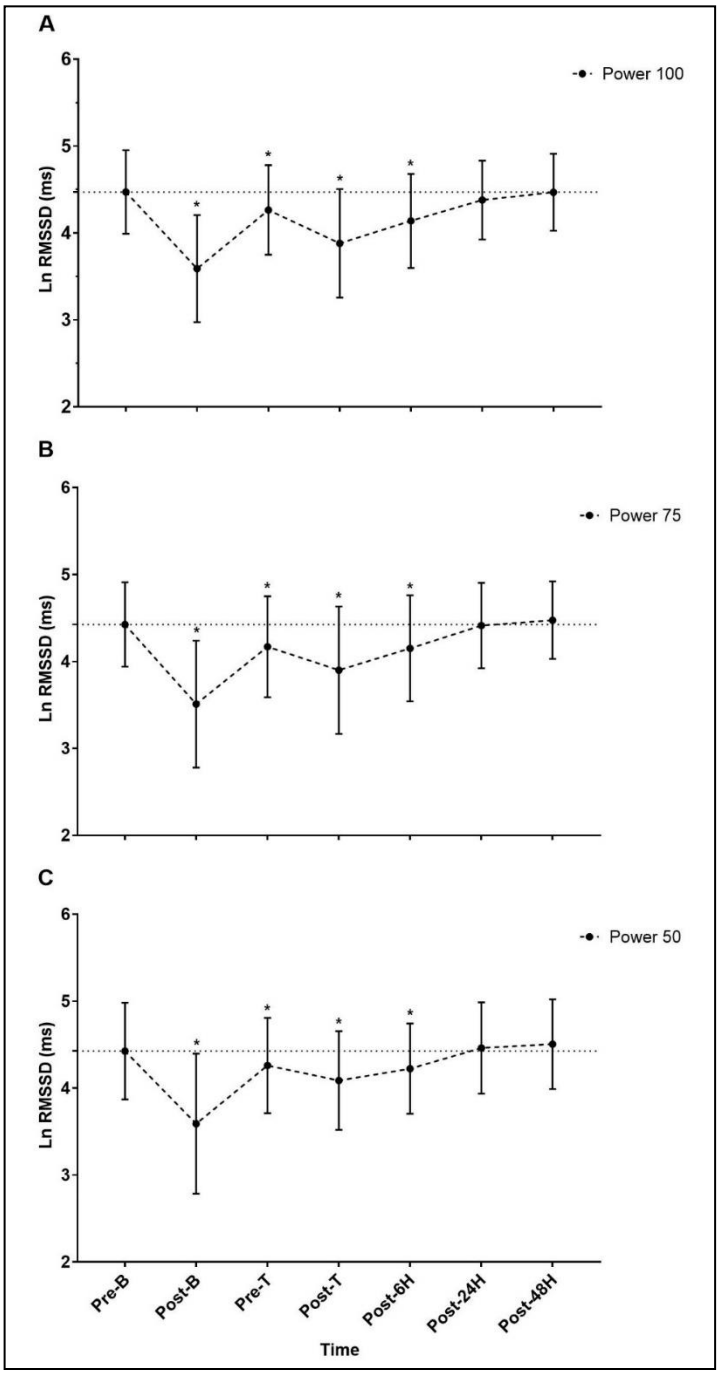


Figure 66. Changes in Ln RMSSD parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.3.4. HF(nu)

There was an overall treatment effect (p = 0.022) and an overall time effect (p = 0.002) on HF(nu). No significant group x time interaction for HF(nu) was observed (p = 0.337). Simple main effects for treatment showed that HF(nu) was significantly different between treatments (P100 vs P75 vs P50) at Pre-B (p = 0.050, (P100 vs P75: p = 0.089; P100 vs P50: p = 0.385) and Pre-T (p = 0.025, (P100 vs P75: p = 0.068 ; P100 vs P50: p = 1.000) (Figure 67).

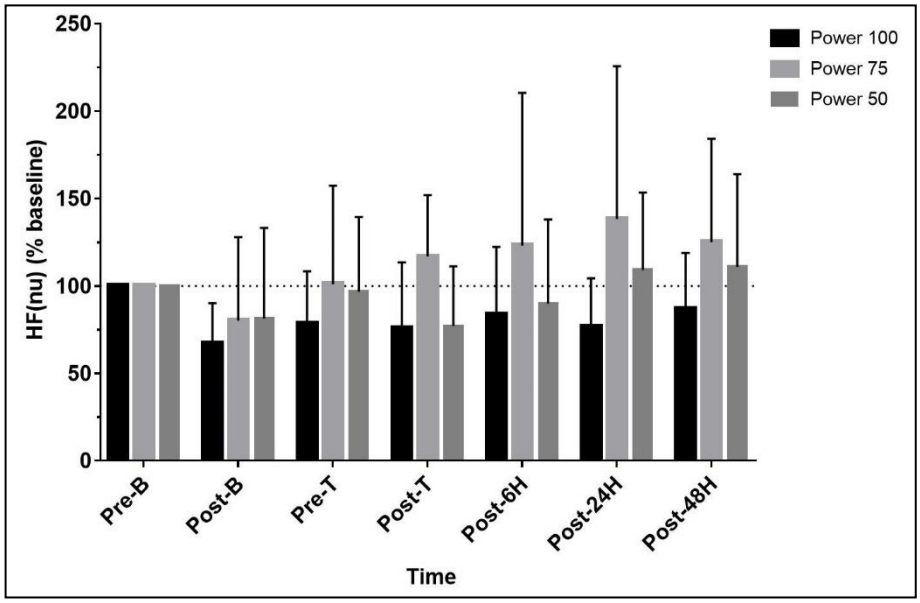


Figure 67. Comparison of P100, P75 and P50 on HF(nu) values (n = 11).

Simple main effects over time revealed that HF(nu) did not significantly differ between time points in P75 (P = 0.122) and P50 (P = 0.079) except in P100 (P = 0.049) trial.

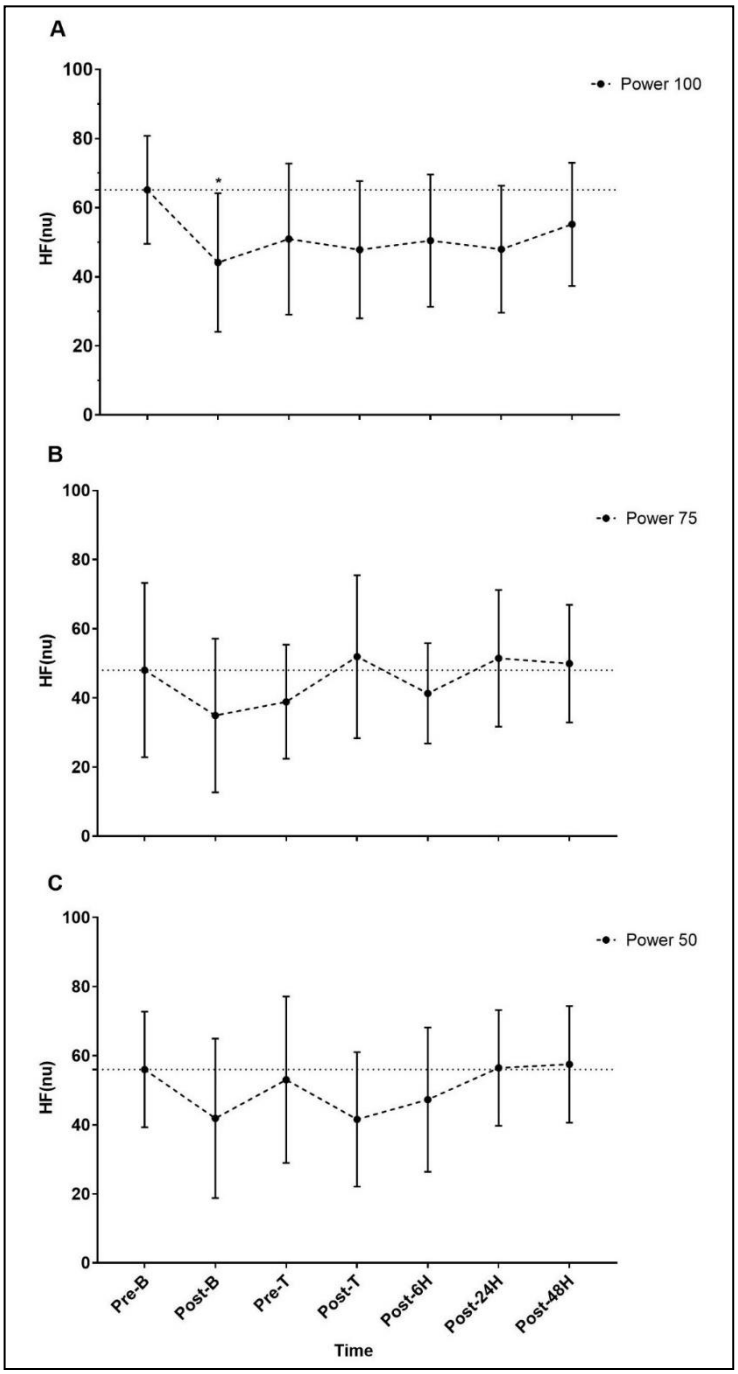


Figure 68. Changes in HF(nu) parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In P100, no significant time differences were observed at Pre-T (p = 0.649, ES = -0.75), Post-T (p = 0.407, ES = -0.97), Post-6H (p = 1.000, ES = -0.84), Post-24H (p = 0.628, ES = -1.01) and Post-48H (p = 1.000, ES = -0.59) except at Post-B (p = 0.031, ES = -1.17) compared to Pre-B. In P75, no significant time differences were observed at all the time points (Post-B (p = 1.000, ES = -0.55), Pre-T (p = 1.000, ES = -0.43), Post-T (p = 1.000, ES = 0.16), Post-6H (p = 1.000, ES = -0.33), Post-24H (p = 1.000, ES = 0.15) and Post-48H (p = 1.000, ES = 0.09)) compared to Pre-B. Similarly, P50 also showed no significant time difference at all the time points (Post-B (p = 1.000, ES = -0.70), Pre-T (p = 1.000, ES = -0.14), Post-T (p = 0.722, ES = -0.79), Post-6H (p = 1.000, ES = -0.46), Post-24H (p = 1.000, ES = 0.03) and Post-48H (p = 1.000, ES = 0.09)) compared to Pre-B value. These results suggest that cardiac parasympathetic modulation decreased following the M-Beast protocol in all 3 training loads and ARE protocols of P100 and P50 gradually returned to Pre-B values. Interestingly, ARE protocol of P75 did not decrease the cardiac parasympathetic modulation. According to the ES results, cardiac parasympathetic modulation of P50 and P75 recovered at Post-24H, whereas P100's level did not recover at Post-48H (Figure 68).

6.2.2.3.5. LF(nu)

There was an overall treatment effect (p = 0.022) and an overall time effect (p = 0.002) on LF(nu). No significant group x time interaction for LF(nu) was observed (p = 0.334). Simple main effects for treatment showed that LF(nu) was significantly different between treatments (P100 vs P75 vs P50) at Pre-B (p = 0.050, (P100 vs P75: p = 0.090; P100 vs P50: p = 0.385) and Pre-T (p = 0.024, (P100 vs P75: p = 0.068 ; P100 vs P50: p = 1.000) (Figure 69).

Simple main effects over time revealed that LF(nu) did not significantly differ between time points in P75 (P = 0.130) and P50 (P = 0.079) except P100 (P = 0.049) trial. In P100, no significant time differences were observed at Pre-T (p = 0.643, ES = 0.75), Post-T (p = 0.413, ES = 0.97), Post-6H (p = 1.000, ES = 0.84), Post-24H (p =

0.623, ES = 1.01) and Post-48H (p = 1.000, ES = 0.59) except at Post-B (p = 0.031, ES = 1.17) compared to Pre-B. In P75, no significant time differences were observed at all the time points (Post-B (p = 1.000, ES = 0.55), Pre-T (p = 1.000, ES = 0.43), Post-T (p = 1.000, ES = -0.16), Post-6H (p = 1.000, ES = 0.31), Post-24H (p = 1.000, ES = -0.15) and Post-48H (p = 1.000, ES = -0.09)) compared to Pre-B. Similarly, P50 trail also showed no significant time differences at all the time points (Post-B (p = 1.000, ES = 0.70), Pre-T (p = 1.000, ES = 0.14), Post-T (p = 0.743, ES = 0.79), Post-6H (p = 1.000, ES = 0.46), Post-24H (p = 1.000, ES = -0.03) and Post-48H (p = 1.000, ES = -0.09)) compared to Pre-B. These results indicate that cardiac sympathetic modulation increased following the M-Beast protocol in all 3 training loads and ARE protocols of P100 and P50 gradually returned to Pre-B values. Interestingly, ARE protocol of P75 did not increase the cardiac sympathetic modulation. According to the ES results, cardiac parasympathetic modulation of P50 and P75 recovered at Post-24H, whereas P100's level did not recover at Post-48H (Figure 70).

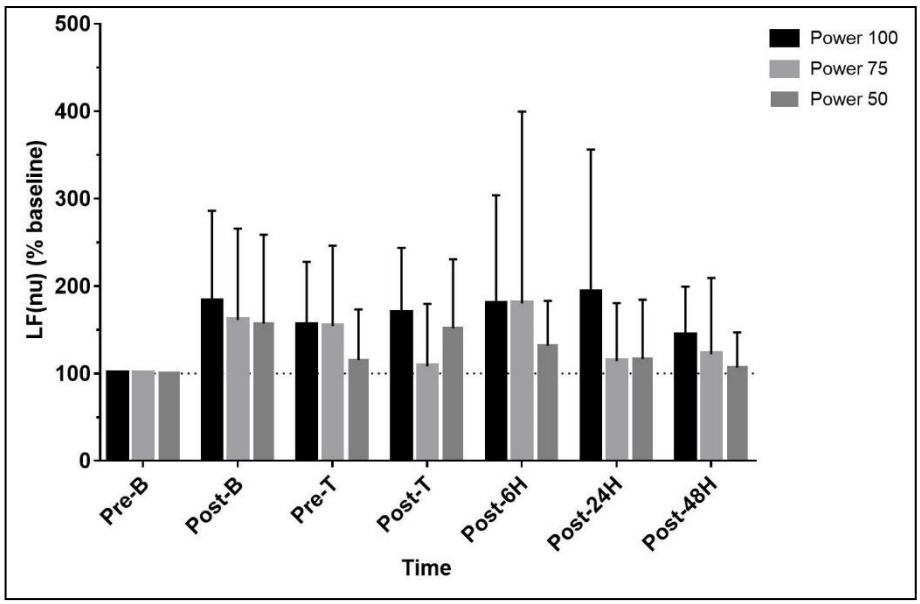


Figure 69. Comparison of P100, P75 and P50 on LF(nu) values (n = 11).

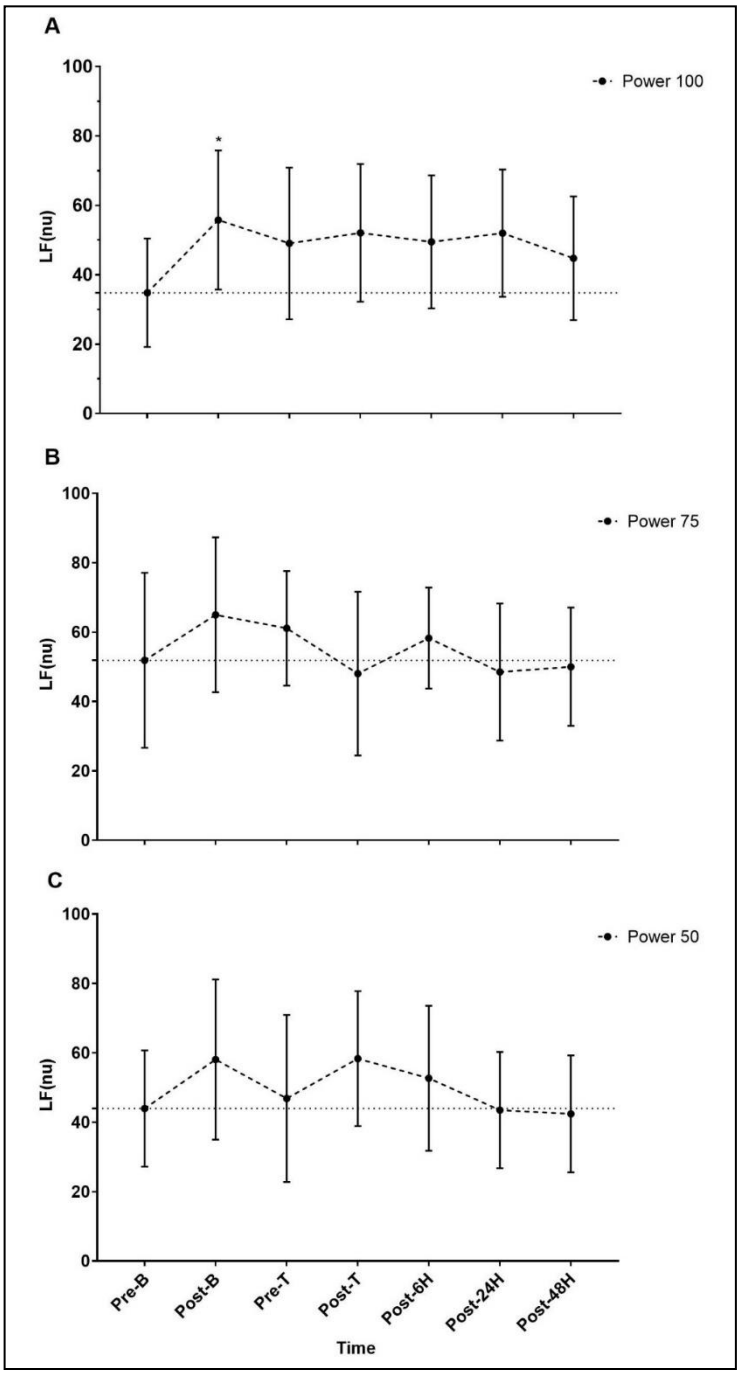


Figure 70. Changes in LF(nu) parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.3.6. LF/HF ratio

There was an overall treatment effect on LF/HF ratio (p = 0.021) and an overall time effect (p = 0.001) on LF/HF ratio. No significant group x time interaction for LF/HF ratio was observed (p = 0.418). Simple main effects for treatment showed that LF/HF ratio was significantly different between treatments (P100 vs P75 vs P50) at Pre-T (p = 0.030, (P100 vs P75: p = 0.067; P100 vs P50: p = 1.000)) (Figure 71).

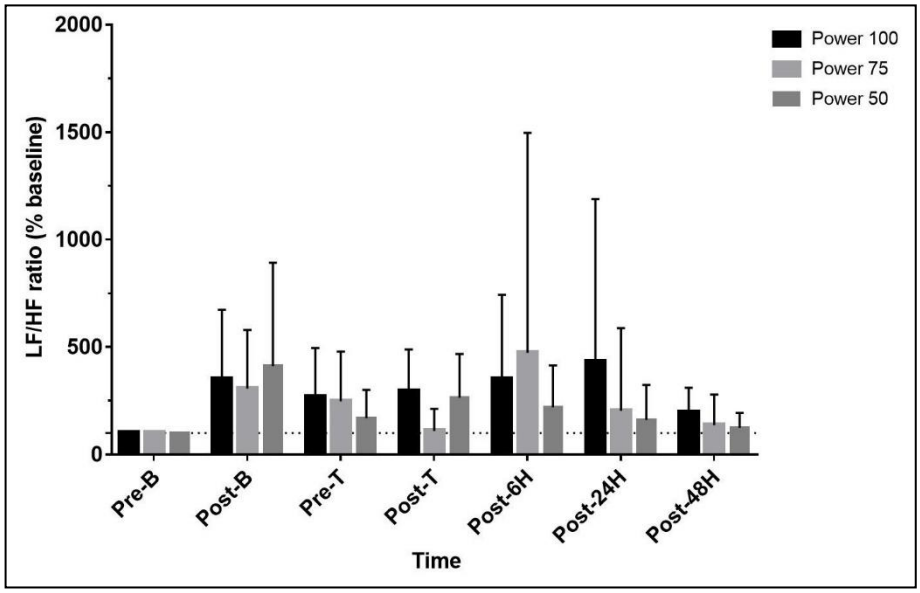


Figure 71. Comparison of P100, P75 and P50 on LF/HF ratio values (n = 11).

Simple main effects over time revealed that LF/HF ratio tended to be different between time points in P100 (P = 0.058) trial, but not in P75 (P = 0.117) and P50 (P = 0.070) trials. In P100, no significant time differences were observed at Pre-T (p = 0.560, ES = 0.82), Post-T (p = 0.300, ES = 0.89), Post-6H (p = 1.000, ES = 0.88), Post-24H (p = 1.000, ES = 0.99) and Post-48H (p = 1.000, ES = 0.62) except at Post-B (p = 0.038, ES = 1.08) compared to Pre-B. In P75, no significant time differences were observed at all the time points (Post-B (p = 1.000, ES = 0.57), Pre-T (p = 1.000, ES = 0.08), Post-T (p = 1.000, ES = -0.27), Post-6H (p = 1.000, ES = -0.13), Post-24H (p = 1.000, ES = -0.22) and Post-48H (p = 1.000, ES = -0.39)) compared to Pre-B.

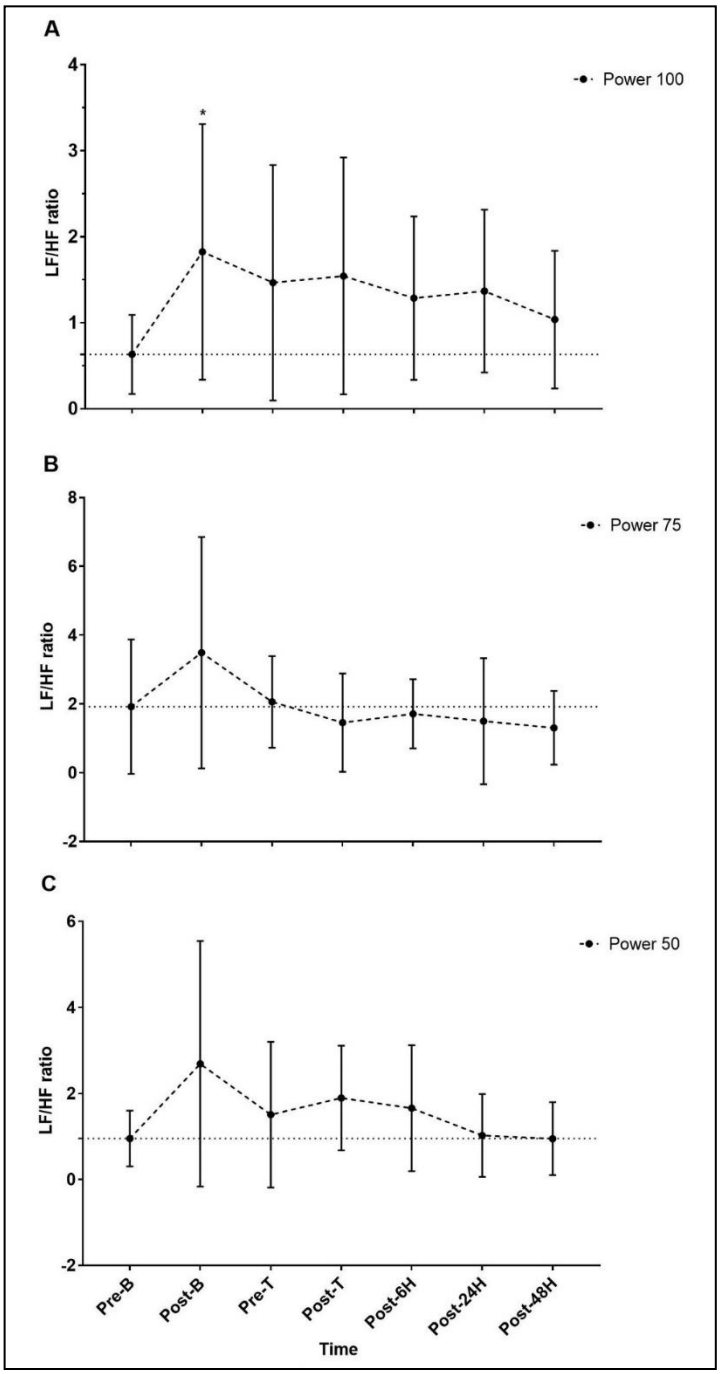


Figure 72. Changes in LF/HF ratio parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Similarly, P50 trial also showed no significant time differences at all the time points (Post-B (p = 1.000, ES = 0.84), Pre-T (p = 1.000, ES = 0.43), Post-T (p = 0.743, ES = 0.97), Post-6H (p = 1.000, ES = 0.62), Post-24H (p = 1.000, ES = 0.09) and Post-48H (p = 1.000, ES = -0.01)) compared to Pre-B. These results revealed that cardiac sympathovagal balance shifted to cardiac sympathetic modulation following the M-Beast protocol in all three trials and P100 and P50 and it gradually returned to Pre-B values. Interestingly, ARE protocol of P75 did not shift to cardiac sympathetic modulation. According to the ES results, cardiac sympathovagal balance of P75 remained unchanged from Post-T, whereas P50 needed longer time (Post-48H) to recover. Although, P100 did not recover at Post-48H (Figure 72).

6.2.2.3.7. Total power

There was no overall treatment effect on TP (p = 0.673). However, there was an overall time effect on TP (p < 0.001). No significant group x time interaction for TP was observed (p = 0.805; Figure 73).

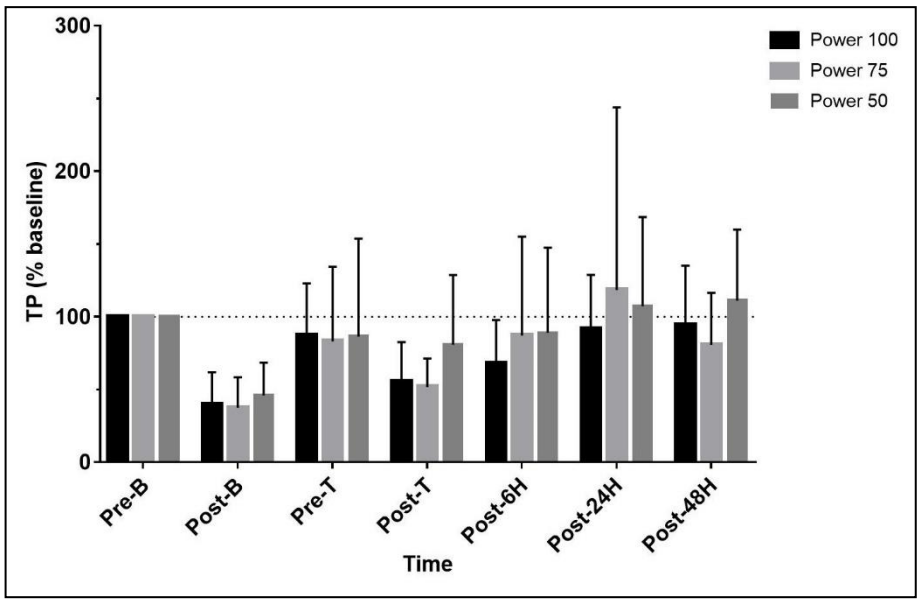


Figure 73. Comparison of P100, P75 and P50 on TP values (n = 11).

Simple main effects over time revealed that TP differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100, significant time differences were observed at Post-B ($p = 0.013$, ES = -0.90) and Post-T ($p = 0.054$, ES = -0.65) except at Pre-T ($p = 1.000$, ES = -0.35), Post-6H ($p = 0.403$, ES = -0.61) Post-24H ($p = 1.000$, ES = -0.10) and Post-48H ($p = 1.000$, ES = -0.38) compared to Pre-B. Similarly, P75 trial also showed significant differences at Post-B ($p = 0.009$, ES = -0.88) and Post-T ($p = 0.006$, ES = -0.46) except at Pre-T ($p = 1.000$, ES = -0.37), Post-6H ($p = 1.000$, ES = -0.28) Post-24H ($p = 1.000$, ES = -0.26) and Post-48H ($p = 1.000$, ES = -0.38) compared to Pre-B. However, in P50, no significant time differences were observed at Pre-T ($p = 1.000$, ES = -0.51), Post-T ($p = 1.000$, ES = -0.38), Post-6H ($p = 1.000$, ES = -0.61),Post-24H ($p = 1.000$, ES = -0.06) and Post-48H ($p = 1.000$, ES = -0.09) except at Post-B ($p = 0.009$, ES = -0.96) compared to Pre-B value. These results revealed that total autonomic activity increased following the M-Beast protocol in all three trials and P100 and P75 and it gradually returned to Pre-B values. Interestingly, ARE protocol of P50 did not decrease the total autonomic activity. Moreover, total autonomic activity recovered to baseline (Pre-B) at Post-6H for P100 and P75, whereas P50 remained unchanged from Pre-T (Figure 74). According to the ES results, total autonomic activity did not recover at Post-48H in all 3 training loads. Even though not fully recovered, P50 showed better recovery level than P100 and P75 at Post-48H.

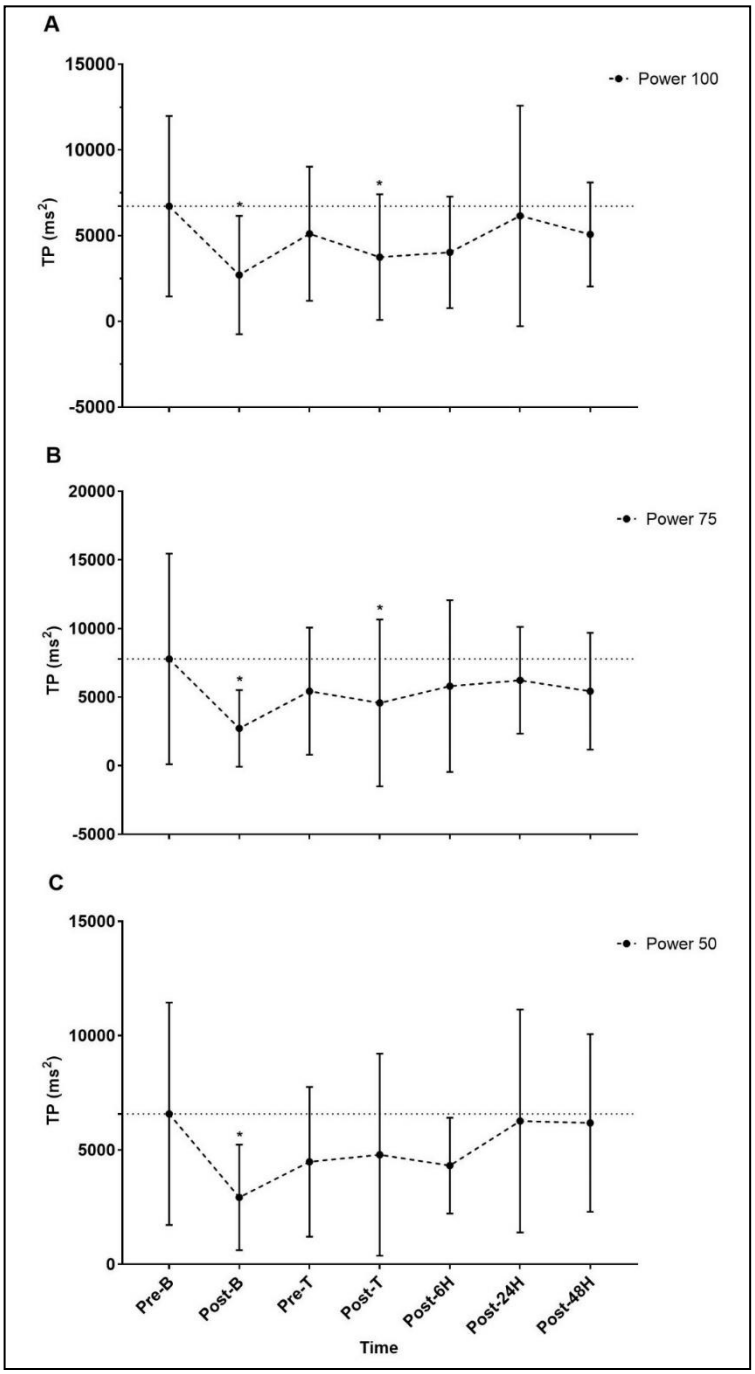


Figure 74. Changes in TP parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.3.8. SD1

There was no overall treatment effect on SD1 ($p = 0.684$). However, there was an overall time effect on SD1 ($p < 0.001$) and significant group x time interaction for SD1 was observed ($p = 0.049$). Simple main effects for treatment showed that SD1 there was no significant difference between treatments (P100 vs P75 vs P50) at the Pre-B ($p = 0.754$), Post-B ($p = 0.806$), Pre-T ($p = 0.572$), Post-T ($p = 0.199$), Post-6H ($p = 0.847$), Post-24H ($p = 0.365$) and Post-48H ($p = 0.491$) (Figure 75).

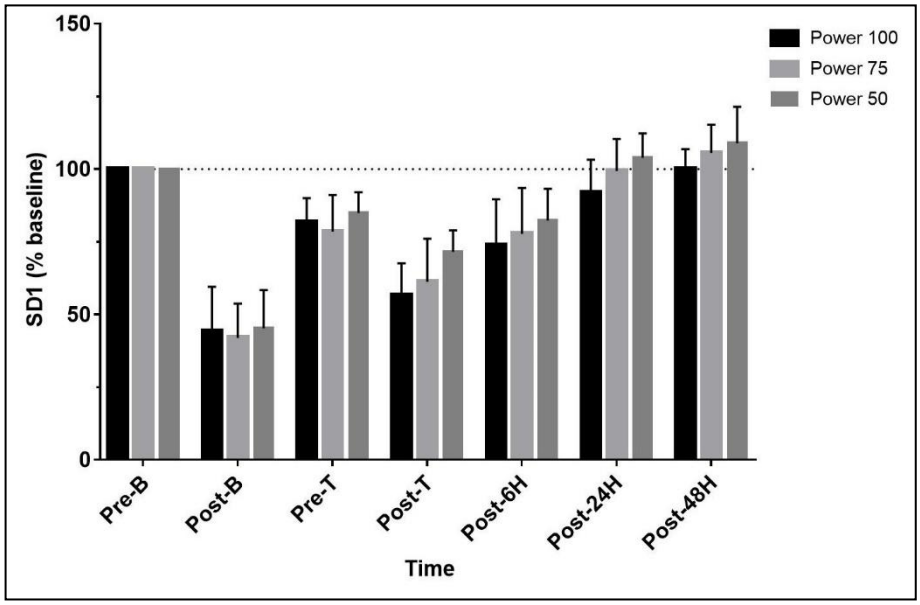


Figure 75. Comparison of P100, P75 and P50 on SD1 values (n = 11).

Simple main effects over time revealed that SD1 differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100, significant time differences were observed at Post-B ($p = 0.002$, ES = -1.35), Pre-T ($p = 0.007$, ES = -0.38), Post-T ($p < 0.001$, ES = -0.94), Post-6H ($p = 0.038$, ES = -0.57) except at Post-24H ($p = 0.767$, ES = -0.21) and Post-48H ($p = 1.000$, ES = -0.04) compared to Pre-B.

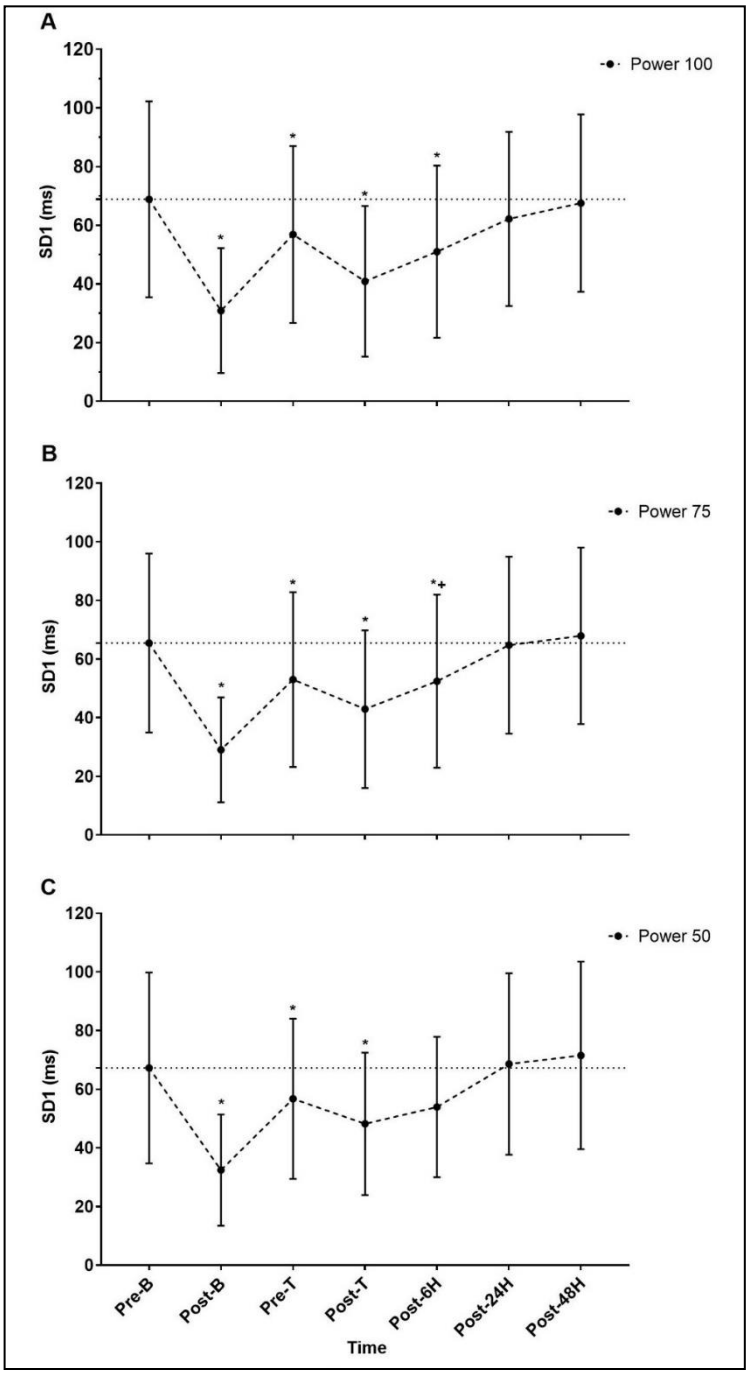


Figure 76. Changes in SD1 parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis. *+ Significant trend time difference compared to Pre-B ($p \leq 0.06$) from post-hoc Bonferroni analysis.

In P75, significant time differences were observed at Post-B ($p < 0.001$, ES = -1.46), Pre-T ($p = 0.019$, ES = -0.41), Post-T ($p < 0.001$, ES = -0.78) and tendency towards significance at Post-6H ($p = 0.061$, ES = -0.43), except at Post-24H ($p = 1.000$, ES = -0.02) and Post-48H ($p = 1.000$, ES = 0.08) compared to Pre-B. In P50, significant time differences were observed at Post-B ($p = 0.001$, ES = -1.31), Pre-T ($p = 0.029$, ES = -0.35), Post-T ($p = 0.002$, ES = -0.66) except at Post-6H ($p = 0.080$, ES = -0.47), Post-24H ($p = 1.000$, ES = 0.04) and Post-48H ($p = 0.134$, ES = 0.13) compared to Pre-B.

These results revealed that cardiac parasympathetic modulation decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, cardiac parasympathetic modulation recovered to baseline (Pre-B) at Post-6H for P50, whereas P100 and P75 needed longer time (Post-24H) to recover. According to the ES results, cardiac parasympathetic modulation of P50 recovered at Post-24H, whereas P75 needed longer time (Post-48H) to recover. Furthermore, cardiac parasympathetic modulation of P100 did not recover at Post-48H (Figure 76).

6.2.2.3.9. SD2

There was no overall treatment effect on SD2 ($p = 0.539$). However, there was an overall time effect on SD2 ($p < 0.001$). No significant group x time interaction for SD2 was observed ($p = 0.351$; Figure 77).

Simple main effects over time revealed that SD2 differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100, significant time differences were observed at Post-B ($p = 0.010$, ES = -1.03) and Post-T ($p = 0.034$, ES = -0.74), except at Pre-T ($p = 0.661$, ES = -0.35), Post-6H ($p = 0.566$, ES = -0.59), Post-24H ($p = 1.000$, ES = -0.25) and Post-48H ($p = 1.000$, ES = -0.39) compared to Pre-B.

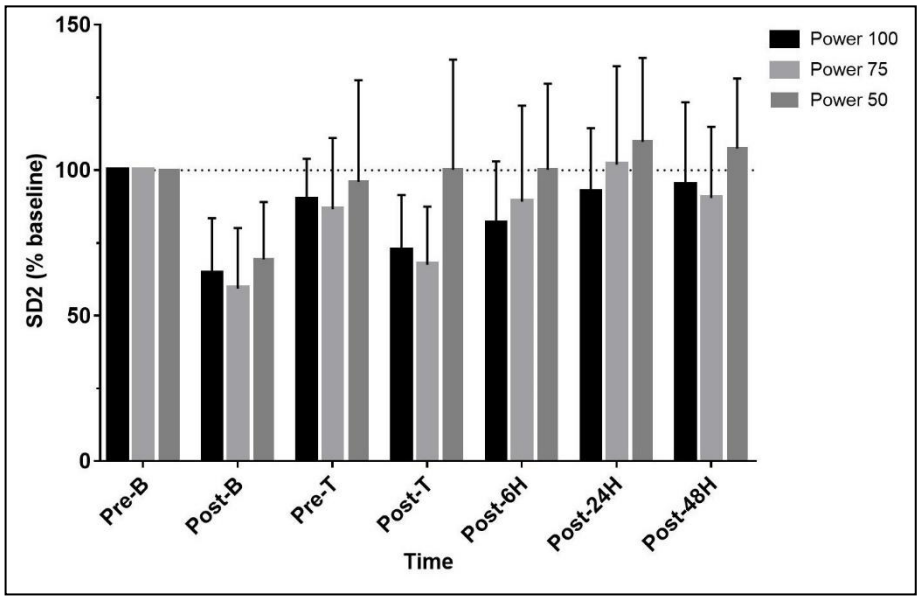


Figure 77. Comparison of P100, P75 and P50 on SD2 values (n = 11).

Similarly, P75 also showed significant time differences at Post-B (p = 0.011, ES = -1.14) and Post-T (p = 0.012, ES = -0.77), except at Pre-T (p = 1.000, ES = -0.41), Post-6H (p = 1.000, ES = -0.29), Post-24H (p = 1.000, ES = 0.01) and Post-48H (p = 1.000, ES = -0.16) compared to Pre-B. In P50, no significant time differences were observed at Pre-T (p = 1.000, ES = -0.30), Post-T (p = 1.000, ES = 0.01), Post-6H (p = 1.000, ES = -0.25), Post-24H (p = 1.000, ES = 0.13) and Post-48H (p = 1.000, ES = 0.06) except for Post-B (p = 0.026, ES = -0.83) compared to Pre-B. These results shown that SD2 decreased following the M-Beast protocol in all 3 training loads and ARE protocols of S100, and S75 gradually returned to Pre-B.

Interestingly, ARE protocol of P50 did not decrease the SD2. Moreover, SD2 recovered to baseline (Pre-B) at Post-6H for P100 and P75, whereas P50 remains recovered from Pre-T (Figure 78). According to the ES results, SD2 of P50 and P75 recovered (P50 showed better recovery level than P75) at Post-24H, whereas P100's level did not recover at Post-48H.

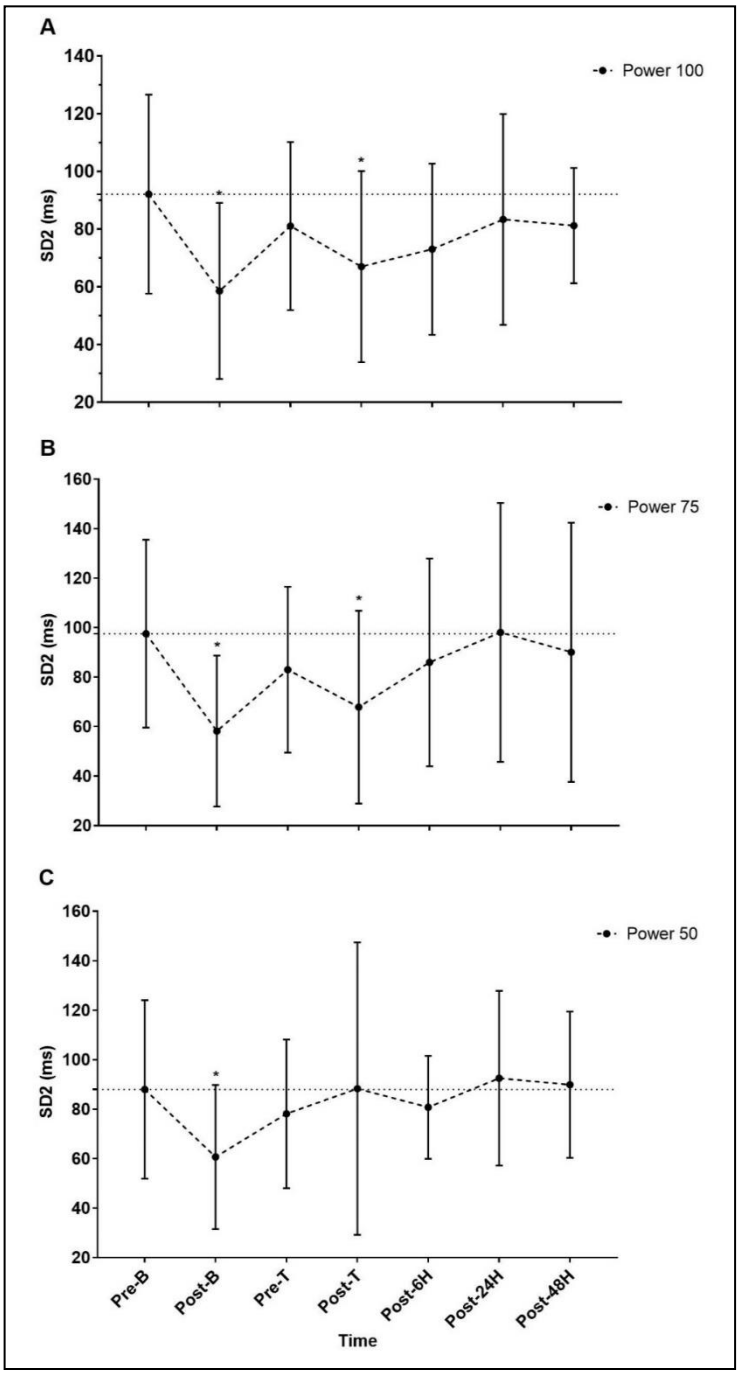


Figure 78. Changes in SD2 parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.2.3.10. SD2/SD1

There was no overall treatment effect on SD2/SD1 ($p = 0.220$). However, there was an overall time effect on SD2/SD1 ($p < 0.001$). No significant group x time interaction for SD2/SD1 was observed ($p = 0.994$; Figure 79).

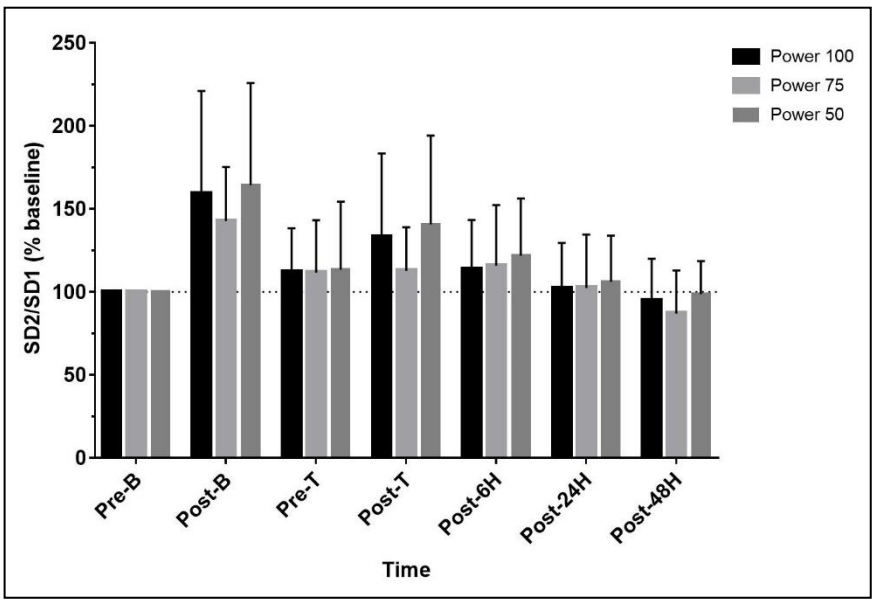


Figure 79. Comparison of P100, P75 and P50 on SD2/SD1 values (n = 11).

Simple main effects over time revealed that SD2/SD1 differed significantly between time points in P100 ($P < 0.001$), P75 ($P = 0.001$) and P50 ($P < 0.001$) trial. However, significant time differences were observed at P100 (Post-B ($p = 0.102$, ES = 1.50), Pre-T ($p = 1.000$, ES = 0.48), Post-T ($p = 0.700$, ES = 0.98), Post-6H ($p = 1.000$, ES = 0.48), Post-24H ($p = 1.000$, ES = 0.01), Post-48H ($p = 1.000$, ES = -0.24)) and P50 (Post-B ($p = 0.142$, ES = 1.22), Pre-T ($p = 1.000$, ES = 0.29), Post-T ($p = 0.731$, ES = 0.94), Post-6H ($p = 1.000$, ES = 0.60), Post-24H ($p = 1.000$, ES = 0.09), Post-48H ($p = 1.000$, ES = -0.21)) at all the time points compared to Pre-B. In P75, significant time differences were observed at Pre-T ($p = 1.000$, ES = 0.20), Post-T ($p = 1.000$, ES = 0.34), Post-6H ($p = 1.000$, ES = 0.31), Post-24H ($p = 1.000$, ES = -0.01), Post-48H ($p = 1.000$, ES = -0.51) except at Post-B ($p = 0.031$, ES = 1.01) compared to Pre-B (Figure 80).

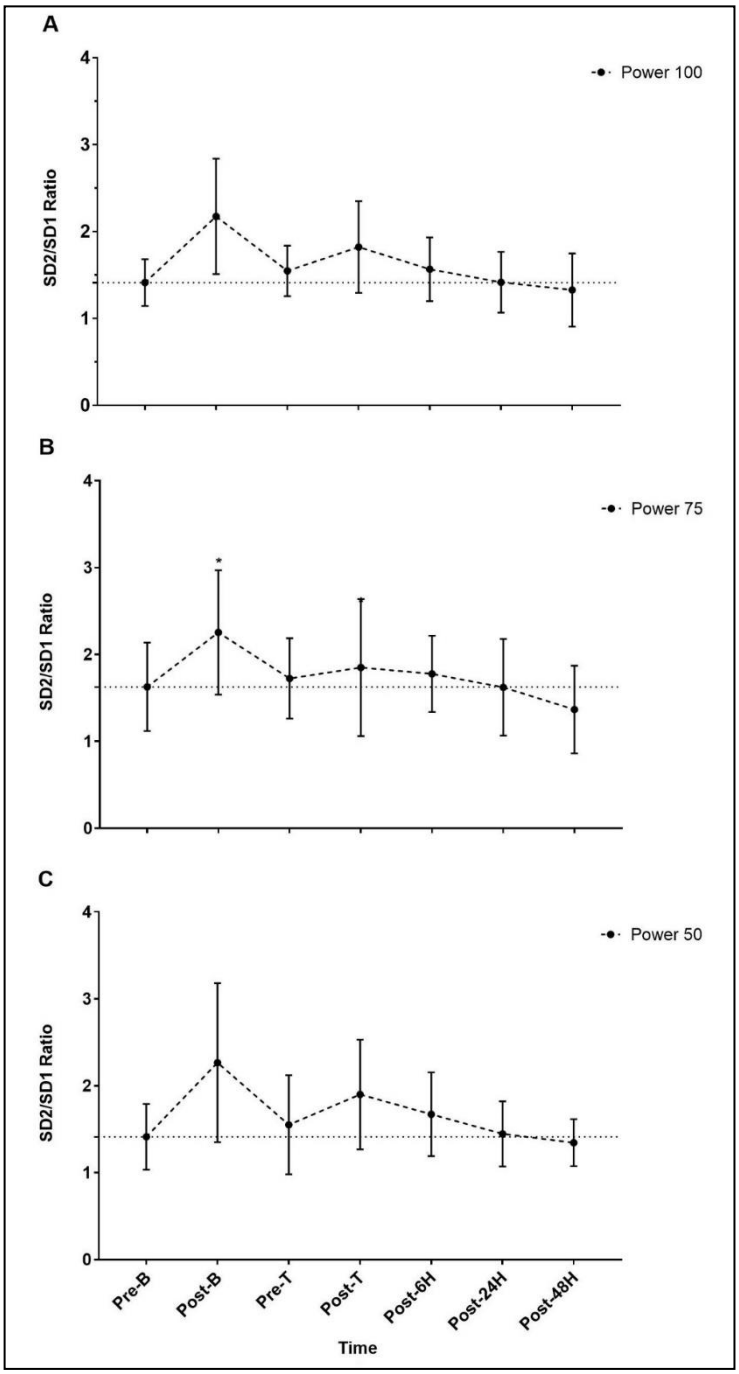


Figure 80. Changes in SD2/SD1 parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results indicate that cardiac sympathovagal balance shifted to cardiac sympathetic modulation following the M-Beast protocol ARE protocols for all training loads, and it gradually returned to Pre-B values. According to the ES results, cardiac sympathovagal balance of P75 recovered at Post-24H, whereas P100's and P50's level recovered at Post-48H.

6.2.2.3.11. Stress Score index (SS)

There was no overall treatment effect on SS ($p = 0.539$). However, there was an overall time effect on SS ($p < 0.001$). No significant group x time interaction for SS was observed ($p = 0.351$; Figure 81).

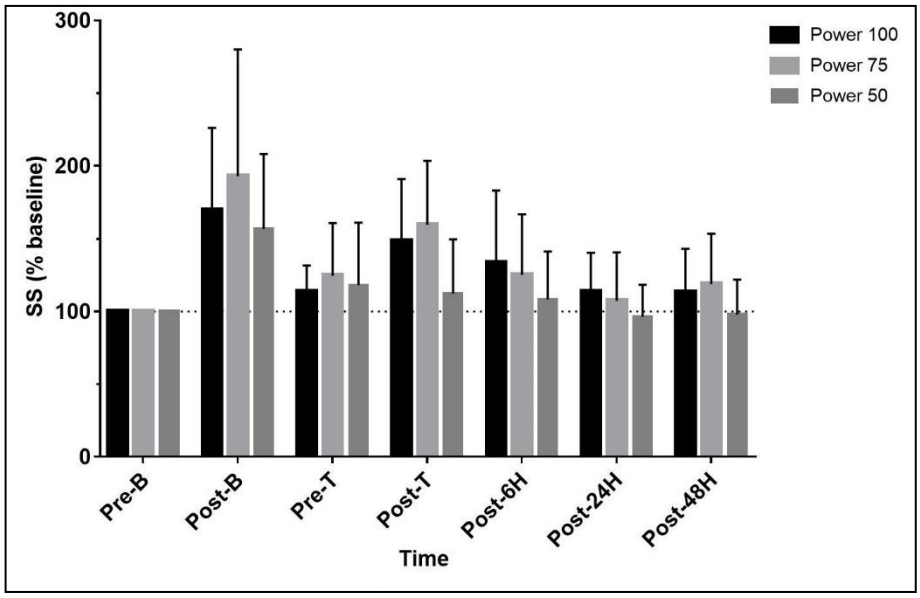


Figure 81. Comparison of P100, P75 and P50 on SS values (n = 11)

Simple main effects over time revealed that SS differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. In P100 significant time differences were observed at Post-B ($p = 0.010$, ES = 1.26) and Post-T ($p = 0.034$, ES = 0.85), except at Pre-T ($p = 0.661$, ES = 0.27), Post-6H ($p = 0.566$, ES = 0.60), Post-24H ($p = 1.000$, ES = 0.24) and Post-48H ($p = 1.000$, ES = 0.14) compared to Pre-B. Similarly, P75 also showed significant time differences at Post-B ($p = 0.011$,

ES = 1.15) and Post-T (p = 0.012, ES = 0.98), except at Pre-T (p = 1.000, ES = 0.47), Post-6H (p = 1.000, ES = 0.48), Post-24H (p = 1.000, ES = 0.12) and Post-48H (p = 1.000, ES = 0.44) compared to Pre-B. In P50, no significant time differences were observed at Pre-T (p = 1.000, ES = 0.19), Post-T (p = 1.000, ES = 0.19), Post-6H (p = 1.000, ES = -0.01), Post-24H (p = 1.000, ES = -0.18) and Post-48H (p = 1.000, ES = -0.15) except for Post-B (p = 0.026, ES = 0.79) compared to Pre-B. These results revealed that the SS increased following the M-Beast protocol in all 3 training loads and ARE protocols of P100 and P75 and it gradually returned to Pre-B values. Interestingly, ARE of P50 did not increase the SS. Furthermore, the SS recovered to baseline (Pre-B) at Post-6H for P100 and P75, whereas in P50 it did not change from Pre-T (Figure 82). According to the ES results, SS of P50 recovered at Post-6H, whereas P100's and P75's level did not recover at Post-48H.

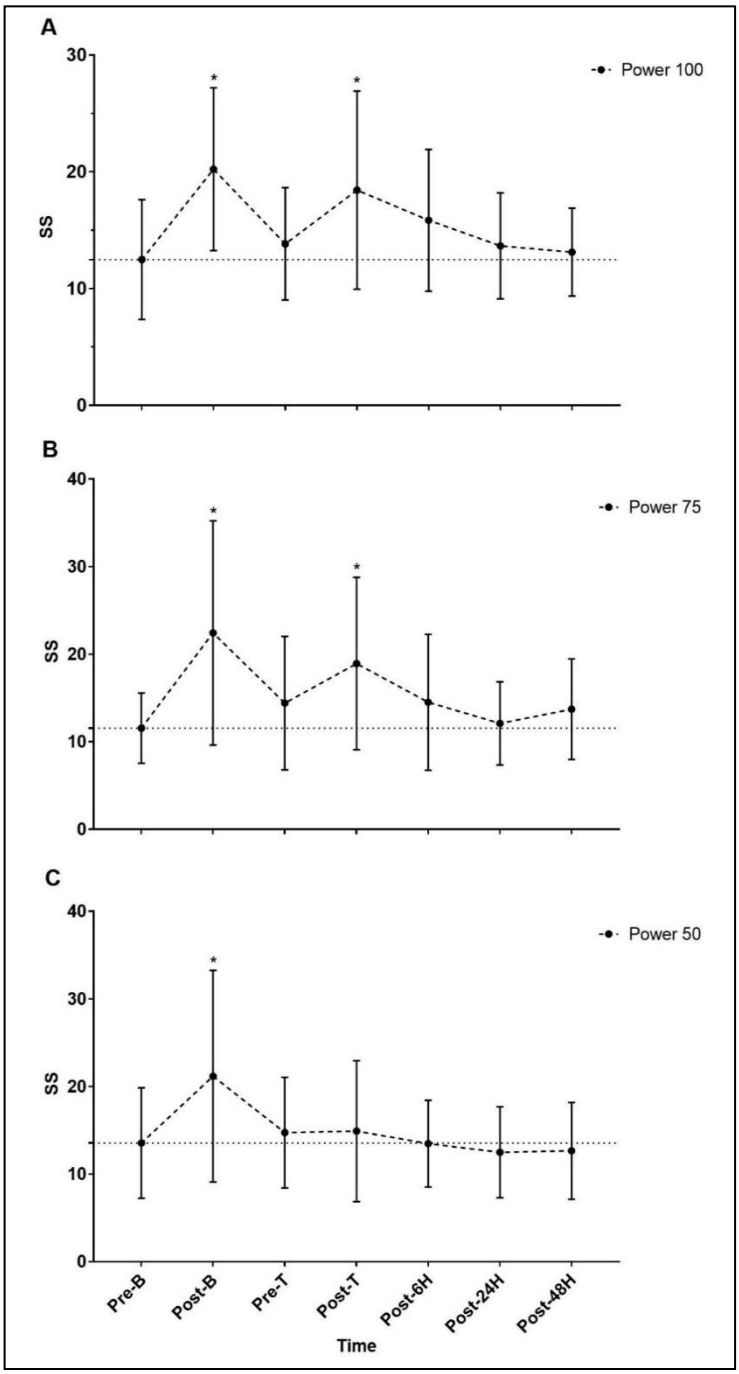


Figure 82. Changes in SS parameter in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.3. Performance variables / Physical functions

6.2.3.1. Strength 100 versus Power 100 training

The results from the performance variables for the comparison between S100 and P100 are reported below.

6.2.3.1.1. Bench press relative peak power

There was no overall treatment effect on BP RPP ($p = 0.736$). However, there was an overall time effect on BP RPP ($p < 0.001$) and significant treatment x time interaction for BP RPP ($p = 0.019$). Simple main effects for treatment showed that BP RPP was significantly different in the strength modality at Pre-B ($p = 0.021$) and Post-T ($p = 0.034$; Figure 83) compared to the power modality. This indicates that S100 modality for AREs decreased more in BP RPP than P100 modality.

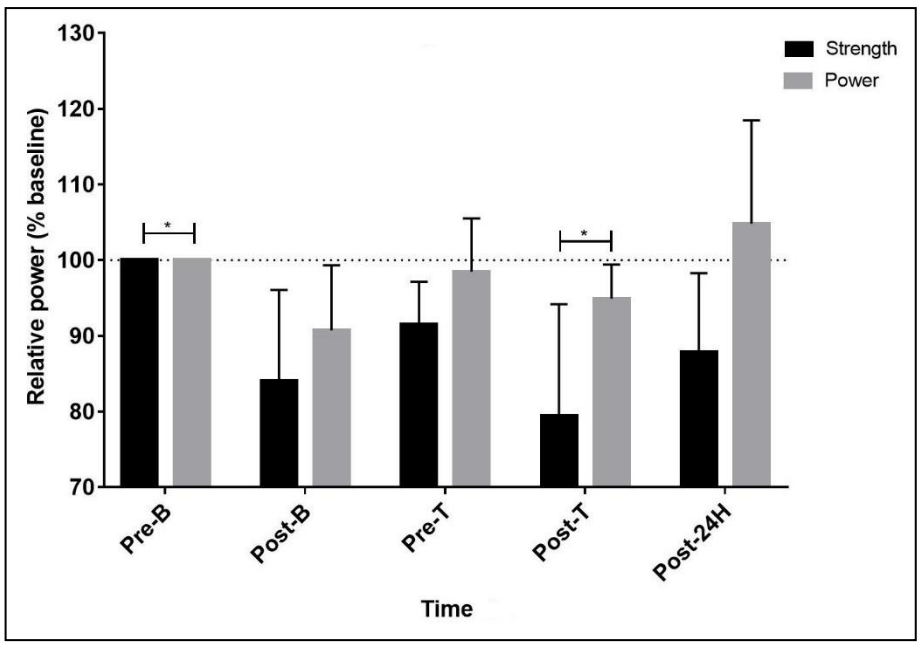


Figure 83. Comparison between S100 and P100 on BP relative peak power (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

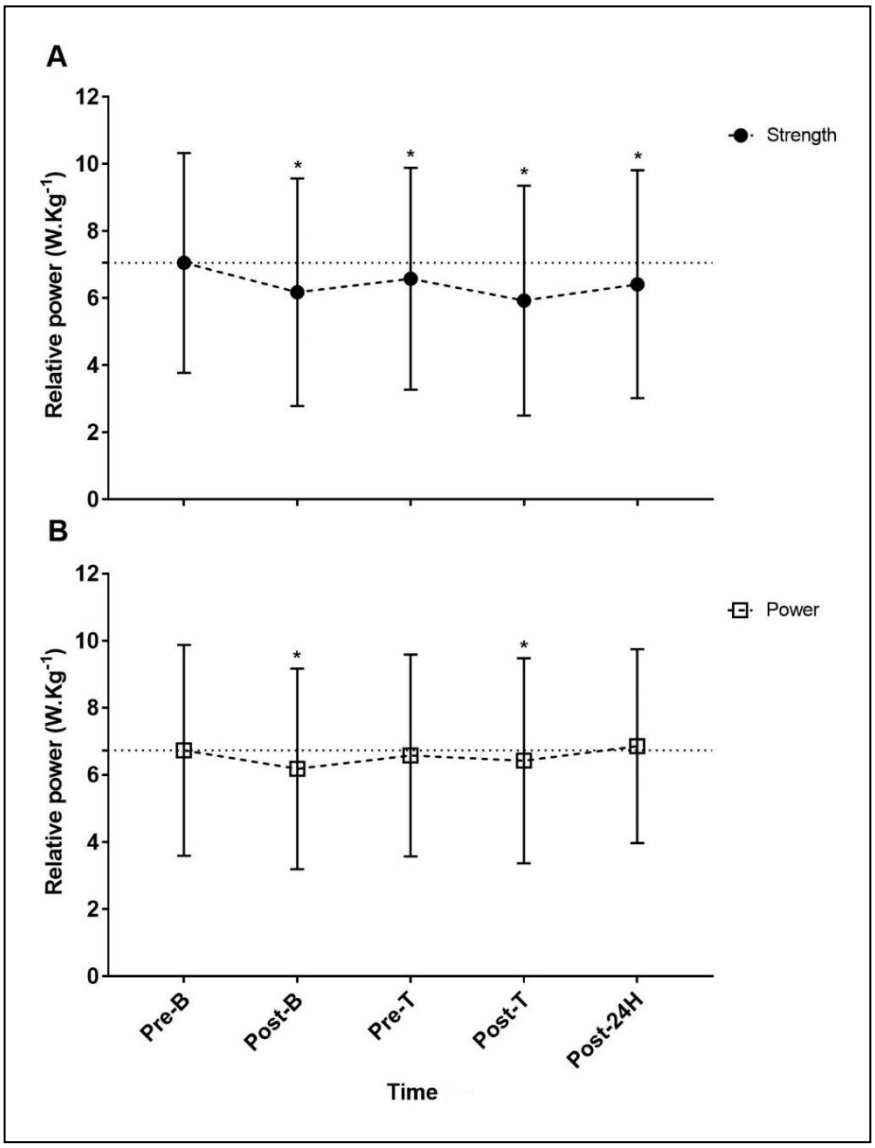


Figure 84. Changes in mean BP relative power values in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Simple main effects over time revealed that BP RPP differed significantly between time points in S100 ($P < 0.001$) and P100 ($P = 0.001$) trials. Compared to Pre-B, significant time differences were observed at Post-B (S100: $p = 0.001$, ES = -0.26; P100: $p = 0.003$, ES = -0.18) and Post-T (S100: $p = 0.001$, ES = -0.34; P100: $p = 0.002$, ES = -0.10) in both protocols and Pre-T ($p = 0.001$, ES = -0.14) and Post-24 (p

= 0.009, ES = -0.19) in S100. Interestingly, there was no significant difference at Pre-T (p = 1.000, ES = -0.05) and Post-24H (p = 1.000, ES = 0.04) in P100.These results revealed that power production decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, BP RPP recovered to baseline (Pre-B) at Post-24H for P100, but not for S100 at Post-24H. According to the ES results, BP RPP of P100 recovered at post-24H, whereas S100's level did not recover at Post-24H (Figure 84).

6.2.3.1.2. Countermovement jump height

There was no overall treatment effect on CMJ height (p = 0.622). However, there was an overall time effect on CMJ height (p < 0.001). No significant group x time interaction for CMJ height was observed (p = 0.273; Figure 85).

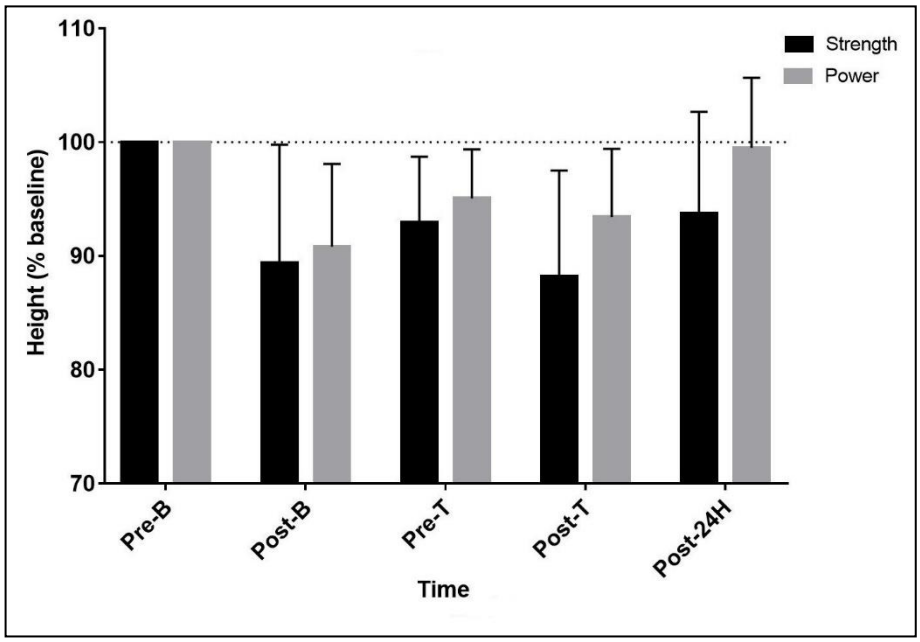


Figure 85. Comparison between S100 and P100 on CMJ height values (n = 11).

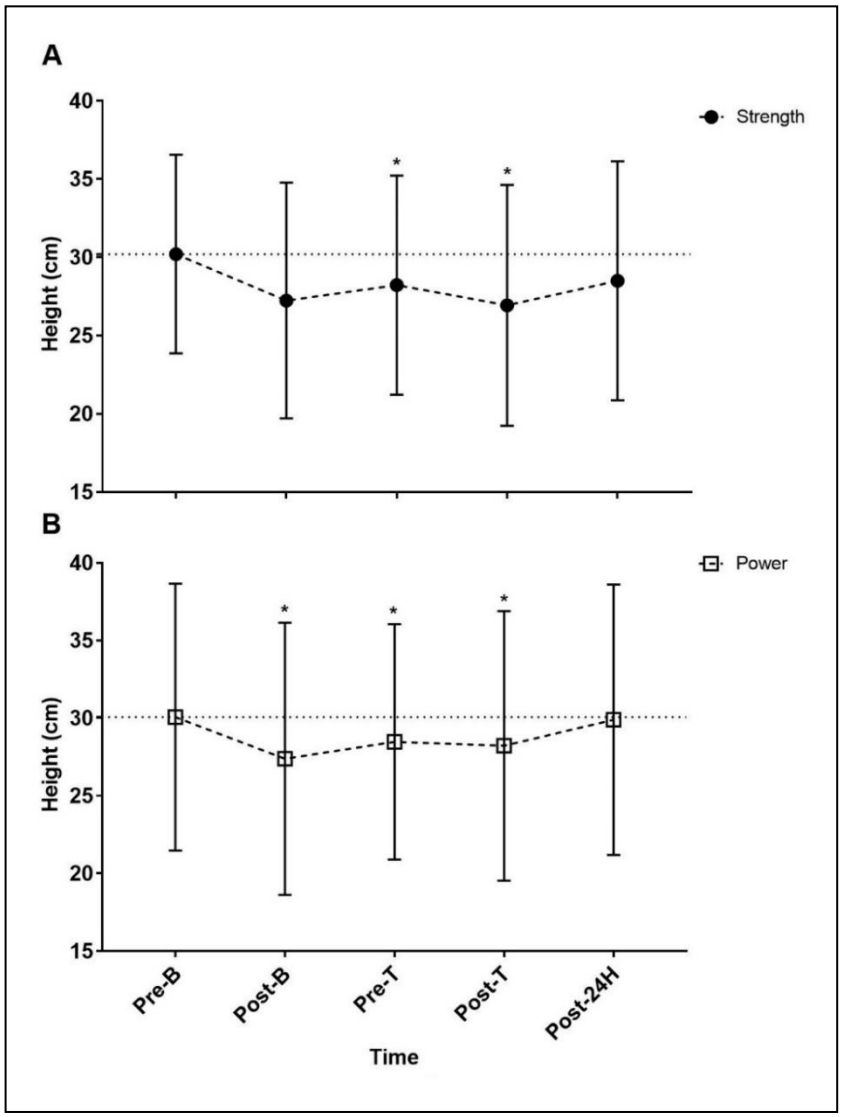


Figure 86. Changes in mean CMJ height values in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Simple main effects over time revealed that CMJ height differed significantly between time points in S100 ($P = 0.003$) and P100 ($P < 0.001$) trials. Compared to Pre-B, significant time differences were observed at Post-B ($p = 0.026$, ES = -0.31), Pre-T ($p = 0.039$, ES = -0.20) and Post-T ($p = 0.045$, ES = -0.21) in P100 protocol and Pre-T ($p = 0.021$, ES = -0.30) and Post-T ($p = 0.020$, ES = -0.46) in S100 protocol. But, no significant time difference was observed at Post-B ($p = 0.099$, ES = -0.43) in S100

protocol compare to Pre-B. At Post-24H, there were no significant differences in both protocols (S100: p = 0.434, ES = -0.24; P100: p = 1.000, ES = -0.02) compared to Pre-B (Figure 86). These results revealed that jump height decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, CMJ height performance recovered to baseline (Pre-B) at Post-24H for both training modalities (S100 and P100). According to the ES results, CMJ height performance of both training modalities did not recover at post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H.

6.2.3.1.3. Countermovement jump relative peak power

There was no overall treatment effect on CMJ RPP output (p = 0.273). However, there was an overall time effect on CMJ RPP output (p = 0.001) and significant group x time interaction for CMJ RPP output was observed (p = 0.018; Figure 87). Simple main effects for treatment showed that CMJ RPP was significantly different in the strength modality at Post-24H (p = 0.016) compared to the power modality.

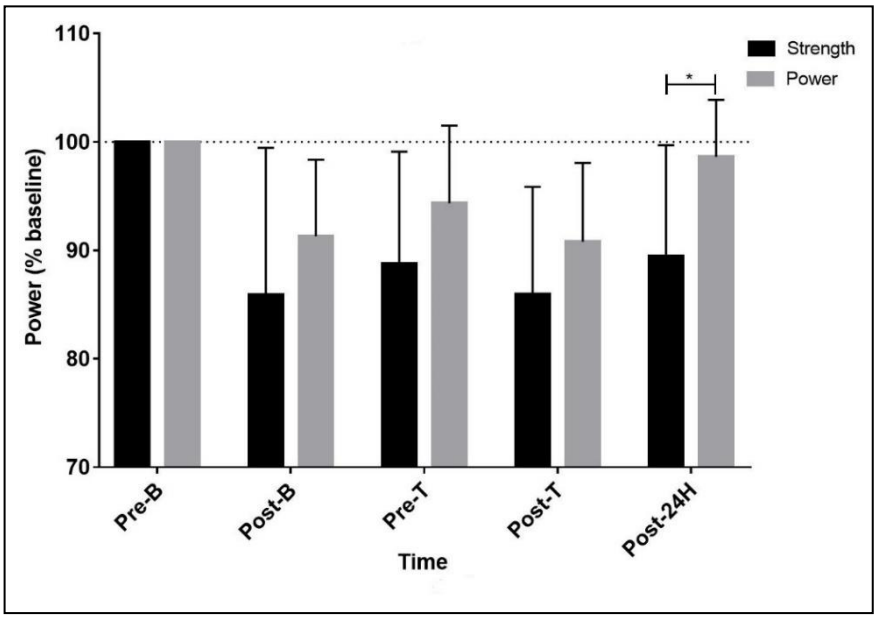


Figure 87. Comparison between S100 and P100 on CMJ relative peak power (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that CMJ RPP differed significantly between time points in S100 ($P = 0.002$) and P100 ($P < 0.001$) trials. Compared to Pre-B, no significant time differences were shown at Post-B ($p = 0.069$, ES = -0.93), Pre-T ($p = 0.081$, ES = -0.74), except at Post-T ($p = 0.015$, ES = -0.97) in S100. In P100, significant time differences were shown at Post-B ($p = 0.034$, ES = -0.53) and Post-T ($p = 0.036$, ES = -0.52), except at Pre-T ($p = 0.287$, ES = -0.34) compared to Pre-B. However, no significant time differences were shown at Post-24H (S100: $p = 0.104$, ES = -0.72; P100: $p = 1.000$, ES = -0.07) on both protocols compared to Pre-B (Figure 88). These results revealed that level of power production decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, level of power production recovered to baseline (Pre-B) at Post-24H for both training modalities. According to the ES results, level of power production of both training modalities were not yet recovered at Post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H.

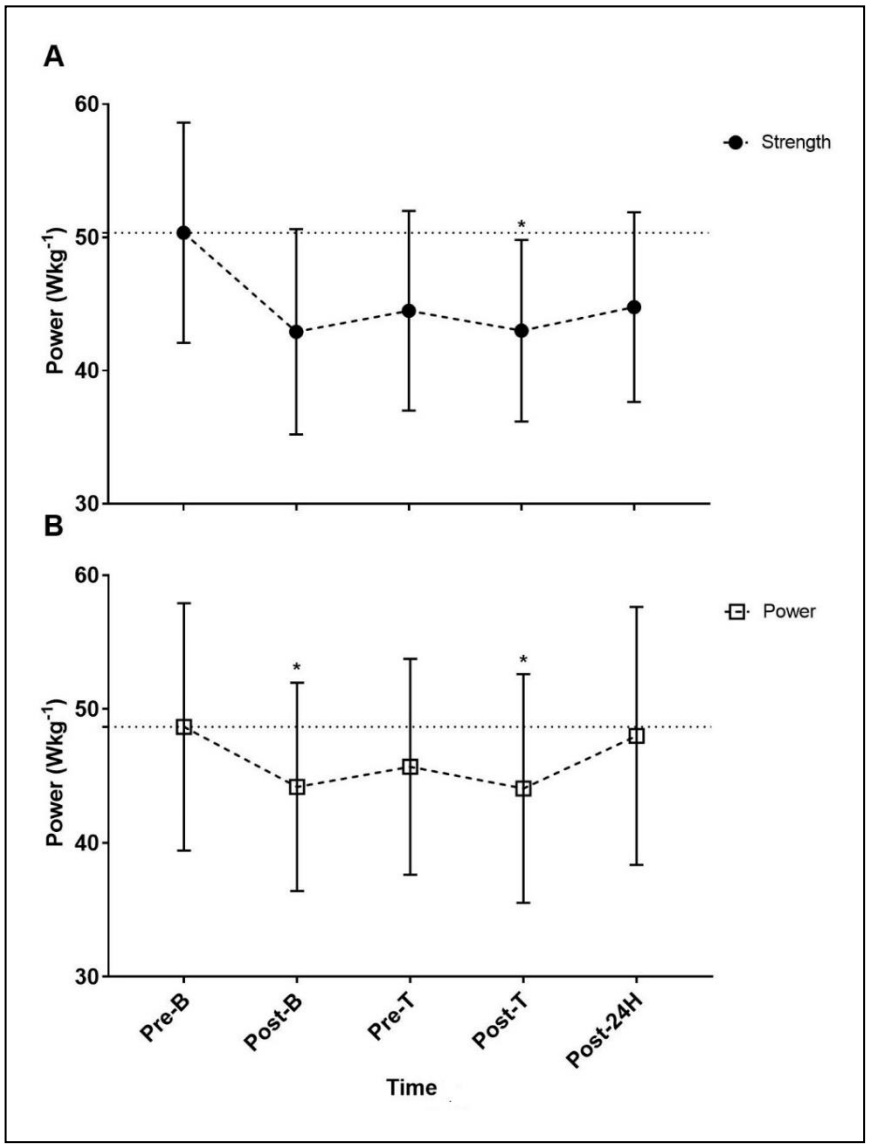


Figure 88. Changes in mean CMJ relative peak power values in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.3.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the performance variables for the comparison between S100, S75 and S50 are reported below.

6.2.3.2.1. Bench press relative peak power

There was no overall treatment effect on BP RPP ($p = 0.567$). However, there was an overall time effect on BP RPP ($p < 0.001$) and significant group x time interaction for BP RPP was observed ($p = 0.007$; Figure 89). Simple main effects for treatment showed that BP RPP was significantly different between treatments (S100 vs S75 vs S50) at Pre-B ($p = 0.033$, (S100 vs S75: $p = 0.405$; S100 vs S50: $p = 0.008$)), Post-T ($p = 0.016$, (S100 vs S75: $p = 0.468$; S100 vs S50: $p = 0.045$)) and Post-24H ($p = 0.039$, (S100 vs S75: $p = 0.862$; S100 vs S50: $p = 0.040$)).

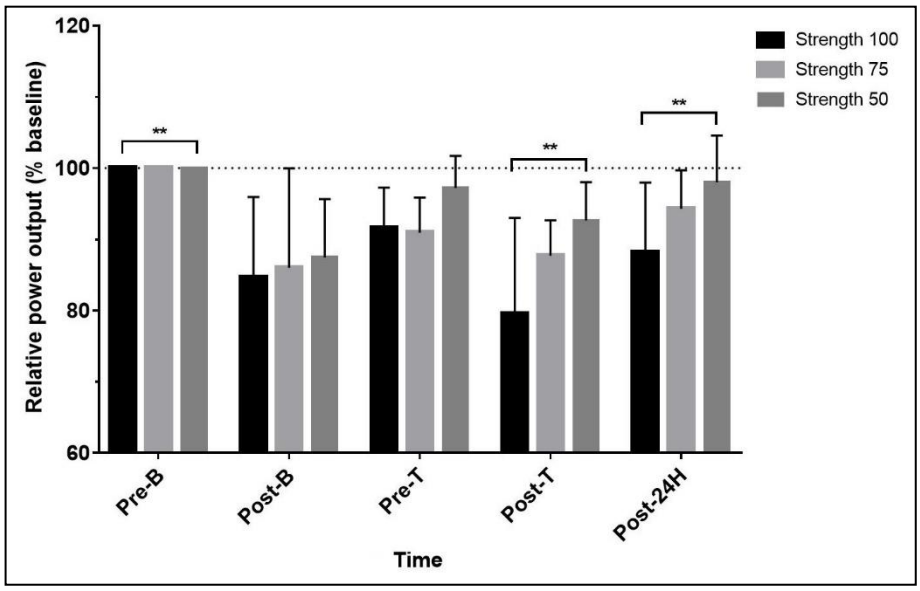


Figure 89. Comparison of S100, S75 and S50 on BP relative peak power values (n = 13). ** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that BP RPP different significantly between time points in S100 ($P < 0.001$), S75 ($P = 0.001$) and S50 ($P < 0.001$) trial. In S100, significant difference was showed at Post-B ($p < 0.001$, ES = -0.29), Pre-T ($p = 0.002$, ES = -0.17), Post-T ($p < 0.001$, ES = -0.38 and Post-24H ($p = 0.004$, ES = -0.22) compared to Pre-B.

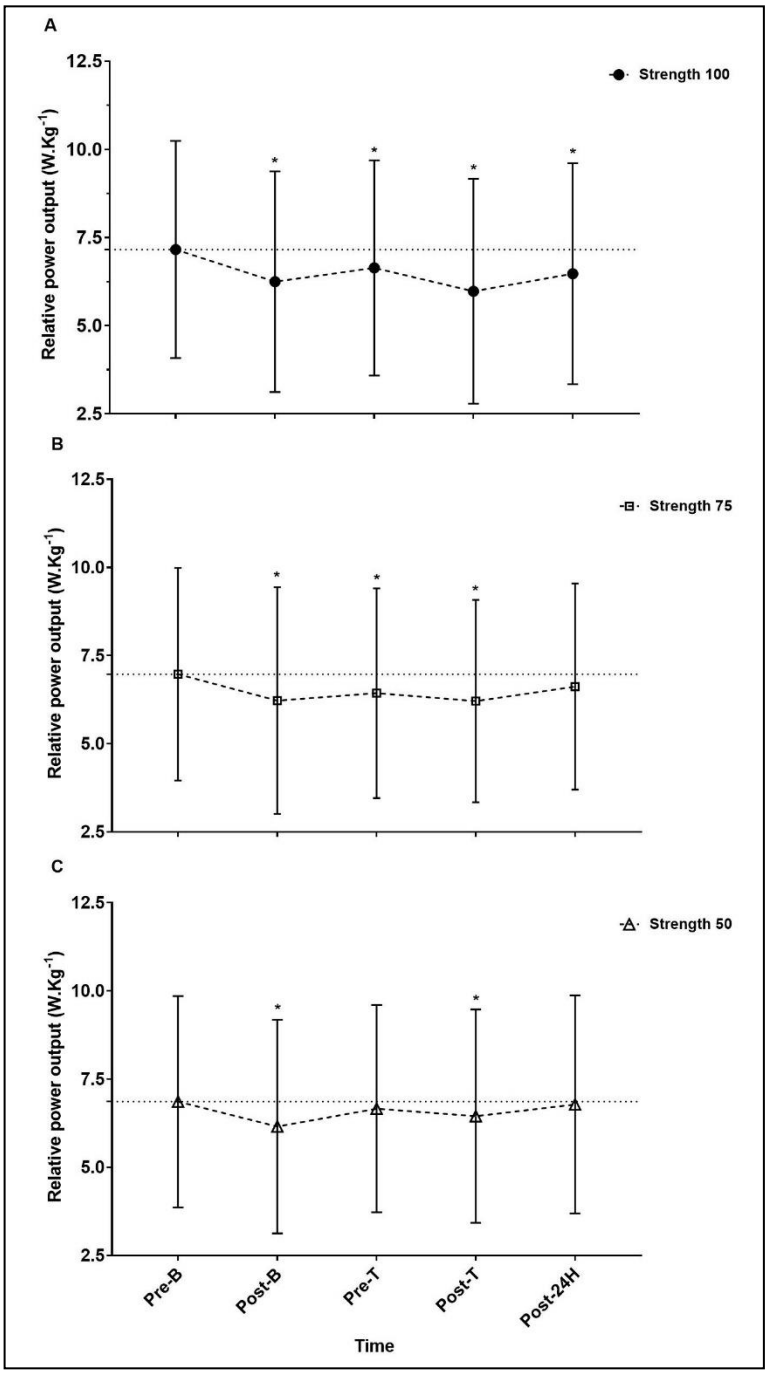


Figure 90. Changes in mean BP relative peak power values in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In S75, significant time differences were observed at Post-B ($p = 0.007$, ES = -0.24), Pre-T ($p < 0.001$, ES = -0.18), Post-T ($p < 0.001$, ES = -0.26) except at Post-24H ($p = 0.067$, ES = -0.12) compared to Pre-B. In S50, significant time differences were observed at Post-B ($p < 0.001$, ES = -0.23) and Post-T ($p < 0.001$, ES = -0.14), except at Pre-T ($p = 0.159$, ES = -0.07) and Post-24H ($p = 1.000$, ES = -0.03) compared to Pre-B. These results revealed that peak power decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, level of peak power recovered to baseline (Pre-B) at Post-24H for S75 and S50, whereas S100 did not recover at Post-24H (Figure 90). According to the ES results, level of peak power of all 3 training loads did not recover at Post-24H. Even though not fully recovered, S50 showed better recovery and S100 showed least recovery at Post-24H.

6.2.3.2.2. Countermovement jump height

There was no overall treatment effect on CMJ height ($p = 0.447$). However, there was an overall time effect on CMJ height ($p < 0.001$). No significant group x time interaction for CMJ height was observed ($p = 0.202$; Figure 91).

Simple main effects over time revealed that CMJ height different significantly between time points in S100 ($P < 0.001$), S75 ($P = 0.002$) and S50 ($P = 0.002$) trial. In S100, no significant time differences were observed at Post-B ($p = 0.072$, ES = -0.39) and Post-24H ($p = 0.389$, ES = -0.23) compared to Pre-B. In addition, significant difference and significant trend was showed at Post-T ($p = 0.007$, ES = -0.46) and Pre-T ($p = 0.063$, ES = -0.24), respectively, compared to Pre-B. In S75, no significant time differences were observed at Pre-T ($p = 1.000$, ES = -0.08), Post-T ($p = 0.119$, ES = -0.26), Post-24H ($p = 0.205$, ES = -0.16) except at Post-B ($p = 0.019$, ES = -0.32), compared to Pre-B. In S50, no significant time differences were observed at all the time points (Post-B ($p = 0.193$, ES = -0.20), Pre-T ($p = 1.000$, ES = -0.09), Post-T ($p = 0.874$, ES = -0.14) and Post-24H ($p = 1.000$, ES = 0.04)) compared to Pre-B. These results revealed that jump height decreased following the M-Beast protocol and

ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, jump height recovered to baseline (Pre-B) at Post-24H for S100, whereas S75 and S50 remained recovered from Pre- T (Figure 92). According to the ES results, jump height of S50 recovered at Post-24H, whereas S100's and S75's level were not yet recovered at Post-24H. Even though not fully recovered, S75 showed better recovery level than S50 at Post-24H.

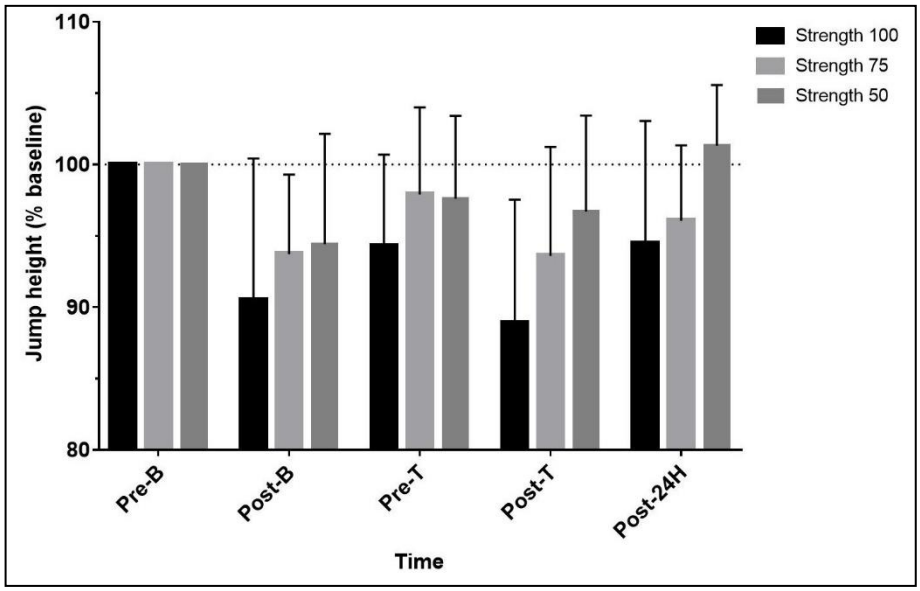


Figure 91. Comparison of S100, S75 and S50 on CMJ height values (n = 13).

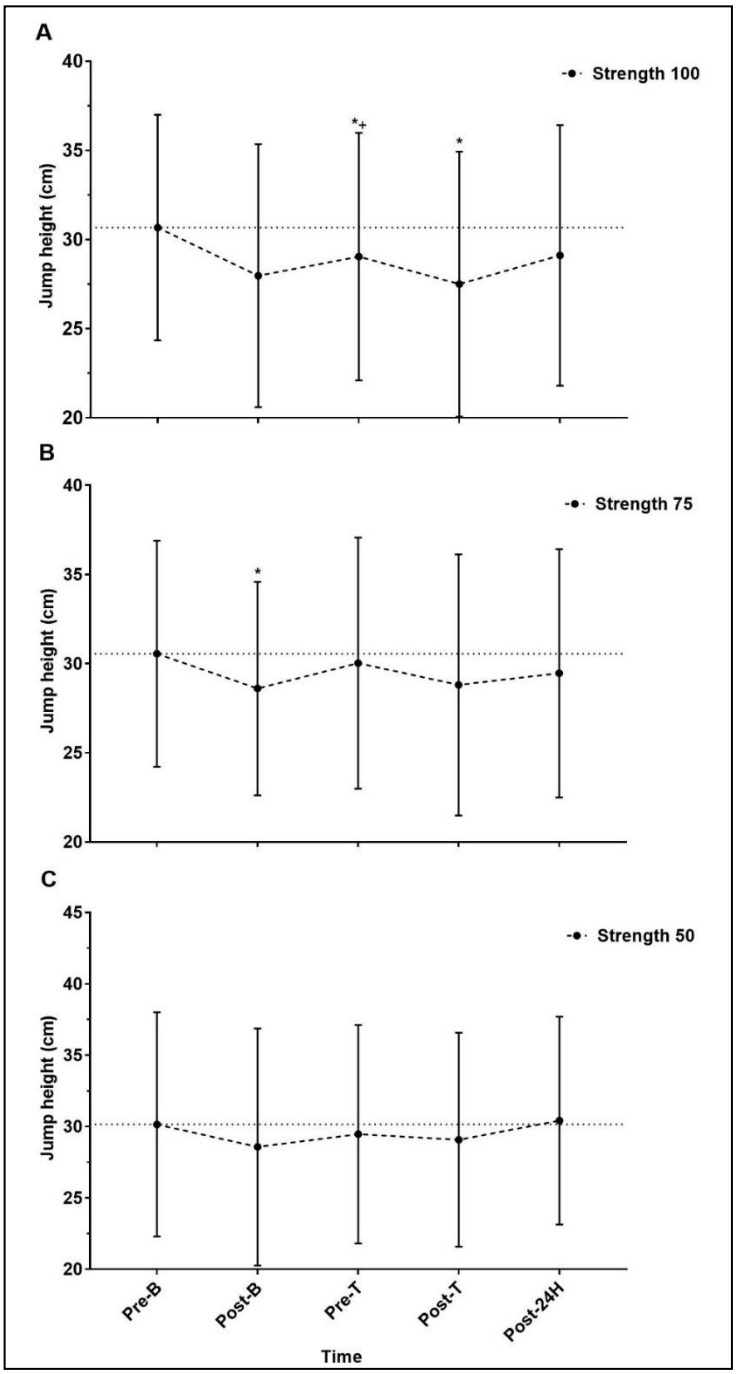


Figure 92. Changes in mean CMJ height values in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis. *+ Significant trend time difference compared to Pre-B ($p \leq 0.06$) from post-hoc Bonferroni analysis.

6.2.3.2.3. Countermovement jump relative peak power

There was no overall treatment effect on CMJ RPP ($p = 0.249$). However, there was an overall time effect on CMJ RPP ($p < 0.001$) and significant group x time interaction for CMJ RPP was observed ($p = 0.019$). Simple main effects for treatment showed that CMJ RPP was significantly different between treatments (S100 vs S75 vs S50) at the Pre-B ($p = 0.052$, (S100 vs S75: $p = 0.163$; S100 vs S50: $p = 0.296$)), Post-24H ($p = 0.003$, (S100 vs S75: $p = 0.909$; S100 vs S50: $p = 0.055$)), and there was a significant trend at Post-T ($p = 0.057$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.029$)). (Figure 93)

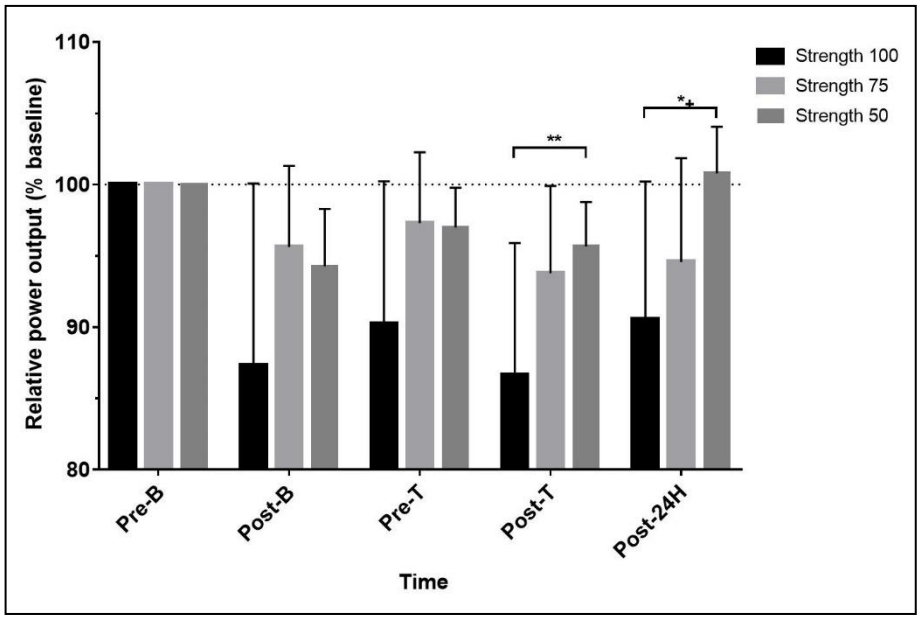


Figure 93. Comparison of S100, S75 and S50 on CMJ relative peak power values (n = 13). ** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$). *+ Significant trend pairwise comparison differences in S50 compared to S100 ($p \leq 0.06$).

Simple main effects over time revealed that CMJ RPP differed significantly between time points in S100 ($P = 0.001$), S75 ($P = 0.006$) and S50 ($P < 0.001$) trial. In S100, significant time differences were observed at Post-B ($p = 0.042$, ES = -0.87) and Post-T ($p = 0.005$, ES = -0.96) except at Pre-T ($p = 0.070$, ES = -0.67) and Post-24H ($p = 0.068$, ES = -0.68) compared to Pre-B.

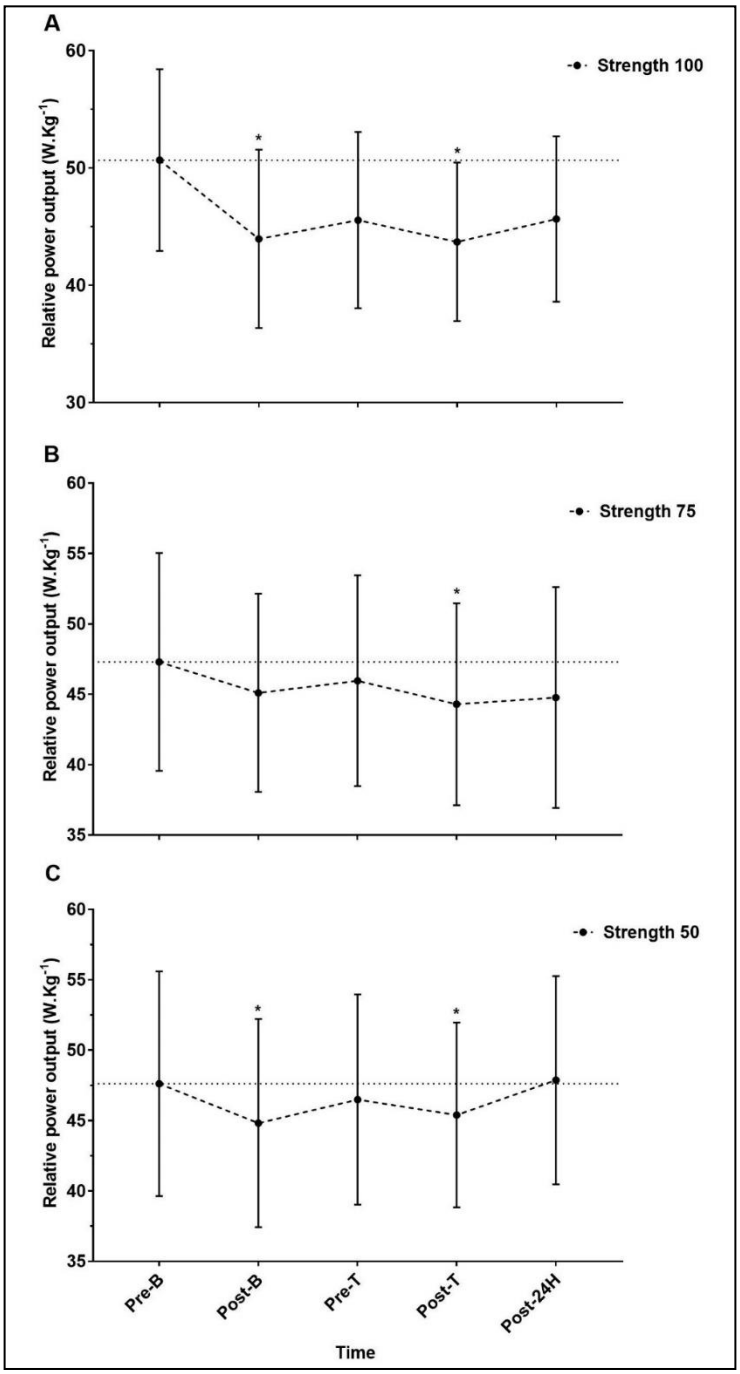


Figure 94. Changes in mean CMJ relative peak power values in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In S75, no significant time differences were observed at Post-B (p = 0.133, ES = -0.30), Pre-T (p = 0.646, ES = -0.18), Post-24H (p = 0.121, ES = -0.32) except at Post-T (p = 0.035, ES = -0.40) compared to Pre-B. In S50, significant time differences were observed at Post-B (p = 0.003, ES = -0.36) and Post-T (p = 0.009, ES = -0.30) except at Pre-T (p = 0.286, ES = -0.15) and Post-24H (p = 1.000, ES = 0.03) compared to Pre-B (Figure 94). These results revealed that peak power decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, peak power recovered to baseline (Pre-B) at Post-24H for all three training loads. According to the ES results, peak power of S50 recovered at Post-24H, whereas S75's and S100's level were not yet recovered at Post-24H. Even though not fully recovered, S75 showed better recovery level than S100 at Post-24H.

6.2.3.3. Power 100 versus Power 75 versus Power 50 training

The results from the performance variables for the comparison between P100, P75 and P50 are reported below.

6.2.3.3.1. Bench press relative peak power

There was no overall treatment effect on BP RPP (p = 0.787). However, there was an overall time effect on BP RPP (p < 0.001). No significant group x time interaction for BP RPP was observed (p = 0.631; Figure 95).

Simple main effects over time revealed that BP RPP different significantly between time points in P100 (P = 0.001), P75 (P < 0.001) and P50 (P < 0.001) trial. In P100, significant time differences were observed at Post-B (p = 0.003, ES = -0.18) and Post-T (p = 0.002, ES = -0.10) except at Pre-T (p = 1.000, ES = -0.05) and Post-24H (p = 1.000, ES = 0.04) compared to Pre-B. In P75, significant time differences were observed at Post-B (p = 0.001, ES = -0.24) and Post-T (p = 0.004, ES = -0.14) except at Pre-T (p = 0.128, ES = -0.07) and Post-24H (p = 1.000, ES = 0.03) compared to Pre-B value. In P50, significant time differences were observed at Post-B (p =

0.003, ES = -0.31) and Post-T (p = 0.013, ES = -0.12) except at Pre-T (p = 1.000, ES = -0.05) and Post-24H (p = 1.000, ES = 0.01) compared to Pre-B value.

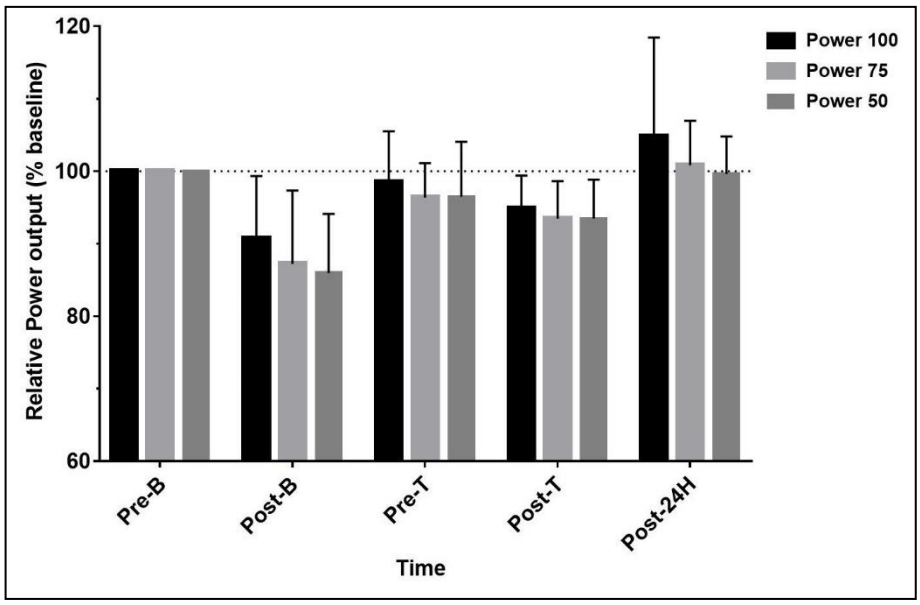


Figure 95. Comparison between P100, P75 and P50 on BP relative peak power values (n = 11)

These results revealed that peak power decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, BP RPP recovered to baseline (Pre-B) at Post-24H for all 3 training loads. According to the ES results, BP RPP of all 3 training loads recovered at Post-24H (Figure 96).

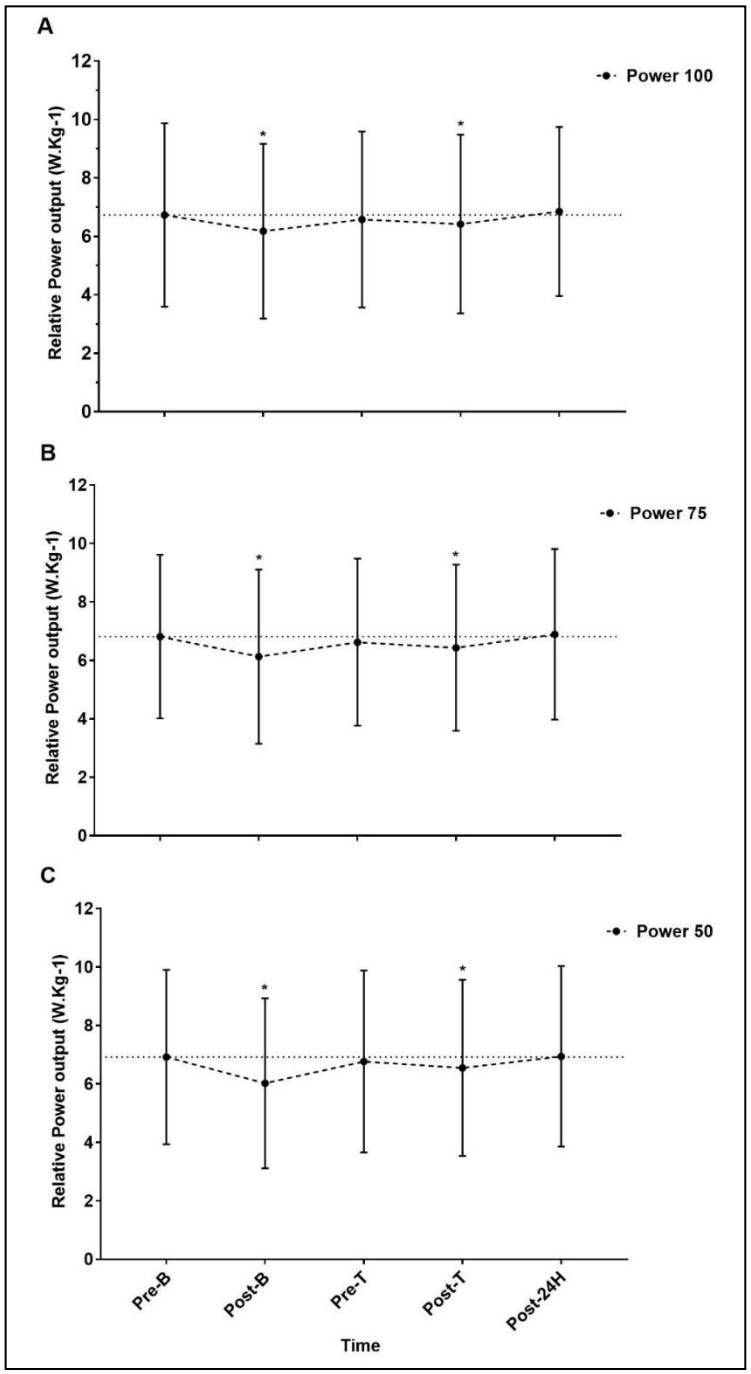


Figure 96. Changes in mean BP relative peak power values in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.3.3.2. Countermovement jump height

There was no overall treatment effect on CMJ height (p = 0.148). However, there was an overall time effect on CMJ height (p = 0.002). No significant group x time interaction for CMJ height was observed (p = 0.941; Figure 97).

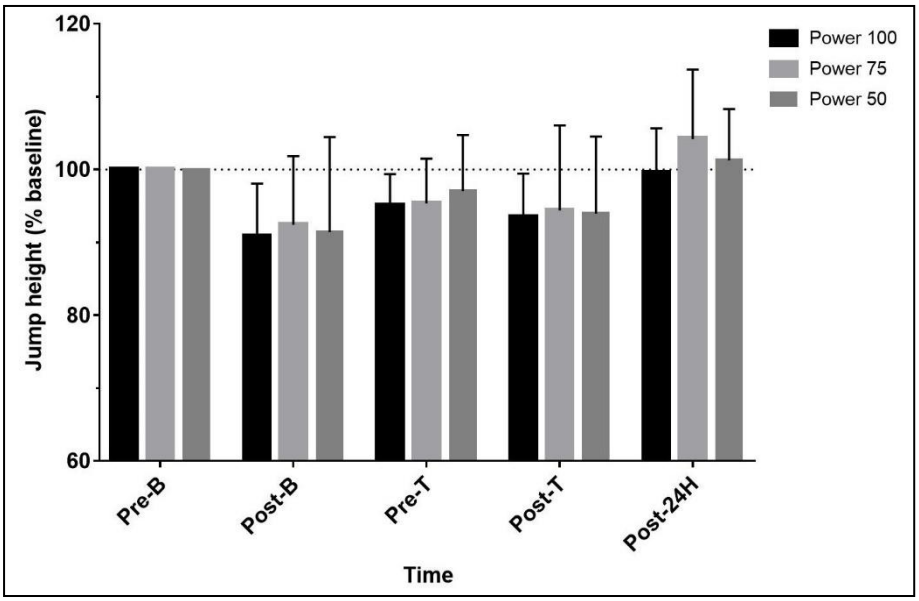


Figure 97. Comparison between P100, P75 and P50 on CMJ height values (n = 11).

Simple main effects over time revealed that CMJ height differed significantly between time points in P100 (P < 0.001), P75 (P = 0.027) and P50 (P = 0.008) trial. In P100, significant time differences were observed at Post-B (p = 0.026, ES = -0.31), Pre-T (p = 0.039, ES = -0.20) and Post-T (p = 0.045, ES = -0.21) except at Post-24H (p = 0.968, ES = -0.02) compared to Pre-B. In P75, no significant time differences were observed at all the time points (Post-B (p = 0.215, ES = -0.20), Pre-T (p = 0.312, ES = -0.13), Post-T (p = 1.000, ES = -0.16) and Post-24H (p = 1.000, ES = 0.13)) compared to Pre-B. Similarly, P50 also showed no significant time differences at all the time points (Post-B (p = 0.481, ES = -0.27), Pre-T (p = 1.000, ES = -0.10), Post-T (p = 0.682, ES = -0.23) and Post-48H (p = 1.000, ES = 0.03)) compared to Pre-B.

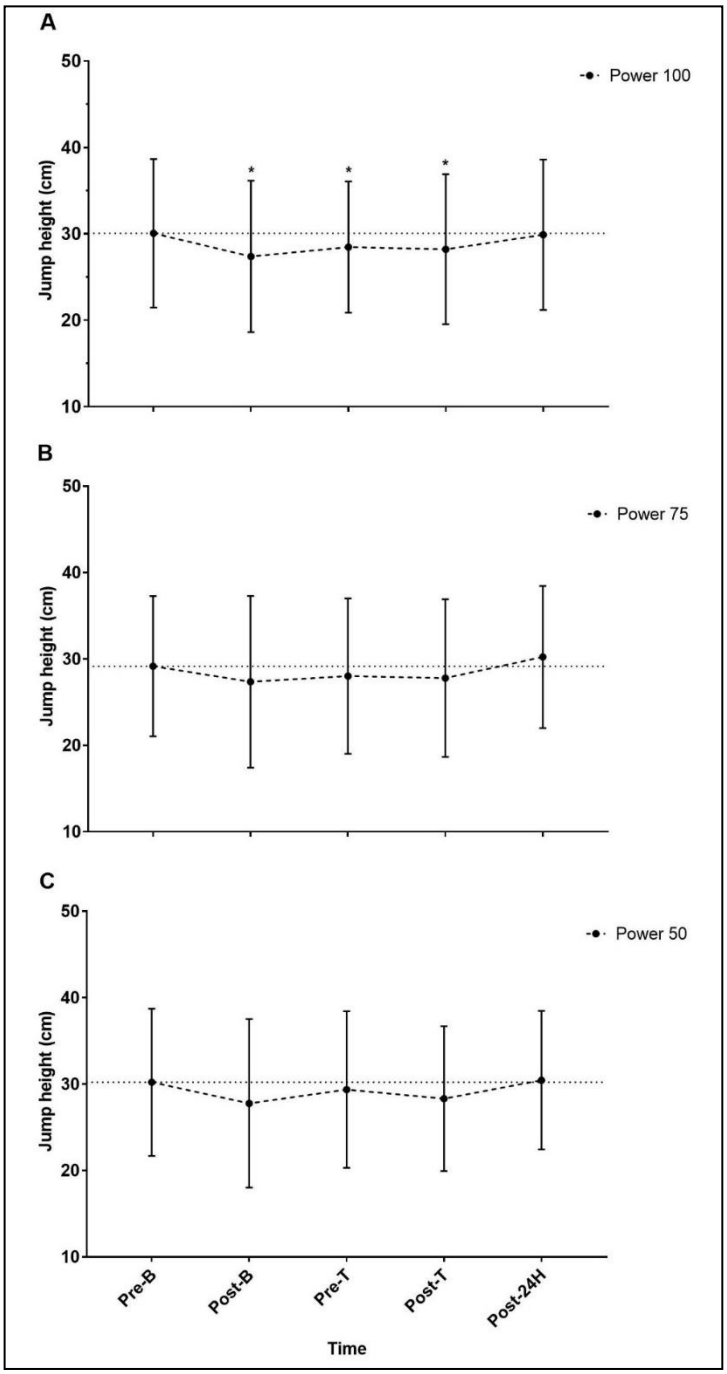


Figure 98. Changes in mean CMJ height values in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that jump height decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B. Interestingly, jump height recovered to baseline (Pre-B) at Post-6H for P100, whereas P75 and P50 remained recovered from Pre-T. According to the ES results, jump height of P75 and P50 recovered at Post-24H, whereas P100's level did not recover at Post-24H (Figure 98).

6.2.3.3.3. Countermovement jump relative peak power

There was no overall treatment effect on CMJ RPP ($p = 0.403$). However, there was an overall time effect on CMJ RPP ($p < 0.001$). No significant group x time interaction for CMJ RPP was observed ($p = 0.146$; Figure 99).

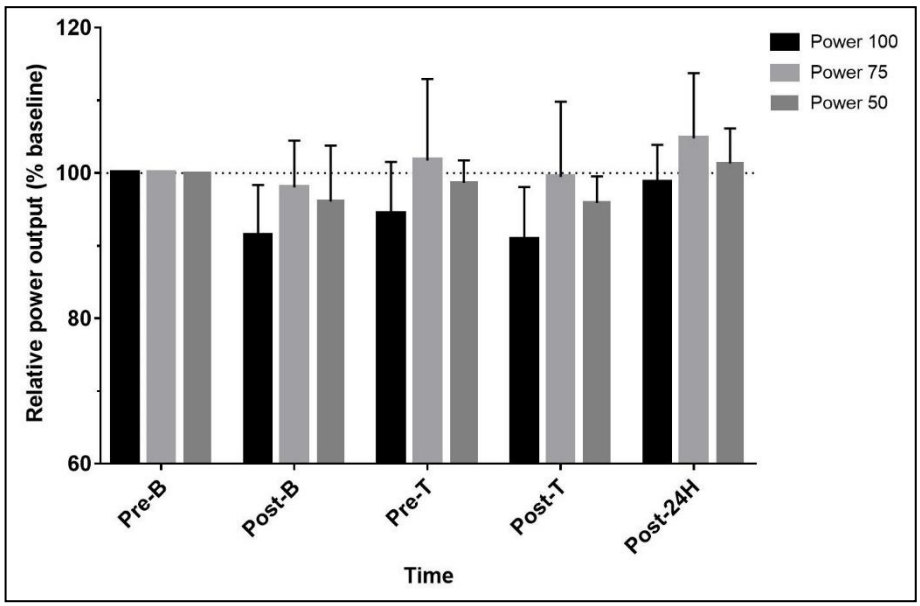


Figure 99. Comparison between P100, P75 and P50 on CMJ relative peak power values (n = 11).

Simple main effects over time revealed that CMJ RPP different significantly between time points in P100 ($P < 0.001$) and P50 ($P = 0.006$) except P75 ($P = 0.244$) trial.

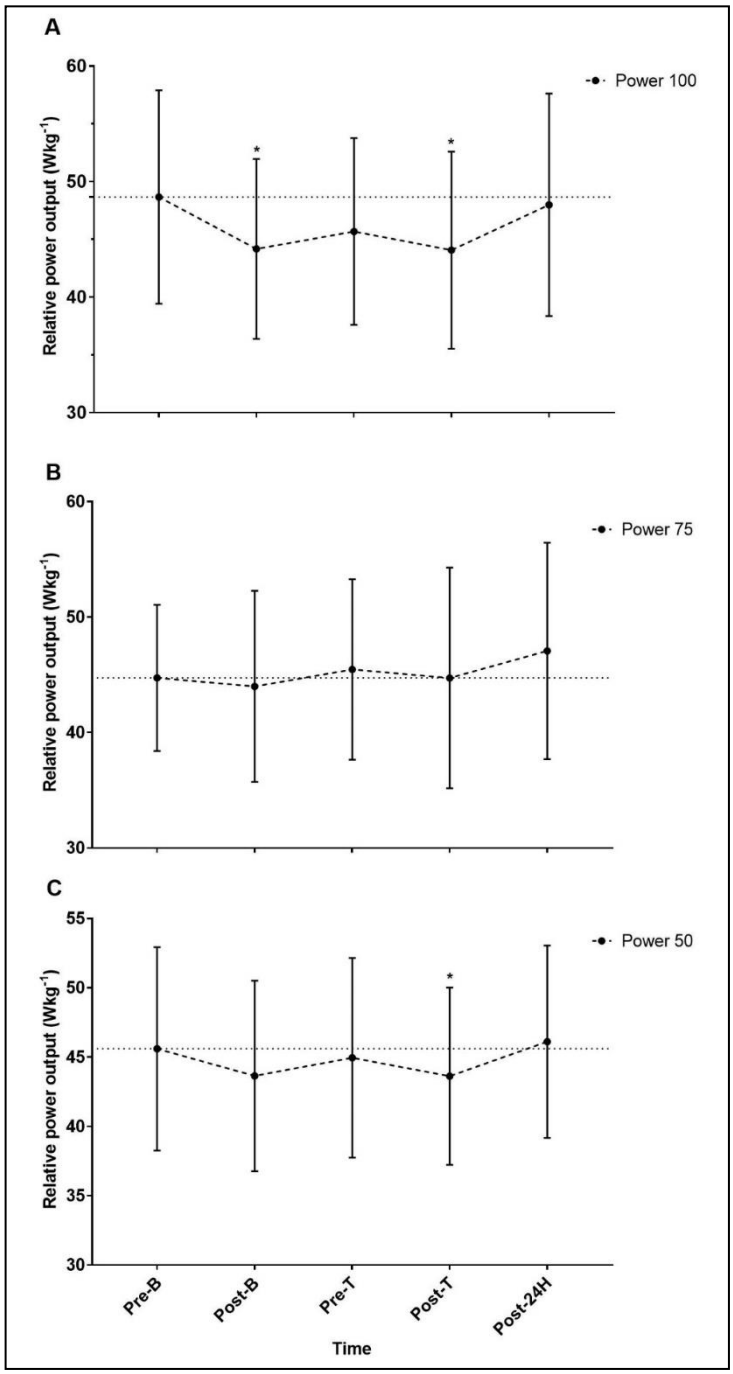


Figure 100. Changes in mean CMJ relative peak power values in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In P100, significant time differences were observed at Post-B (p = 0.034, ES = -0.53) and Post-T (p = 0.036, ES = -0.52) except at Pre-T (p = 0.287, ES = -0.34) and Post-24H (p = 1.000, ES = -0.07) compared to Pre-B. In P75, no significant time differencse were observed at all the time points (Post-B (p = 1.000, ES = -0.10), Pre-T (p = 1.000, ES = 0.10), Post-T (p = 1.000, ES = 0.00) and Post-24H (p = 1.000, ES = 0.29) compared to Pre-B. In P50, no significant time differences were observed at Post-B (p = 0.900, ES = -0.28), Pre-T (p = 1.000, ES = -0.09), Post-24H (p = 1.000, ES = 0.07) except at Post-T (p = 0.054, ES = -0.29) compared to Pre-B.

These results revealed that CMJ RPP performance decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, CMJ RPP performance recovered to baseline (Pre-B) at Post-24H for P100 and P50, whereas P75 remain recovered from Pre-T. According to the ES results, CMJ RPP performance of P75 remain recovered from Pre-T and P50 was recovered at Post-24H, whereas P100's level did not recover at Post-24H (Figure 100).

6.2.4. Neuromuscular fatigue

6.2.4.1. Strength 100 versus Power 100 training

The results from the neuromuscular fatigue indicating variables of comparison between S100 and P100 are reported below.

6.2.4.1.1. Maximal voluntary isometric contractions peak force

There was no overall treatment effect on MVC peak force (p = 0.640). However, there was an overall time effect on MVC peak force (p < 0.001). No significant group x time interaction for MVC peak force was observed (p = 0.219; Figure 101).

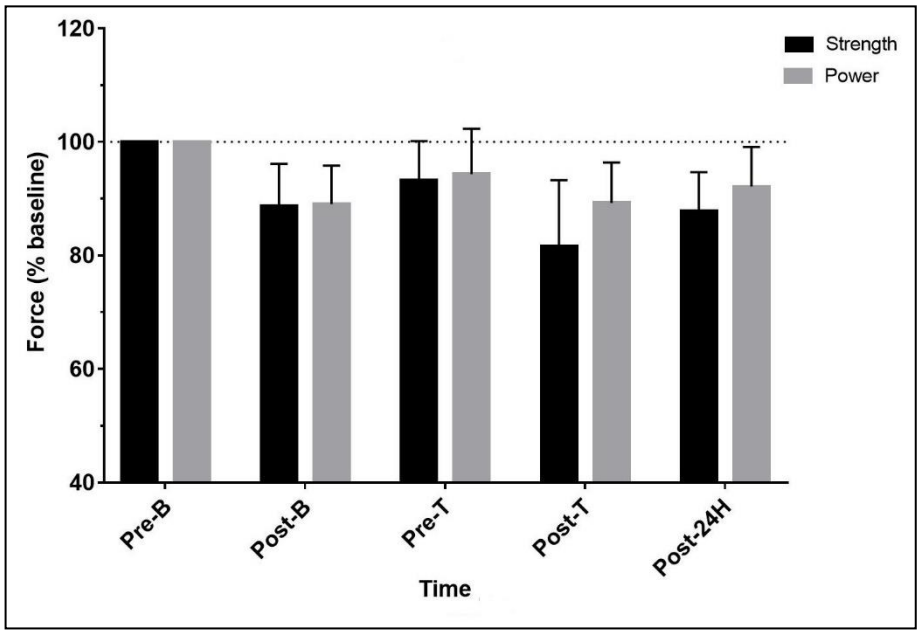


Figure 101. Comparison between S100 and P100 on MVC peak force values (n = 9)

Simple main effects over time revealed that MVC peak force differed significantly between time points in S100 ($P < 0.001$) and P100 ($P = 0.009$) protocol. Compared to Pre-B, significant time differences were shown at Post-B (S100: $p = 0.038$, ES = -0.33; P100: $p = 0.014$, ES = -0.38) in both training modalities compared to respective Pre-B. No significant time differences were observed at Pre-T (S100: $p = 0.240$, ES = -0.26; P100: $p = 0.846$, ES = -0.27) in both training modalities and at Post-T (S100: $p = 0.084$, ES = -0.76) in S100 compared to their respective Pre-B. There was a tendency towards significant difference at Post-T (P100: $p = 0.063$, ES = -0.45) in P100. Interestingly, there was a significant difference compared to Pre-B in S100 ($p = 0.020$, ES = -0.43) at Post-24H, but not for P100 ($p = 0.087$, ES = -0.26) compared to their respected Pre-B values (Figure 102). These results revealed that peak force decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, peak force recovered to baseline (Pre-B) at Post-24H for P100, whereas S100's level did not recover at Post-24H. According to the ES results, peak force of both training

modalities were not yet recovered at post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H.

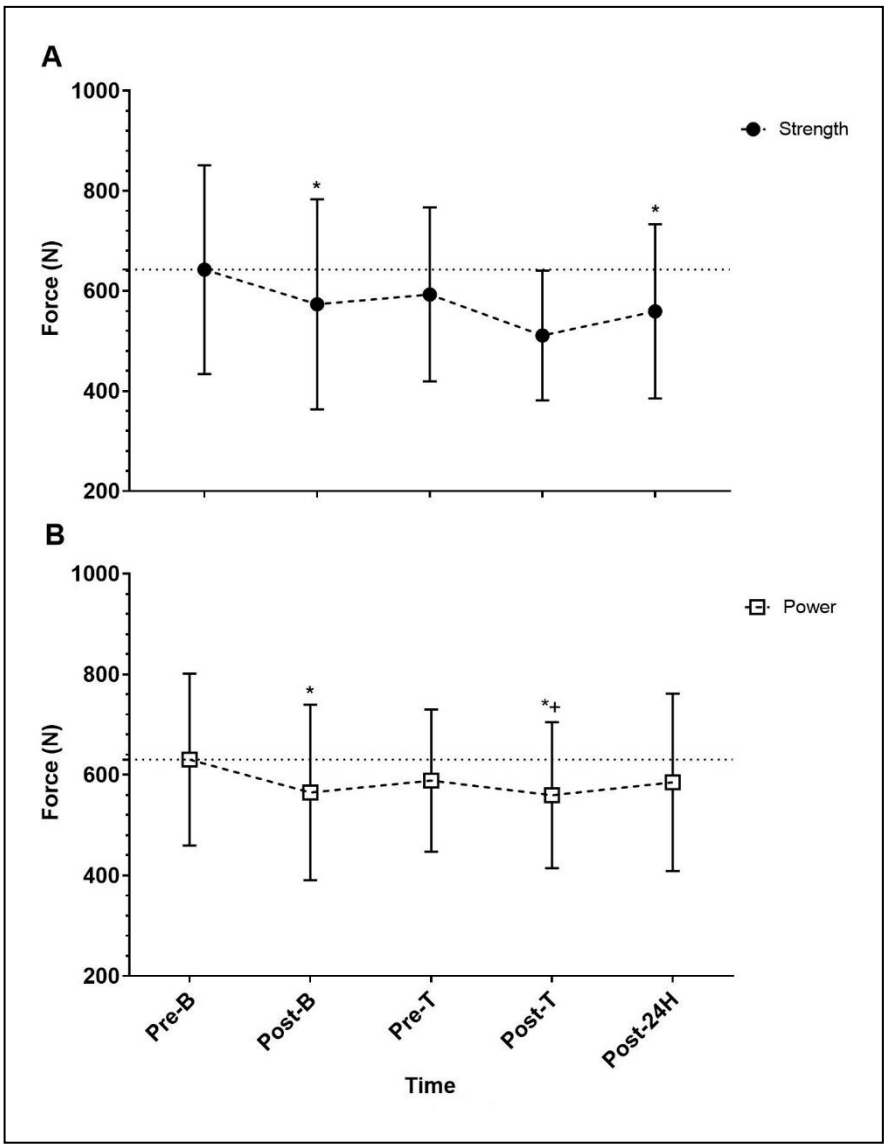


Figure 102. Changes in mean MVC peak force values in (A) S100 and (B) P100 protocols (n = 09). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis. *+ Significant trend time difference compared to Pre-B ($p \leq 0.06$) from post-hoc Bonferroni analysis.

6.2.4.1.2. Rate of force development (0–200 ms) in maximal voluntary isometric contraction

There was no overall treatment effect on RFD^{200MVC} ($p = 0.095$). However, there was an overall time effect on RFD^{200MVC} ($p = 0.022$) and significant group x time interaction for RFD^{200MVC} was observed ($p = 0.018$; Figure 103). Simple main effects for treatment showed that RFD^{200MVC} was significantly lower in the strength modality at Post-24H ($p = 0.022$) of the trials.

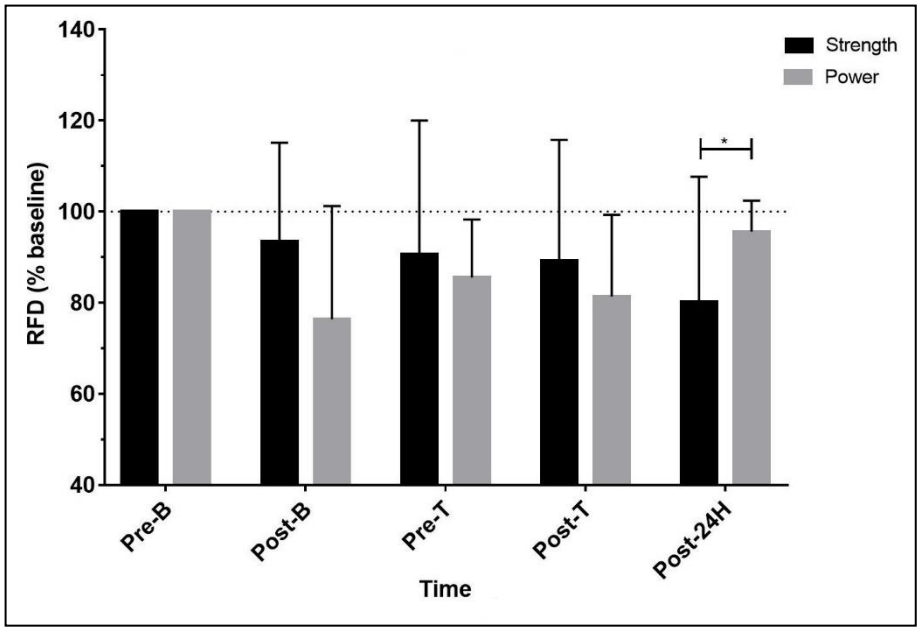


Figure 103. Comparison between S100 and P100 on RFD^{200MVC} values (n = 09). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that RFD^{200MVC} differed significantly between time points in P100 ($P = 0.021$), but not for S100 ($P = 0.147$) trial. No significant time differences were observed at Post-B (S100: $p = 1.000$, ES = -0.19; P100: $p = 0.326$, ES = -0.82), Pre-T (S100: $p = 1.000$, ES = -0.34; P100: $p = 0.124$, ES = -0.67), Post-T (S100: $p = 1.000$, ES = -0.48; P100: $p = 0.163$, ES = -0.86) and Post-24H (S100: $p = 0.386$, ES = -0.63; P100: $p = 0.999$, ES = -0.17) compared to Pre-B in both training modalities (Figure 104). These results revealed that RFD decreased

following the M-Beast protocol and ARE protocols for both training modalities. Following the M-Beast protocol and ARE protocols, P100 gradually returned to Pre-B value, while S100 remained depressed. According to the ES results, RFD of both training modalities were not yet recovered at Post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H.

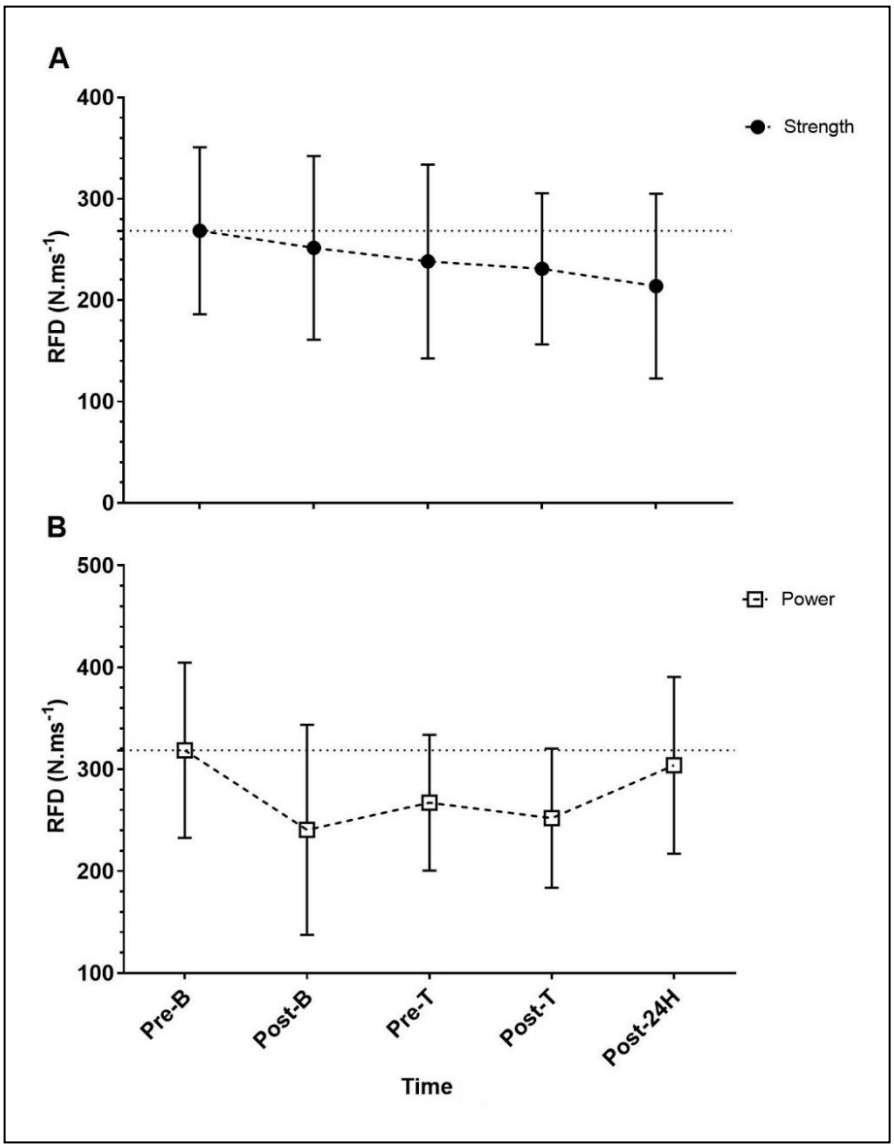


Figure 104. Changes in mean RFD^{200MVC} values in (A) S100 and (B) P100 protocols (n = 09).

6.2.4.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the neuromuscular fatigue indicating variables for the comparison between S100, S75 and S50 are reported below

6.2.4.2.1. Maximal voluntary isometric contractions peak force

There was no overall treatment effect on MVC peak force ($p = 0.896$). However, there was an overall time effect on MVC peak force ($p < 0.001$) and significant group x time interaction for MVC peak force was observed ($p = 0.012$; Figure 105).

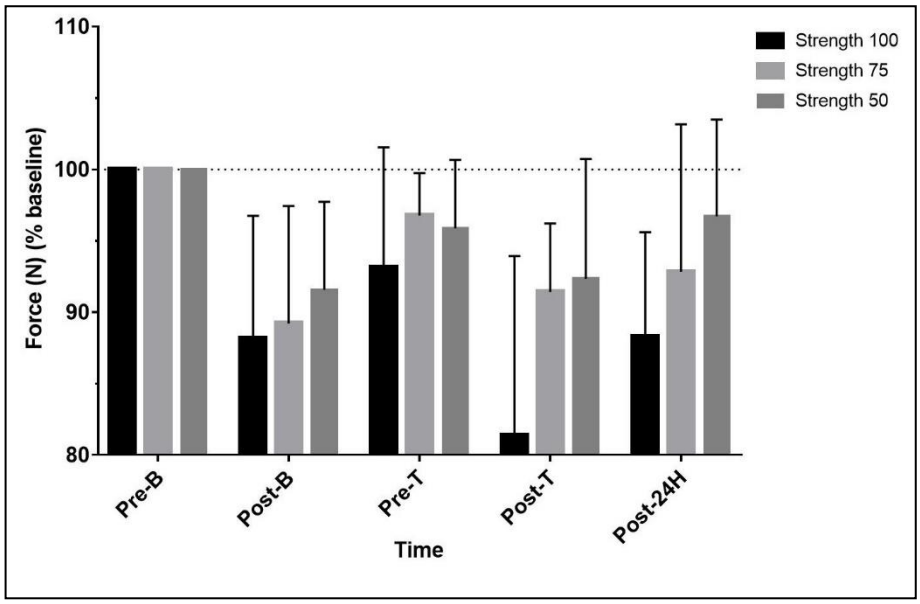


Figure 105. Comparison of S100, S75 and S50 on MVC peak force values (n = 11).

Simple main effects over time revealed that MVC peak force different significantly between time points in S100 ($P < 0.001$), S75 ($P = 0.013$) and S50 ($P < 0.001$) trial. In S100, significant time differences were observed at Post-B ($p = 0.017$, ES = -0.35), Post-T ($p = 0.012$, ES = -0.66), Post-24H ($p = 0.006$, ES = -0.39) except at Pre-T ($p = 0.226$, ES = -0.25) compared to Pre-B.

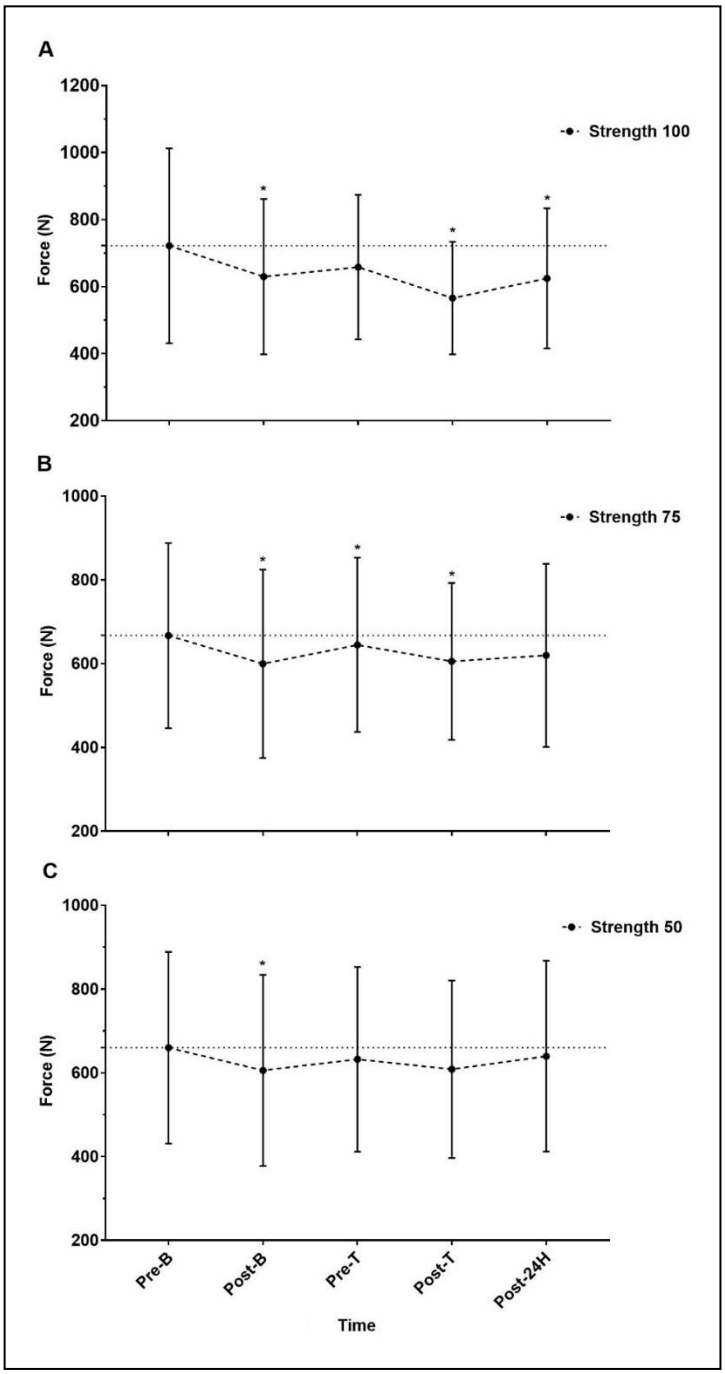


Figure 106. Changes in mean MVC peak force values in (A) S100, (B) S75 and (C) S50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In S75, significant time differences were observed at Post-B (p = 0.022, ES = -0.30), Pre-T (p = 0.052, ES = -0.10) and Post-T (p = 0.002, ES = -0.30) except at Post-24H (p = 0.514, ES = -0.22) compared to Pre-B. In S50, no significant time difference were observed at Pre-T (p = 0.185, ES = -0.12), Post-T (p = 0.184, ES = -0.23), Post-24H (p = 1.000, ES = -0.09) except at Post-B (p = 0.015, ES = -0.24) compared to Pre-B. These results revealed that peak force decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, peak force recovered to baseline (Pre-B) at Pre-T for S50 and remained recovered, whereas S75 needed longer time (Post-24H) to recover. Although, S100 was not yet recovered at Post-24H (Figure 106). According to the ES results, peak force of all 3 training loads were not yet recovered at Post-24H. Even though not fully recovered, S50 showed better recovery and S100 showed least recovery at Post-24H.

6.2.4.2.2. Rate of force development (0–200 ms) in maximal voluntary isometric contraction

There was no overall treatment effect on RFD^{200MVC} (p = 0.138). However, there was an overall time effect on RFD^{200MVC} (p = 0.006). No significant group x time interaction for RFD^{200MVC} was observed (p = 0.712; Figure 107).

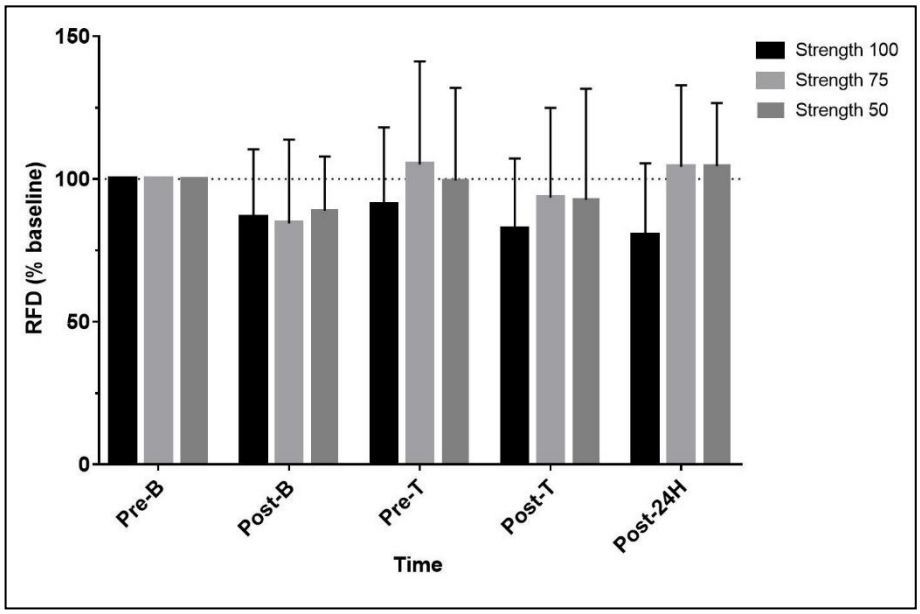


Figure 107. Comparison of S100, S75 and S50 on RFD^{200MVC} values (n = 11)

Simple main effects over time revealed that RFD^{200MVC} did not differ significantly between time points in S75 (P = 0.330) and S50 (P = 0.156) except at S100 (P = 0.052) trial. All 3 training loads ((S100: Post-B (p = 0.831, ES = -0.51), Pre-T (p = 1.000, ES = -0.22), Post-T (p = 0.164, ES = -0.72) and Post-24H (p = 0.110, ES = -0.50)), (S75: Post-B (p = 0.809, ES = -0.33), Pre-T (p = 1.000, ES = 0.01), Post-T (p = 1.000, ES = -0.23) and Post-24H (p = 1.000, ES = -0.05)) and (S50: Post-B (p = 0.504, ES = -0.39), Pre-T (p = 1.000, ES = -0.26), Post-T (p = 0.969, ES = -0.50) and Post-24H (p = 1.000, ES = -0.06))) showed no significant time differences between time points compared to respective Pre-B. Even though all 3 training loads showed no significant difference compared to their respected Pre-B values, RFD decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. According to the ES results, RFD of all 3 training loads were not yet recovered at Post-24H. Even though not fully recovered, S75 and S50 showed similar better recovery level than S100 at Post-24H (Figure 108).

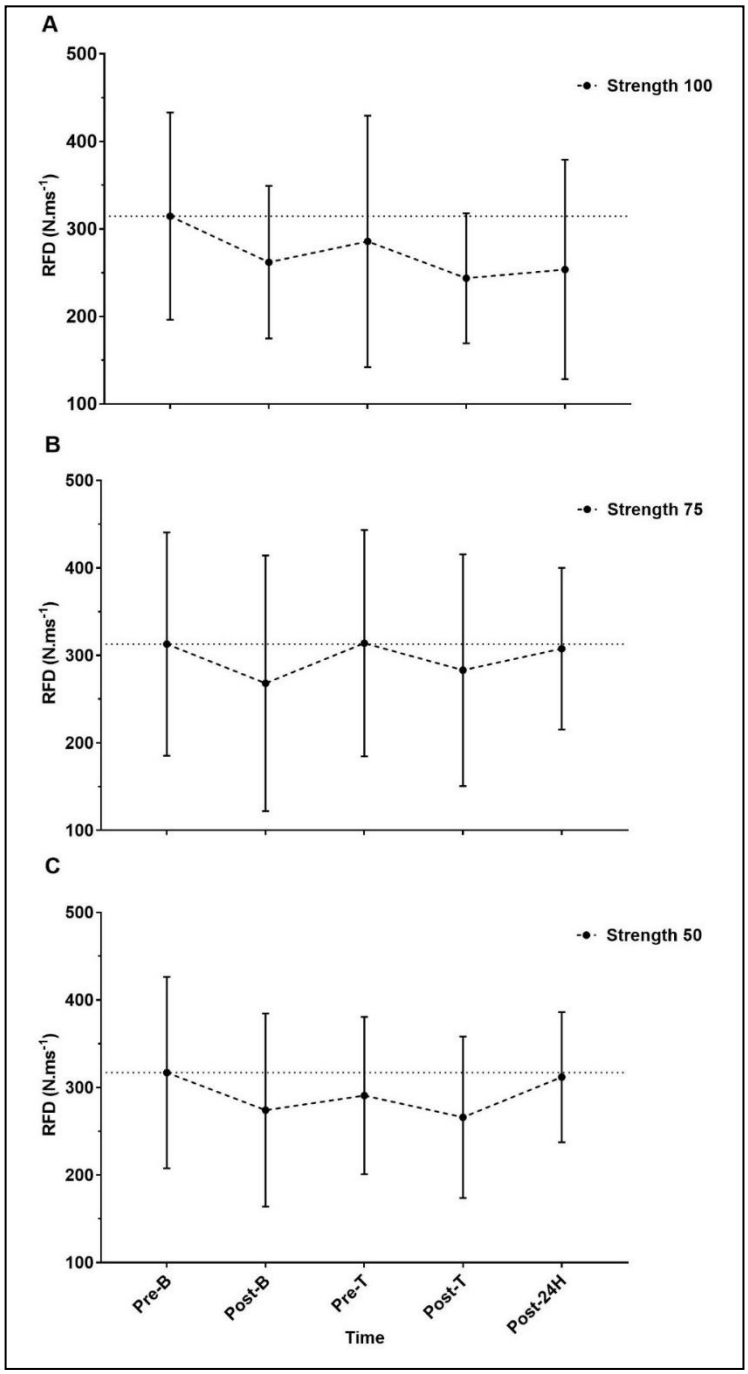


Figure 108. Changes in mean RFD^{200MVC} values in (A) S100, (B) S75 and (C) S50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.4.3. Power 100 versus Power 75 versus Power 50 training

The results from the neuromuscular fatigue indicating variables for the comparison between P100, P75 and P50 are reported below.

6.2.4.3.1. Maximal voluntary isometric contractions peak force

There was no overall treatment effect on MVC peak force ($p = 0.616$). However, there was an overall time effect on MVC peak force ($p < 0.001$) and significant group x time interaction for MVC peak force was observed ($p = 0.036$; Figure 109).

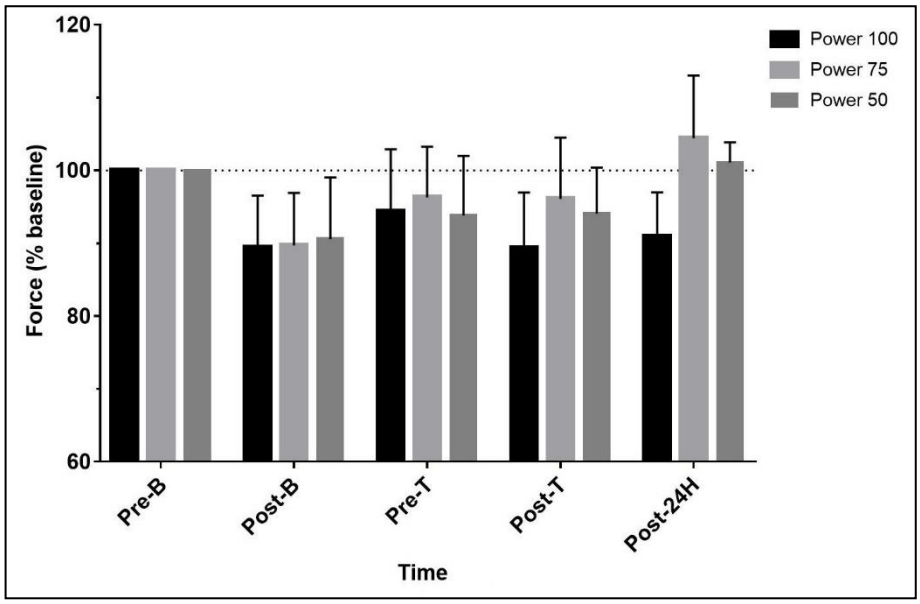


Figure 109. Comparison of P100, P75 and P50 on MVC peak force values (n = 08)

Simple main effects over time revealed that MVC peak force different significantly between time points in P100 ($P = 0.024$), P75 ($P = 0.001$) and P50 ($P = 0.015$) trial. In P100, significant time differences were observed at Post-B ($p = 0.044$, ES = -0.34) and Post-24H ($p = 0.028$, ES = -0.29) except at Pre-T ($p = 1.000$, ES = -0.25) and Post-T ($p = 0.153$, ES = -0.42) compared to Pre-B. In P75, no significant time differences were observed at all the time points (Post-B ($p = 0.116$, ES = -0.40), Pre-T ($p = 1.000$, ES = -0.15), Post-T ($p = 1.000$, ES = -0.16), Post-24H ($p = 1.000$, ES

= 0.13)) compared to Pre-B. Similarly, P50 also showed no significant time differences were observed at all the time points (Post-B (p = 0.153, ES = -0.34), Pre-T (p = 0.783, ES = -0.24), Post-T (p = 0.386, ES = -0.23) and Post-24H (p = 1.000, ES = 0.03)) compared to Pre-B.

These results revealed that peak force decreased following the M-Beast protocol in all 3 training loads and ARE protocols for P100 and P75 training loads, and it gradually returned to Pre-B values. Interestingly, P50 did not decrease the peak force following the ARE protocol. Interestingly, peak force remained recovered throughout the protocol of P75 and P50, whereas P100 was not yet recovered at Post-24H. According to the ES results, peak force of P75 and P50 were recovered at Post-24H, whereas P100's level did not recover at Post-24H (Figure 110).

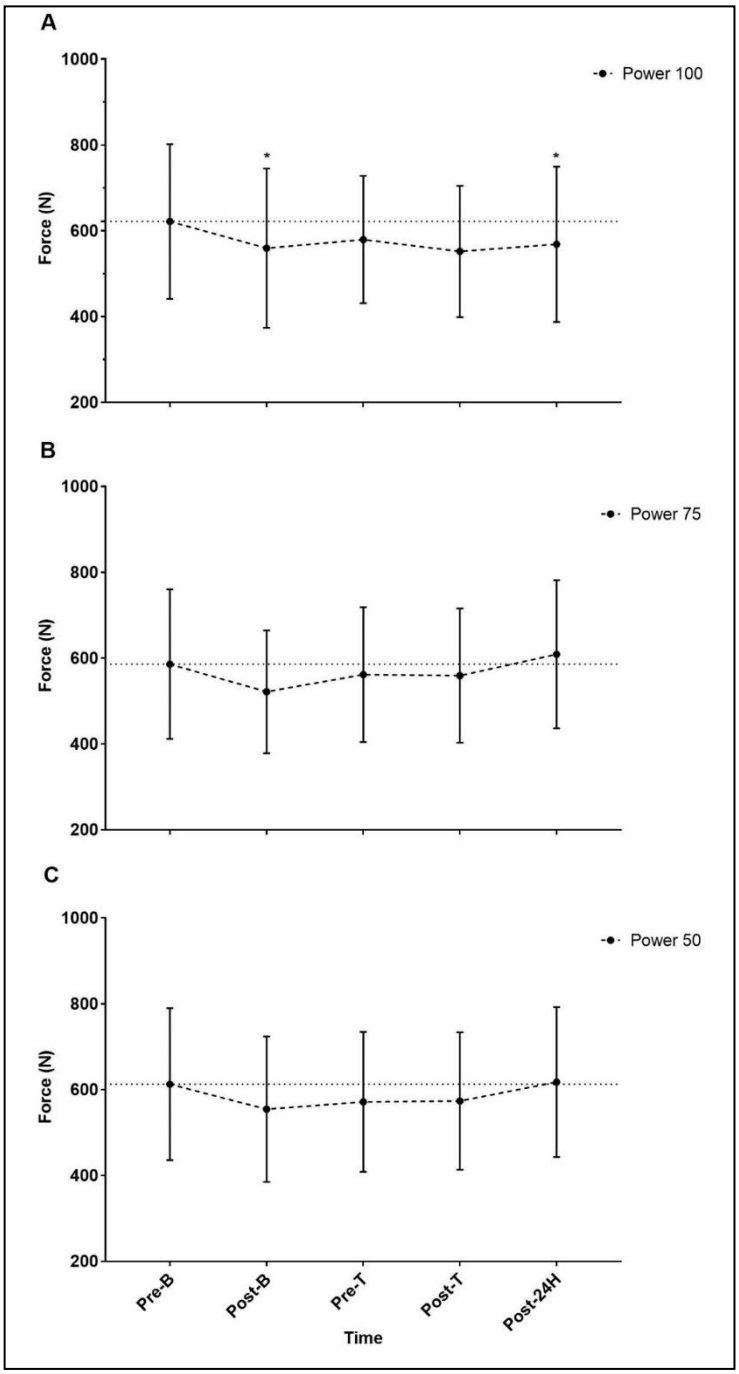


Figure 110. Changes in mean MVC peak force values in (A) P100, (B) P75 and (C) P50 protocols (n = 08). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.4.3.2. Rate of force development (0–200 ms) in maximal voluntary isometric contraction

There was no overall treatment effect on RFD^{200MVC} ($p = 0.888$). However, there was an overall time effect on RFD^{200MVC} ($p < 0.001$). No significant group x time interaction for RFD^{200MVC} was observed ($p = 0.449$; Figure 111).

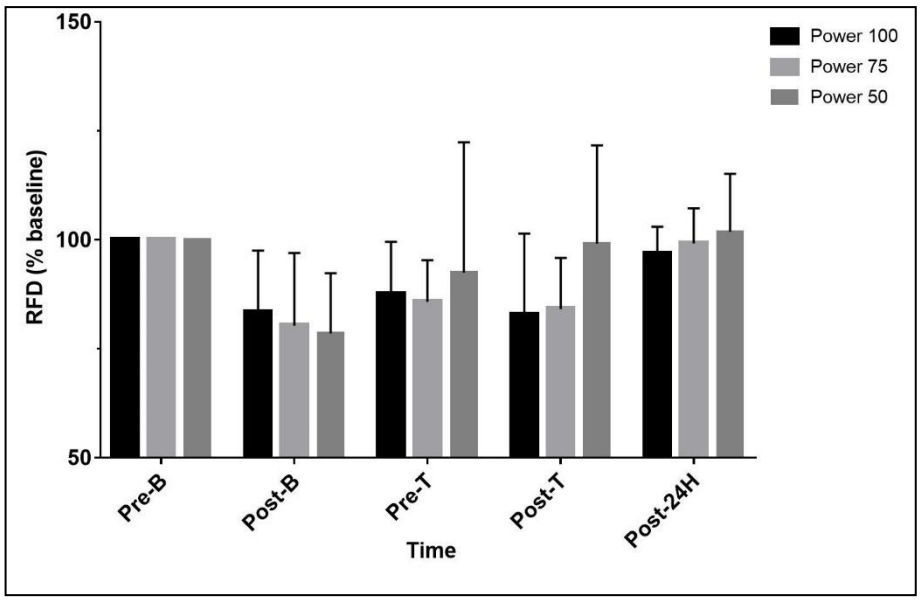


Figure 111. Comparison of P100, P75 and P50 on RFD^{200MVC} values (n = 08).

Simple main effects over time revealed that RFD^{200MVC} differed significantly between time points in P100 ($P = 0.043$) and P75 ($P = 0.001$) except in P50 ($P = 0.104$) trial. In P100, (Post-B ($p = 0.159$, ES = -0.58), Pre-T ($p = 0.285$, ES = -0.54), Post-T ($p = 0.393$, ES = -0.74) and Post-24H ($p = 1.000$, ES = -0.11)) and P75 trial (Post-B ($p = 0.238$, ES = -0.78), Pre-T ($p = 0.094$, ES = -0.41), Post-T ($p = 0.145$, ES = -0.50) and Post-24H ($p = 1.000$, ES = 0.00)), showed no significant difference at all the time points compared to Pre-B value. In P50, no significant time difference were observed at Pre-T ($p = 1.000$, ES = -0.20), Post-T ($p = 1.000$, ES = -0.04) and Post-24H ($p = 1.000$, ES = 0.11), except at Post-B ($p = 0.056$, ES = -0.68) compared to Pre-B value.

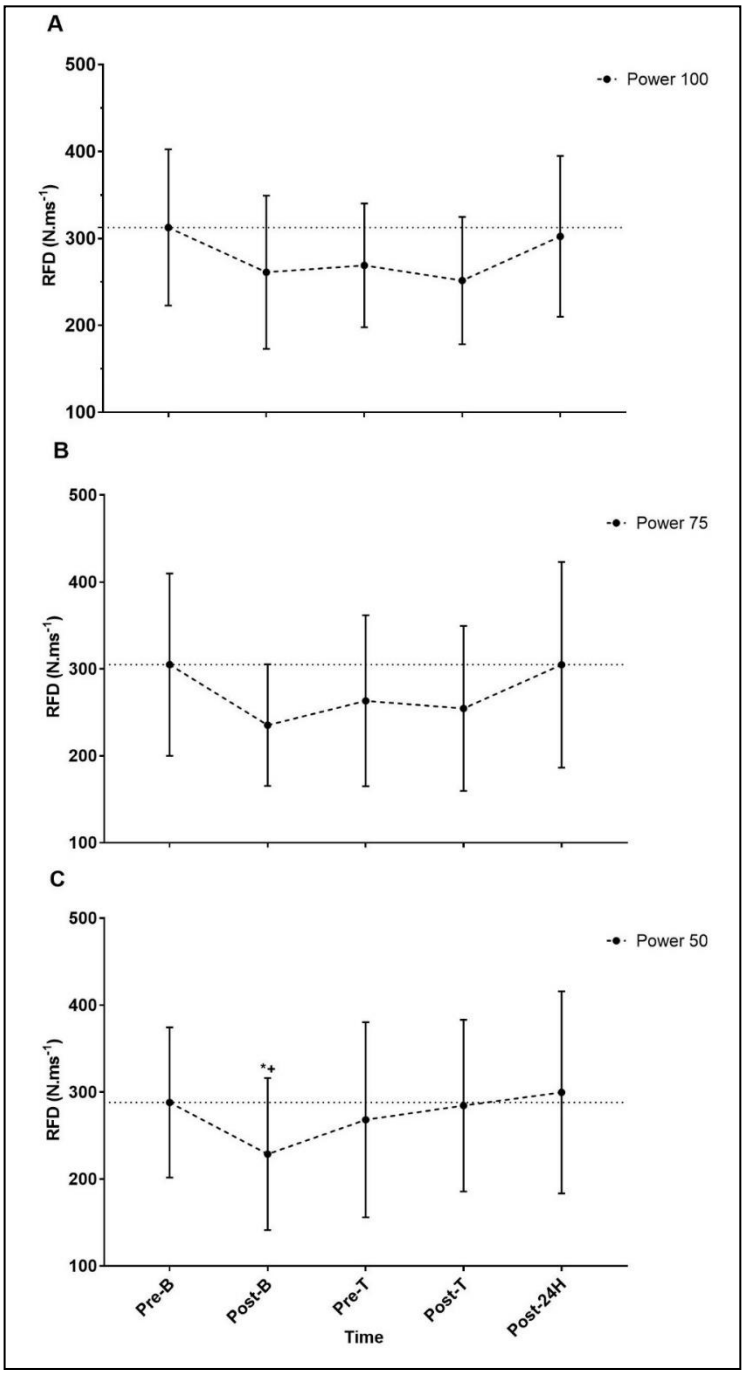


Figure 112. Changes in mean RFD^{200MVC} values in (A) P100, (B) P75 and (C) P50 protocols (n = 08). *+ Significant trend time difference compared to Pre-B ($p \leq 0.06$) from post-hoc Bonferroni analysis.

These results revealed that RFD decreased following the M-Beast protocol in all 3 training loads and ARE protocols for P100 and P75 training loads, and it gradually returned to Pre-B values. Interestingly, P50 did not decrease the RFD following the ARE protocol. According to the ES results, RFD of P75 and P50 recovered at Post-24H, whereas P100's level did not recover at Post-24H.Among the fully recovered, P50 showed better recovery level than P75 at Post-24H (Figure 112).

6.2.5. Central fatigue

6.2.5.1. Strength 100 versus Power 100 training

The results from the central fatigue indicating variables for the comparison between S100 and P100 are reported below.

6.2.5.1.1. Central activation ratio

There was no overall treatment effect on CAR ($p = 0.865$). However, there was an overall time effect on CAR ($p < 0.001$) and significant group x time interaction for CAR was observed ($p = 0.005$; Figure 113).

Simple main effects over time that CAR differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. Compared to Pre-B, significant time differences were observed at all the time points (Post-B :$P = 0.003$, ES = -2.54; Pre-T :$P = 0.002$, ES = -2.31; Post-T :$P = 0.014$, ES = -2.10 and Post-24 :$P = 0.026$, ES = -2.12) in S100 and Post-B ($P = 0.004$, ES = -2.55) and Post-T ($P = 0.009$, ES = -2.07) in P100. In P100, Pre-T ($P = 0.155$, ES = -1.34) and Post-24H ($P = 0.512$, ES = -1.03) showed no significant difference compared to their respective Pre-B.

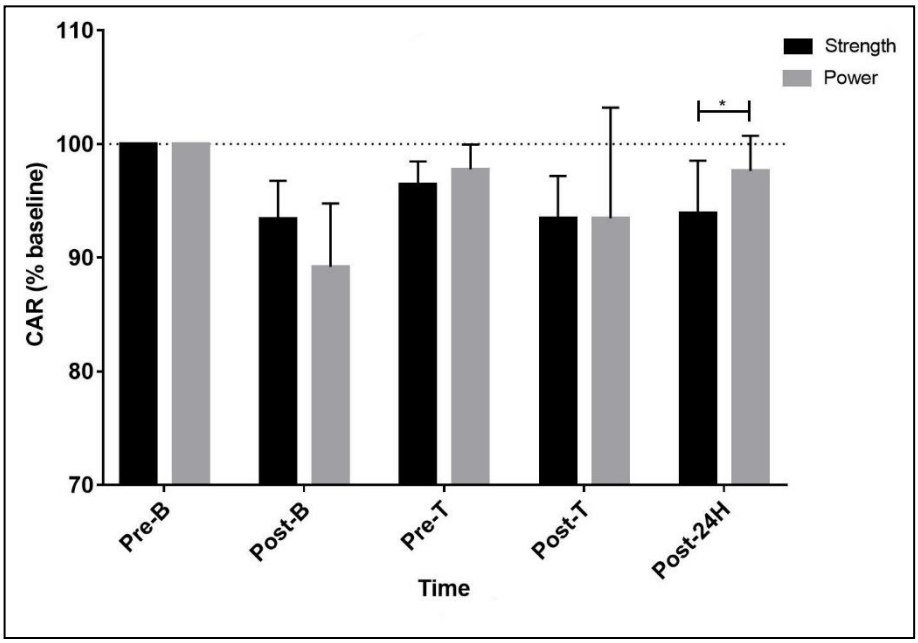


Figure 113. Comparison between S100 and P100 on CAR values (n = 09). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

These results revealed that CAR decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. Interestingly, CAR recovered to baseline (Pre-B) at Post-24H for P100, whereas S100 level did not recover at Post-24H. According to the ES results, CAR of both training modalities were not yet recovered at Post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H. (Figure 114).

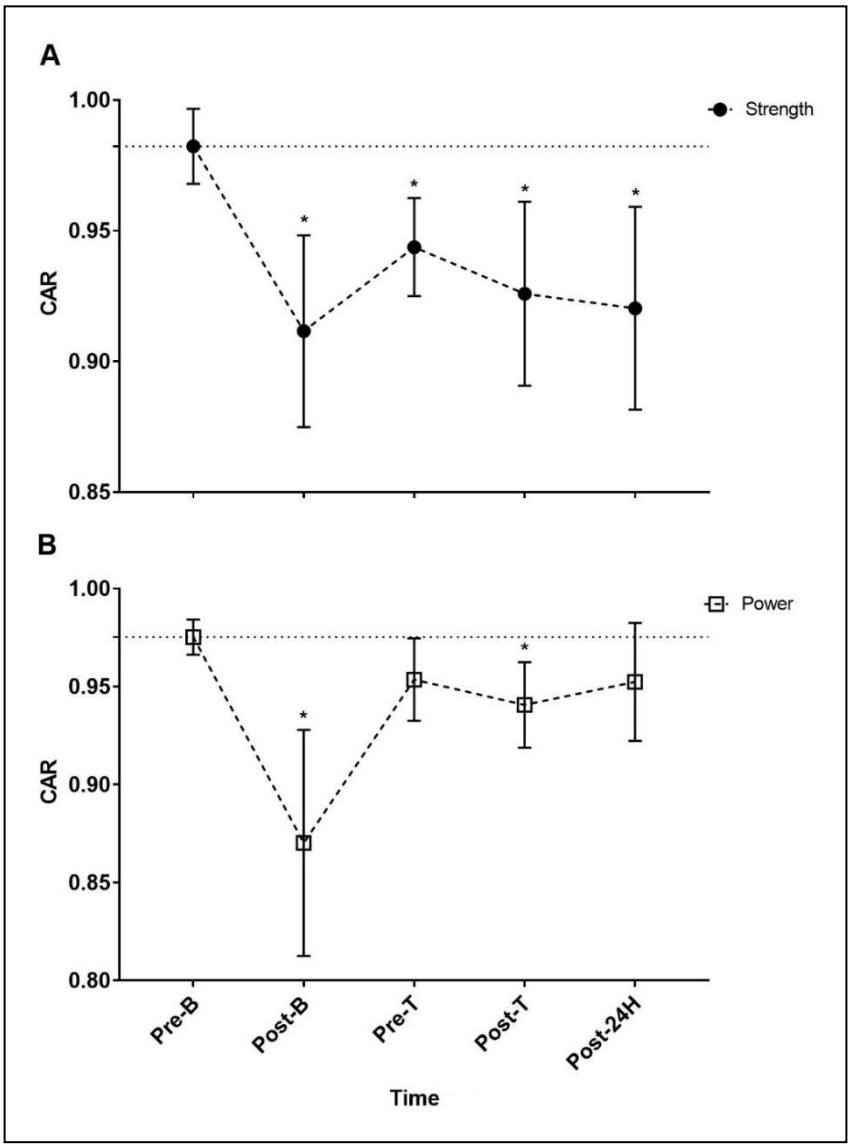


Figure 114. Changes in mean CAR values in (A) S100 and (B) P100 protocols (n = 09). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.5.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the central fatigue indicating variables for the comparison between S100, S75 and S50 are reported below

6.2.5.2.1. Central activation ratio

There was no overall treatment effect on CAR ($p = 0.276$). However, there was an overall time effect on CAR ($p < 0.001$) and significant group x time interaction for CAR was observed ($p = 0.002$). Simple main effects for treatment showed that CAR was significantly different between treatments (S100 vs S75 vs S50) at the Post-B ($p = 0.032$, (S100 vs S75: $p = 0.042$; S100 vs S50: $p = 1.000$)) of the trials (Figure 115)

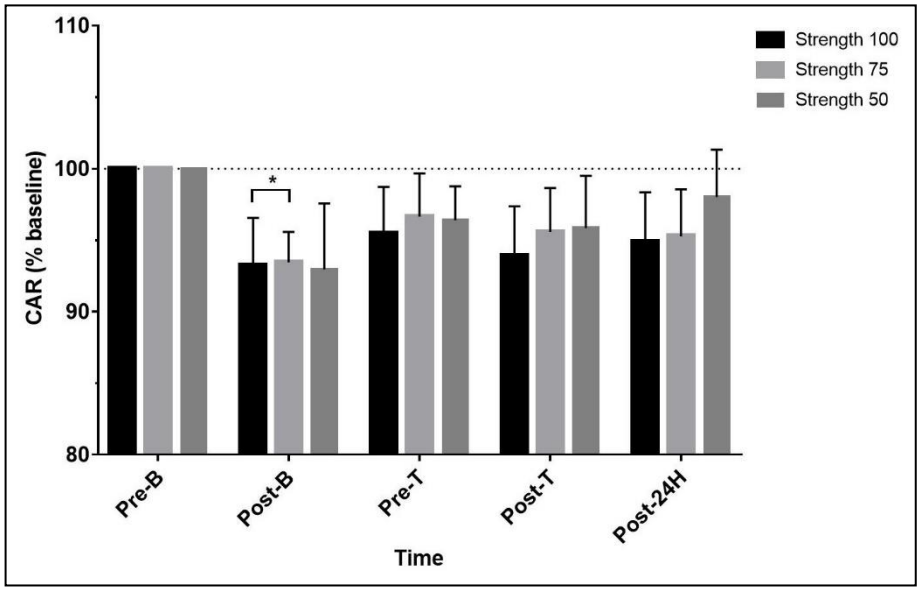


Figure 115. Comparison of S100, S75 and S50 on CAR values (n = 11). * Significant pairwise comparison differences in S75 compared to S100 ($p \leq 0.05$)

Simple main effects over time revealed that CAR differed significantly between time points in S100 ($P = 0.001$), S75 ($P = 0.018$) and S50 ($P < 0.001$) trial. In S100, significant time differences were observed at Post-B ($p = 0.003$, ES = -2.53) and Post-T ($p = 0.029$, ES = -2.24) except at Pre-T ($p = 0.114$, ES = -1.81) and Post-24H ($p = 0.904$, ES = -2.34) compared to Pre-B. In S75, no significant time differences were observed at Post-B ($p = 0.463$, ES = -4.18), Pre-T ($p = 1.000$, ES = -1.47) and Post-24H ($p = 1.000$, ES = -2.03) except at Post-T ($p = 0.053$, ES = -1.93) compared to Pre-B.

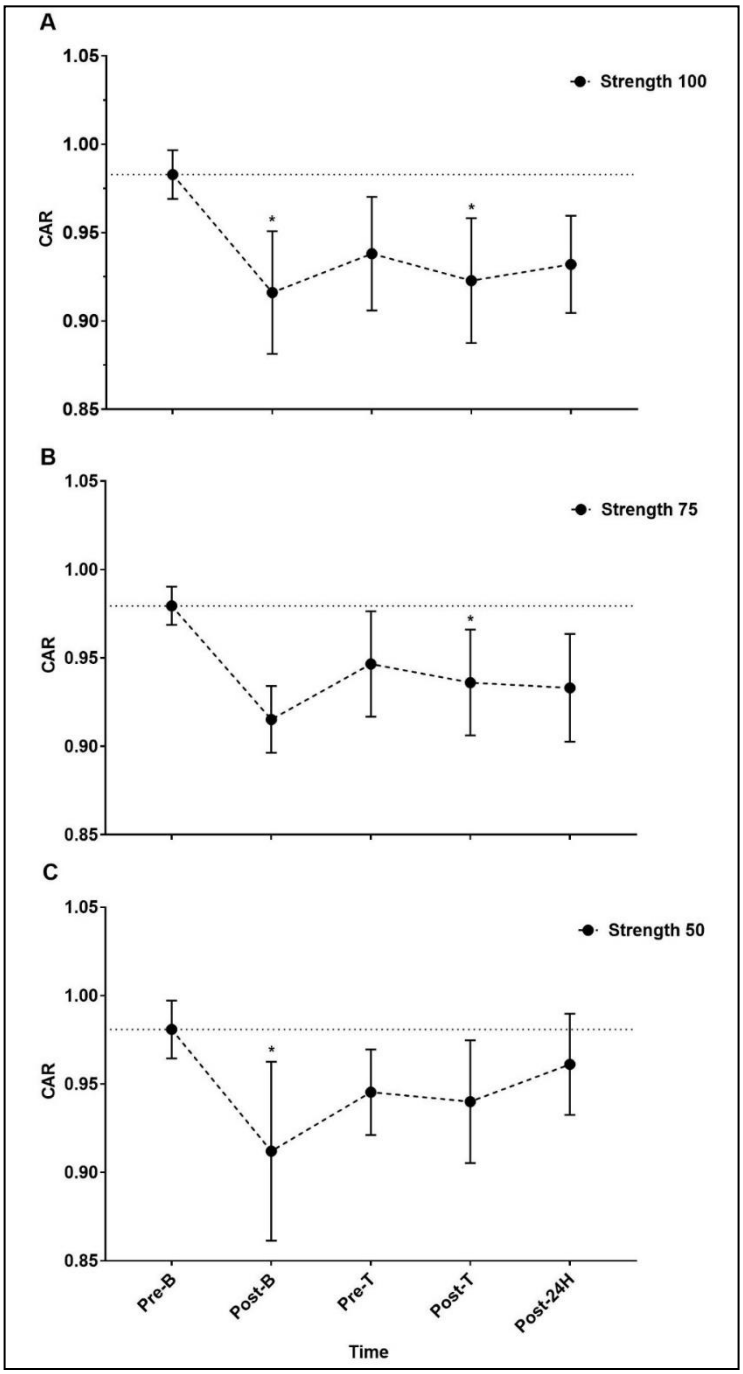


Figure 116. Changes in mean CAR values in (A) S100, (B) S75 and (C) S50 protocols (n = 11).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Similarly, S50 also showed no significant time differences at Pre-T (p = 0.412, ES = -1.72), Post-T (p = 1.000, ES = -1.50) and Post-24H (p = 1.000, ES = -0.85) except at Post-B (p = 0.007, ES = -1.83) compared to Pre-B. These results revealed that CAR decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, CAR remained recovered from the Pre-T for S50, whereas S75 and S100 needed longer time (Post-24H) to recover (Figure 116). According to the ES results, CAR of all 3 training loads were not yet recovered at Post-24H. Even though not fully recovered, S50 showed better recovery and S100 showed least recovery at Post-24H.

6.2.5.3. Power 100 versus Power 75 versus Power 50 training

The results from the central fatigue indicating variables for the comparison between P100, P75 and P50 are reported below

6.2.5.3.1. Central activation ratio

There was no overall treatment effect on CAR (p = 0.672). However, there was an overall time effect on CAR (p = 0.025). No significant group x time interaction for CAR was observed (p = 0.459; Figure 117).

Simple main effects over time revealed that CAR differed significantly between time points in P75 (P = 0.045) and P50 (P = 0.046), except in P100 (P = 0.145). In P100, no significant time differences were observed at all the time points (Post-B (p = 0.530, ES = -2.37), Pre-T (p = 1.000, ES = -1.34), Post-T (p = 1.000, ES = -0.93) and Post-24H (p = 1.000, ES = -0.96)) compared to Pre-B. Similarly, P75 also showed no significant time differences at all the time points (Post-B (p = 0.267, ES = -1.82), Pre-T (p = 0.181, ES = -1.17), Post-T (p = 0.470, ES = -2.47) and Post-24H (p = 0.621, ES = -0.56)) compared to Pre-B.

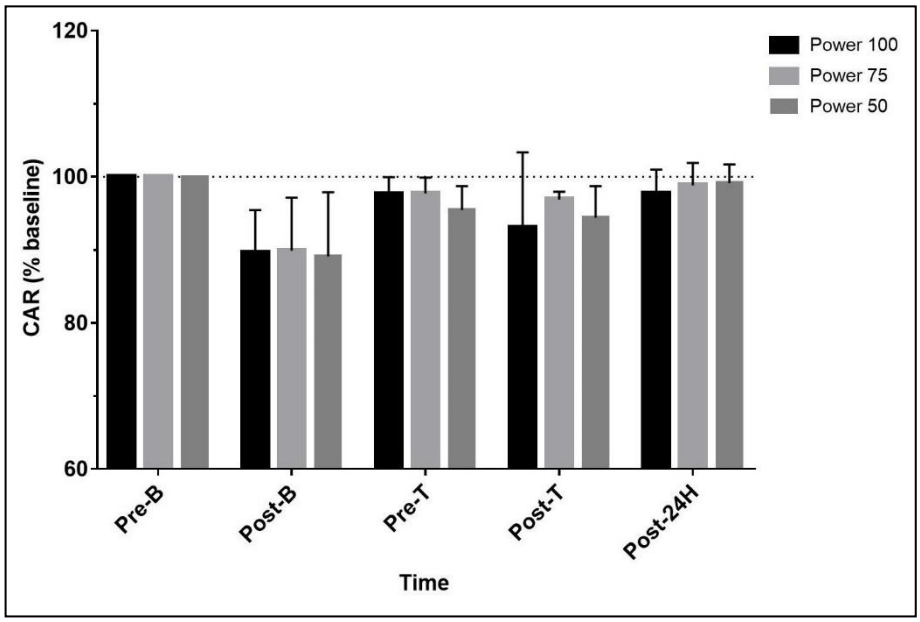


Figure 117. Comparison of P100, P75 and P50 on CAR values (n = 08).

In the same way, there was no significant time differences were observed at all the time points (Post-B (p = 0.443, ES = -1.71), Pre-T (p = 0.314, ES = -1.95), Post-T (p = 0.528, ES = -1.88) and Post-24H (p = 1.000, ES = -0.49) in P50, compared to Pre-B (Figure 119). These results revealed that CAR decreased following the M-Beast protocol and ARE protocols for all training loads, and it gradually returned to Pre-B values. Interestingly, all 3 training loads remained recovered throughout the protocols. According to the ES results, CAR of all 3 training loads were not yet recovered at Post-24H. Even though not fully recovered, P50 showed better recovery and P100 showed least recovery at Post-24H.

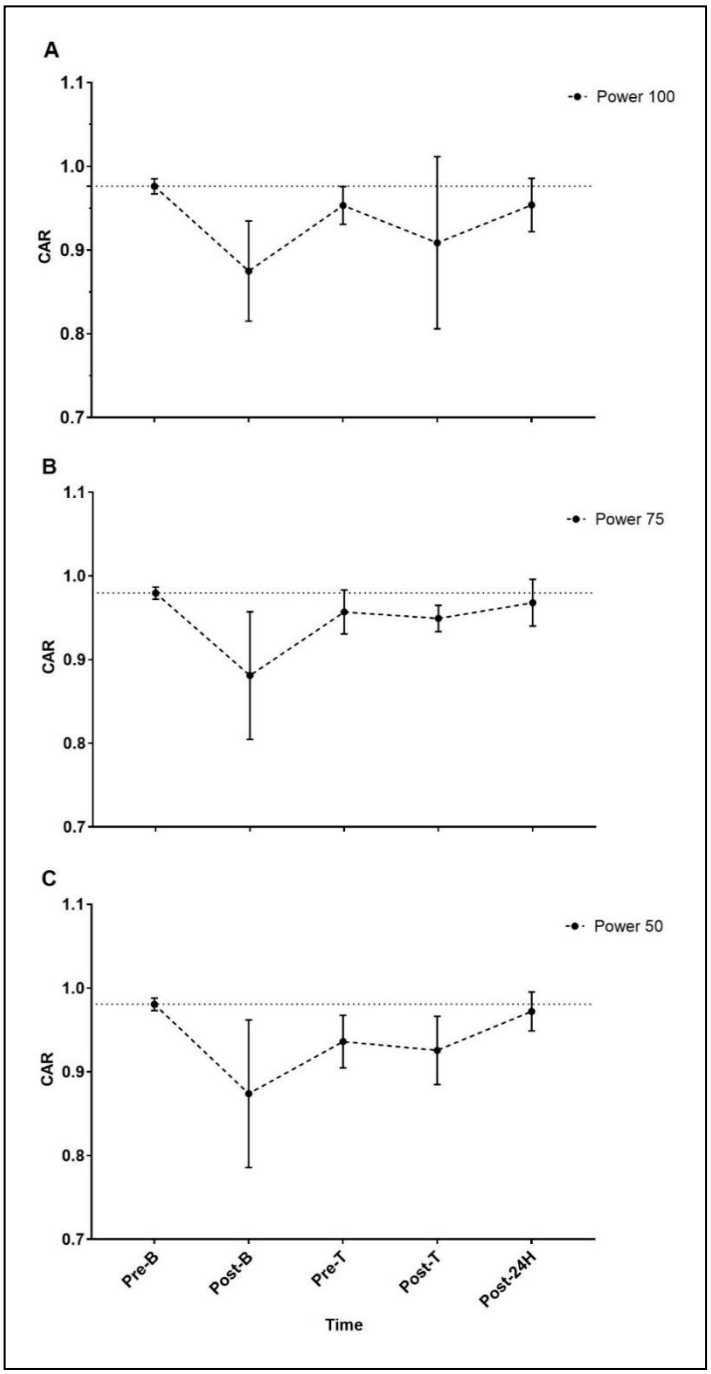


Figure 118. Changes in mean CAR values in (A) P100, (B) P75 and (C) P50 protocols (n = 08). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.6. Peripheral fatigue

6.2.6.1. Strength 100 versus Power 100 training

The results from the peripheral fatigue indicating variables for the comparison between S100 and P100 are reported below.

6.2.6.1.1. Tetanic force

There was neither an overall treatment effect ($p = 0.432$) nor an overall time effect ($p = 0.594$) on tetanic force. No significant group x time interaction for tetanic force was observed ($p = 0.599$; Figure 119).

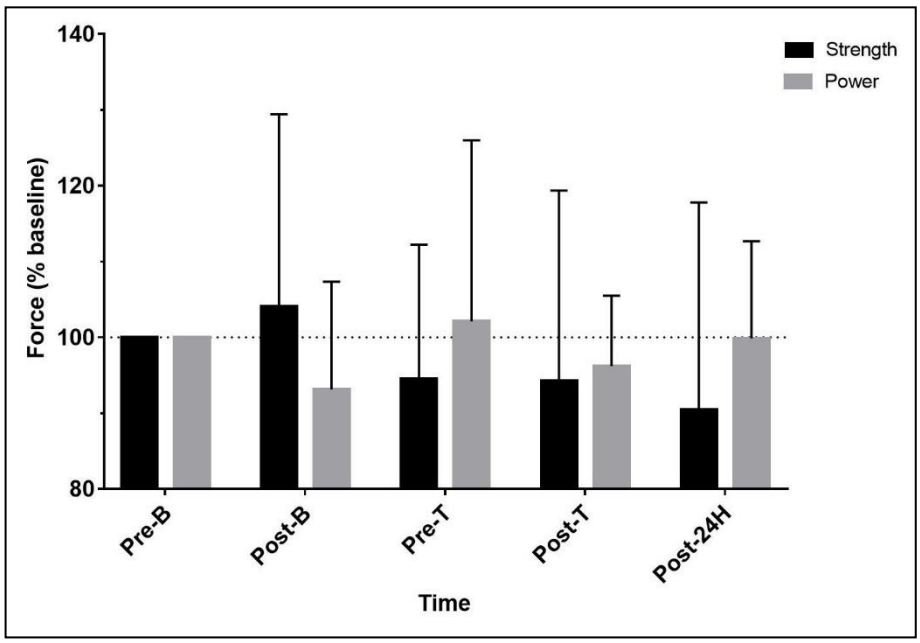


Figure 119. Comparison between S100 and P100 on Tetanic force values (n = 09)

6.2.6.1.2. Maximum rate of force development on Tetanic contraction

There was neither an overall treatment effect ($p = 0.628$) nor an overall time effect ($p = 0.161$) on RFD^{tet}. No significant group x time interaction for RFD^{tet} was observed ($p = 0.999$; Figure 120).

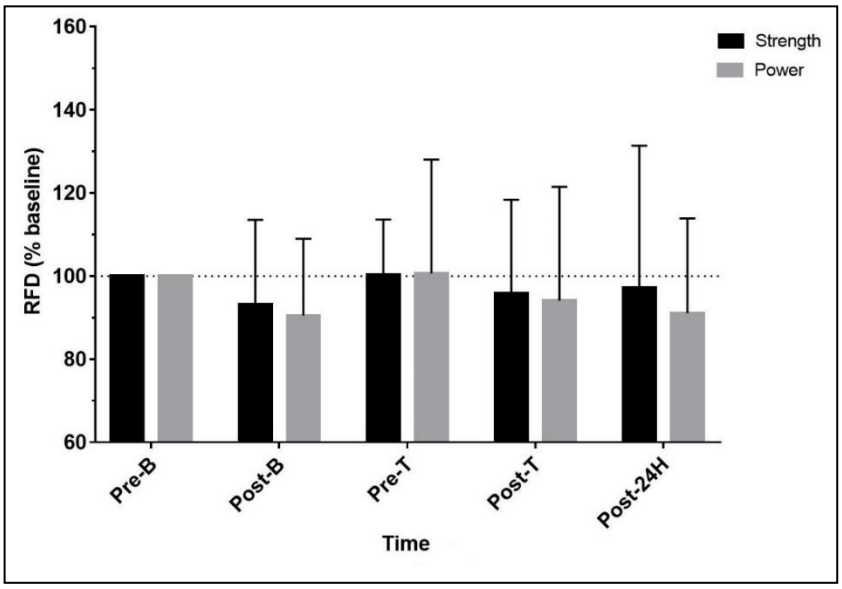


Figure 120. Comparison between S100 and P100 on RFD^{tet} values (n = 09).

6.2.6.1.3. Maximum rate of force relaxation on Tetanic contraction

There was neither an overall treatment effect (p = 0.772) nor an overall time effect (p = 0.974) on RFR^{tet}. No significant group x time interaction for RFR^{tet} was observed (p = 0.496; Figure 121).

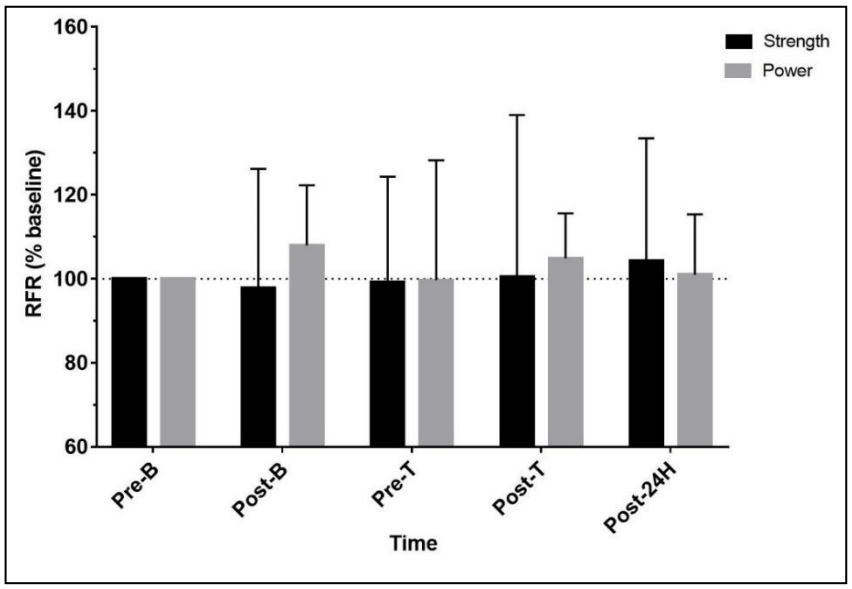


Figure 121. Comparison between S100 and P100 on RFR^{tet} values (n = 09)

6.2.6.1.4. Twitch force

There was neither an overall treatment effect (p = 0.251) nor an overall time effect (p = 0.525) on twitch force. No significant group x time interaction for twitch force was observed (p = 0.888; Figure 122).

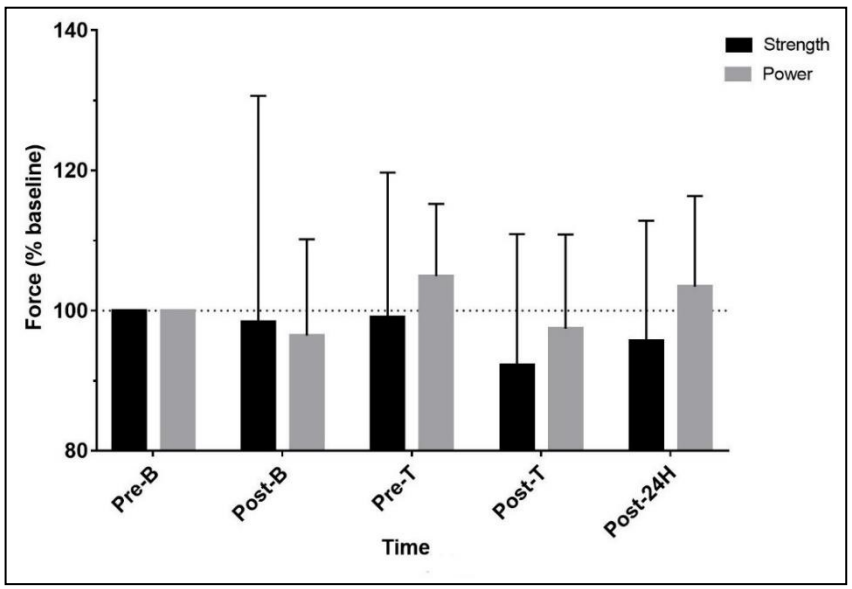


Figure 122. Comparison between S100 and P100 on twitch force values (n = 09)

6.2.6.1.5. $T_{1/2}$

There was neither an overall treatment effect (p = 0.634) nor an overall time effect (p = 0.141) on half-time of force relaxation. No significant group x time interaction for half-time of force relaxation was observed (p = 0.541; Figure 123).

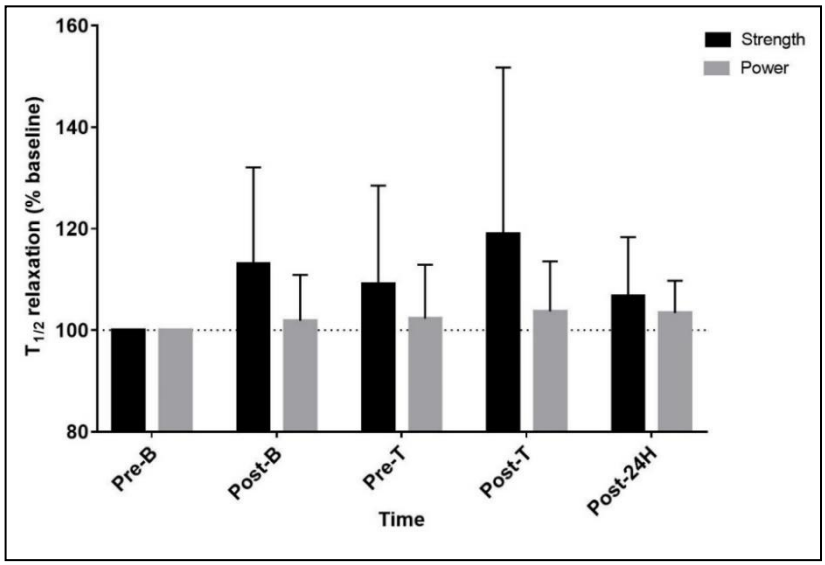


Figure 123. Comparison between S100 and P100 on half-time of force relaxation values (n = 09).

6.2.6.1.6. Twitch-to-tetanic ratio

There was neither an overall treatment effect (p = 0.767) nor an overall time effect (p = 0.194) on twitch/tetanic ratio. No significant group x time interaction for twitch/tetanic ratio was observed (p = 0.740; Figure 124).

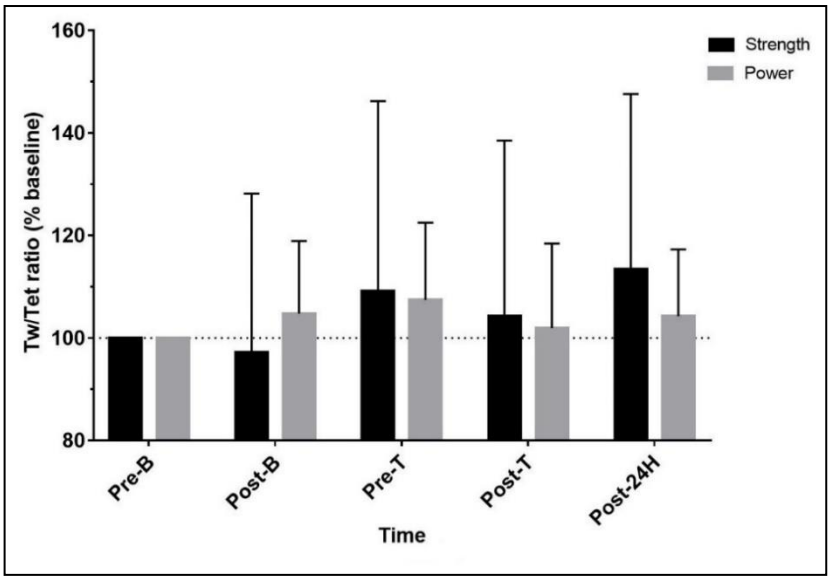


Figure 124. Comparison between S100 and P100 on twitch-to-tetanus ratios values (n = 09)

6.2.6.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the peripheral fatigue indicating variables for the comparison between S100, S75 and S50 are reported below

6.2.6.2.1. Tetanic force

There was no overall treatment effect on tetanic force (p = 0.236). However, there was an overall time effect on tetanic force (p = 0.046). No significant group x time interaction for tetanic force was observed (p = 0.191; Figure 125), where simple main effects over time revealed that there was no significant different between time points in S100 (P = 0.075), S75 (P = 0.232) and S50 (P = 0.170).

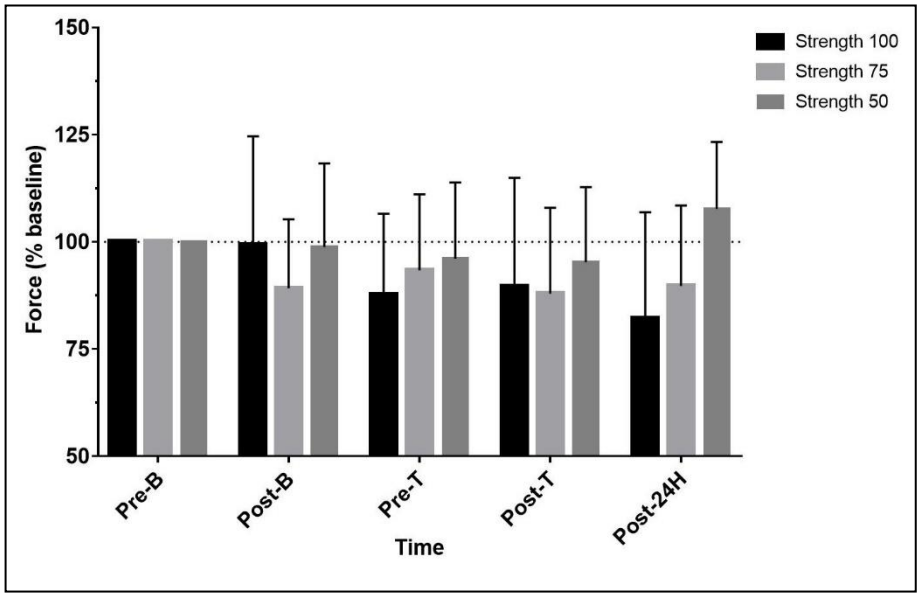


Figure 125. Comparison of S100, S75 and S50 on tetanic force values (n = 11).

6.2.6.2.2. Maximum rate of force development on tetanic contraction

There was neither an overall treatment effect (p = 0.298) nor an overall time effect (p = 0.261) on RFD^{tet}. No significant group x time interaction for RFD^{tet} was observed (p = 0.078; Figure 126).

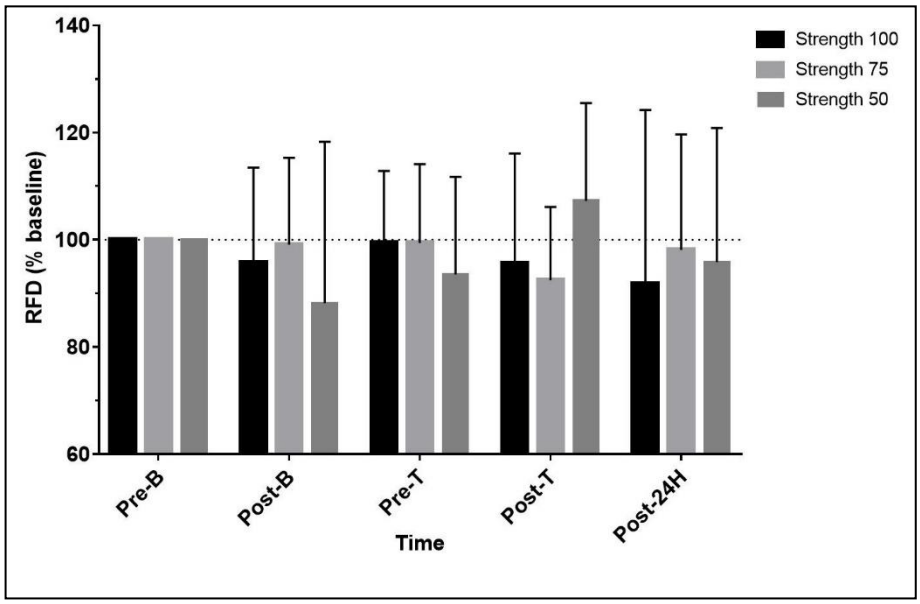


Figure 126. Comparison of S100, S75 and S50 on RFD^{tet} values (n = 11).

6.2.6.2.3. Maximum rate of force relaxation on Tetanic contraction

There was neither an overall treatment effect (p = 0.406) nor an overall time effect (p = 0.566) on RFR^{tet}. No significant group x time interaction for RFR^{tet} was observed (p = 0.815; Figure 127).

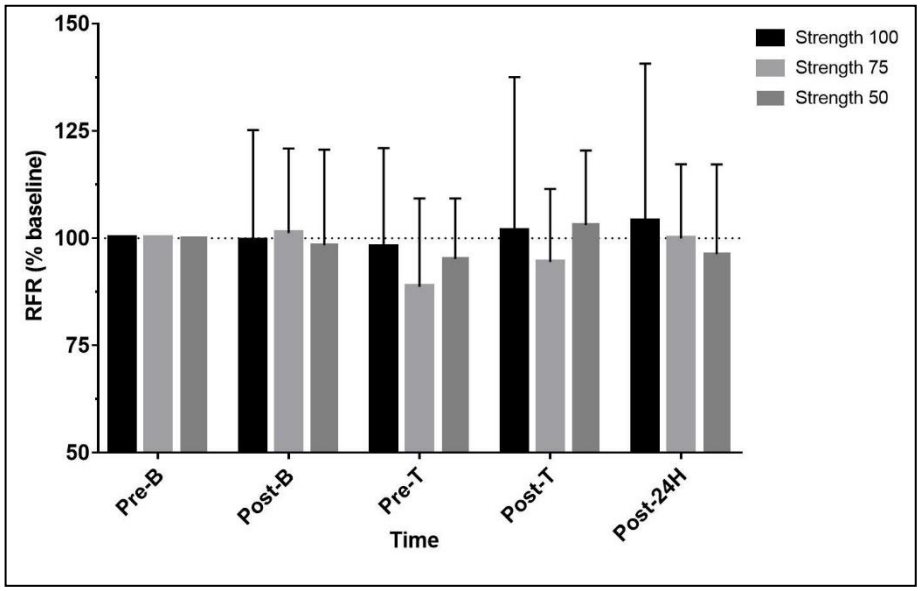


Figure 127. Comparison of S100, S75 and S50 on RFR^{tet} values (n = 11)

6.2.6.2.4. Twitch force

There was neither an overall treatment effect (p = 0.578) nor an overall time effect (p = 0.171) on twitch force. No significant group x time interaction for twitch force was observed (p = 0.854; Figure 128)

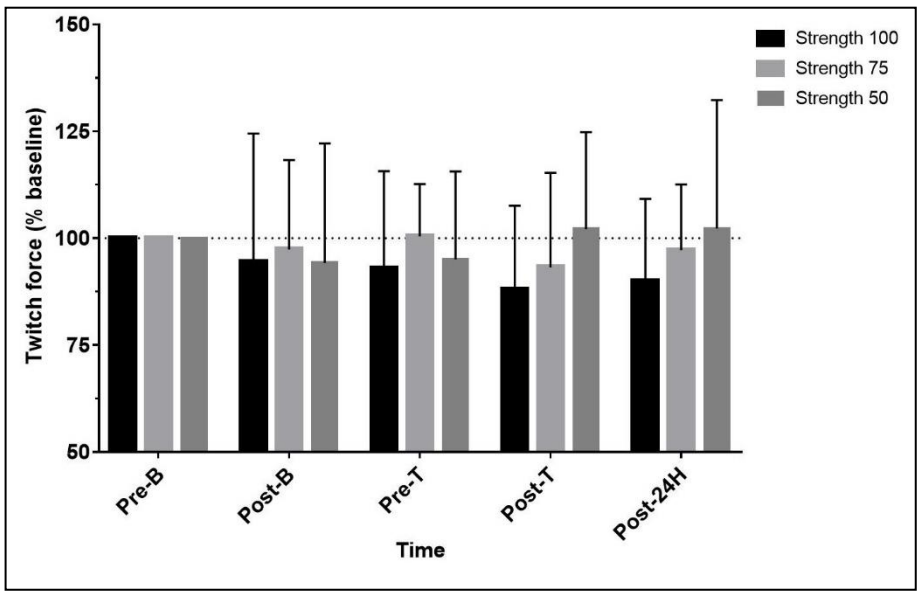


Figure 128. Comparison of S100, S75 and S50 on twitch force values (n = 11).

6.2.6.2.5. $T_{1/2}$

There was no overall treatment effect on half-time of force relaxation (p = 0.843). However, there was an overall time effect on half-time of force relaxation (p = 0.006). No significant group x time interaction for half-time of force relaxation was observed (p = 0.270; Figure 129). Simple main effects over time revealed that there was a trend towards significance between time points in S50 (P = 0.064) trial and no significant differences were showed in S100 (P = 0.279) and S75 (P = 0.381) between time points.

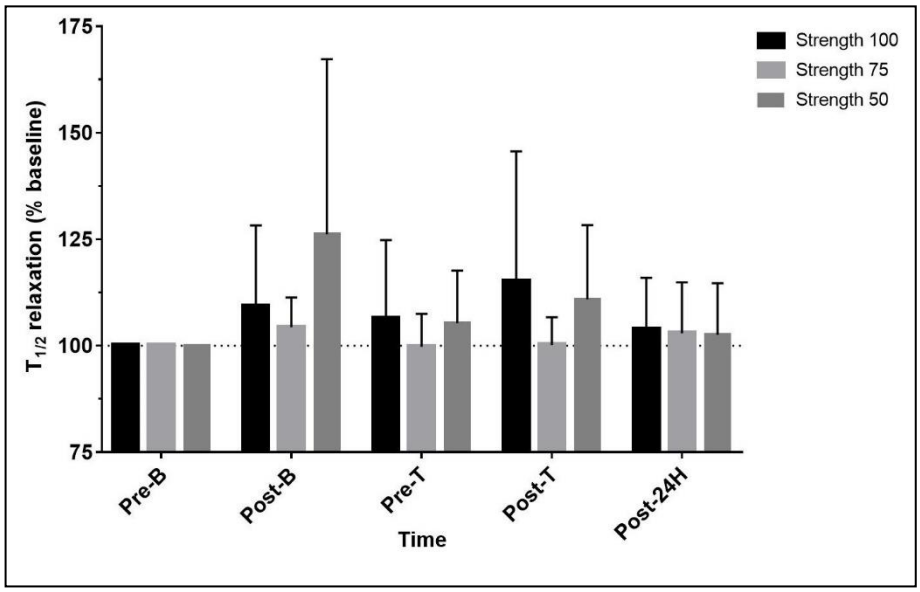


Figure 129. Comparison of S100, S75 and S50 on $T_{1/2}$ values (n = 11).

6.2.6.2.6. Twitch-to-tetanus ratios

There was neither an overall treatment effect (p = 0.564) nor an overall time effect (p = 0.567) on twitch-to-tetanus ratios. No significant group x time interaction for twitch-to-tetanus ratios was observed (p = 0.310; Figure 130)

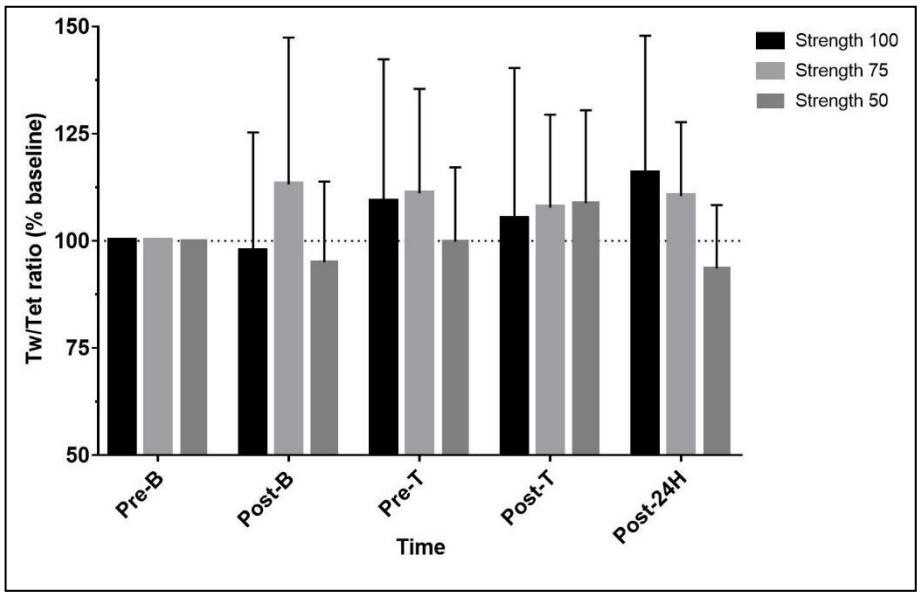


Figure 130. Comparison of S100, S75 and S50 on Twitch/Tetanus ratio values (n = 11)

6.2.6.3. Power 100 versus Power 75 versus Power 50 training

The results from the peripheral fatigue variables for the comparison between P100, P75 and P50 are reported below.

6.2.6.3.1. Tetanic force

There was an overall treatment effect on tetanic force (p = 0.035). However, there was no overall time effect on tetanic force (p = 0.244). No significant group x time interaction for tetanic force was observed (p = 0.952; Figure 131). Simple main effects over time revealed that there was no significant different between time points in P100 (P = 0.625), P75 (P = 0.590) and P50 (P = 0.299).

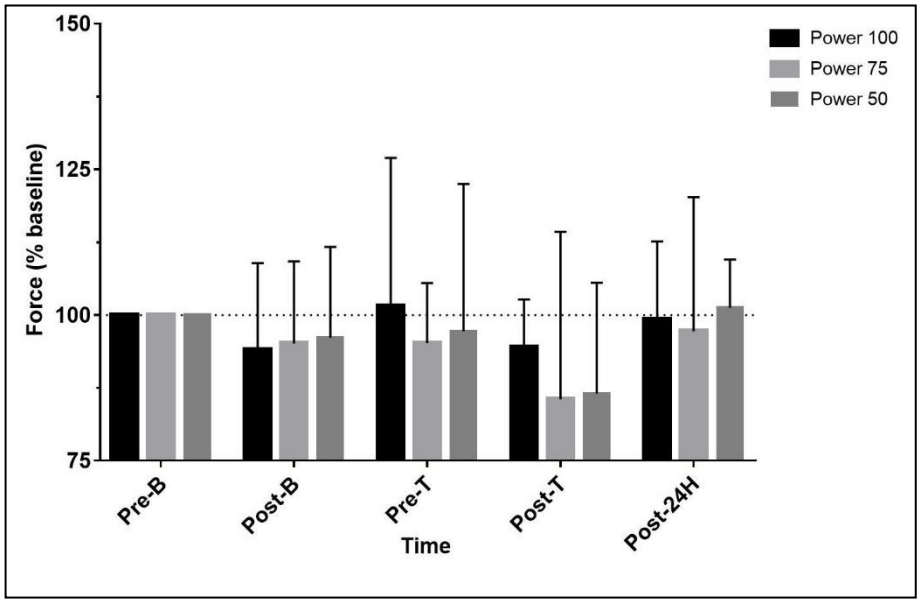


Figure 131. Comparison of P100, P75 and P50 on tetanic force values (n = 08).

6.2.6.3.2. Maximum rate of force development on Tetanic contraction

There was neither an overall treatment effect (p = 0.115) nor an overall time effect (p = 0.332) on RFD^{tet}. No significant group x time interaction for RFD^{tet} was observed (p = 0.697; Figure 132).

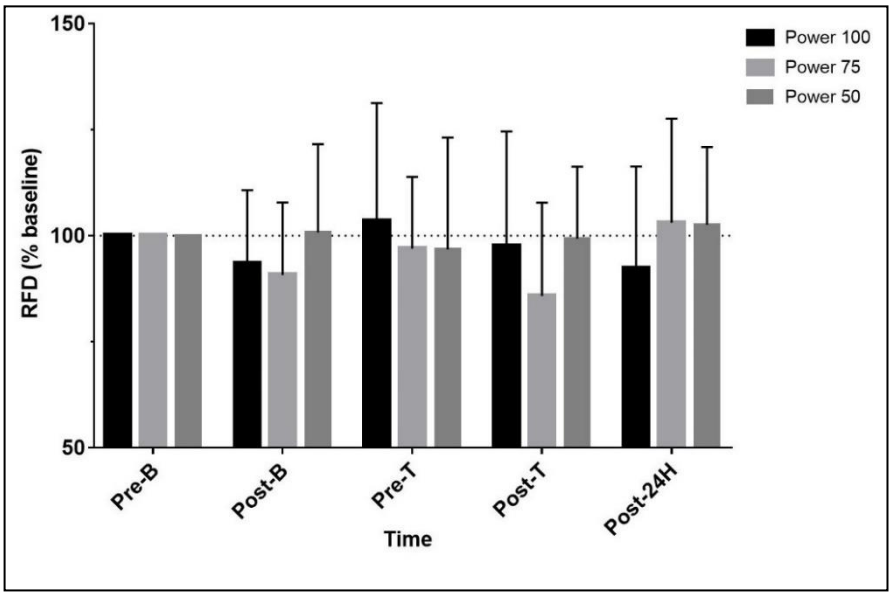


Figure 132. Comparison of P100, P75 and P50 on RFD^{tet} values (n = 08)

6.2.6.3.3. Maximum rate of force relaxation on Tetanic contraction

There was neither an overall treatment effect (p = 0.289) nor an overall time effect (p = 0.670) on RFR^{tet}. No significant group x time interaction for RFR^{tet} was observed (p = 0.914; Figure 133).

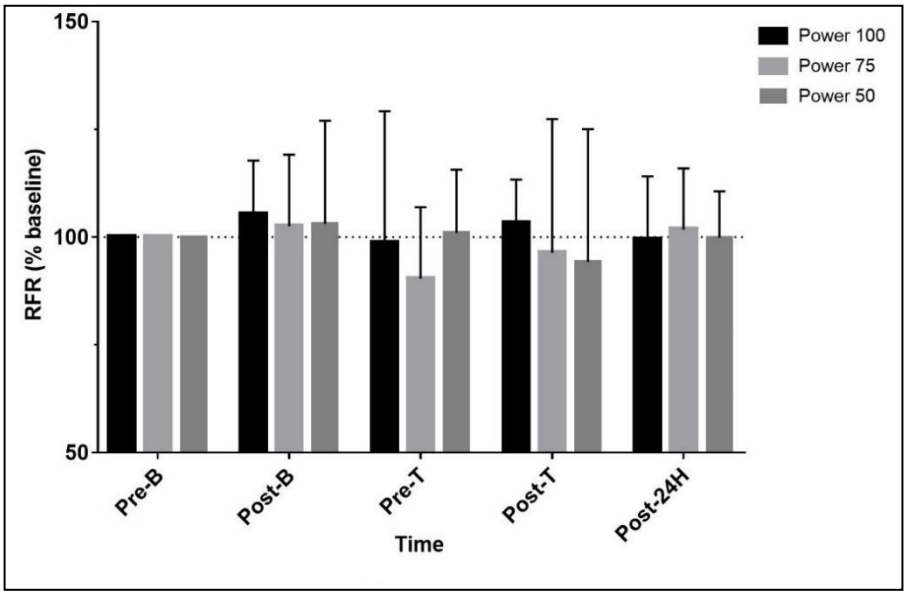


Figure 133. Comparison of P100, P75 and P50 on RFR^{tet} values (n = 08).

6.2.6.3.4. Twitch force

There was neither an overall treatment effect (p = 0.763) nor an overall time effect (p = 0.180) on twitch force. No significant group x time interaction for twitch force was observed (p = 0.478; Figure 134).

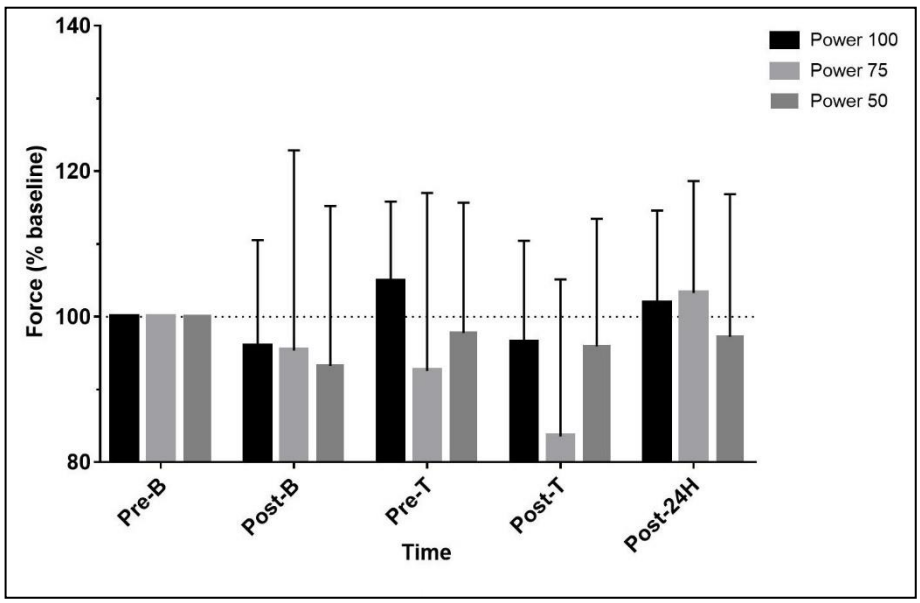


Figure 134. Comparison of P100, P75 and P50 on twitch force values (n = 08).

6.2.6.3.5. $T_{1/2}$

There was neither an overall treatment effect (p = 0.735) nor an overall time effect (p = 0.933) on half-time of force relaxation. No significant group x time interaction for half-time of force relaxation was observed (p = 0.228; Figure 135).

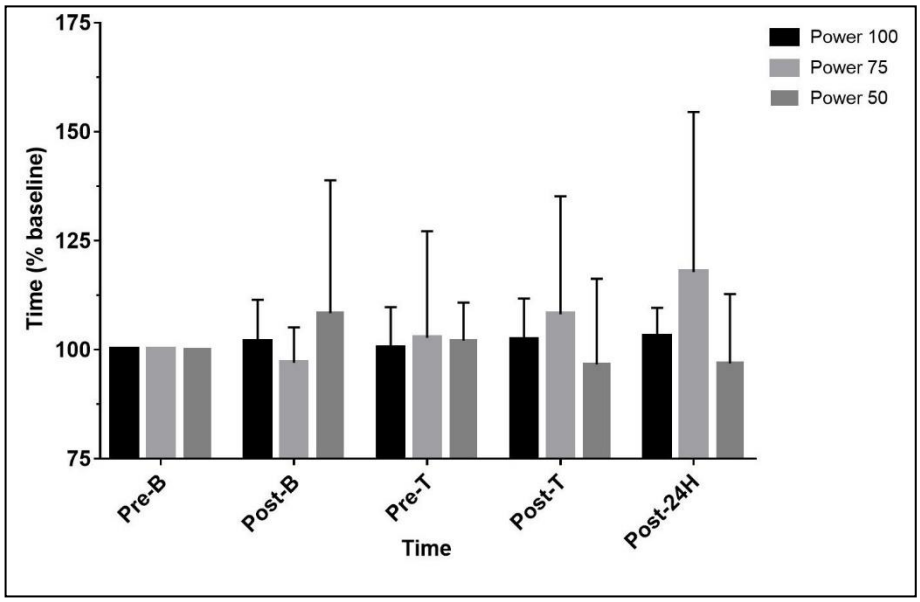


Figure 135. Comparison of P100, P75 and P50 on $T_{1/2}$ values (n = 08).

6.2.6.3.6. Twitch-to-tetanus ratio

There was an overall treatment effect on twitch-to-tetanus ratio (p = 0.014). However, there was an overall time effect on twitch-to-tetanus ratio (p = 0.777). No significant group x time interaction for twitch-to-tetanus ratio was observed (p = 0.391; Figure 136).

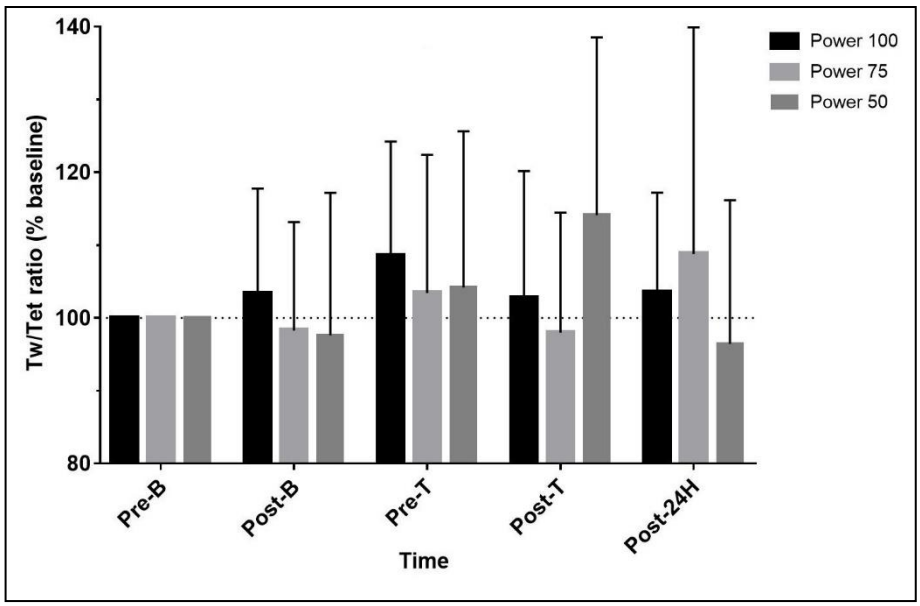


Figure 136. Comparison of P100, P75 and P50 on twitch/tetanic ratio values (n = 08).

6.2.7. Perceptual Responses

6.2.7.1. Strength 100 versus Power 100 training

The results from the perceptual response variables for the comparison between S100 and P100 are reported below.

6.2.7.1.1. Muscle pain (DOMS)

There was no overall treatment effect on DOMS ($p = 0.092$). However, there was an overall time effect on DOMS ($p < 0.001$). No significant group x time interaction for DOMS was observed ($p = 0.357$; Figure 137).

Simple main effects over time revealed that DOMS differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trial. In S100, significant time differences were observed at all the time points (Post-B ($p < 0.001$, ES = 3.68), Pre-T ($p < 0.001$, ES = 3.78), Post-T ($p < 0.001$, ES = 4.61), Post-6H ($p < 0.001$, ES = 6.14) and Post-24H ($p < 0.001$, ES = 10.05)) compared to Pre-B.

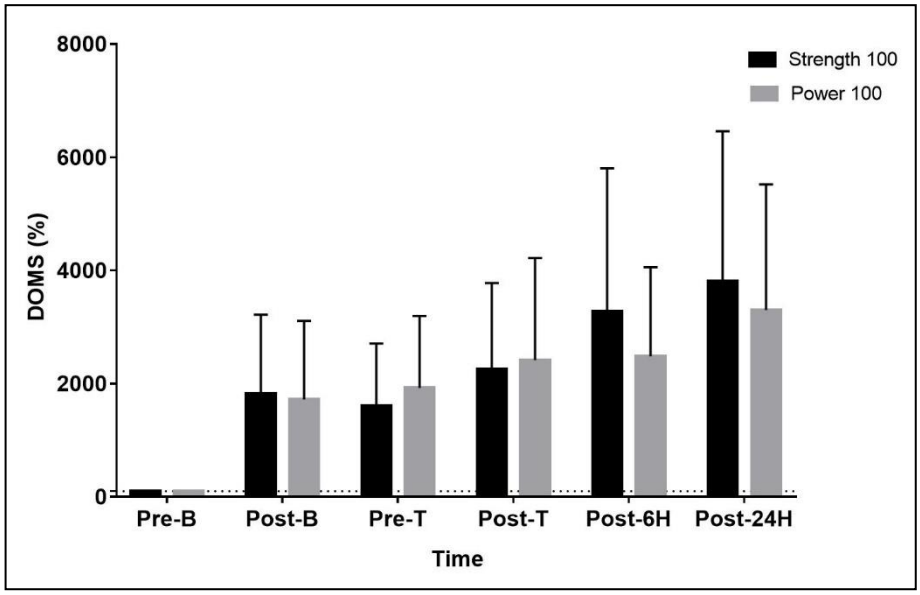


Figure 137. Comparison between S100 and P100 on mean DOMS values (n = 11).

Similarly, P100 also showed significant time differences at all the time points (Post-B ($p < 0.001$, ES = 4.63), Pre-T ($p < 0.001$, ES = 4.12), Post-T ($p < 0.001$, ES = 3.74), Post-6H ($p < 0.001$, ES = 5.32) and Post-24H ($p < 0.001$, ES = 5.35)) compared to Pre-B (Figure 138). These results indicate that sensation of muscle pain was gradually increased following the M-Beast protocol and ARE protocols and remained significantly higher at Post-24H on both training modalities suggesting that sensation of muscle pain was not recovered to baseline (Pre-B) at Post-24H. Similarly, ES results also revealed that sensation of muscle pain was presented and did not yet recovered to Pre-B at Post-24H. Even though not fully recovered, P100 showed better recovery level than S100 at Post-24H.

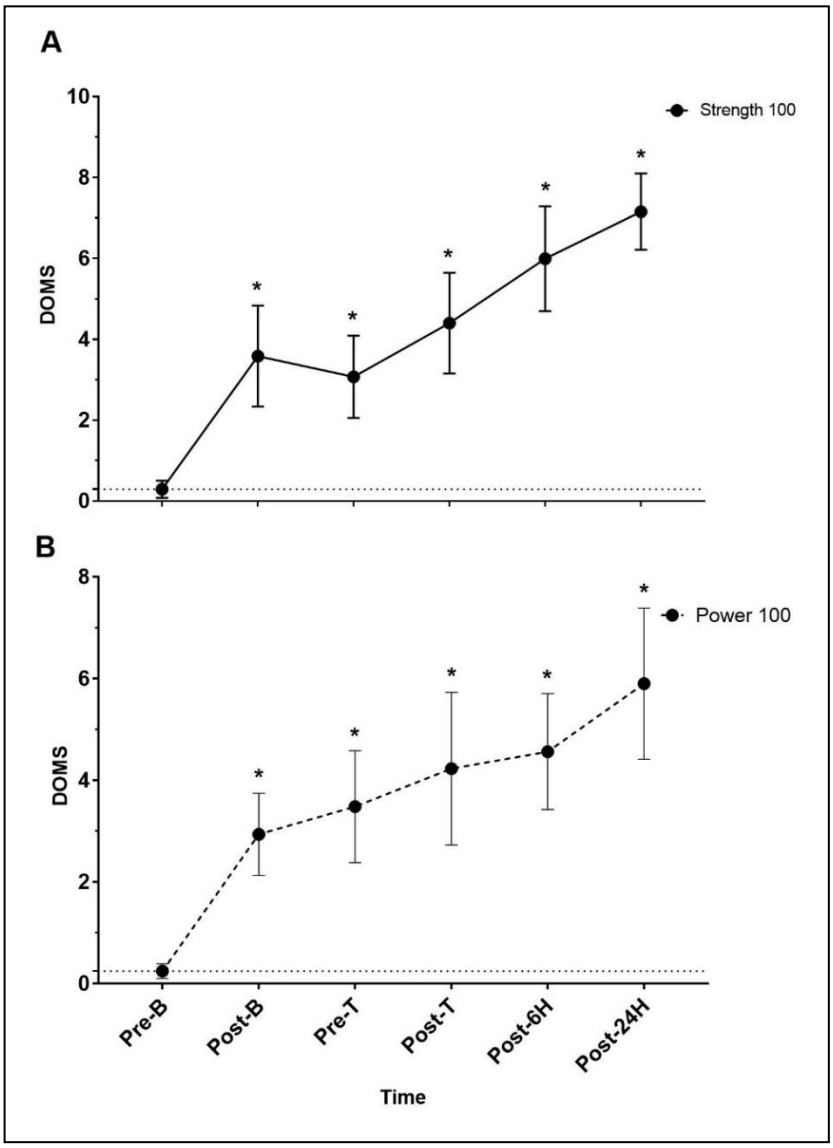


Figure 138. Changes in mean DOMS values in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.7.1.2. Profile of Mood States (POMS)

There was neither an overall treatment effect ($p = 0.363$) nor an overall time effect ($p = 0.185$) on POMS. No significant group x time interaction for POMS was observed ($p = 0.149$; Figure 139). POMS results showed that training stress from the training modalities did not significantly affect the mood states of the participants.

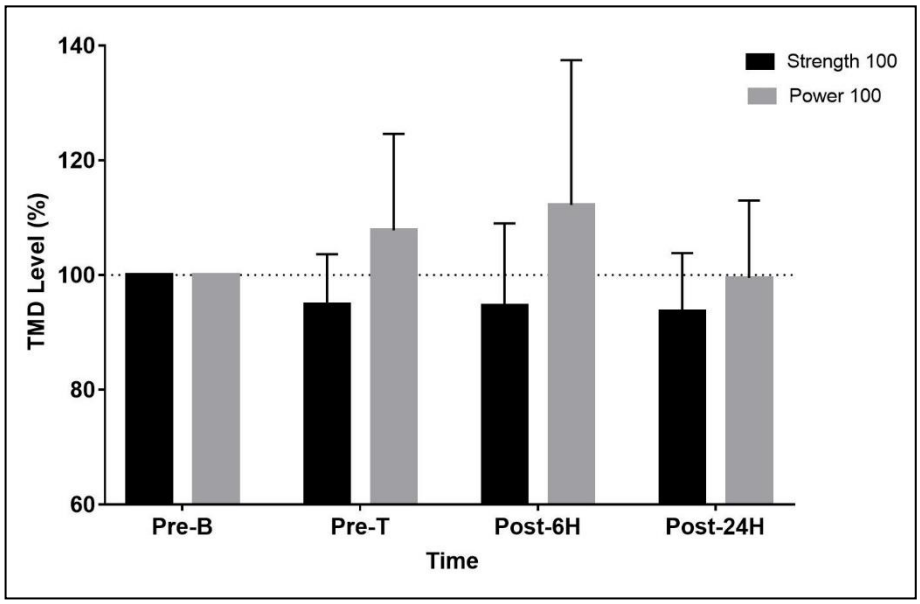


Figure 139. Comparison between S100 and P100 on mean POMS values (n = 11).

6.2.7.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the perceptual response variables for the comparison between S100, S75 and S50 are reported below

6.2.7.2.1. Muscle pain (DOMS)

There was an overall treatment effect ($p = 0.050$) and overall time effect ($p < 0.001$) on DOMS. There was no significant treatment x time interaction for DOMS ($p = 0.503$). Simple main effects for treatment showed that DOMS was significantly different between treatments (S100 vs S75 vs S50) at Post-T ($p = 0.045$, (S100 vs S75: $p = 0.094$; S100 vs S50: $p = 0.208$)), Post-6H ($p = 0.010$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.025$)) and Post-24H ($p = 0.001$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.013$)) (Figure 140).

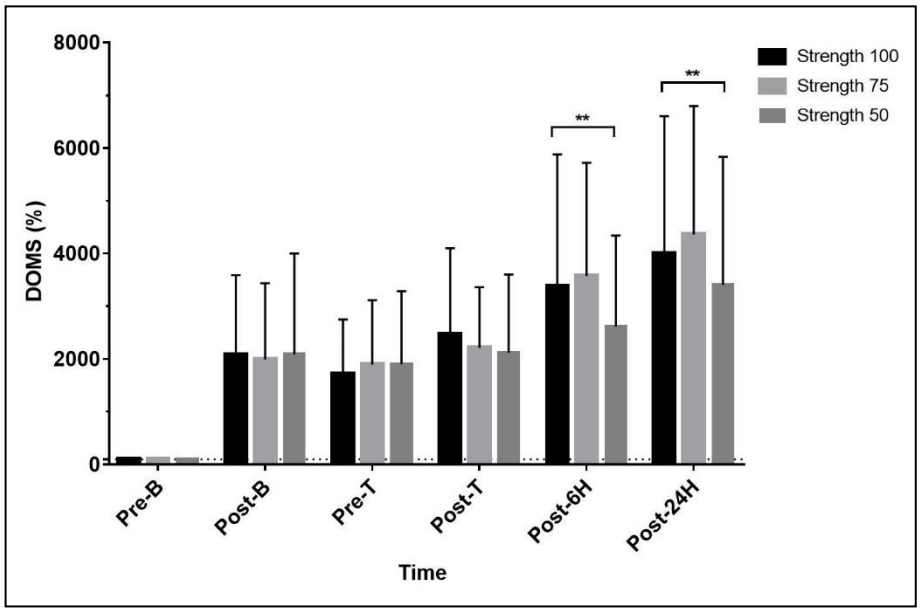


Figure 140. Comparison of S100, S75 and S50 on DOMS values (n = 13). ⌐** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that DOMS differed significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P < 0.001$) trials. In S100, significant time differences were observed at all the time points (Post-B ($p < 0.001$, ES = 4.02), Pre-T ($p < 0.001$, ES = 4.06), Post-T ($p < 0.001$, ES = 4.97), Post-6H ($p < 0.001$, ES = 5.59) and Post-24H ($p < 0.001$, ES = 10.33)) compared to Pre-B. Similarly, S75 (Post-B ($p < 0.001$, ES = 4.23), Pre-T ($p < 0.001$, ES = 3.95), Post-T ($p < 0.001$, ES = 8.51), Post-6H ($p < 0.001$, ES = 6.66) and Post-24H ($p < 0.001$, ES = 9.14)) and S50 (Post-B ($p < 0.001$, ES = 3.13), Pre-T ($p < 0.001$, ES = 3.65), Post-T ($p < 0.001$, ES = 5.40), Post-6H ($p < 0.001$, ES = 6.39) and Post-24H ($p < 0.001$, ES = 5.56)) also showed significant time differences at all the time points compared to Pre-B (Figure 141). These results showed that sensation of muscle pain not yet recovered to respected baseline (Pre-B) in all 3 training loads at Post-24H. According to the ES results, sensation of muscle pain of all 3 training loads were not yet recovered at Post-24H. Even though not fully recovered, S50 showed better recovery and S100 showed least recovery level at Post-24H.

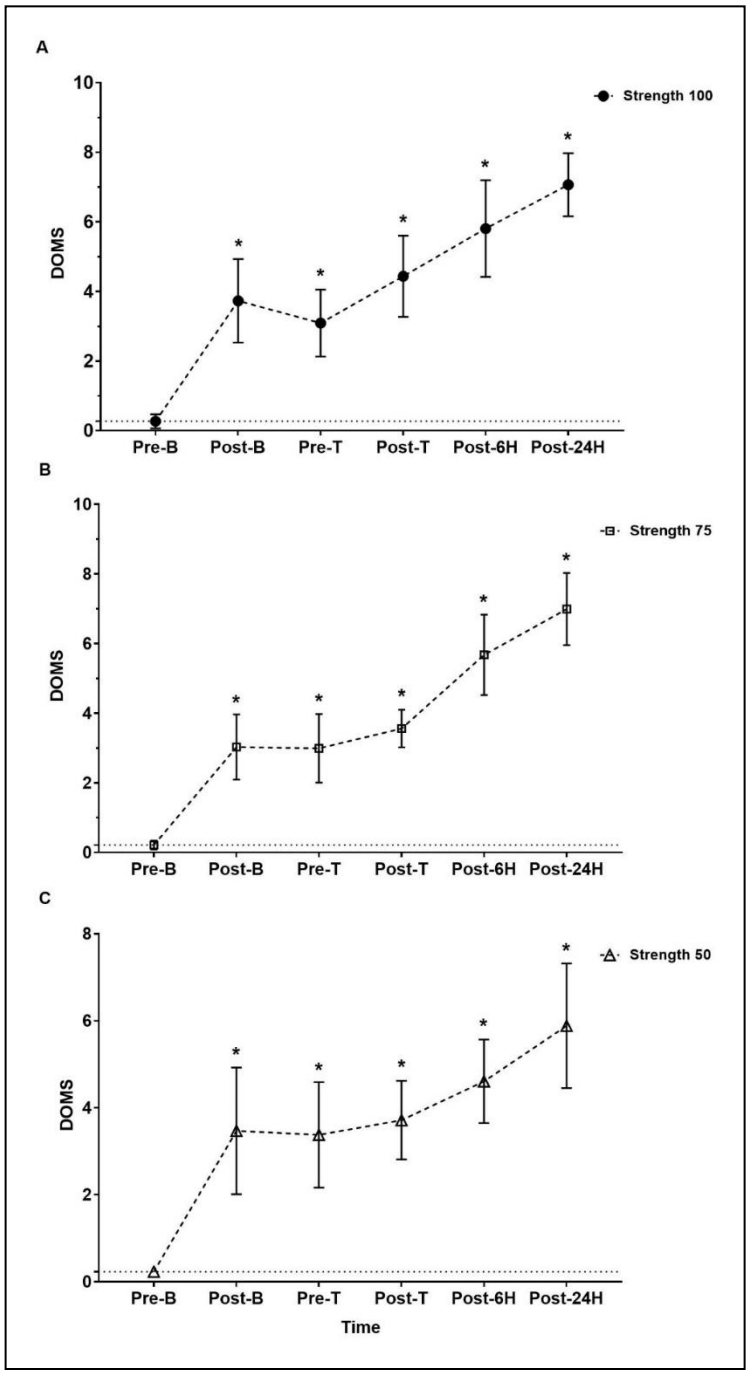


Figure 141. Changes in mean DOMS values in (A) S100, (B) S75 and S50 protocols (n = 13).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.7.2.2. Profile of Mood States (POMS)

There was neither an overall treatment effect (p = 0.616) nor an overall time effect (p = 0.446) on POMS. There was significant group x time interaction for POMS was observed (p = 0.016; Figure 142). Simple main effects over time revealed that POMS not different significantly between time points in S100 (P = 0.349) and S75 (P = 0.304), However, there was significant trend in S50 (P = 0.064) trial.

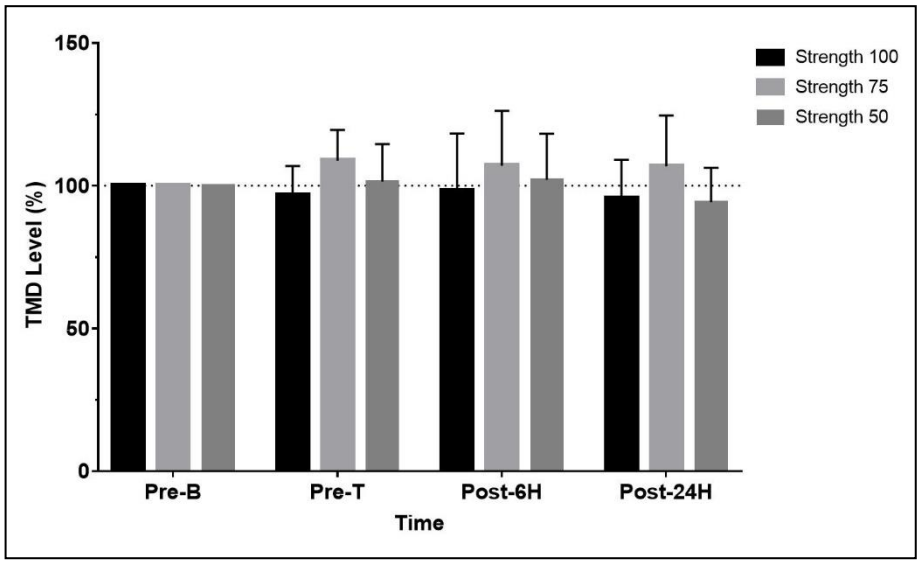


Figure 142. Comparison of S100, S75 and S50 on TMD values (n = 13).

6.2.7.3. Power 100 versus Power 75 versus Power 50 training

The results from the perceptual response variables for the comparison between P100, P75 and P50 are reported below.

6.2.7.3.1. Muscle pain (DOMS)

There was an overall treatment effect on DOMS (p = 0.020), as well as an overall time effect on DOMS (p < 0.001). There was no significant treatment x time interaction for DOMS (p = 0.433). Simple main effects for treatment showed that DOMS was significantly different between treatments (P100 vs P75 vs P50) at Post-24H (p = 0.003, (P100 vs P75: p = 1.000; P100 vs P50: p = 0.005)) of the trials (Figure 143).

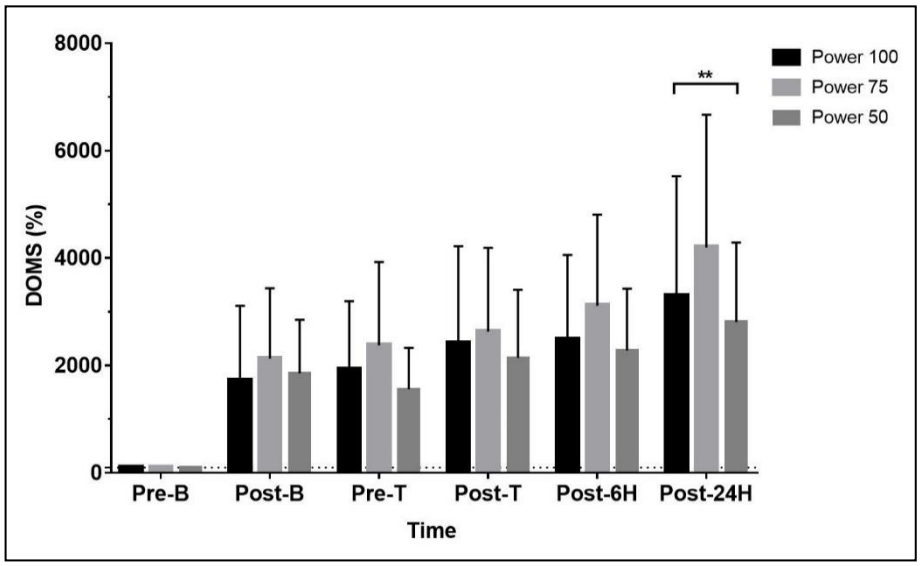


Figure 143. Comparison of P100, P75 and P50 on DOMS values (n = 11). ** Significant pairwise comparison differences in P50 compared to P100 ($p \leq 0.05$).

Simple main effects over time revealed that DOMS differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trials. In P100, significant time differences were observed at all the time points (Post-B ($p < 0.001$, ES = 4.63), Pre-T ($p < 0.001$, ES = 4.12), Post-T ($p < 0.001$, ES = 3.74), Post-6H ($p < 0.001$, ES = 5.32) and Post-24H ($p < 0.001$, ES = 5.35)) compared to Pre-B. Similarly, P75 (Post-B ($p < 0.001$, ES = 3.68), Pre-T ($p < 0.001$, ES = 4.43), Post-T ($p < 0.001$, ES = 5.42), Post-6H ($p < 0.001$, ES = 5.88) and Post-24H ($p < 0.001$, ES = 5.43)) and P50 (Post-B ($p < 0.001$, ES = 5.25), Pre-T ($p < 0.001$, ES = 5.94), Post-T ($p < 0.001$, ES = 5.61), Post-6H ($p < 0.001$, ES = 7.36) and Post-24H ($p < 0.001$, ES = 5.91)) trials also showed significant time differences at all the time points compared to respected Pre-B values (Figure 144). These results showed that sensation of muscle pain was not yet recovered to respected baseline (Pre-B) in all 3 training loads at Post-24H. According to the ES results, sensation of muscle pain of all 3 training loads were not yet recovered at Post-24H.

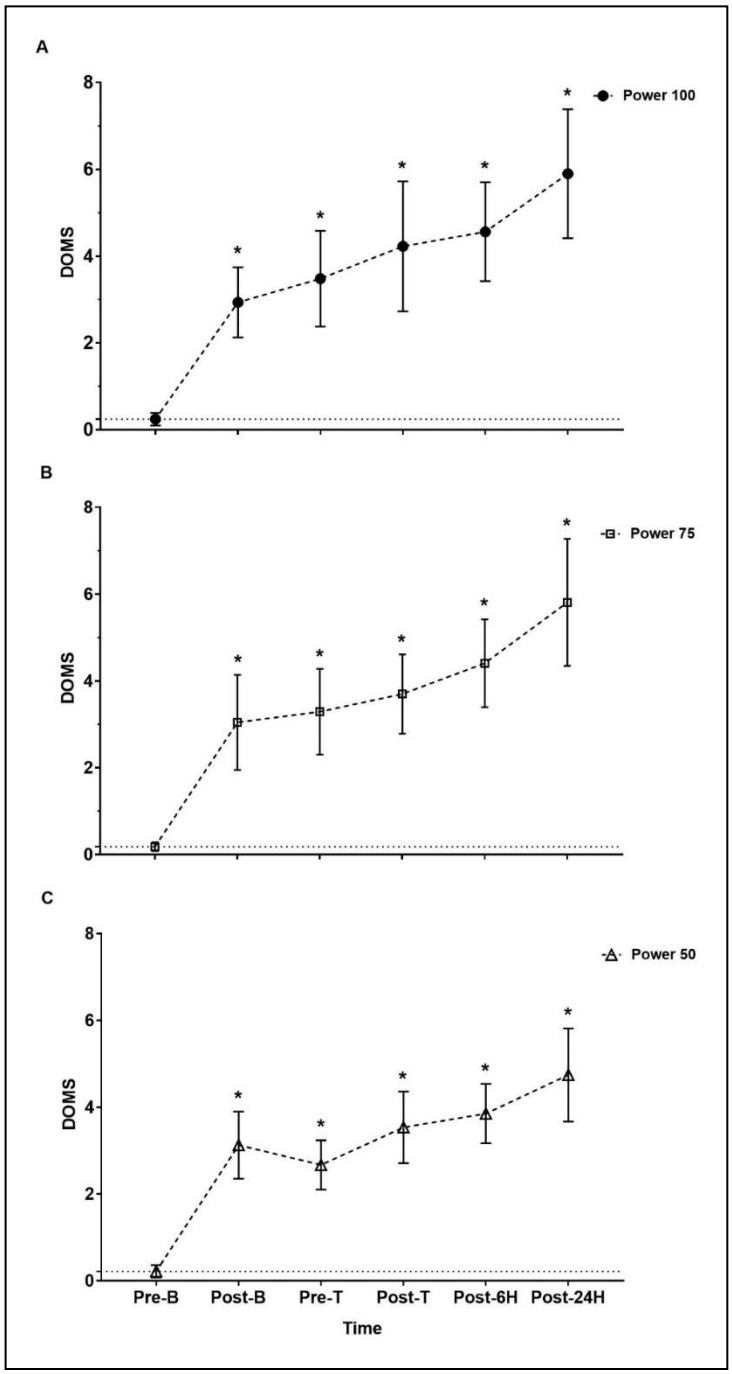


Figure 144. Changes in mean DOMS values in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

6.2.7.3.2. Profile of Mood States (POMS)

There was neither an overall treatment effect (p = 0.343) nor an overall time effect (p = 0.287) on POMS. No significant group x time interaction for POMS was observed (p = 0.817; Figure 145).

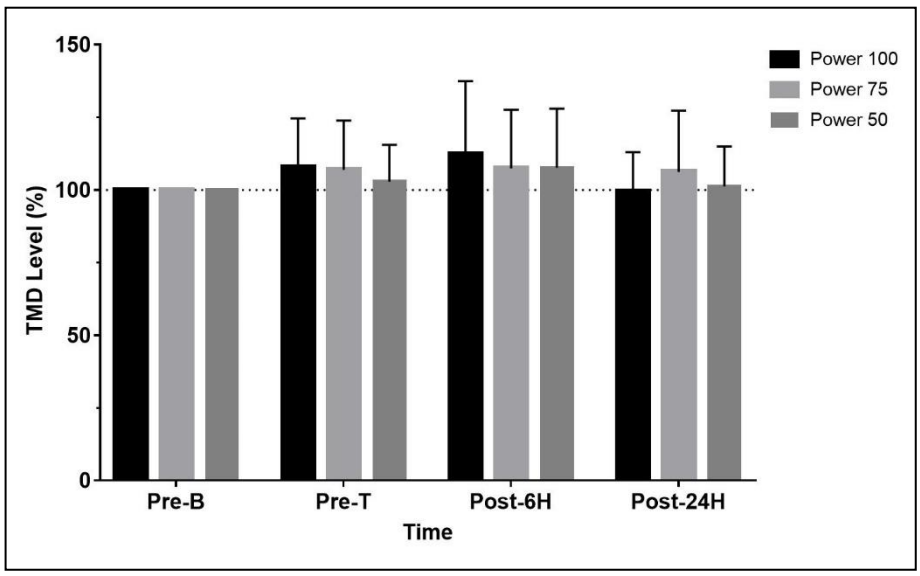


Figure 145. Comparison of P100, P75 and P50 on TMD values (n = 11)

6.2.8. Other

6.2.8.1. Strength 100 versus Power 100 training

The results from the perceived exertion and blood lactate concentration for the comparison between S100 and P100 are reported below

6.2.8.1.1. Borg Scale of Perceived Exertion

There was an overall treatment effect on perceived exertion (p = 0.002), as well as an overall time effect on perceived exertion (p < 0.001). There was significant treatment x time interaction for perceived exertion (p = 0.004), where simple main effects for treatment showed that Borg scale of perceived exertion was significantly lower in the strength modality at Post-T (p = 0.002) compared to the power modality (Figure 146).

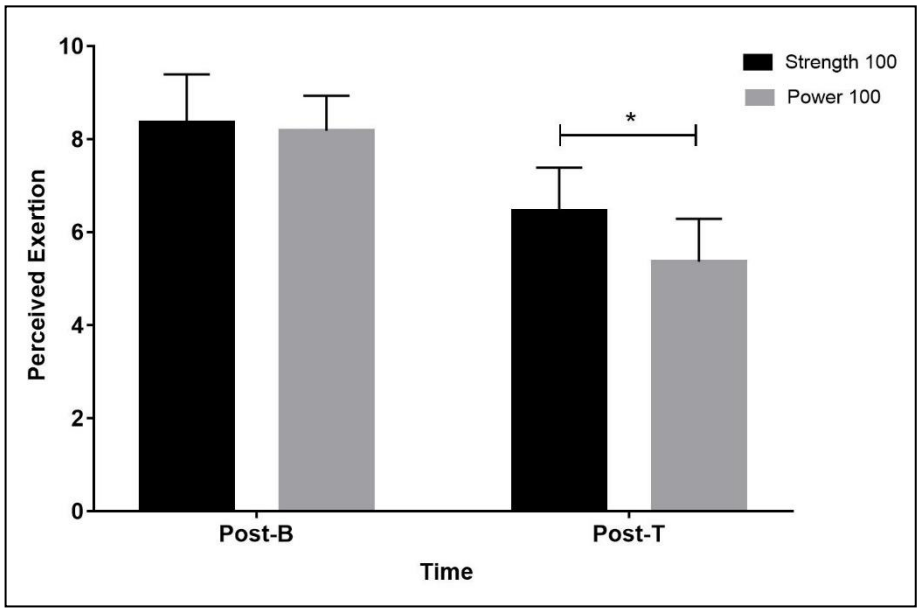


Figure 146. Comparison between S100 and P100 on mean BORG values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that perceived exertion differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. In S100, significant time difference was observed at Post-T ($p < 0.001$, ES = -1.94) compared to Post-B. Similarly, P100 showed significant time difference at Post-T ($p < 0.001$, ES = -3.35) compared to Post-B (Figure 147).

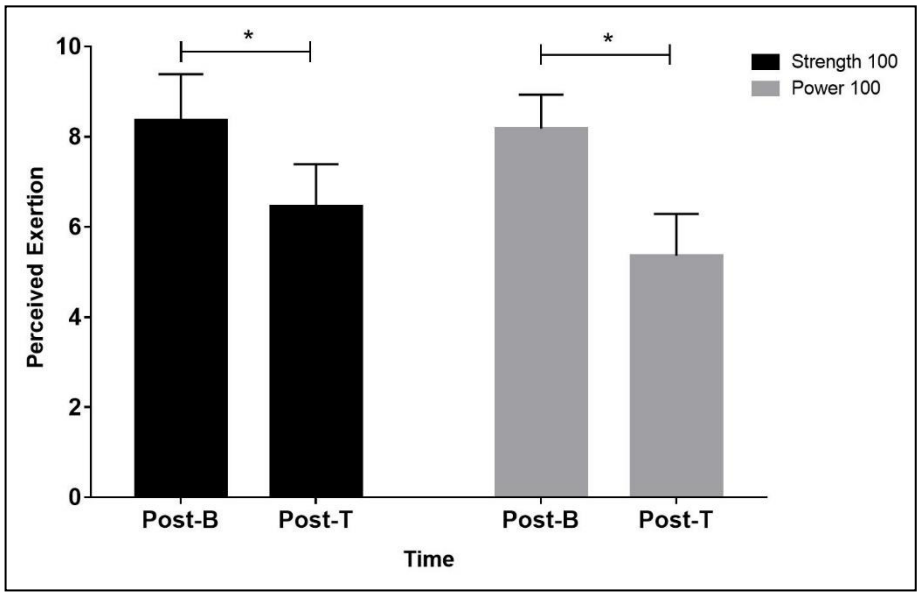


Figure 147. Changes in mean perceived exertion values in S100 and P100 protocols (n = 11).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

These results revealed that perceived exertion level was significantly higher following M-Beast protocol than ARE protocol in both training modalities. Interestingly, data also showed that S100 creates higher sensation level of exertion than P100.

6.2.8.1.2. Blood lactate concentrations

There was an overall treatment effect on blood lactate concentration ($p = 0.024$), as well as an overall time effect on blood lactate concentration ($p < 0.001$). There was no significant treatment x time interaction for blood lactate concentration ($p = 0.672$; Figure 148).

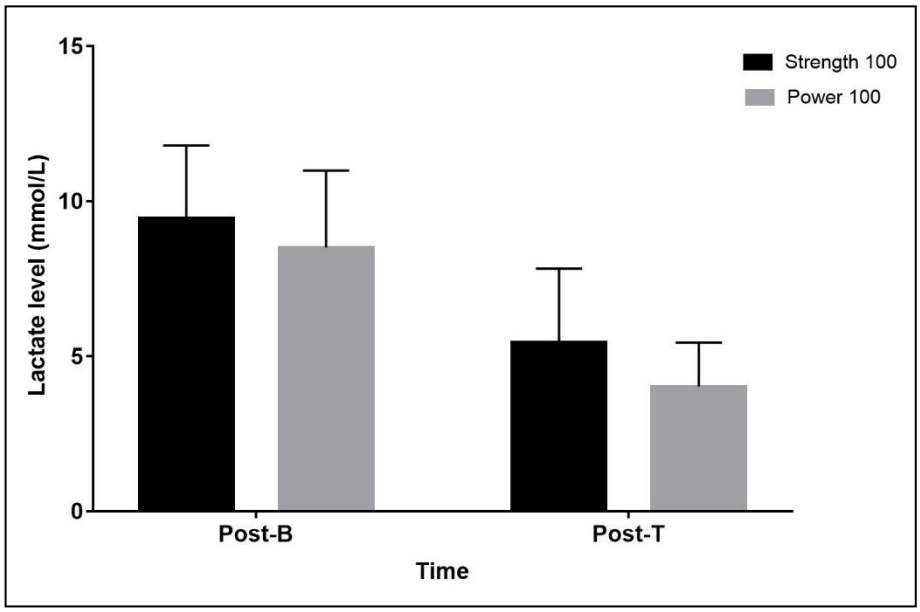


Figure 148. Comparison between S100 and P100 on mean Lactate level values (n = 11).

Simple main effects over time revealed that blood lactate concentration different significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trial. In S100, significant time difference was shown at Post-T ($p < 0.001$, ES = -1.69) compared to Post-B. Similarly, P100 also showed significant time difference at Post-T ($p < 0.001$, ES = -2.21) compared to Post-B (Figure 149). These results revealed that blood lactate concentration level was significantly higher following M-Beast protocol than ARE in both trials. However, data showed that there was no significant difference in blood lactate concentration between S100 and P100.

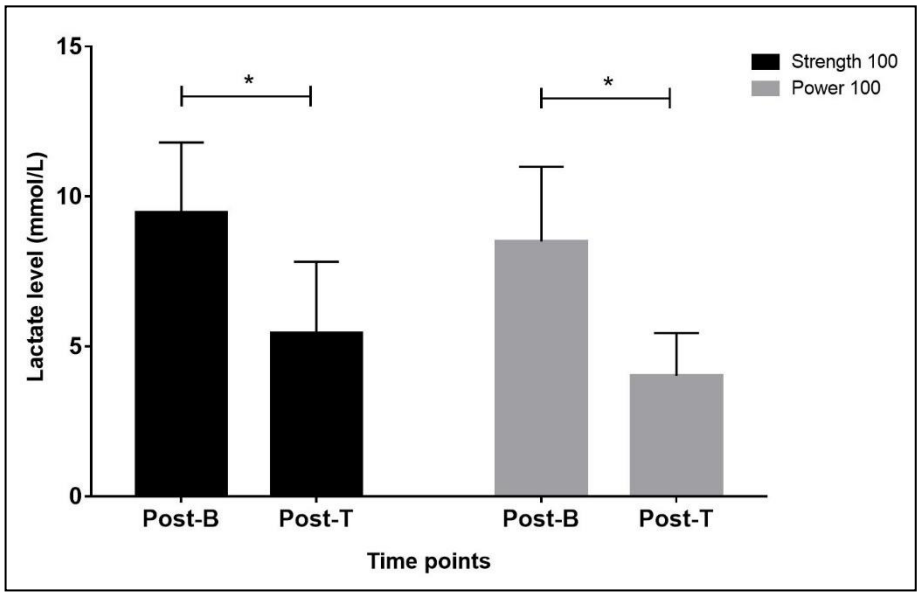


Figure 149. Changes in mean blood lactate accumulation values in S100 and P100 protocols (n = 11).

6.2.8.2. Strength 100 versus Strength 75 versus Strength 50 training

The results from the perceived exertion and blood lactate concentration for the comparison between S100, S75 and S50 are reported below

6.2.8.2.1. Borg Scale of Perceived Exertion

There was an overall treatment effect on perceived exertion ($p < 0.001$), as well as an overall time effect on perceived exertion ($p < 0.001$). There was significant treatment x time interaction for perceived exertion ($p < 0.001$), where simple main effects for treatments (S100 vs S75 vs S50) at Post-T ($p < 0.001$, (S100 vs S75: $p = 0.012$; S100 vs S50: $p < 0.001$)) of the trials (Figure 150)

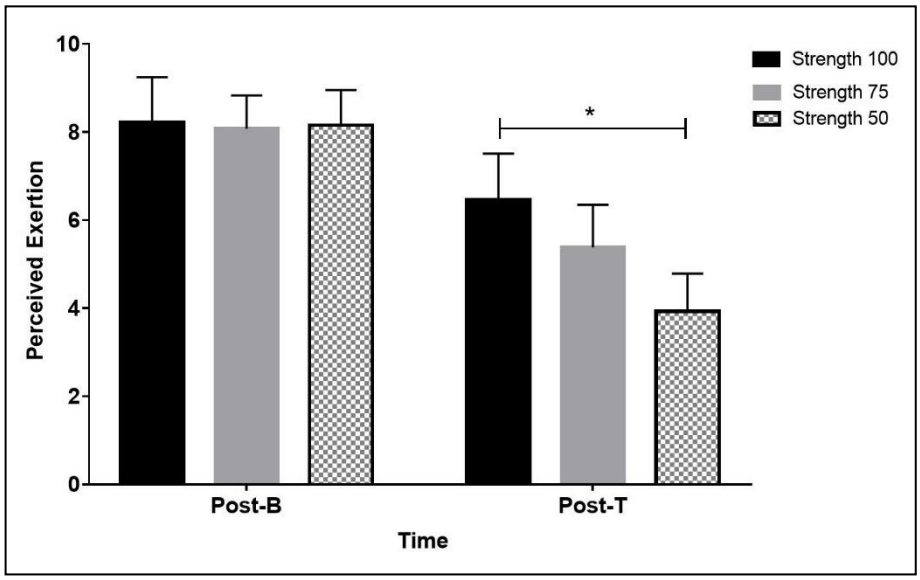


Figure 150. Comparison of strength 100, strength 75 and strength 50 on perceived exertion values (n = 13). * Significant pairwise comparison differences in S75 and S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that perceived exertion differed significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P < 0.001$) trial. Compared to Post-B in each trial, significant time differences were showed in respected Post-T ((S100: $P < 0.001$, ES = -1.72), (S75: $P < 0.001$, ES = -3.11) and (S50: $P < 0.001$), ES = -5.08). These results revealed that perceived exertion level was significantly higher following M-Beast protocol than ARE in all 3 trials (Figure 151). Interestingly, data also showed that S100 ARE protocol created the greatest sensation level of exertion and S50 ARE protocol created the least sensation level of exertion (Figure 150).

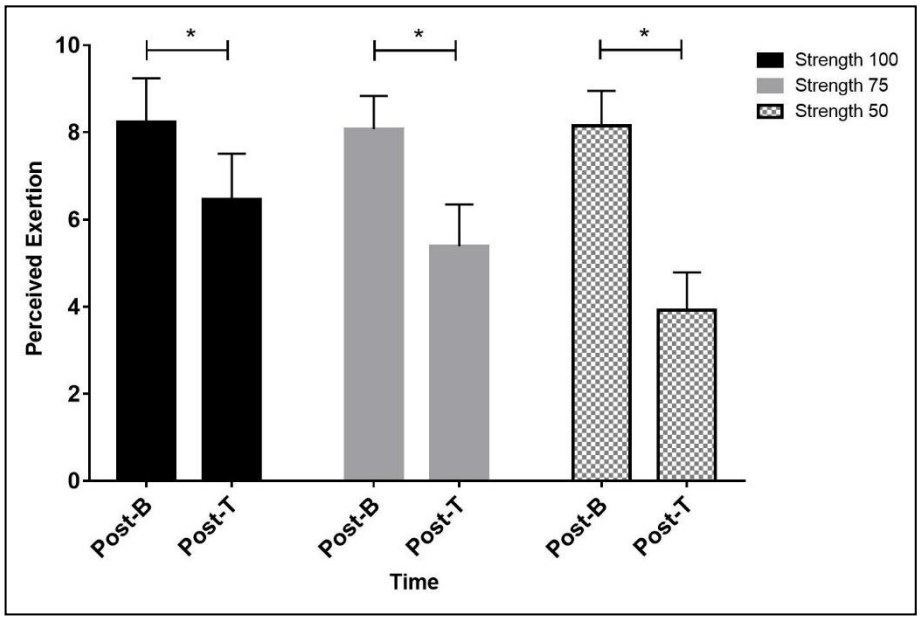


Figure 151. Changes in mean perceived exertion values in S100, S75 and S50 protocols (n = 13). * Significant difference between time points in S100, S75 and S50 ($p \leq 0.05$).

6.2.8.2.2. Blood lactate concentrations

There was no overall treatment effect on blood lactate concentration ($p = 0.221$). However, there was an overall time effect on blood lactate concentration ($p < 0.001$). No significant group x time interaction for blood lactate concentration was observed ($p = 0.141$; Figure 152).

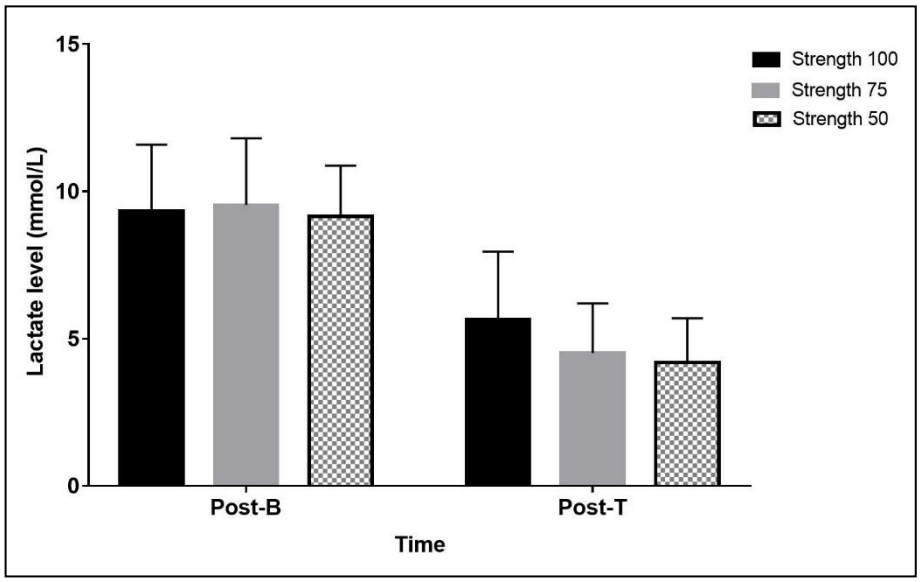


Figure 152. Comparison of S100, S75 and S50 on blood lactate concentration values (n = 13).

Simple main effects over time revealed that blood lactate concentration differed significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P < 0.001$) trial. Compared to Post-B, in each trial, significant difference was shown in respected Post-T ((S100: $P < 0.001$, ES = -1.62), (S75: $P < 0.001$, ES = -2.52) and (S50: $P < 0.001$), ES = -3.08). These results revealed that blood lactate concentration level was significantly higher following M-Beast protocol than ARE in all 3 trials. Interestingly, data also showed that there was no significant difference in the level of muscle metabolism between the ARE training loads (Figure 153).

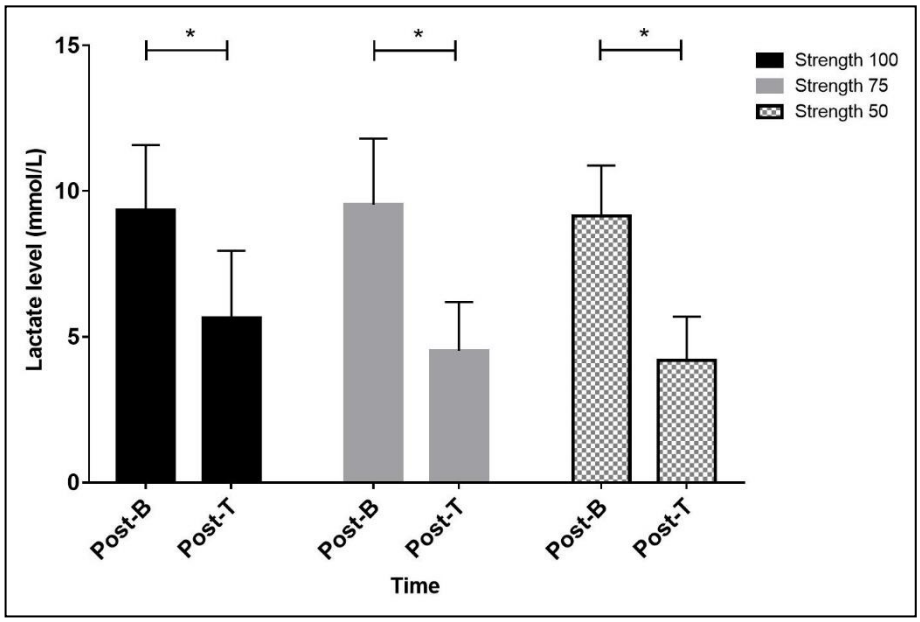


Figure 153. Changes in mean blood lactate concentration values in S100, S75 and S50 protocols (n = 13). * significant difference between time points in S100, S75 and S50

6.2.8.3. Power 100 versus Power 75 versus Power 50 training

The results from the perceived exertion and blood lactate concentration for the comparison between P100, P75 and P50 are reported below

6.2.8.3.1. Borg Scale of Perceived Exertion

There was an overall treatment effect on perceived exertion ($p < 0.001$), as well as an overall time effect on perceived exertion ($p < 0.001$). There was significant treatment x time interaction for perceived exertion ($p = 0.002$), where simple main effects for treatment showed that perceived exertion was significantly different between treatments (P100 vs P75 vs P50) at Post-T ($p < 0.001$, (P100 vs P75: $p = 0.201$; P100 vs P50: $p = 0.001$)) of the trials (Figure 154)

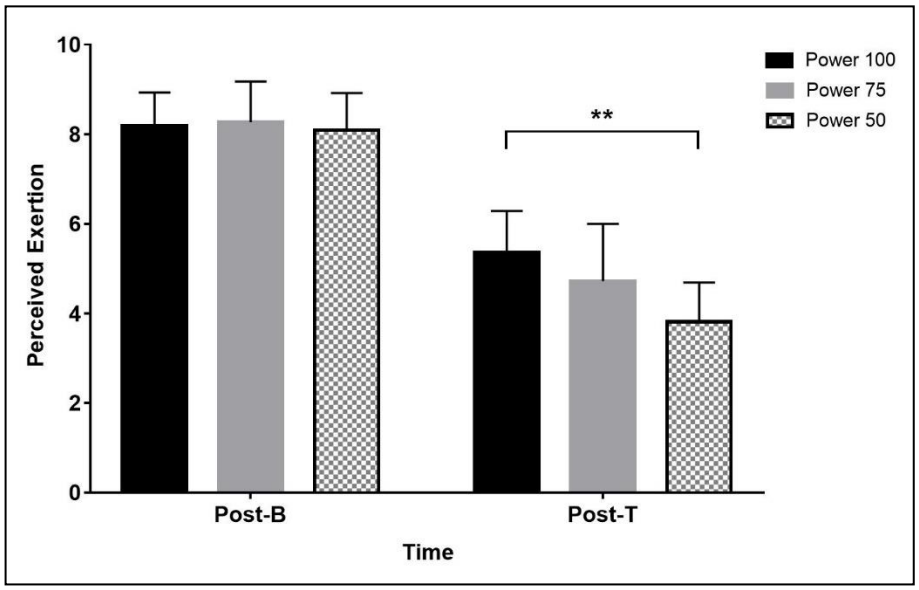


Figure 154. Comparison of P100, P75 and P50 on perceived exertion values (n = 11). ** Significant pairwise comparison differences between P50 and P100 ($p \leq 0.05$).

Simple main effects over time revealed that perceived exertion differed significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P < 0.001$) trial. Compared to Post-B in each trial, significant difference was shown in respected Post-T ((P100: $P < 0.001$, ES = -3.35), (P75: $P < 0.001$, ES = -3.21) and (P50: $P < 0.001$), ES = -5.01). These results revealed that perceived exertion was significantly higher following M-Beast protocol than ARE in all 3 training loads. Interestingly, results also showed that P100 ARE created the greatest sensation level of exertion and P50 ARE created the least sensation level of exertion (Figure 155).

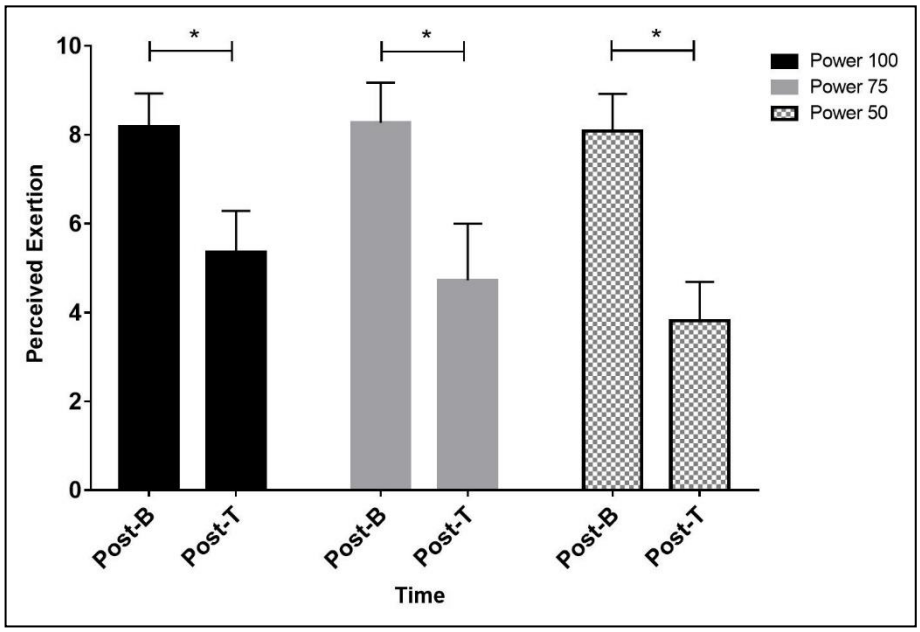


Figure 155. Changes in mean perceived exertion values in P100, P75 and P50 protocols (n = 11). * significant difference between time points in P100, P 75 and P50

6.2.8.3.2. Blood lactate concentration

There was no overall treatment effect on blood lactate concentration ($p = 0.619$). However, there was an overall time effect on blood lactate concentration ($p < 0.001$). No significant group x time interaction for blood lactate concentration was observed ($p = 0.495$; Figure 156).

Simple main effects over time revealed that blood lactate concentration different significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P = 0.001$) trial. Compared to Post-B in each trial, significant difference was shown in respected Post-T ((P100: $P < 0.001$, ES = -2.21), (P75: $P < 0.001$, ES = -2.58) and (P50: $P = 0.001$), ES = -1.93). These results revealed that blood lactate concentration level was significantly higher following M-Beast protocol than ARE in all 3 training loads. Interestingly, data also showed that there was no significant difference in the level of muscle metabolism between the training loads (Figure 157).

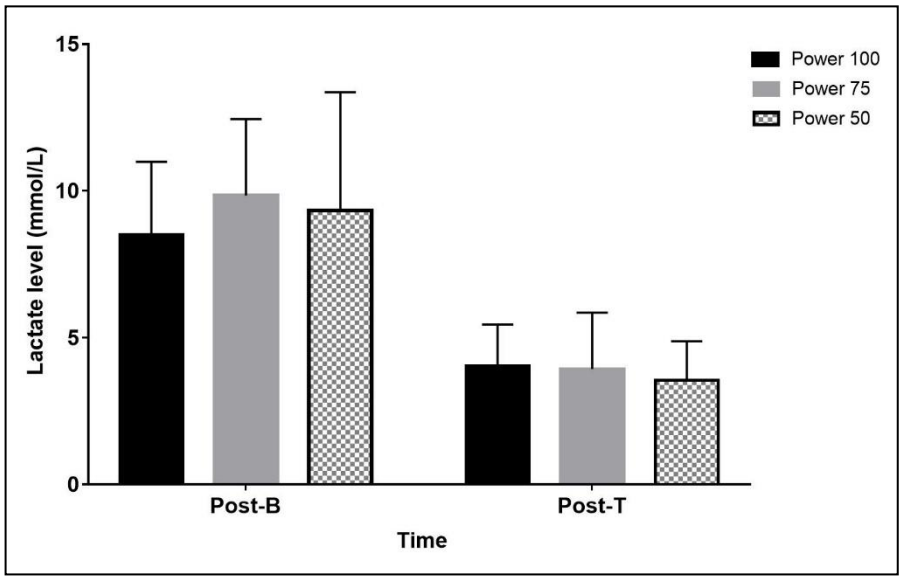


Figure 156. Comparison of P100, P75 and P50 on blood lactate concentration values (n = 11).

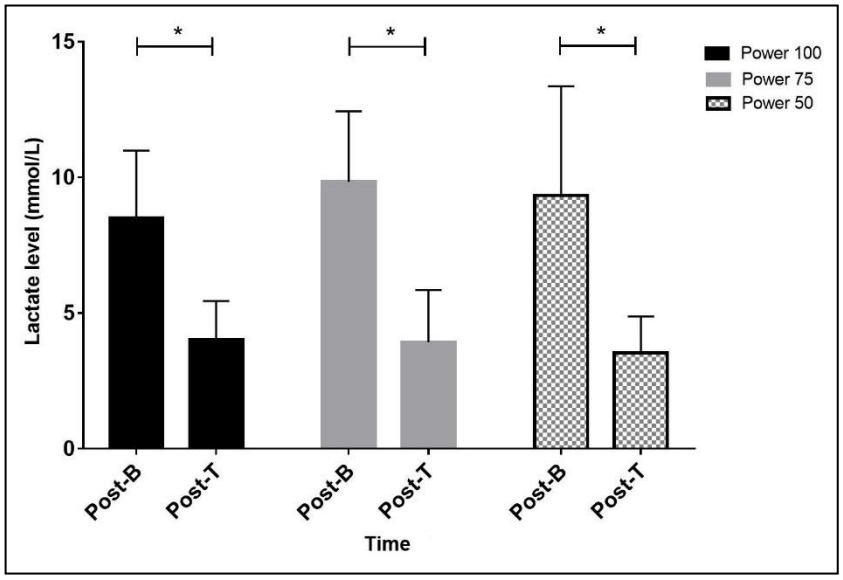


Figure 157. Changes in mean blood lactate concentration values in P100, P75 and P50 protocols (n = 11). * significant difference between time points in P100, P75 and P50

6.2.9. Time-course of recovery monitoring using different monitoring tools.

6.2.9.1. Time-course of recovery monitoring using different monitoring tools for strength training loads

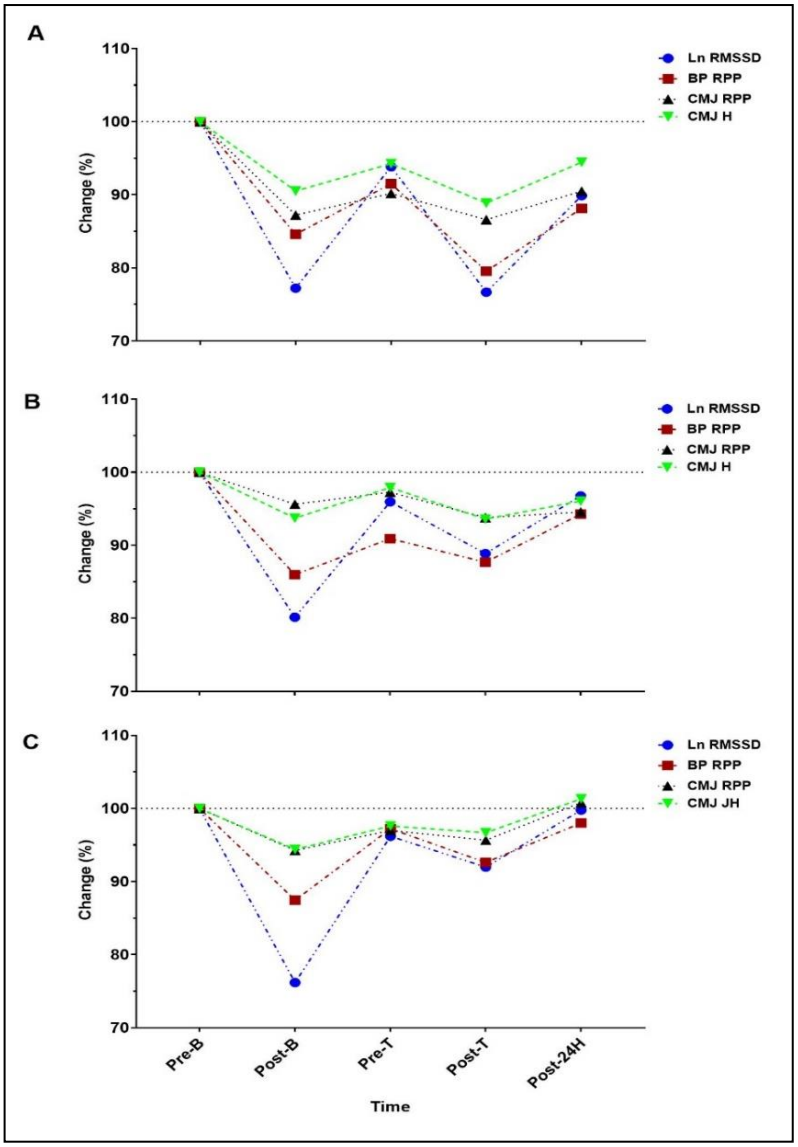


Figure 158. Time-course of recovery monitoring using difference monitoring tools for strength training loads ((A) S100, (B) S75 and (C) S50).

Abbreviations: BP RPP = Bench press relative peak power output, CMJ RPP = Countermovement jump relative peak power output. CMJ JP = Countermovement jump height, Ln RMSSD = natural logarithm of the square root of the mean sum of the squared differences

6.2.9.2. Time-course of recovery monitoring using difference monitoring tools for power training loads

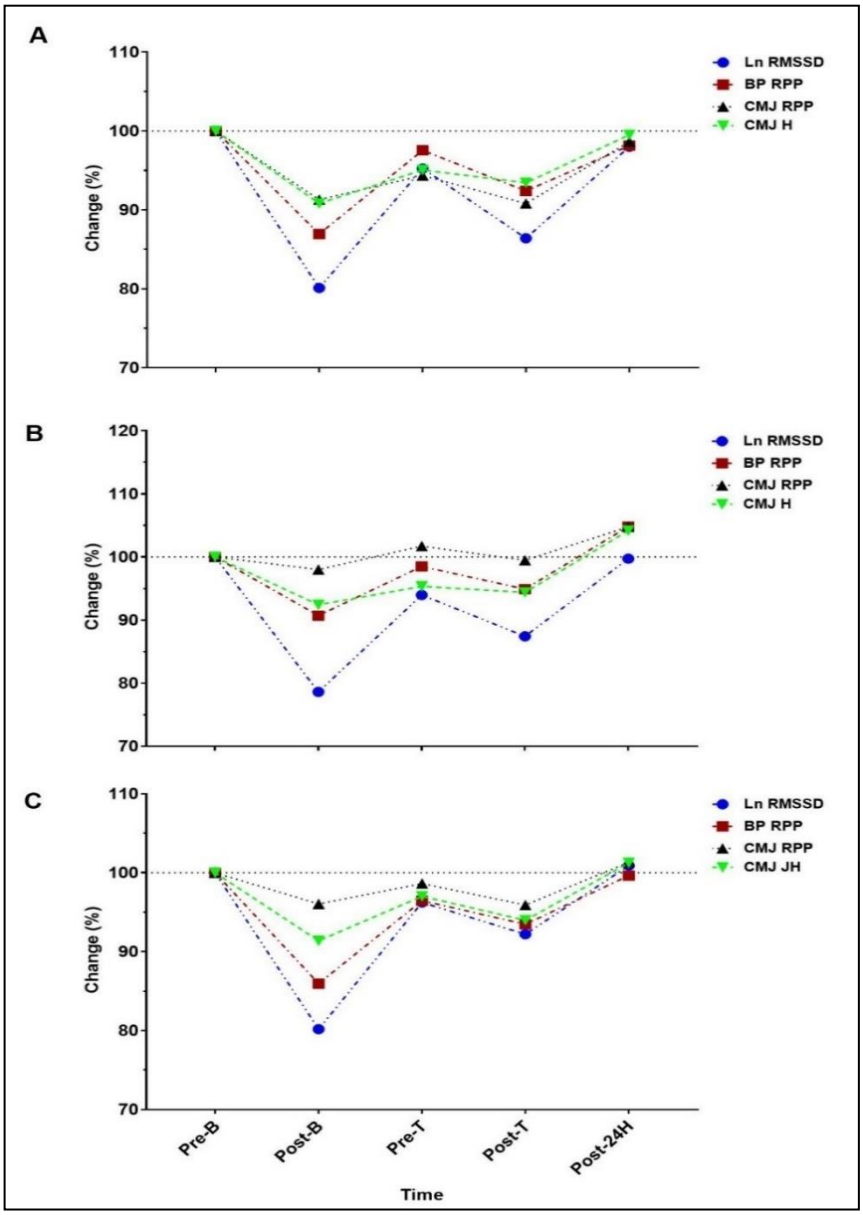


Figure 159. Time-course of recovery monitoring using difference monitoring tools for power training loads ((A) P100, (B) P75 and (C) P50).

Abbreviations: BP RPP = Bench press relative peak power output, CMJ RPP = Countermovement jump relative peak power output. CMJ JP = Countermovement jump height, Ln RMSSD = natural logarithm of the square root of the mean sum of the squared differences

VII – DISCUSSION

VII. DISCUSSION

This chapter will discuss, substantiate, and compare present doctoral thesis findings with previous research. The chapter is organized into two sections: discussion regarding the findings of Study 1 (systematic review and meta-analysis study) and Study 2 (experimental study)

7.1. STUDY 1

The main aim of this systematic review with meta-analysis was to provide essential information regarding the recovery status of cardiac autonomic activity following an ARE session, particularly identifying the moderating factors that affect HRV parameters. The principal findings demonstrated a significant decrease in cardiac parasympathetic modulation and an increase in cardiac sympathetic modulation about 30 min following an ARE session. Moreover, overall autonomic modulation showed a significant decrease after an ARE session. The reduction of RMSSD and HF(nu) parameters indicates a withdrawal of cardiac parasympathetic modulation (155, 161, 169), and the increase in the LF(nu) parameter suggests the domination of cardiac sympathetic modulation (155, 161) after an ARE session. Furthermore, an increase in the LF/HF ratio suggests a shift in sympathovagal balance towards sympathetic domination (155, 169), and a reduction in the SDNN value indicates a decrease in overall autonomic modulation (161). Overall, our systematic review with meta-analysis suggests that the early recovery phase is still predominated by cardiac sympathetic activity.

Our results are in accordance with the review article conducted by Kingsley *et al.* (38), which examined the ARE on HRV parameters. In their study, they reviewed 10 studies (153 young healthy adults) published before September 2013 in MEDLINE and PUBMED databases and the results showed an increase in LF(nu)

and LF/HF ratio parameters and a decrease in HF(nu) parameter, indicating a decrease in cardiac parasympathetic modulation and domination of cardiac sympathetic modulation following an ARE session (38). However, there several studies (> 15) have been published on this subject since 2014, and there appeared to be some discrepancies in some of the research findings. Moreover, it is important to acknowledge that the interpretation of the LF parameter and the LF/HF ratio has recently been argued. Some authors consider the LF parameter to be a cardiac sympathetic modulation marker, while others believe that it reflects both sympathetic and parasympathetic modulation. With regards to the LF/HF ratio, some authors interpret this variable as a cardiac sympathetic modulation marker, while others suggest that it is a reflection of sympathovagal balance (161).

A physiological explanation for the increase in cardiac sympathetic modulation and decrease in cardiac parasympathetic modulation during the recovery phase of resistance training may be that there was a decrease in plasma volume as a result of an acute cardiovascular imbalance (216). This imbalance may be a result of the blood entering (leaking) into the interstitial cellular space, therefore decrease the blood flow back to the heart (venous return) (265), which would change the sensitivity of the arterial baroreflex in order to maintain the blood pressure changes caused by a decrease in stroke volume (which is a consequence of an increase in heart rate after resistance exercise) (216). This creates a greater activation of metaboreceptors and mechanoreceptors, thus providing adequate blood flow in order to meet the metabolic demands of the active muscles (41, 216, 227). Also, there may be an increase in peripheral vascular resistance in arterial vessels supplying visceral organs, where redistributed blood flows to the active muscles during the recovery process (41, 216, 227). Moreover, Buchheit *et al.* (266) have suggested that the levels of fast-twitch muscle fiber recruitment, catecholamine release and accumulation of lactate, hydrogen ions and inorganic phosphate may play a role in decreasing cardiac parasympathetic modulation, thereby increasing cardiac sympathetic modulation. Thus, evaluating HRV

variables can be useful in determining cardiac autonomic stress, which may be beneficial for fitness trainers or coaches to use as a monitoring tool for measuring the effect of the training load on the cardiac autonomic system following an ARE session.

Our subgroup analyses revealed that training volume is an important moderating factor for RMSSD, LF(nu), and HF(nu) parameters. The number of sets is a moderating factor for RMSSD parameter, while exercise intensity and rest between sets are moderating factors for HF(nu) parameter. These aforementioned moderating factors affect the recovery process of cardiac autonomic modulation following a resistance training session. Therefore, fitness trainers and coaches could monitor and adjust the training load by measuring the changes in cardiac autonomic modulation using HRV variables, such as RMSSD (training volume, number of sets per exercise) and HF(nu) (number of exercises, reset between sets). The following sections provide a more detailed discussion of each of the subgroup analyses.

7.1.1. Subjects characteristics

Subgroup analyses did not show a significant difference in gender (between males and females) for RMSSD, HF(nu), LF(nu) and LF/HF ratio parameters. These findings agree with Kingsley *et al.* (212), who concluded that changes in HRV parameters (Ln RMSSD, Ln HF, Ln LF and Ln LF/HF ratio, Ln TP) in response to an ARE (post 25-30 minutes) were not influenced by gender differences. The BMI subgroup analyses also demonstrated no significant effect on RMSSD, HF(nu), LF(nu) and LF/HF ratio parameters. Similarly, Macêdo *et al.* (42) reported that changes in HRV parameters (SDNN, RMSSD and pNN50) in response to ARE (post 30 minutes) were not affected by body weight. However, Beske *et al.* (267) reported that lower cardiovagal baroreflex gain was marginally related to higher body fat percentage. But if we consider that lower cardiovagal baroreflex sensitivity elicits a weaker response to the changes in systolic blood pressure, then lower cardiovagal

baroreflex sensitivity does not effectively change the heart rate (268). Therefore, higher body fat mass may have a minimal effect on cardiac sympathetic modulation and, thus, may only trigger a minimal change in heart rate. Likewise, the analyses of the training status subgroup demonstrated no significant effect on RMSSD, HF(nu), LF(nu), and LF/HF ratio variables. These findings are again in accordance with Kingsley *et al.* (210), who concluded that changes in HRV parameters in response to the ARE were not influenced by training status. In summary, our study showed that gender, BMI, and training status do not play a role in cardiac autonomic modulation changes following an ARE sessions. Therefore, trainers and coaches may not need to specialize a resistance training session based on an individual's gender, BMI level or training status. However, we believe that further investigations on the relationship between BMI and HRV parameters related to an ARE session are needed.

7.1.2. Training characteristics

7.1.2.1. Number of repetitions, sets, and exercises per workout

There were no significant differences among the number of repetitions subgroups for RMSSD, LF(nu), HF(nu), and LF/HF ratio parameters. Interestingly, a significant difference was demonstrated between subgroups for the RMSSD parameter and the number of sets and number of exercises, but this significant difference was not demonstrated for the LF(nu) and HF(nu) parameters. Additionally, SMD results showed that the RMSSD parameter was affected greatly by an ARE session that included exactly 3 sets per exercise but was not affected greatly when there were <3 sets per exercise. Our findings conform with Figueiredo *et al.* (227), who reported a reduced cardiac sympathetic modulation response with a lower number of sets of resistance training compared to a higher number sets. Therefore, performing >3 sets per exercise generates a higher sympathetic stress and may delay the recovery process compared to performing <3 sets per exercise.

SMD data also demonstrated that the RMSSD parameter may be affected by the number of exercises with a higher effect shown for exactly 6 exercises, although this did not reach statistical significance (p_{diff} = 0.07). Thus, performing 3 sets per exercise, and possibly 6 exercises per session, generates a greater withdrawal of cardiac parasympathetic modulation after an ARE session. It remains to be determined whether the number of exercises truly has an effect on RMSSD.

7.1.2.2. Rest between sets

The rest period only had an effect on the HF(nu) parameter. SMD data showed that HF(nu) was greatly affected by an ARE session that included <2 min of rest between sets but was less affected when there was exactly 2 min or >2 min of rest between sets. Goessler *et al.* (231) suggested that at least 2 min of rest between sets reduces the postexercise cardiac sympathetic modulation following ARE. Therefore, having <2 min of resting time between sets generates greater withdrawal of cardiac parasympathetic modulation, and 2 or more minutes of rest between sets creates lesser withdrawal of cardiac parasympathetic modulation, independent of the other variables of resistance training. These results indicate that having <2 min of rest between sets delays the recovery process following an ARE session compared to ≥2 min of rest between sets.

7.1.2.3. Exercise intensity

Based on our subgroup analysis, the exercise intensity (low, moderate or high) in an ARE session is not a moderating factor for RMSSD, LF(nu), or LF/HF ratio. Figueiredo *et al.* (41) showed no differences between the intensity levels in a training session (60%, 70% and 80% of 1RM) and RMSSD. Additionally, Rezk *et al.* (216) demonstrated no difference in LF(nu) or HF(nu) when comparing 40% and 80% 1RM training sessions. This lack of difference is interesting because it suggests that, although ARE has an effect on cardiac sympathetic and parasympathetic modulations, different intensity levels work independently from other covariables related to resistance training, and does not significantly affect cardiac autonomic

modulation. However, in our study, only the HF(nu) parameter showed a significant difference between exercise intensity subgroups. Surprisingly, our SMD results showed that low exercise intensity had the greatest effect and high exercise intensity had the least effect on HF(nu). One possible explanation for the difference in results shown between the HF(nu) and RMSSD parameters (both of which represent cardiac parasympathetic modulation) is that the included studies in each subgroup were different but the tendency of the findings was the same: a lower intensity had a higher effect. This may be a consequence of having a longer training duration of lower intensity. Another explanation may be that respiration control influences HF(nu) during HRV measurements (38, 169). The normal respiratory rate in healthy human adults is within the range of 12 – 20 breaths min^{-1} (269-271). Chang *et al.* (206) reported that a respiratory rate of 12 breaths min^{-1} produces the amplitude of the RR interval to be located at the conjunction between standard LF band and HF band in 53 healthy volunteers. In our study, we controlled the breathing rate (12 breaths min^{-1}) by instructing participants to breathe following a visual guide (Elite HRV, Asheville, North Carolina, USA) to help minimize respiratory rate factor on HRV parameters between visits and participants.

Furthermore, it has been shown that compared to spontaneous breathing, voluntary controlled 15 breaths min^{-1} increased the HF parameter and slow breathing rate decreased HF and increased LF parameters (272, 273). On the other hand, Patwardhan *et al.* (274) showed that higher breathing rate (18 and 21 breaths min^{-1}) created a greater decrease in HF parameter compared to spontaneous breathing in the resting condition. Therefore, respiration rate may influence the changes of HF and LF parameters and it may misinterpret the changes of cardiac parasympathetic and sympathetic modulation. Also, in our review, the included studies where lower exercise intensities were performed used a higher training volume, and the included studies where higher exercise intensities were performed used a lower training volume. All these factors should be taken into consideration when interpreting the outcomes of our meta-analysis. Furthermore, our results

indicated that there is a direct relationship between higher training volume and greater cardiac sympathetic activation and withdrawal of cardiac parasympathetic modulation.

7.1.2.4. Training volume

There was a significant difference between subgroups based on training volumes in the RMSSD, LF(nu) and HF(nu) parameters. Our results are consistent with the findings of Figueiredo *et al.* (227) who suggested that higher training volume increases the recruitment of additional motor units, thus minimizing the likelihood of muscular failure during the concentric phase of lifting (concentric failure) and triggering a progressive activation of the cardiac sympathetic modulation (275). Moreover, our SMD results revealed that higher training volume had a greater effect and lower training volume had the lesser effect on RMSSD, HF(nu) and LF(nu) parameters.

Our results indicated that higher training volume produces a greater activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation. In other words, when the human body experiences a higher level of resistance training volume, the magnitude of activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation is higher than it is with lower training volume. On the other hand, previous studies have reported that a low volume of high-intensity resistance training greatly improves strength, muscle size (276, 277), force production and rate of force development (278) compared to a high volume of moderate- or low-intensity resistance training. Thus, our meta-analysis suggests that a low training volume of high-intensity ARE would enable athletes to have the optimal training load or stimulus without creating a large change in cardiac autonomic modulation, thus allowing for an early recovery without ultimately sacrificing training adaptation and performance.

7.2. STUDY 2

The second study in this PhD thesis is based on some of the findings of the first study. To the best of the authors' knowledge, most studies that examine resistance training and recovery limit the comparison between pre-resistance training level with post-training session time points (e.g., up to 6, 24 and 48 hours) (120, 153, 279-283). However, on a practical level, many trainers who use the concept of periodization strongly believe that microcycles are the most important period because daily training interventions form the basis of the overall training plan. The training microcycle usually lasts about a week, and within these training cycles, athletes are performing several training sessions. Therefore, when it comes to recovery, it is important to understand the changes and recovery during the whole training microcycle and not just in one training session. Thus, Study 2 had three main objectives, the first of which was to evaluate and compare the changes and recovery of HRV parameters and other (objective and subjective) responses induced by strength training and power training under the fatigue conditions within the micro training cycle. The second objective was to evaluate and compared the changes and recovery of HRV parameters and other objective and subjective responses induced by different training loads (Exercise intensity (% 1RM) × Training volume) of strength and power training modalities under the fatigue conditions within the micro training cycle. The final objective was to identify the optimal training loads based on HRV parameters, need to maintain adequate recovery within the micro training cycle in strength and power training modalities. The following discusses the major results of each objective.

With respect to the findings of the first objective, during the microcycle, an intensive fatigue session (M-BEAST) and subsequent ARE training session (strength or power resistance training modality) negatively affects the cardiac autonomic activity by decreasing the cardiac parasympathetic modulation (pNN50, Ln RMSSD, HF(nu), SD1, SD2) and overall autonomic modulation (SDNN, TP) while increasing the cardiac sympathetic modulation (LF(nu), LF/HF

ratio) and stress index (SS) level. In addition, the strength training modality created higher training stress on cardiac autonomic modulation (greater activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation) compared to the power training modality in the subsequent ARE training session. These changes recovered sooner (although not fully) in the power training modality compared to the strength training modality within the microcycle. Interestingly, LF(nu), HF(nu) and LF/HF ratio parameters showed different indications (and to some extent contradictory) of cardiac sympathetic modulation, cardiac parasympathetic modulation and cardiac sympathovagal balance, respectively, compared to other HRV parameters indications.

Performance markers (BP RPP, CMJ height and CMJ RPP) showed impaired performance levels after both the intense fatigue session and subsequent ARE training session, suggesting the occurrence of physical fatigue, which gradually returned to baseline values during recovery. Specifically, the strength training modality created higher physical fatigue (worser performance) than power training modality and that this fatigue recovered sooner with power training modality compared to strength training modality within the micro training cycle (24H following the ARE session). In addition, a similar pattern of change was observed in the HRV parameters, except for LF(nu), HF(nu) and LF/HF ratio.

Neuromuscular fatigue level can be determined by the changes in MVC peak force and RFD^{200MVC} level (248, 279, 284-286). A reduction of MVC peak force and RFD^{200MVC} following the intensive fatigue session and subsequent ARE training sessions showed that neuromuscular fatigue was presented. Similar to the aforementioned markers, the strength training modality created a higher level of neuromuscular fatigue than the power training modality, and these changes recovered sooner in the power training modality compared to the strength training modality 24H following the subsequent ARE session. Central fatigue (CAR variable) was present following the intensive fatigue session and subsequent ARE training session. However, central fatigue recovered to baseline 24H following the

ARE power session, whereas it did not after the strength training modality. However, peripheral fatigue markers (RFD^{tet}, RFR^{tet}, Twitch force, Tetanic force, twitch/tetanic ratio and $T_{1/2}$) showed neither an overall treatment effect nor an overall time effect suggesting that both training modalities did not significantly affect the respected peripheral fatigue levels.

With regards to the perceptual responses, muscle soreness (DOMS) increased following the intensive fatigue session and subsequent ARE training session. Specifically, the strength training modality created a higher level of muscle soreness compared to the power training modality. Muscle soreness did not recovered after 24H following the ARE sessions and remained higher after strength training compared to power training. Interestingly, the total mood disturbance (POMS) level remained unchanged following the intensive fatigue session and subsequent ARE training session. Perceived exertion (BORG) and blood lactate concentration results showed an increase following the intensive fatigue session and subsequent ARE training session. Interestingly, perceived exertion and blood lactate concentration levels were higher after strength training compared to power training.

With regards to the findings of the second objective, 100% load of strength and power training sessions showed the highest training stress and 50% load of strength and power training showed the lowest stress on the cardiac autonomic system. Most importantly, in strength training modality, the cardiac autonomic modulation recovered to baseline sooner following 50% training load and 100% training load took longer to return to baseline after both strength and power training sessions within the microcycle. Similar results were observed in the performance parameters and neuromuscular fatigue (central and peripheral) variables where 50% training load returned to baseline earlier than with 100% training load. Although some monitoring markers ((Strength modality: TP, BP RPP, MVC peak force, RFD^{MVC200}, CAR, DOMS), (Power modality: TP, CAR)) did not show a complete recovery within the microcycle, However, 50% training load

showed a better recovery level, and 100% training loads were the least recovered among the three training loads in those monitoring markers. Overall, these results demonstrate that most of the HRV parameters (except LF(nu), HF(nu) and LF/HF ratio) are sensitive to the training stress of the different training loads of the strength or power resistance training modalities and recovery periods.

Results from the third objective revealed that 75% of strength training load and 100% power training load provided not only maximum training stress but also adequate recovery at the end of the microcycle based on HRV parameters (e.g., Ln RMSSD).

7.2.1. Acute responses following the intensive fatigue session and ARE session

The intensive fatigue session was conducted using the M-Beast protocol (262), which was adapted from a football-specific fatigue protocol simulating a player's performance during a soccer match, to induce fatigue in participants at the start of the microcycle. The present study showed a decrease in cardiac autonomic activity and performance levels and an increase in neuromuscular fatigue, biochemical and psychological stresses following the intensive fatigue session.

A previous study in 10 middle-aged males demonstrated a decrease in cardiac parasympathetic modulation (RMSSD, HF), overall autonomic modulation (SDNN, TP) and an increase in cardiac sympathetic modulation (LF/HF) following a soccer match (287). Several studies also indicated a decrease in cardiac parasympathetic modulation (Ln RMSSD, HF(nu)), overall autonomic modulation (TP) and an increase in cardiac sympathetic modulation (LF(nu), LF/HF ratio) after an endurance exercise session (219, 224, 288, 289). For example, Nuuttila *et al.* (289) demonstrated a decrease in cardiac parasympathetic modulation (Ln RMSSD) following four different endurance sessions (90 min low-intensity, 30min moderate-intensity, 6 × 3 min high-intensity interval and 10 × 30s supramaximal-intensity interval exercises on a treadmill) in 24 recreationally endurance-trained male participants. Heffernan *et al.* (288) reported that following (post 30 min) the

30-min of continuous upright stationary cycling at 65% of peak oxygen uptake session, cardiac parasympathetic modulation (HF(nu)), overall autonomic modulation (TP) decreased, and cardiac sympathetic modulation (LF(nu), LF/HF ratio) was increased compared to baseline values in 14 male participants. Moreover, Kliszczewicz *et al.* (224) observed a decrease in cardiac parasympathetic modulation (Ln RMSSD and Ln HF) following (post 15 -30 min) 20-min treadmill running in 85% of their HR_{max} session in 10 physically fit males and Teixeira *et al.* (219) demonstrated a decrease in cardiac parasympathetic modulation (HF(nu)) and an increase in cardiac sympathetic modulation (LF(nu), LF/HF ratio) after performed 30 min of exercise on a cycle ergometer at 75% of VO_2 peak in 20 young normotensive participants (10 women and 10 men).

These studies show that intensive fatigue or endurance exercise sessions reduce cardiac autonomic modulation. Similar to these findings, our intensive fatigue and ARE sessions also showed a decrease in cardiac parasympathetic modulation and cardiac autonomic modulation and an increase in the cardiac sympathetic modulation following the ARE session. For more discussion on cardiac autonomic activity following an ARE session, please review section (7.1) related to study 1 (Page number 316).

Performance is another important factor affected by fatigue. It has been demonstrated that a decrease in performance capacity can be partially explained by neuromuscular fatigue, muscle soreness, stiffness and energetic stores (1, 79). Similar to our findings, previous studies have also demonstrated a decrease in performance markers (CMJ, BP performance) following acute endurance exercises (286, 289-291) and ARE session (280, 292, 293). In relation to endurance sessions, Brownstein *et al.* (286) studied 16 male semi-professional soccer players after performing a 90-min soccer match and observed that CMJ performance decreased. Leeder *et al.* (291) showed that after performing repeated intermittent-sprint exercise (Loughborough Intermittent Shuttle Test) session, CMJ performance decreased. Furthermore, Wiewelhove *et al.* (290) reported a similar reduction in

CMJ performance after sprint interval training session (4 × 6 × 5 sec sprint running session) in 16 well-trained intermittent sport players. Also, Gonzalez-Badillo *et al.* (292) demonstrated that CMJ performance significantly reduced after performing ARE session (BP and squat exercises, 4 sets of 8 repetitions or 4 sets of 4 repetitions with 80% 1RM, 2min rest between sets), Flatt *et al.* (280) showed a reduction in CMJ and BP performance following an ARE session, consisting of 6 sets to failure at 90% of 10 RM in squat, BP and pull-down exercises. Neuromuscular fatigue may be the main mechanism responsible for the decrease in performance markers, as the CMJ test has been established as a measure for neuromuscular fatigue (16, 294). Previous studies demonstrated that a CMJ is the most reliable and valid test for evaluating the explosive power production of the lower body (295, 296), and researchers commonly use the CMJ test to monitor neuromuscular fatigue and recovery status (297). CMJ without arm swing test is more sensitive in detecting acute changes in neuromuscular fatigue and athlete readiness compared to CMJ with arm swing (298, 299).

In the present study, MVC peak force and RFD^{200MVC} data showed that neuromuscular fatigue was presented following the intensive fatigue session and ARE sessions and suggests the probable mechanism for the reduction in the performance markers. Previous studies that examined intensive fatigue sessions (286, 291) and ARE sessions (279, 285) also demonstrated a decrease in MVC peak force and RFD^{200MVC}. In relation to intensive endurance sessions, Brownstein *et al.* (286) showed that MVC and RFD values decreased after performing a 90-min soccer match in 16 male semi-professional soccer players. Moreover, Leeder *et al.* (291) demonstrated that MVC performance decreased after performing repeated intermittent-sprint exercise (Loughborough Intermittent Shuttle Test) session. Thamm *et al.* (279) reported a decrease in MVC peak force and RFD^{200MVC} following (1H after the ARE session) an ARE session consisting of five sets of 10 repetitions at 70% of 1RM with 2 minutes inter-set rest in 10 young men. Similarly, Ahtiainen *et al.* (285) showed a decrease in MVC peak force after performing a ARE

session (4 sets × 12 repetitions at 100% of the 12RM) in 8 strength-trained athletes and 8 non-athletes. Therefore, these studies suggest the presence of neuromuscular fatigue as result of a reduction in contractile function (peripheral fatigue), and/or the capacity of the central nervous system (central fatigue) (286). Our present study showed that CAR (central fatigue) significantly declined following the intensive fatigue session and ARE session. However, there was no change in peripheral fatigue markers. In general, fatigue caused by prolonged activity has shown to decrease voluntary activation (300). Contrary to our findings, high-intensity exercise can result in contractile mechanisms disturbances (300, 301). Similarly, a decrease in power output of dynamic activities was also associated with peripheral fatigue, especially with muscle shortening velocity (302, 303). Furthermore, fatigue decreased the activity of the central motor drive, consequently affecting the ability to generate muscle power and RFD (302, 304-306).

When considering an athlete's fatigue, it is important to consider the perceptual responses as well. In this study, the VAS was used to determine the participants' perception of muscle soreness (DOMS), which increased at the end of intense fatigue session, and it increased further after ARE session. These findings agree with the previous findings related to intensive endurance training that showed, participants' perception of muscle soreness was increased after performing 10 × 30s supramaximal-intensity interval or 90 min low-intensity running on a treadmill (289). Leeder *et al.* (291) also showed an increased perception of muscle soreness after performing repeated intermittent sprint exercise session (Loughborough Intermittent Shuttle Test) in 8 well-trained male team-sport athletes. Moreover, Brownstein *et al.* (286) showed an increased perception of muscle soreness following a 90-min soccer match in 16 male semi-professional soccer players. With regards to the ARE session, Flatt *et al.* (280) and Chen *et al.* (153) demonstrated that perception of muscle soreness was increased following six sets to failure with 90% of 10 repetition maximum in the squat, BP, and pull-down exercises and after performing 2-hour weight training program (4

exercises (back squat, seated shoulder press, dead lift, and front squat) were used and intensity for each training started from 60% maximal effort 3 times, 70% maximal effort 3 times, 80% maximal effort 3 times, 90% maximal effort 2 times, 95% maximal effort 1 time with ~90-second rest on each pull), respectively. Additionally, blood lactate concentration increased following the intensive fatigue session and ARE session, suggesting the accumulation of toxic metabolic waste products inside the muscles and also the higher contribution of anaerobic metabolism for energy production (307). The accumulation of lactic acid may explain the acute muscle pain rather than muscle soreness following the intensive fatigue session and ARE session (308). On the other hand, the perceived exertion scale showed that participants identified that the intensive fatigue session was harder than the ARE session. Interestingly, total mood (POMS questionnaire) were similar between and within the intensive fatigue and ARE sessions.

In summary, the intensive fatigue session and ARE session acutely alters cardiovascular, neuromuscular, metabolic, performance and perceptual responses. Identifying and quantifying these individual training responses is important in order to monitor and adjust the training load. Most importantly, the present study results demonstrated that some of the HRV parameters (pNN50, SDNN, Ln RMSSD, SD1 and SS) used have the capability of identifying and quantifying the training responses related to the intensive fatigue session and ARE session.

Acute changes of the present study also showed that the same training session affects differently at the individual and intra-individual level. Similarly, other studies have shown that the effect on the participant varies according to the protocol of the training session, as well as the training modality (120, 309). Therefore, the next section discusses the acute effects of different training modalities (strength vs. power) on cardiovascular, neuromuscular, metabolic, performance and perceptual responses.

7.2.2. Comparison of acute effects between strength training and power training modalities

The present study showed that the strength training modality generated greater activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation, a decrease in performance, higher neuromuscular fatigue, biochemical and psychological stress compared to the power training modality. These changes suggest that strength training had a greater effect on cardiovascular, neuromuscular, metabolic, performance and perceptual responses than power training. It is important to note that both strength (S100) and power (P100) training modalities had the same training volume (4 sets × 5 repetitions). The only differences between modalities were that strength training consisted of 90% of 1RM with 4 minutes rest between sets and the power training was executed with an optimal load for each exercise with 3 minutes rest between sets.

The activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation following ARE sessions in our study are in line with the study conducted by Lima *et al.* (265), who reported a greater increase in cardiac sympathetic modulation 70% 1RM compared to 50% 1RM exercise session. However, our meta-analysis study and several other studies (41, 216) indicated that there was no significant difference of effect from the different intensities. This controversy may be explained by the variations in training volume used in each study, where studies that utilised lower exercise intensities had higher training volume and studies that used higher exercise intensities performed lower training volume. However, our training volume was consistent between the different intensities, as well as in the study conducted by Lima *et al.* (265). Therefore, our present findings suggest that changes in cardiac autonomic modulation following the ARE may depend on exercise intensity if training volume is held constant. On the other hand, our meta-analysis data suggested that there was no significant difference of effect for the cardiac autonomic modulation when considering the rest between sets (< 2 min, equal to 2 min or > 2 min. Moreover, Goessler *et al.* (231)

also suggested that at least 2 min of rest between sets reduces the postexercise cardiac sympathetic modulation following ARE. In the present study, 4 min of rest between sets was used for strength and 3 min for the power training modality, which was recommended by the NSCA guidelines (1).

We observed a greater reduction in performance (CMJ height, CMJ RPP and BP RPP) following strength training modality compared power training modality. Helland *et al.* (293) also showed greater impairment in CMJ performance following strength (5 RM) compared to power (50% of 5 RM) resistance training sessions. Similarly, Freitas *et al.* (241) observed a greater reduction in CMJ and BP power output following a high-resistance circuit training session that used 85% (6RM) of 1RM compared to a power circuit training with 45% of 1RM. According to the present study, the data demonstrated higher level of neuromuscular fatigue (MVC peak force and RFD^{200MVC}) and central fatigue (CAR) following the strength training than power training modality, suggesting that these are the probable mechanisms responsible for the reduced performance markers (100-103). On the other hand, performing high-intensity resistance training results in an increased rate of energy consumption through phosphagen breakdown and activation of glycogenolysis, which consequently reduces ATP and muscle glycogen concentrations of the body compared to low-intensity resistance training (310). Therefore, a higher level of neuromuscular fatigue and limitation of energy supply to the muscles may contribute to the lower performance in strength training modality compared to the power training modality (100, 302, 310).

Similarly, perceived exertion (RPE scale) was significantly higher following the strength training modality compared to the power training modality. This finding may largely be explained by the greater requirement of muscle tension development, thus increasing the demand for neuromuscular activation (motor unit recruitment and firing frequency) (260). Moreover, our perceived exertion findings agree with Day *et al.* (260), who reported that the resistance training protocol with higher intensity (90% of 1RM) elicited higher perceived exertion level

compared to 70% of 1RM and 50% of 1RM. Lagally *et al.* (307) also demonstrated higher perceived exertion following one set of biceps curl exercise at 90% 1RM compared to 30% and 60% 1RM. We showed higher blood lactate concentration with the strength training modality compared to power training modality, which coincides with the studies conducted by Lagally *et al.* (307), Thornton *et al.* (311) and DaSilva *et al.* (312). This suggests that the accumulation of toxic metabolic waste products inside the muscles and also the contribution of anaerobic metabolism to energy production may have been greater in the strength training compared to power training modality (307). Interestingly, there were no differences in POMS questionnaire and DOMS between the training modalities.

In summary, the strength training modality generated greater acute alterations to cardiovascular, neuromuscular, metabolic, performance and perceptual responses compared to the power training modality. Interestingly, HRV parameters like pNN50, SDNN, Ln RMSSD, TP, SD1, SD2 and SS showed the capability of identifying and quantifying the training effect of strength and power training modalities similar to other well established fatigue/training load monitoring tools in the sports field.

7.2.3. Recovery following the intensive fatigue session and ARE sessions

It is well-recognized that recovery is a process of restoration of the physiological and psychological condition following an intervention (151). Many systems are involved (removing or recycling the accumulated metabolites in skeletal muscles, body temperature and fluid levels returning to previous levels, and activating neuroendocrine-immune responses to restore homeostasis) to return the body to the previous level of homeostasis or even a higher level of homeostasis, as also known as the supercompensation stage (1, 93, 151, 313). The ANS regulates these physiological processes at different levels, and the cardiovascular system plays a key role in the recovery process (151). Most importantly, the recovery of cardiac autonomic modulation is associated with the recovery of cardiovascular

homeostasis. Therefore, monitoring the cardiac autonomic modulation after exercise ensures an adequate balance between training stress and recovery for optimal microcycle.

The present study showed that participants did not recover to their respected baseline value in (cardiovascular, neuromuscular, metabolic, performance and perceptual responses) 48H after the M-BEAST fatigue session. The recovery of cardiac autonomic modulation was similar to the results presented by Furlan *et al.* (314) and Niewiadomski *et al.* (315). For example, Furlan *et al.* (314) demonstrated that 48H after a single bout of a maximal dynamic exercise session, cardiac sympathetic modulation (increased) and cardiac parasympathetic modulation (decreased) did not recover to baseline values. Niewiadomski *et al.* (315) also showed a suppression of cardiac parasympathetic modulation 48H following 30-min of submaximal exercise (85% HR_{max} intensity) on a cycle ergometer. Moreover, a systematic review conducted by Stanley *et al.* (151) reported that, in a given aerobic-based training session, cardiac autonomic modulation returned to baseline up to 24H using low-intensity, between 24 to 48H with threshold intensity and at least 48H following high-intensity exercise. Similarly, Leeder *et al.* (291) showed impaired performance, increased neuromuscular fatigue and muscle soreness even after 48H following the Loughborough intermediate shuttle test. Our findings agree with these findings, and it is clear that high-intensity aerobic session induced fatigue and required more than 48H to recover the cardiac parasympathetic modulation, as suggested by Stanley *et al.* (151).

Similar to our study results, gradual domination of cardiac parasympathetic modulation and withdrawal of cardiac sympathetic modulation, and return to baseline values around 24H to 48H following the ARE session were observed in several studies (153, 280, 281). For an example, Flatt *et al.* (280) demonstrated that Ln RMSSD parameter decreased (i.e., withdrawal of cardiac parasympathetic modulation) and gradually recovered close to pre-training value 48H after performing an ARE protocol consisted of six sets to momentary muscular failure in

four exercises (90s rest between sets and 2 min rest between exercises) with 90% of 10RM. Also, Chen *et al.* (153) observed that cardiac parasympathetic modulation significantly decreased and cardiac sympathetic modulation marginally elevated within 24H and returned to baseline 48H following 2 hours of resistance training session in 7 weightlifters. In contrast, Thamm *et al.* (279) investigated the effects of two ARE protocols (i: 5 sets, 10 repetitions at 70% of 1RM with 3 min rest between sets and ii: 15 sets, 1 repetition at 100% of 1RM with 3 min rest between sets) and reported that RMSSD significantly decreased while other HRV parameters (LF, HF, LF/HF) remained unchanged after each protocol. Interestingly, RMSSD returned to baseline within 30 minutes after these ARE sessions, suggesting the recovery of cardiac parasympathetic modulation. However, there was a significant increase in LF 48H after the 2nd ARE session, which may indicate a significant increase in cardiac sympathetic modulation. In general, it is important to note that there is controversy regarding the interpretation and accuracy of the LF parameter, which will be discussed later in this section. Another point to note here is that the aforementioned studies investigated the effects of a single ARE session and the time course of recovery. However, to the best of our knowledge, our study is the first to investigate the effects of the intensive fatigue session which was followed by an ARE training session within the microcycle. Nevertheless, there is one study that is conducted a 6-day overload microcycle with eleven strength, or high-intensity interval training sessions and demonstrated that Ln RMSSD parameter remained decreased compared to pre-training values until 48H from the last training session (281). These findings suggest a prolonged decrease in cardiac parasympathetic modulation and increase cardiac sympathetic modulation in young, healthy adults following ARE session.

Several studies, including the present study, monitored changes in HRV parameters for a prolonged amount of time (24H -72H) after the ARE session. In our study, post-30 minutes showed the lowest cardiac parasympathetic modulation and post-48H demonstrated the highest level when comparing the pre-

ARE value. A similar pattern was reported in Flatt *et al.* (280) (Lowest - 10 min after ARE, Highest – 48H after ARE) and Chen *et al.* (153) (Lowest - 10 min after ARE, Highest – 72H after ARE) studies. However, there are other studies that presented contradictory or no significant changes in cardiac parasympathetic or sympathetic modulations following an ARE session (41, 225). This controversy may be because these studies measured immediately following the ARE session (which may be influenced by breathing pattern) and not following more time points in the recovery stage.

With regards to performance and neuromuscular markers, our study showed that they returned or were closer to baseline values around 24H following the ARE session. Neuromuscular fatigue level is influenced by central (decreased motoneuron firing frequency and/or a number of functioning motor units) and peripheral (impaired muscle contractile activity, leading to loss of muscle fibre force caused by impaired neuromuscular transmission, impaired excitation-contraction coupling or failure of muscle action potentials and decrease of Ca2+ release from the sarcoplasmic reticulum) factors (316). However, the present study's central and peripheral fatigue data also indicated that there was a significant training effect on central fatigue and neuromuscular fatigue, but not peripheral fatigue. Recovery of performance (CMJ height, CMJ RPP, BP RPP) and neuromuscular markers (MVC and RFD) were similar to the previously reported studies (279, 280, 293). Thamm *et al.* (279) demonstrated that MVC value decreased following the ARE sessions and gradually returning to baseline following 48H. Moreover, Flatt *et al.* (280) also showed there was impairment in performance and neuromuscular markers (CMJ, BP and squat velocity) following an ARE session (six sets to failure with 90% of 10RM in the squat, BP, and pull-down exercises), and those changes returned close to baseline following 48H. Similar results were demonstrated in CMJ height, squat jump peak power and RFD, BP peak power output and several other performance markers after performing two ARE protocols in a randomized cross-over controlled study conducted by Helland *et al.* (293).

Perception of muscle soreness gradually increased throughout the microcycle in both training modalities and remained above the respected baseline (Pre-B) value even after 24H from the ARE session. Similar results were reported by Flatt *et al.* (280) and Thamm *et al.* (279) following the ARE sessions where muscle soreness stayed above baseline after 48H.

The present study showed that between 24H - 48H from the ARE session, most of the recovery markers returned to or was close to baseline values. These study results demonstrate that training intensity and modality plays an important part in the recovery rate following training stresses. Therefore, the next section discusses the comparison of fatigue recovery time between strength and power training modality on cardiovascular, neuromuscular, metabolic, performance and perceptual responses.

7.2.4. Comparison of fatigue recovery time between strength training and power training modalities

Overall, the recovery following the power training modality returned to baseline with the cardiac sympathetic and parasympathetic modulation parameters, performance markers, neuromuscular fatigue and psychological stress compared to the strength training modality. These results indicate that high intensity ARE session creates a higher level of stress on the cardiac autonomic modulation, thus in turn would take more time to remove accumulated metabolites in skeletal muscles, return to normal body temperature, recover fluids to pre-levels and activate neuroendocrine-immune responses to restore homeostasis (1, 93, 151, 313). Thus, this would explain the slow recovery following the strength training modality, whereas a relatively less intensity ARE session (i.e., power training modality) favoured quicker recovery.

Furthermore, there is a higher rate of energy utilization in the strength training modality compared to the power training modality (100, 302, 310, 311). Not only does energy come from muscle glycogen, but also from muscle adenosine

triphosphate (ATP) and creatine phosphate (311). Thus, the recovery process consists of restoring the levels of ATP and creatine phosphate via aerobic metabolism, as well as the redistribution of compartmental ions (e.g., sodium and potassium) and repair tissue damage (311). Resting metabolic rate (RMR) indicators the daily energy needs for an individual while awake in a postabsorptive, thermoneutral state (317, 318). Dolezal (319) and Williamson *et al.* (320) demonstrated that RMR remained higher up to 48H after performing high or moderate-intensity ARE session, which suggests greater muscle damage from a high- or moderate-intensity ARE session and raised energy requirement for the degradation and resynthesis of damaged muscle fibres even up to 48H from the training session (311, 319, 321).

Neuromuscular fatigue recovery is an important factor when considering the performance improvement. Moreover, the magnitude of exercise-induced neuromuscular fatigue also plays a major role in the time to full recovery. In the present study, we made sure that training volume (number of sets and number of repetitions) was the same in both training modalities with similar exercises. Results showed that greater impairment of MVC peak force and RFD^{MVC200} following the ARE session of strength training modality compared to power training modality. This may be because higher concentric force creates greater mechanical stress on muscle tissues (293), whereas higher eccentric force induces neuromuscular fatigue. Thus, this may explain why slower recovery was observed with strength training modality (i.e., higher-intensity) compared to power training modality (i.e., lower-intensity). The concentric phase of the strength training modality was performed at low velocity with higher intensity compared to the power training modality (293, 322-324)

The uniqueness of the present study is that we followed recovery not only after an intensive training session but also after the subsequent ARE session within the microcycle. We've demonstrated that subsequent power training session elicits full recovery compared to the subsequent strength training session. Thus,

monitoring the recovery of the whole microcycle and not just after a single training session is essential, particularly during the competitive season when there are more than one session/game. However, in a periodized training program, the microcycle is dedicated to specific training objective. Previous studies and NSCA guidelines recommend a specific percentage of 1RM range for different resistance training goals (≥85% of 1RM for Strength training and 75-85% of 1RM or optimal load for power training) (1). Therefore, changing the 1RM percentage used for RT exercises or modifying the training modality based on the recovery status from the previous training stress of the athlete in the subsequent ARE session might be problematic and it might be important to consider changing the training volume. Lastly, in our study, HRV may be the non-invasive and field-friendly monitoring tool as they are sensitive to the training stress (i.e., Training load and modality) and recovery timeline (Figure 158 and Figure 159).

7.2.5. Comparison of acute effect and recovery time of fatigue between different training loads

Our meta-analysis study showed a significant effect of ARE training volumes on HRV, where higher training volume had a greater effect and lower training volume had a lesser effect on cardiac autonomic modulation. Interestingly, the meta-analysis demonstrated greater cardiac autonomic stress with a higher number of sets, but the number of repetitions did not significantly HRV parameters. Therefore, in the experimental study, we wanted to examined the training load based on the number of sets (4 sets = 100%, 3 sets = 75% and 2 sets = 50%) in the subsequent ARE session to investigate further the effect of training load on recovery within the microcycle in both strength and power training modalities, as well as to identify the optimal training load to minimize fatigue for the subsequent training.

Figueiredo *et al.* (227) demonstrated that higher training volume creates substantial cardiac autonomic stress following the ARE session. In their study, they

compared 3 different training volumes (1 vs 3 vs 5 sets) consisting of 8 exercises with 70% of 1RM, 2 min rest between exercises and sets and 8 to 10 repetitions per set. The highest volume (5 sets) had the greatest impact on cardiac autonomic modulation. Moreover, Gonzalez-Badillo *et al.* (292) reported that higher training volume elicits a significant decrease in cardiac parasympathetic modulation compared to lower training volume. These findings agree with our results where we observed that S100 and P100 induced greater cardiac sympathetic modulation and lesser cardiac parasympathetic modulation, and in turn, S50 and P50 provoked lower cardiac sympathetic modulation and higher cardiac parasympathetic modulation. Apart from the previously discussed physiological explanations behind these changes, the recruitment of additional motor units may also minimize the likelihood of muscular failure during the concentric phase of lifting (concentric failure) and may trigger a progressive activation of the cardiac sympathetic modulation (275).

In our experimental study, the performance and neuromuscular fatigue markers showed a similar pattern of results as the HRV (Ln RMSSD) parameters (Figure 158 and Figure 159). The highest performance impairment and neuromuscular fatigue were observed with S100 and P100, whereas S50 and P50 showed the lowest impairment in performance and neuromuscular fatigue based on ES analysis. This is in line with Gonzalez-Badillo *et al.* (292), who conducted a study comparing 2 protocols of BP and squat exercises: i) 3 sets with 4 repetitions and ii) 3 sets with 8 repetitions with 80% of 1RM. According to their findings, CMJ height, BP and squat velocities significantly decreased after a high volume training protocol compared to a low volume protocol. However, González-Hernández *et al.* (325) reported that a lower number of sets (3 sets) in an ARE session reduced the movement velocity and CMJ height, suggesting greater performance impairment and neuromuscular fatigue compared to a higher number of sets (6 sets). This controversy may be explained by methodological difference where González-Hernández *et al.* (325) utilized 3 sets of repetitions to muscle failure versus 6 sets of

half the maximum possible number of repetitions per set with the 10RM while maintaining the equal training volume. We used a constant number of repetitions and manipulated the training volume using the number of sets in the ARE session.

When considering perceptual responses, muscle soreness and perceived exertion were higher after S100 and P100 and lower after S50 and P50 ARE training sessions. Interestingly, DOMS markers did not recover to the baseline values even after 24H from the ARE session. However, DOMS remained closer to the respected baseline value in lower compared to the higher training volumes in both training modalities. This is in line with Bartolomei *et al.* (326),who conducted a study with 12 experienced resistance trained men comparing 2 protocols of squat exercises: i) 8 sets of 3 repetitions (Low volume) at 90% of 1RM with 3 min rest between sets (Low training load) and ii) 8 sets of 10 repetitions (High volume) at 70% of 1RM with 3 min rest between sets (High training load). According to their findings, muscle pain and soreness did not recover to the baseline values even after 24H from the ARE session. Interestingly, their results shown that muscle pain and soreness level remained closer to the baseline in lower compared to the higher volume/training load protocol. However, it's important to mention that in their study exercise intensity was not equal (90% vs 70% of 1RM). Yet, training load showed similar results as training volume. On the other hand, a study conducted by Paschalis *et al.* (327) reported that both higher (isokinetic quadriceps eccentric exercise - 12 sets of 10 maximal eccentric voluntary efforts with 2 minutes rest between sets.) and lower (continuous eccentric exercise at 50% of the individual subject's eccentric peak torque until volume was equal to higher intensity protocol) exercise intensity protocols increased DOMS and remained above the respected baseline values even after 24H following the training session, suggesting delayed recovery. Furthermore, their results showed that DOMS remained closer to the respected baseline value with the lower compared to higher intensity protocol. When considering these studies, it is plausible to propose that training volume plays an important role on the effect and recovery of the DOMS variable following

an ARE session. The POMS questionnaire did not show any significant difference in the total mood difference between the trials in both ARE modalities. The smaller sample size might be problematic when utilizing these types of questionnaires to understand the proper psychological picture.

When considering the recovery time from training stresses, it was clear that not only training intensity /modality but also training volume plays a crucial part in both training modalities. In addition, our results clearly demonstrate that lower training volume tended to recover sooner than higher training volumes.

7.2.6. Controversial interpretations and accuracy of HRV variables

Overall, HRV is a non-invasive, indirect method for monitoring cardiac autonomic modulation. It has especially useful for monitoring the training stress and recovery of high-performance athletes (151, 328-331). There are a number of HRV parameters that reflect cardiac parasympathetic modulation, cardiac sympathetic modulation, as well as the function of the overall autonomic modulation (155, 161-163, 169, 176-178, 180, 182, 187, 188). However, our study presented contradictory findings between LF(nu), HF(nu) and LF/HF ratio parameters compared to other HRV parameters presented in the Study 2. As an example, Ln RMSSD and HF(nu) parameters often are considered as a reflection of cardiac parasympathetic modulation activity (161, 169). Yet, when comparing the recovery of strength and power training modalities (S100 vs P100) following the ARE session, Ln RMSSD parameter recovered sooner with P100 than S100 modality, but not in the HF(nu) parameter. Similar incidents have been shown in several studies (225, 279). For example, Thamm *et al.* (279) showed that RMSSD value was higher post-30 min and post-1H of the ARE session with the maximum strength (MAX) protocol compared to hypertrophic (HYP) protocol. However, HF parameter of HYP protocol remained higher compared to the MAX protocol at the same aforementioned time points. Furthermore, 24H and 48H after the ARE session, data showed that these indications were the other way around (i.e., higher

RMSSD parameter with HYP protocol compared to MAX protocol but higher HF parameter with MAX protocol than HYP protocol). Similar controversy was observed study conducted by Figueiredo *et al.* (225) in 11 prehypertensive men with at least 6 months of RT experience. Their study protocol consisted of similar strength training protocols with 2 different rest intervals between sets and exercises (1 min vs 2 min rest intervals). Their results showed that RMSSD parameter recovered to the respective baseline values, but not for HF(nu) parameter, in both protocols 60 min following the strength training session.

One explanation may be respiration control because it has an effect on LF(nu), HF(nu), and LF/HF ratio during HRV measurements (38, 169). Also, the interpretation of some parameters may contribute to the controversy. Some authors (165, 332) consider the LF and LF(nu) parameters to reflect cardiac sympathetic modulation marker, while others (333, 334) believe that it reflects both cardiac sympathetic and parasympathetic modulation. Moreover, some studies suggest that LF/HF ratio represents sympathovagal balance (133, 161, 219, 335-338). One of the reasons for this controversy is that some studies (339, 340) report a decrease in the LF parameter during conditions such as exercise and myocardial ischemia, which goes against the expected increase in cardiac sympathetic modulation in these situations (133, 136, 161, 335, 339-342). Similarly, the suggestion that the HF parameter indicates cardiac parasympathetic modulation has also been challenged by some studies (335, 343, 344).

There are several assumptions to consider: i) cardiac sympathetic modulation is a major factor responsible for the LF peak and cardiac parasympathetic modulation is exclusively responsible for the HF peak of the heart rate power spectrum, ii) disease or physiological challenges provoke reciprocal changes in cardiac sympathetic and parasympathetic modulation and iii) there is a simple linear interaction between the effects of cardiac sympathetic and cardiac parasympathetic modulation on HRV. Pagani *et al.* (345) proposed that LF/HF ratio could be used to quantify the sympathovagal balance (165, 346). However,

this proposal was challenged by other researchers (335, 339, 347, 348), as they argued that: i) LF parameter does not purely reflect the cardiac sympathetic modulation, ii) cardiac parasympathetic and sympathetic modulations are complex, non-linear, and frequently non-reciprocal and iii) confounding respiration mechanics and resting HR creates uncertainty regarding cardiac parasympathetic and sympathetic modulation's contribution to the LF/HF ratio (169).

When considering the use of HRV parameters for training load and fatigue monitorization, accuracy of the information provided by a particular testing method is the most important factor. Even though monitoring HRV is non-invasive, comfortable, affordable, and field user-friendly, interpretation of some HRV parameters (HF(nu), LF(nu), LF/HF ratio, etc..) is still controversial and may be affected by external factors (like breathing pattern of the person during the measuring time). Therefore, it's more stable HRV parameters that are less affected by respiration fluctuation should be considered for monitoring the training load and fatigue status of the athletes.

7.2.7. Use of Ln RMSSD parameter for training load and fatigue monitoring

Some researchers suggest that HRV is not widely accepted to monitor cardiac sympathetic modulation but for cardiac parasympathetic modulation (133). Among the HRV parameters, pNN50, HF(nu) and RMSSD parameters are widely used. As we discussed in the prior section, use of HF(nu) parameter for cardiac parasympathetic modulation is controversial. Between pNN50 and RMSSD, RMSSD is the most widely used parameter and the primary time domain parameter is used to evaluate the cardiac parasympathetic modulation (169). The recommended minimum recording time is 5 minutes (short-term), but studies have shown that ultra-short-term duration, like 10s, the 30s and 60s, is reliable (169, 171-174), which is more time effective field-use. Most importantly, the RMSSD parameter is less affected by fluctuations in respiration and is a more stable

parameter. Therefore it is a more robust indicator of cardiac parasympathetic effect (175, 176) and a promising method for monitoring individual adaptation to training at resting and during post-exercise recovery conditions (173). In summary, RMSSD parameter is a suitable monitoring tool for investigating and quantity the training effect, stress or training load and recovery.

7.2.8. Association between Ln RMSSD parameter and other subjective and objective markers

The present study showed that there were several significant correlations with the changes in Ln RMSSD parameter (see Appendix 13.2.3 Table 25 - 30, which shows the results of association results). However, correlations were inconsistent across recovery time course with the changes in Ln RMSSD.

Limited studies investigated the association between the changes in performance, neuromuscular, central fatigue, peripheral fatigue, and perceptual markers with changes in Ln RMSSD parameter following an ARE session and during the recovery course. Interestingly, Gonzalez-Badillo *et al.* (292) reported a significant, yet moderate correlation (r = -0.55) in a relative loss of CMJ height only at Post-6H with changes in Ln RMSSD parameter following ARE sessions. Their study analysed the time course of recovery up to 48H following two resistance exercise protocols (i: 3 sets of 4 repetitions and ii: 3 sets of 8 repetitions, with 80% of 1RM in BP and squat exercises) with nine physically active male volunteers. Flatt *et al.* (280) showed no significant association between the changes in neuromuscular performance (CMJ peak power and mean concentric BP and squat velocity with load corresponding to 1.0 ms^{-1}), perceptual recovery (perceived recovery and soreness scales) markers with the changes in Ln RMSSD parameter after performing an ARE session consisting of 6 sets to failure with 90% of 10 RM in the squat, BP, and pull-down exercises. These 10 male adults had more than one-year of RT experience and, during the study, above recovery markers were tested before, after, 24H and 48H after the ARE session.

It is clear that these studies, including the present study, show inconsistent and contradictory findings related to the association between the changes in stress and recovery markers with Ln RMSSD parameters. This may be because there are limited studies that have investigated this area and that there are methodological differences. Thus, further investigation examining the association between the changes in Ln RMSSD and other stress and recovery markers are needed to gain a better understanding. Even though there was no significant, strong and consistent association between the changes in Ln RMSSD and other markers in the present study, we did observe an association between the performance and neuromuscular fatigue markers with Ln RMSSD parameter (Figure 158 and Figure 159).

7.2.9. The optimal training load for strength and power training for adequate recovery within microcycle

There is evidence that lower resistance training volume has the capability to increases muscle strength (349, 350). Furthermore, Carpinelli *et al.* (351) stated that there is little scientific evidence to suggest that greater volume of resistance exercise is necessary to increase strength and induce hypertrophy. Moreover, a meta-regression study conducted by Krieger *et al.* (350) reported that there was no significant difference of effect between 1 set per exercise and 4 to 6 sets per exercise or between 2 to 3 sets per exercise and 4 to 6 sets per exercise. Nonetheless, 2 to 3 sets per resistance exercise created significantly greater ES than 1 set and associated with 46% greater strength gains (350). These findings suggest that manipulation of the number of sets as conducted in the present study may not significantly affect the performance improvement while maintaining the training goals of the resistance-training program.

Based on the Ln RMSSD parameter results presented in our study, the ideal strength training load would be 75% (90% of 1RM, 3 sets, 5 repetitions with 4 min rest between sets) and 100% power training load (optimal load, 4 sets, 5 repetitions with 3 min rest between sets) if the athlete is fatigued in order to achieve adequate

recovery within the microcycle (e.g., for the subsequent ARE session 48H after an intensive fatigue session). These optimal loads would ensure adequate training stimulus for adaptation while accounting for full recovery. However, the effectiveness of such a selection will need to be explored in future studies.

VIII - LIMITATIONS

VIII. LIMITATIONS

There were some limitations in the presented studies, which may affect the interpretation of the reported results.

Regarding Study 1, there were a limited number of studies included in the systematic review with meta-analysis due to the lack of research on ARE interventions that measured HRV parameters as an outcome variable. Following an extensive search of the literature through electronic databases, we reviewed reference lists of books written on the subject to identify more studies. A larger number of studies on ARE interventions that investigated HRV parameters could have generated more accurate results. Future studies should try to fill this knowledge gap in the literature. In addition, some of the included studies had a small sample size (range: 8 - 34), which may not have provided a complete understanding of how HRV parameters are affected by ARE. However, the effect sizes of the meta-analysis normalized the sample size effect and gave a clearer representation of the effect of ARE on HRV parameters.

Moreover, there was a presence of heterogeneity in several moderating factors (RMSSD, HF(nu), LF(nu) and LF/HF ratio) pre-post intervention studies, which was likely due to the methodological diversity (differences in the way that studies were conducted) of the included studies. However, we evaluated the methodological quality using the "Study quality assessment tools" provided by the National Heart, Lung, and Blood Institute (235) and the quality of the studies were high for pre-post interventions (8.18 ± 0.53, out of a possible 12 points). Furthermore, we conducted subgroups analysis to explore the heterogeneity according to the guidelines of Cochrane Handbook for Systematic Reviews of Interventions (233).

Similarly, different equipment, software ((Equipment: Polar HR monitors (RS800cx, RS800, S810i), ECG (TEB, D10) monitor, Modified CM5 configuration with a Biopac data acquisition system), (Software: Kubios HRV analysis, Matlab, Acqknowledge, WinCPRS)) and data analysing methods (Abnormal beat-to-beat interval identification methods and Ectopic/artefacts beats correction methods) were used to obtain HRV parameters in the different studies and this could affect the accuracy of the provided data of the study. However, all the equipment and software used in these studies were well recognised and utilized in past studies to gather and analyse HRV data.

In Study 2, the small sample size in the experimental study may limit the generalizability of the present study findings and may have prevented the identification of potentially significant changes between RT modalities and training loads. Because it will be difficult to find significant relationships from the small sample size, as statistical tests usually require a larger sample size to ensure a representative distribution of the population.

On the other hand, all the tests were performed in sequence before (Figure 7 - 46 minutes to complete, including warm-up) and after (Figure 8 - 30 minutes to complete) the training protocol, which may have affected the results of the testing variables. Specifically, the influence of one test may have effects on the results of a subsequent one. However, the order of the tests was kept the same for all the visits throughout the study for all the participants. Therefore, one test's influence on another may not change (i.e., consistent) throughout the study.

Moreover, HRV parameters provide only an indirect insight into cardiac autonomic modulation. Holter ECG is the gold standard for measuring NN intervals and the analysis of HRV parameters (352, 353). Regular monitoring of HRV parameters using Holter ECG devices in the field and training environment is difficult and impractical for the trainers and coaches. In the present study, we used a Polar H10 HR sensor, validated against the Holter ECG device (243) and a

reliable, more practical, commercially available, cost-effective and user-friendly device for regular use to monitor HRV parameters (353).

Furthermore, it is uncertain whether performance and subjective markers accurately reflect changes in the athlete's fatigue and recovery status. Because participant's intrinsic motivation level and mentality may affect the accuracy of the markers. Therefore, results may under- or over-estimate fatigue or recovery level. However, when conducting the testing of performance markers, we verbally encouraged the participants during every attempt to improve the motivation level and instructed them to fill out the questionnaires (subjective markers) honestly.

IX – CONCLUSIONS

IX. CONCLUSIONS

Based on the results obtained and the objectives proposed by the present doctoral thesis, the conclusions are made below concerning athletes or physically active people with similar characteristics to those presented in each investigation.

9.1. GENERAL CONCLUSIONS

There was a decrease of overall autonomic modulation, withdrawal of cardiac parasympathetic modulation and activation of cardiac sympathetic modulation following an ARE session (after around 30 min) in healthy individuals. Interestingly, there was a greater effect of training volume on the activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation around 30 min after resistance exercises in healthy individuals. Furthermore, the number of sets, the intensity of exercise, and amount of rest between sets played an important role on HRV parameters.

Moreover, the strength training modality created a greater disturbance on the cardiac autonomic modulation by decreasing parasympathetic modulation, overall autonomic modulation and increasing the cardiac sympathetic modulation compared to the power training modality. A similar effect was observed following higher compared to lower training loads in both training modalities. Interestingly, there was greater neuromuscular (mainly central) fatigue and higher performance impairment following strength compared to power training modality, and likewise with higher compared to lower training loads in both training modalities.

Concerning the recovery time from the subsequent training session after an intensive fatigue condition within the micro training cycle, cardiac autonomic modulation following the power training modality recovered sooner than the strength training modality. A similar recovery pattern was observed in

neuromuscular (i.e. central) fatigue and performance markers. Moreover, lower training loads also showed shorter recovery time compared to higher training loads in both training modalities.

Finally, the present results showed that 75% of strength training load and 100% of power training load may be considered the optimal training load to achieve adequate recovery within the microcycle when athletes are still fatigued from the previous training session based on Ln RMSSD (HRV) parameter.

9.2. SPECIFIC CONCLUSIONS

The specific conclusions of the studies comprising the present thesis are presented below.

Study 1

- Overall autonomic modulation was decreased following an ARE session around 30 minutes in healthy individuals
- There was a withdrawal of cardiac parasympathetic and activation of cardiac sympathetic modulations following ARE session
- Higher training volume had a greater effect, and lower training volume had a lesser effect on cardiac parasympathetic and cardiac sympathetic modulations.
- The ARE's number of sets, the intensity of exercise, and amount of rest between sets are moderating factors on HRV.
- Characteristics of the athletes like gender, BMI and training status do not significantly influence the changes in HRV parameters as a response to ARE session.

Study 2

- Strength training modality created a greater decrease in pNN50, SDNN, Ln RMSSD, TP, SD1 and SD2 parameters, and an increase in SS parameter compared to the power training modality.
- Greater performance impairment was shown in BP RPP marker following the strength training modality compared to power training modality.
- HRV parameters (pNN50, Ln RMSSD and SD1), performance markers (CMJ RPP), neuromuscular (including central) fatigue markers ($RFD^{200MVC}$and CAR) related to power training modality returned to the respected baseline values sooner or closer to the respected baseline values within the testing period compared to the strength training modality.
- 100% training load of strength training modality created a greater decrease in pNN50, SDNN, Ln RMSSD, TP, SD1 and SD2 parameters, and an increase in SS parameter compared to 75% and 50% training load of strength training modality trial following the intensive fatigue session
- 100% training load of strength training modality created a greater increase in SD2/SD1 ratio parameter compared to 50% training load of strength training modality trial following the intensive fatigue session.
- Greater performance impairment was shown in BP RPP and CMJ RPP markers following the 100% training load of strength training modality compared to 50% training load of strength training modality trial following the intensive fatigue session.
- Neuromuscular (MVC peak force, RFD^{200MVC}), Central (CAR) and peripheral fatigue (tetanic force, RFD^{tet}, RFR^{tet}, twitch force, $T_{1/2}$, twitch-to-tetanus ratio) markers did not change after 100% training load of strength training modality compared to 75% and 50% training load of strength training modality trial following the intensive fatigue session.

- 100% training load of power training modality did not affect pNN50, SDNN, Ln RMSSD, TP, SD1 and SD2 parameters, as well as SD2/SD1 ratio and SS parameters compared to 75% and 50% training load of power training modality trial following the intensive fatigue session.
- Significant performance impairment was not shown in BP RPP, CMJ height and CMJ RPP markers and neuromuscular fatigue level in the MVC peak force and RFD^{200MVC} markers following the 100% training load of power training modality compared to 75% and 50% training load of power training modality trial following the intensive fatigue session.
- Central (CAR) and peripheral fatigue (tetanic force, RFD^{tet}, RFR^{tet}, twitch force, $T_{1/2}$, twitch-to-tetanus ratio) markers also did not change following 100% training load of power training modality compared to 75% and 50% training load of power training modality trial following the intensive fatigue session.
- 50% training load of strength training modality returned HRV parameters (pNN50, SDNN, Ln RMSSD, TP, SD1, SD2, SD2/SD1 ratio and SS), performance markers (BP RPP, CMJ height and CMJ RPP), and neuromuscular (including central) fatigue markers (MVC peak force and RFD^{200MVC}, CAR) to Pre-B value (recover) sooner or closer to the respected Pre-B values within the testing period than 100% and 75% training loads of strength training modality following the intensive fatigue session.
- 50% training load of power training modality returned HRV parameters (SDNN, Ln RMSSD, TP, SD1, SD2, LF/HF ratio, SD2/SD1 ratio and SS) and neuromuscular (mainly central) fatigue markers (RFD^{200MVC}, CAR) to Pre-B value (recover) sooner or closer to the respected Pre-B values within the testing period than 100% and 75% training loads of power training modality following the intensive fatigue session.

- 75% of training load related to strength training modality and 100% of training load of power training modality could be considered the optimal training load for adequate recovery within the microcycle when an athlete was under the influence of fatigue due to previous training stress based on Ln RMSSD (HRV) parameter.

X – PRACTICAL APPLICATIONS

X. PRACTICAL APPLICATIONS

Based on the results of the studies presented in this doctoral thesis, the following recommendations may be helpful to coaches, sports scientists and athletes.

- Strength training modality was found to be more demanding and fatigue-inducing than power training modality. Therefore, strength training is more suitable when an athlete is well-recovered from the previous training-induced fatigue session. If the athlete is still not fully recovered, then power training may be more appropriate to achieve adequate recovery within the microcycle.
- Higher training volume of a resistance training protocol (strength or power) is more stressful and fatigue-inducing than lower training volume protocols. Therefore, from a fatigue-management perspective within the microcycle, lower training volume should be used when athletes are not well-recovered from the previous training session. In addition, training volume can be modified by changing the number of sets
- HRV parameters, specifically the Ln RMSSD parameter, is sensitive to the training stress produced by different training modalities (strength and power) and training loads. Moreover, HRV is a non-invasive, comfortable, affordable, and field user-friendly testing method. Hence, it's use is ideal to monitor the internal training load.
- Ln RMSSD parameter is sensitive to the time-course recovery profile following a training session and maybe the ideal parameter follow the evolution of recovery.

- If an athlete is still fatigued from the previous training stress, it would be ideal to manipulate the training load for the subsequent training session to achieve adequate recovery within the microcycle. Moreover, 75% of strength training load and 100% of power training load could be considered the optimal training load for adequate recovery within the microcycle when an athlete is still fatigued.

XI – FUTURE RESEARCH LINES

XI. FUTURE RESEARCH LINES

The scientific literature on HRV in sports is relatively young compared to other disciplines in Sports Science, and very little scientific literature has been found on resistance training and HRV. Furthermore, more in-depth studies on how HRV can monitor resistance training, specifically for training load manipulation and recovery, is needed. Based on the results obtained in the present thesis, the following research lines can provide more understanding on resistance training and HRV:

- To investigate the chronic effects (long term) of periodized strength and power training programs on performance improvement using resistance training load manipulation based on fatigue-related HRV parameters.

- To investigate the acute effect of different training loads and training modalities on HRV parameters and establish a load-HRV relationship.

- To investigate the recovery time and fatigue status using HRV parameters following training-induced fatigue of different training loads and training modalities.

- To investigate the correlation between HRV parameters and other fatigue monitoring markers (Ln RMSSD with jump performance (CMJ, Vertical jump), sprint performance (20m, repeated sprint), muscle contractile properties etc..)

- To determine the most reliable HRV parameter that could be used to monitor recovery and training-induced fatigue levels.

XII – REFERENCES

XII. REFERENCES

1. Haff GG, Triplett NT. Essentials of strength training and conditioning 4th edition: Human kinetics; 2015.

2. Brown LE. Strength training: Human Kinetics; 2007.

3. Stone MH, Fleck SJ, Triplett NT, Kraemer WJJSM. Health-and performance-related potential of resistance training. 1991;11(4):210-31.

4. Suchomel TJ, Nimphius S, Stone MHJSm. The importance of muscular strength in athletic performance. 2016;46(10):1419-49.

5. Deschenes MR, Kraemer WJJAJoPM, Rehabilitation. Performance and physiologic adaptations to resistance training. 2002;81(11):S3-S16.

6. Faigenbaum AD, Kraemer WJ, Blimkie CJ, Jeffreys I, Micheli LJ*, et al.* Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23:S60-S79.

7. Grgic J, Schoenfeld BJ, Skrepnik M, Davies TB, Mikulic PJSM. Effects of rest interval duration in resistance training on measures of muscular strength: a systematic review. 2018;48(1):137-51.

8. Cormie P, McGuigan MR, Newton RU. Developing maximal neuromuscular power: part 2 - training considerations for improving maximal power production. Sports Med. 2011;41(2):125-46.

9. Baker DJS, Journal C. Acute and long-term power responses to power training: Observations on the training of an elite power athlete. 2001;23(1):47-56.

10. Vanrenterghem J, Nedergaard NJ, Robinson MA, Drust BJSm. Training load monitoring in team sports: a novel framework separating physiological and biomechanical load-adaptation pathways. 2017;47(11):2135-42.

11. Kellmann M, Kölling S. Recovery and Stress in Sport: A Manual for Testing and Assessment: Taylor & Francis; 2019.

12. Halson SLJSm. Monitoring training load to understand fatigue in athletes. 2014;44(2):139-47.

13. Kellmann M, Bertollo M, Bosquet L, Brink M, Coutts AJ*, et al.* Recovery and performance in sport: consensus statement. 2018;13(2):240-5.

14. Bellenger CR, Fuller JT, Thomson RL, Davison K, Robertson EY*, et al.* Monitoring athletic training status through autonomic heart rate regulation: a systematic review and meta-analysis. Sports Med. 2016;46(10):1461-86.

15. Buchheit M, Simpson M, Al Haddad H, Bourdon P, Mendez-Villanueva AJEjoap. Monitoring changes in physical performance with heart rate measures in young soccer players. 2012;112(2):711-23.

16. Gathercole R, Sporer B, Stellingwerff T, Sleivert GJIjosp, performance. Alternative countermovement-jump analysis to quantify acute neuromuscular fatigue. 2015;10(1):84-92.

17. Mooney MG, Cormack S, O'Brien BJ, Morgan WM, McGuigan MJTJoS*, et al.* Impact of neuromuscular fatigue on match exercise intensity and performance in elite Australian football. 2013;27(1):166-73.

18. Roe G, Till K, Darrall-Jones J, Phibbs P, Weakley J*, et al.* Changes in markers of fatigue following a competitive match in elite academy rugby union players. 2016;28(1):2-5.

19. Hecksteden A, Skorski S, Schwindling S, Hammes D, Pfeiffer M*, et al.* Blood-borne markers of fatigue in competitive athletes–results from simulated training camps. 2016;11(2):e0148810.

20. Nédélec M, McCall A, Carling C, Legall F, Berthoin S*, et al.* Recovery in soccer. 2012;42(12):997-1015.

21. Meeusen R, Duclos M, Foster C, Fry A, Gleeson M*, et al.* Prevention, diagnosis and treatment of the overtraining syndrome: Joint consensus statement of the European College of Sport Science (ECSS) and the American College of Sports Medicine (ACSM). 2013;13(1):1-24.

22. Coutts AJ, Reaburn P. Monitoring changes in rugby league players' perceived stress and recovery during intensified training. Perceptual and motor skills. 2008;106(3):904-16.

23. Kallus KW, Kellmann M. The recovery-stress questionnaires: user manual: Pearson Frankfurt, Germany; 2016.

24. Saw AE, Main LC, Gastin PB. Monitoring the athlete training response: subjective self-reported measures trump commonly used objective measures: a systematic review. Br J Sports Med. 2015:bjsports-2015-094758.

25. Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA*, et al.* A new approach to monitoring exercise training. 2001;15(1):109-15.

26. Buchheit M. Sensitivity of monthly heart rate and psychometric measures for monitoring physical performance in highly trained young handball players. International journal of sports medicine. 2015;36(05):351-6.

27. Plews DJ, Laursen PB, Kilding AE, Buchheit M. Evaluating training adaptation with heart-rate measures: a methodological comparison. International journal of sports physiology performance. 2013;8(6):688-91.

28. Plews DJ, Laursen PB, Stanley J, Kilding AE, Buchheit MJSm. Training adaptation and heart rate variability in elite endurance athletes: opening the door to effective monitoring. 2013;43(9):773-81.

29. Dong JGJE, medicine t. The role of heart rate variability in sports physiology. 2016;11(5):1531-6.

30. Aubert AE, Seps B, Beckers FJSm. Heart rate variability in athletes. 2003;33(12):889-919.

31. Buchheit MJFip. Monitoring training status with HR measures: do all roads lead to Rome? 2014;5:73.

32. ChuDuc H, NguyenPhan K, NguyenViet DJAp. A review of heart rate variability and its applications. 2013;7:80-5.

33. Makivi B, Niki Djordjevi M, Willis MS. Heart Rate Variability (HRV) as a tool for diagnostic and monitoring performance in sport and physical activities. J Exerc Physiol Online. 2013;16(3).

34. Kingsley JD, Tai Y-L, Marshall EM, Glasgow A, Oliveira R*, et al.* Autonomic modulation and baroreflex sensitivity after acute resistance exercise: responses between sexes. J Sports Med Phys Fitness. 2019;59(6):1036-44.

35. Kryzhanovsky B, Dunin-Barkowski W, Redko V. Advances in neural computation, machine learning, and cognitive research. Neuroinformatics. 2017.

36. Mayo X, Iglesias-Soler E, Farinas-Rodriguez J, Fernandez-del-Olmo M, Kingsley JD. Exercise Type Affects Cardiac Vagal Autonomic Recovery After a Resistance Training Session. J Strength Cond Res. 2016;30(9):2565-73.

37. Kingsley JD, Mayo X, Tai YL, Fennell C. Arterial Stiffness and Autonomic Modulation After Free-Weight Resistance Exercises in Resistance Trained Individuals. J Strength Cond Res. 2016;30(12):3373-80.

38. Kingsley JD, Figueroa A. Acute and training effects of resistance exercise on heart rate variability. Clin Physiol Funct Imaging. 2016;36(3):179-87.

39. Martínez CAG, Quintana AO, Vila XA, Touriño MJL, Rodríguez-Liñares L*, et al.* Heart rate variability analysis with the R package RHRV: Springer; 2017.

40. Monteiro ER, Novaes JS, Fiuza AG, Portugal E, Triani FS*, et al.* Behavior of Heart Rate Variability After 10 Repetitions Maximum Load Test for Lower Limbs. Int J Exerc Sci. 2018;11(6):834-43.

41. Figueiredo T, Willardson JM, Miranda H, Bentes CM, Reis VM*, et al.* Influence of Load Intensity on Postexercise Hypotension and Heart Rate Variability after a Strength Training Session. J Strength Cond Res. 2015;29(10):2941-8.

42. Macêdo RF, Miranda JM, Maçal AC, Queiroz JCC, Bastos AA*, et al.* Overweight not influence the cardiac autonomic and cardiovascular response after resistance exercise in sedentary adolescents. Motri. 2019;15(S3):3-15.

43. Kingsley JD, McMillan V, Figueroa A. The effects of 12 weeks of resistance exercise training on disease severity and autonomic modulation at rest and after acute leg resistance exercise in women with fibromyalgia. Arch Phys Med Rehabil. 2010;91(10):1551-7.

44. Bouchard C, Rankinen T. Individual differences in response to regular physical activity. Medicine and science in sports and exercise. 2001;33(6 Suppl):S446-51; discussion S52-3.

45. Vesterinen V, Nummela A, Heikura I, Laine T, Hynynen E*, et al.* Individual Endurance Training Prescription with Heart Rate Variability. Medicine and science in sports and exercise. 2016;48(7):1347-54.

46. Borresen J, Lambert MI. Autonomic control of heart rate during and after exercise. Sports medicine. 2008;38(8):633-46.

47. Haff G, Haff EJEOPMM, Coburn JW, eds. Champaign, IL: Human Kinetics. Resistance training program design. 2012:359-401.

48. Williams TD, Tolusso DV, Fedewa MV, Esco MRJSm. Comparison of periodized and non-periodized resistance training on maximal strength: a meta-analysis. 2017;47(10):2083-100.

49. Reimers AK, Knapp G, Reimers C-DJJocm. Effects of exercise on the resting heart rate: a systematic review and meta-analysis of interventional studies. 2018;7(12):503.

50. Fleck SJ, Kraemer W. Designing resistance training programs, 4E: Human Kinetics; 2014.

51. Hoffman J. Physiological Aspects of Sport Training and Performance-2nd Edition: Human Kinetics; 2014.

52. Fragala MS, Cadore EL, Dorgo S, Izquierdo M, Kraemer WJ*, et al.* Resistance Training for Older Adults: Position Statement From the National Strength and Conditioning Association. 2019;33(8):2019-52.

53. Saeidifard F, Medina-Inojosa JR, West CP, Olson TP, Somers VK*, et al.* The association of resistance training with mortality: a systematic review and meta-analysis. 2019;26(15):1647-65.

54. Rutherford DJPGtOM. The Role of Physical Activity and Exercise in Managing Obesity and Achieving Weight Loss. 2017;215.

55. Smith JJ, Eather N, Morgan PJ, Plotnikoff RC, Faigenbaum AD*, et al.* The health benefits of muscular fitness for children and adolescents: a systematic review and meta-analysis. 2014;44(9):1209-23.

56. Figueiredo VC, de Salles BF, Trajano GSJSM. Volume for muscle hypertrophy and health outcomes: the most effective variable in resistance training. 2018;48(3):499-505.

57. Prue P, McGuigan M, Newton RJMSSE. Influence of strength on magnitude and mechanisms of adaptation to power training. 2010;42:1566-81.

58. Cormie P, McBride JM, McCaulley GOJTJoS, Research C. Power-time, force-time, and velocity-time curve analysis of the countermovement jump: impact of training. 2009;23(1):177-86.

59. Baker D, Nance SJTJoS, Research C. The relation between strength and power in professional rugby league players. 1999;13(3):224-9.

60. Haff GG, Nimphius SJS, Journal C. Training principles for power. 2012;34(6):2-12.

61. Moritani TJAjopm. Neural factors versus hypertrophy in the time course of muscle strength gain. 1979;58(3):115-30.

62. Siff MJBis. Biomechanical foundations of strength and power training. Wiley Online Library; 2000. p. 103-39.

63. Loturco I, Nakamura F, Kobal R, Gil S, Pivetti B*, et al.* Traditional periodization versus optimum training load applied to soccer players: effects on neuromuscular abilities. 2016;37(13):1051-9.

64. Soriano MA, Suchomel TJ, Marín PJJSM. The optimal load for maximal power production during upper-body resistance exercises: A meta-analysis. 2017;47(4):757-68.

65. Cormie P, McCaulley GO, Triplett NT, McBride JMJM, sports si*, et al.* Optimal loading for maximal power output during lower-body resistance exercises. 2007;39(2):340-9.

66. Soriano MA, Jiménez-Reyes P, Rhea MR, Marín PJJSM. The optimal load for maximal power production during lower-body resistance exercises: a meta-analysis. 2015;45(8):1191-205.

67. Suchomel TJ, Nimphius S, Bellon CR, Stone MH. The Importance of Muscular Strength: Training Considerations. Sports Med. 2018;48(4):765-85.

68. Balshaw TG, Massey GJ, Maden-Wilkinson TM, Morales-Artacho AJ, McKeown A*, et al.* Changes in agonist neural drive, hypertrophy and pre-training strength all contribute to the individual strength gains after resistance training. 2017;117(4):631-40.

69. Folland JP, Williams AG. Morphological and Neurological Contributions to Increased Strength. Sports Med. 2007;37(2):145-68.

70. Buckner SL, Mouser JG, Jessee MB, Dankel SJ, Mattocks KT*, et al.* What does individual strength say about resistance training status? 2017;55(4):455-7.

71. Narici MV, Roi G, Landoni L, Minetti A, Cerretelli PJEjoap*, et al.* Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. 1989;59(4):310-9.

72. Folland JP, Williams AGJSm. Morphological and neurological contributions to increased strength. 2007;37(2):145-68.

73. Bompa T, Buzzichelli C. Periodization training for sports, 3e: Human kinetics; 2015.

74. Wilson GJ, Murphy AJ, Pryor JFJJoap. Musculotendinous stiffness: its relationship to eccentric, isometric, and concentric performance. 1994;76(6):2714-9.

75. Del Vecchio A, Casolo A, Negro F, Scorcelletti M, Bazzucchi I*, et al.* The increase in muscle force after 4 weeks of strength training is mediated by adaptations in motor unit recruitment and rate coding. 2019;597(7):1873-87.

76. Aagaard P, Simonsen E, Andersen J, Magnusson S, Halkjaer-Kristensen J*, et al.* Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. 2000;89(6):2249-57.

77. Rhea MR, Alderman BLJRqfe, sport. A meta-analysis of periodized versus nonperiodized strength and power training programs. 2004;75(4):413-22.

78. Lloyd RS, Oliver JL. Strength and Conditioning for Young Athletes: Science and Application: Taylor & Francis; 2019.

79. Stone MH, Stone M, Sands WA. Principles and practice of resistance training: Human Kinetics; 2007.

80. Haff GGJS, Journal C. Roundtable discussion: Periodization of training—Part 1. 2004;26(1):50-69.

81. Chandler TJ, Brown LE. Conditioning for strength and human performance: Lippincott Williams & Wilkins; 2008.

82. Bourdon PC, Cardinale M, Murray A, Gastin P, Kellmann M*, et al.* Monitoring athlete training loads: consensus statement. 2017;12(s2):S2-161-S2-70.

83. Collette R, Kellmann M, Ferrauti A, Meyer T, Pfeiffer M. Relation Between Training Load and Recovery-Stress State in High-Performance Swimming. 2018;9(845).

84. Kellmann M, Beckmann J. Sport, recovery, and performance: Interdisciplinary insights: Routledge; 2017.

85. Wilk M, Golas A, Stastny P, Nawrocka M, Krzysztofik M*, et al.* Does tempo of resistance exercise impact training volume? 2018;62(1):241-50.

86. Loturco I, Nakamura FY, Tricoli V, Kobal R, Abad CCC*, et al.* Determining the optimum power load in jump squat using the mean propulsive velocity. 2015;10(10):e0140102.

87. Nádori L, Hortobágyi T, Granek I, National S, Conditioning A. Theoretical and methodological basis of training planning with special considerations within a microcycle. Lincoln, Neb.: National Strength and Conditioning Association; 1989.

88. Foster CJM, sports si, exercise. Monitoring training in athletes with reference to overtraining syndrome. 1998;30(7):1164-8.

89. Brown LE, Bradley-Popovich G, Haff GJSCJ. Nonlinear versus linear periodization models. 2001;23(1):42-4.

90. Tan BJTJoS, Research C. Manipulating resistance training program variables to optimize maximum strength in men: a review. 1999;13(3):289-304.

91. Spiering BA, Kraemer WJ, Anderson JM, Armstrong LE, Nindl BC*, et al.* Resistance exercise biology. 2008;38(7):527-40.

92. Kraemer W, Noble B, Clark M, Culver BJIjosm. Physiologic responses to heavy-resistance exercise with very short rest periods. 1987;8(04):247-52.

93. Peake JMJCOiP. Recovery after exercise: what is the current state of play? 2019;10:17-26.

94. Sahlin KJSM. Metabolic factors in fatigue. 1992;13(2):99-107.

95. Márquez G, Romero-Arenas S, Marín-Pagán C, Vera-Ibañez A, Fernandez Del Olmo M*, et al.* Peripheral and central fatigue after high intensity resistance circuit training. 2017;56(1):152-9.

96. Pareja-Blanco F, Rodríguez-Rosell D, Sánchez-Medina L, Sanchis-Moysi J, Dorado C*, et al.* Effects of velocity loss during resistance training on athletic performance, strength gains and muscle adaptations. 2017;27(7):724-35.

97. Gandevia SCJPr. Spinal and supraspinal factors in human muscle fatigue. 2001.

98. Edwards RH, editor Human muscle function and fatigue. Ciba Found Symp; 1981: Wiley Online Library.

99. Pyne DB, Martin DT. Fatigue-Insights from individual and team sports. Regulation of fatigue in exercise: Nova Publishers; 2011. p. 177-86.

100. Knicker AJ, Renshaw I, Oldham AR, Cairns SPJSm. Interactive processes link the multiple symptoms of fatigue in sport competition. 2011;41(4):307-28.

101. Minett GM, Duffield RJFip. Is recovery driven by central or peripheral factors? A role for the brain in recovery following intermittent-sprint exercise. 2014;5:24.

102. Wan J-j, Qin Z, Wang P-y, Sun Y, Liu XJE*, et al.* Muscle fatigue: general understanding and treatment. 2017;49(10):e384-e.

103. Allen DG, Lamb GD, Westerblad HJPr. Skeletal muscle fatigue: cellular mechanisms. 2008.

104. Bhatti M, Han RP, Vignes RJSt. Muscle forces and fatigue in a digital human environment. 2005:2930-6.

105. Bishop PA, Jones E, Woods AKJTJoS, Research C. Recovery from training: a brief review: brief review. 2008;22(3):1015-24.

106. Kenttä G, Hassmén PJSm. Overtraining and recovery. 1998;26(1):1-16.

107. Budgett RJBjosm. Fatigue and underperformance in athletes: the overtraining syndrome. 1998;32(2):107-10.

108. Bian FJN. Electroencephalogram Analysis of Athletes with Over-training Syndrome. 2018;16(5).

109. Fry RW, Morton AR, Keast DJSm. Overtraining in athletes. 1991;12(1):32-65.

110. McGuigan M. Monitoring training and performance in athletes: Human Kinetics; 2017.

111. Bieuzen F, Bleakley CM, Costello JTJPo. Contrast water therapy and exercise induced muscle damage: a systematic review and meta-analysis. 2013;8(4):e62356.

112. Peake JM, Suzuki K, Coombes JSJTJonb. The influence of antioxidant supplementation on markers of inflammation and the relationship to oxidative stress after exercise. 2007;18(6):357-71.

113. Marqués-Jiménez D, Calleja-González J, Arratibel I, Delextrat A, Terrados NJTOSSJ. Fatigue and recovery in soccer: evidence and challenges. 2017;10(1).

114. Heidari J, Beckmann J, Bertollo M, Brink M, Kallus KW*, et al.* Multidimensional monitoring of recovery status and implications for performance. 2019;14(1):2-8.

115. Gathercole RJ, Sporer BC, Stellingwerff T, Sleivert GGJTJoS, Research C. Comparison of the capacity of different jump and sprint field tests to detect neuromuscular fatigue. 2015;29(9):2522-31.

116. Andersson HM, Raastad T, Nilsson J, Paulsen G, Garthe I*, et al.* Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. 2008;40(2):372-80.

117. Robineau J, Jouaux T, Lacroix M, Babault NJTJoS, Research C. Neuromuscular fatigue induced by a 90-minute soccer game modeling. 2012;26(2):555-62.

118. Djaoui L, Garcia JD-C, Hautier C, Dellal AJAjosm. Kinetic post-match fatigue in professional and youth soccer players during the competitive period. 2016;7(1).

119. Krustrup P, Zebis M, Jensen JM, Mohr MJTJoS, Research C. Game-induced fatigue patterns in elite female soccer. 2010;24(2):437-41.

120. González Badillo JJ, Rodríguez Rosell D, Sánchez Medina L, Ribas Serna J, López López C*, et al.* Short-term Recovery Following Resistance Exercise Leading or not to Failure. 2015.

121. Morán-Navarro R, Pérez CE, Mora-Rodríguez R, de la Cruz-Sánchez E, González-Badillo JJ*, et al.* Time course of recovery following resistance training leading or not to failure. 2017;117(12):2387-99.

122. Bartolomei S, Sadres E, Church DD, Arroyo E, Gordon III JA*, et al.* Comparison of the recovery response from high-intensity and high-volume resistance exercise in trained men. 2017;117(7):1287-98.

123. Main L, Grove JRJEJoSS. A multi-component assessment model for monitoring training distress among athletes. 2009;9(4):195-202.

124. McNair DMJE, Service IT. Profile of mood states. 1992.

125. Saw AE, Main LC, Gastin PBJBjosm. Monitoring the athlete training response: subjective self-reported measures trump commonly used objective measures: a systematic review. 2016;50(5):281-91.

126. Halson SL, Bridge MW, Meeusen R, Busschaert B, Gleeson M*, et al.* Time course of performance changes and fatigue markers during intensified training in trained cyclists. 2002;93(3):947-56.

127. O'Connor PJ, Morgan WP, Raglin JSJM, Sports Si, Exercise. Psychobiologic effects of 3 d of increased training in female and male swimmers. 1991.

128. Coutts AJ, Wallace L, Slattery KJIjosm. Monitoring changes in performance, physiology, biochemistry, and psychology during overreaching and recovery in triathletes. 2007;28(02):125-34.

129. Mäestu J, Jürimäe J, Kreegipuu K, Jürimäe TJTSP. Changes in perceived stress and recovery during heavy training in highly trained male rowers. 2006;20(1):24-39.

130. Muscatello RA, Andujar J, Taylor JL, Corbett BAJJoa, disorders d. Exploring key physiological system profiles at rest and the association with depressive symptoms in autism spectrum disorder. 2021;51(1):15-29.

131. Wang L, Olivas A, Francis Stuart SD, Tapa S, Blake MR*, et al.* Cardiac sympathetic nerve transdifferentiation reduces action potential heterogeneity after myocardial infarction. 2020;318(3):H558-H65.

132. Jänig W. Autonomic nervous system. Hum Physiol: Springer; 1989. p. 333-70.

133. Michael S, Graham KS, Davis GMJFip. Cardiac autonomic responses during exercise and post-exercise recovery using heart rate variability and systolic time intervals—a review. 2017;8:301.

134. Fisher JPJEp. Autonomic control of the heart during exercise in humans: role of skeletal muscle afferents. 2014;99(2):300-5.

135. Raven PB, Fadel PJ, Ogoh SJEp. Arterial baroreflex resetting during exercise: a current perspective. 2006;91(1):37-49.

136. White DW, Raven PBJTJop. Autonomic neural control of heart rate during dynamic exercise: revisited. 2014;592(12):2491-500.

137. Nobrega AC, O'Leary D, Silva BM, Marongiu E, Piepoli MF*, et al.* Neural regulation of cardiovascular response to exercise: role of central command and peripheral afferents. 2014;2014.

138. Michelini LC, O'Leary DS, Raven PB, Nóbrega ACJAJoP-H, Physiology C. Neural control of circulation and exercise: a translational approach disclosing interactions between central command, arterial baroreflex, and muscle metaboreflex. 2015;309(3):H381-H92.

139. Fadel PJSjom, sports si. Reflex control of the circulation during exercise. 2015;25:74-82.

140. Robinson BF, Epstein SE, Beiser GD, Braunwald EJCR. Control of heart rate by the autonomic nervous system: studies in man on the interrelation between baroreceptor mechanisms and exercise. 1966;19(2):400-11.

141. O'Leary DSJJoAP. Autonomic mechanisms of muscle metaboreflex control of heart rate. 1993;74(4):1748-54.

142. Rowell LB, O'Leary DSJJoap. Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. 1990;69(2):407-18.

143. Katayama K, Saito MJTJoPS. Muscle sympathetic nerve activity during exercise. 2019;69(4):589-98.

144. Vieluf S, Hasija T, Jakobsmeyer R, Schreier PJ, Reinsberger CJFip. Exercise-induced changes of multimodal interactions within the autonomic nervous network. 2019;10:240.

145. Peçanha T, Silva-Júnior ND, Forjaz CLdMJCp, imaging f. Heart rate recovery: autonomic determinants, methods of assessment and association with mortality and cardiovascular diseases. 2014;34(5):327-39.

146. Kannankeril PJ, Le FK, Kadish AH, Goldberger JJJJoim. Parasympathetic effects on heart rate recovery after exercise. 2004;52(6):394-401.

147. Coote JHJEp. Recovery of heart rate following intense dynamic exercise. 2010;95(3):431-40.

148. Cunha FA, Midgley AW, Gonçalves T, Soares PP, Farinatti P. Parasympathetic reactivation after maximal CPET depends on exercise modality and resting vagal activity in healthy men. Springerplus. 2015;4:100-.

149. Gourine AV, Ackland GLJP. Cardiac vagus and exercise. 2019;34(1):71-80.

150. Nandi PS, Spodick DHJBhj. Recovery from exercise at varying work loads. Time course of responses of heart rate and systolic intervals. 1977;39(9):958.

151. Stanley J, Peake JM, Buchheit MJSm. Cardiac parasympathetic reactivation following exercise: implications for training prescription. 2013;43(12):1259-77.

152. Thamm A, Freitag N, Figueiredo P, Doma K, Rottensteiner C*, et al.* Can Heart Rate Variability Determine Recovery Following Distinct Strength Loadings? A Randomized Cross-Over Trial. Int J Environ Res Public Health. 2019;16(22).

153. Chen JL, Yeh DP, Lee JP, Chen CY, Huang CY*, et al.* Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. J Strength Cond Res. 2011;25(6):1546-52.

154. Risk M, Bril V, Broadbridge C, Cohen A. Heart rate variability measurement in diabetic neuropathy: review of methods. Diabetes Technol Ther. 2001;3(1):63-76.

155. Ernst G. Heart-rate variability—More than heart beats? Front Public Health. 2017;5:240.

156. Achten J, Jeukendrup AE. Heart rate monitoring. Sports Med. 2003;33(7):517-38.

157. Kang JH, Kim JK, Hong SH, Lee CH, Choi BY. Heart Rate Variability for Quantification of Autonomic Dysfunction in Fibromyalgia. Ann Rehabil Med. 2016;40(2):301-9.

158. Banerjee R, Ghose A, Khandelwal S, editors. A Novel Recurrent Neural Network Architecture for Classification of Atrial Fibrillation Using Single-lead ECG. 2019 27th European Signal Processing Conference (EUSIPCO); 2019: IEEE.

159. Malik M, Camm AJJI. Heart rate variability (Armonk, NY, Futura Pub. Co.). 1995.

160. Hon EHJAJOG. Electronic evaluations of the fetal heart rate patterns preceding fetal death, further observations. 1965;87:814-26.

161. Electrophysiology TFotESoCatNASoPa. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation. 1996;93(5):1043-65.

162. Ernst G. Hidden signals—the history and methods of heart rate variability. Front Public Health. 2017;5:265.

163. Dong JG. The role of heart rate variability in sports physiology. Exp Ther Med. 2016;11(5):1531-6.

164. Germán-Salló Z, Germán-Salló MJPt. Non-linear methods in HRV analysis. 2016;22:645-51.

165. Malliani A, Pagani M, Lombardi F, Cerutti SJC. Cardiovascular neural regulation explored in the frequency domain. 1991;84(2):482-92.

166. Peng CK, Havlin S, Stanley HE, Goldberger ALJCaijons. Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. 1995;5(1):82-7.

167. Shono H, Peng C-K, Goldberger AL, Shono M, Sugimori HJCiB*, et al.* A new method to determine a fractal dimension of non-stationary biological time-serial data. 2000;30(4):237-45.

168. Berntson GG, Thomas Bigger Jr J, Eckberg DL, Grossman P, Kaufmann PG*, et al.* Heart rate variability: origins, methods, and interpretive caveats. 1997;34(6):623-48.

169. Shaffer F, Ginsberg J. An overview of heart rate variability metrics and norms. Front Public Health. 2017;5:258.

170. Penttilä J, Kuusela T, Scheinin HJEjocp. Analysis of rapid heart rate variability in the assessment of anticholinergic drug effects in humans. 2005;61(8):559-65.

171. Salahuddin L, Cho J, Jeong MG, Kim D, editors. Ultra short term analysis of heart rate variability for monitoring mental stress in mobile settings. 2007 29th annual international conference of the ieee engineering in medicine and biology society; 2007: IEEE.

172. Baek HJ, Cho C-H, Cho J, Woo J-MJT, e-Health. Reliability of ultra-short-term analysis as a surrogate of standard 5-min analysis of heart rate variability. 2015;21(5):404-14.

173. Esco MR, Flatt AAJJoss, medicine. Ultra-short-term heart rate variability indexes at rest and post-exercise in athletes: evaluating the agreement with accepted recommendations. 2014;13(3):535.

174. Nakamura FY, Flatt AA, Pereira LA, Ramirez-Campillo R, Loturco I*, et al.* Ultra-short-term heart rate variability is sensitive to training effects in team sports players. 2015;14(3):602.

175. Hill L, Siebenbrock A, Sollers J, Thayer JFJBSI. Are all measures created equal? Heart rate variability and respiration. 2009;45:71-6.

176. Sztajzel JJSmw. Heart rate variability: a noninvasive electrocardiographic method to measure the autonomic nervous system. 2004;134(35-36):514-22.

177. Gerhart H, Tai YL, Fennell C, Mayo X, Kingsley JDJIjoes. Autonomic modulation in older women: using resistance exercise as a countermeasure. 2017;10(2):178.

178. de Vilhena Toledo MA, Junqueira LFJCAR. Cardiac autonomic modulation and cognitive status in Alzheimer's disease. 2010;20(1):11-7.

179. Frenneaux MPJH. Autonomic changes in patients with heart failure and in post-myocardial infarction patients. 2004;90(11):1248-55.

180. Cottin F, Médigue C, Leprêtre P-M, Papelier Y, Koralsztein J-P*, et al.* Heart rate variability during exercise performed below and above ventilatory threshold. 2004;36(4):594-600.

181. Montano N, Ruscone TG, Porta A, Lombardi F, Pagani M*, et al.* Power spectrum analysis of heart rate variability to assess the changes in sympathovagal balance during graded orthostatic tilt. 1994;90(4):1826-31.

182. Shaffer F, McCraty R, Zerr CLJFip. A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. 2014;5:1040.

183. Henriques T, Ribeiro M, Teixeira A, Castro L, Antunes L*, et al.* Nonlinear methods most applied to heart-rate time series: A review. 2020;22(3):309.

184. Lombardi FJC. Chaos theory, heart rate variability, and arrhythmic mortality. 2000;101(1):8-10.

185. Peltola MJAUODM. Analysis of heart rate variability from 24-hour ambulatory electrocardiographic recordings. 2010;1087.

186. Sieci ski S, Kostka PS, Tkacz EJJS. Heart Rate Variability Analysis on Electrocardiograms, Seismocardiograms and Gyrocardiograms on Healthy Volunteers. 2020;20(16):4522.

187. Orellana JN, de la Cruz Torres B, Cachadiña ES, de Hoyo M, Cobo SDJIjosp*, et al.* Two new indexes for the assessment of autonomic balance in elite soccer players. 2015;10(4):452-7.

188. Navarro-Lomas G, Jurado-Fasoli L, Castillo MJ, Femia P, Amaro-Gahete FJJP. Assessment of autonomous nerve system through non-linear heart rate variability outcomes in sedentary healthy adults. 2020;8:e10178.

189. Behbahani S, Dabanloo NJ, Nasrabadi AMJAiB. Ictal Heart Rate Variability Assessment with Focus on Secondary Generalized and Complex Partial Epileptic Seizures. 2013;4(1).

190. Hsu CH, Tsai MY, Huang GS, Lin TC, Chen KP*, et al.* Poincaré plot indexes of heart rate variability detect dynamic autonomic modulation during general anesthesia induction. Acta Anaesthesiol Taiwan. 2012;50(1):12-8.

191. Guzik P, Piskorski J, Krauze T, Schneider R, Wesseling KH*, et al.* Correlations between Poincaré plot and conventional heart rate variability parameters assessed during paced breathing. 2007:0702020009-.

192. Villafaina S, Collado-Mateo D, Domínguez-Muñoz FJ, Gusi N, Fuentes-Garcia JP. Effects of exergames on heart rate variability of women with fibromyalgia: A randomized controlled trial. Scientific Reports. 2020;10(1):5168.

193. Miranda-Mendoza J, Reynoso-Sanchez L, Hoyos-Flores J, Quezada-Chacón J, Naranjo J*, et al.* STRESS SCORE AND LnrMSSD AS INTERNAL LOAD PARAMETERS DURING COMPETITION. 2020;20(77).

194. Pincus SMJPotNAoS. Approximate entropy as a measure of system complexity. 1991;88(6):2297-301.

195. Tarvainen M, Lipponen J, Niskanen J, Ranta-Aho PJKUoEF. Kubios HRV Version 3–User's Guide. 2017.

196. Beckers F, Ramaekers D, Aubert AEJCEAIJ. Approximate entropy of heart rate variability: validation of methods and application in heart failure. 2001;1(4):177-82.

197. Richman JS, Moorman JRJAJoP-H, Physiology C. Physiological time-series analysis using approximate entropy and sample entropy. 2000.

198. Chen X, Solomon IC, Chon KH, editors. Comparison of the use of approximate entropy and sample entropy: applications to neural respiratory signal. 2005 IEEE Engineering in Medicine and Biology 27th Annual Conference; 2006: IEEE.

199. Abad C, Kobal R, Kitamura K, Gil S, Pereira L*, et al.* Heart rate variability in elite sprinters: effects of gender and body position. 2017;37(4):442-7.

200. Kleiger RE, Stein PK, Bigger Jr JTJAoNE. Heart rate variability: measurement and clinical utility. 2005;10(1):88-101.

201. Cacioppo JT, Berntson GG, Binkley PF, Quigley KS, Uchino BN*, et al.* Autonomic cardiac control. II. Noninvasive indices and basal response as revealed by autonomic blockades. 1994;31(6):586-98.

202. Dietrich A, Rosmalen JG, Althaus M, van Roon AM, Mulder LJ*, et al.* Reproducibility of heart rate variability and baroreflex sensitivity measurements in children. 2010;85(1):71-8.

203. da Silva VP, de Oliveira NA, Silveira H, Mello RGT, Deslandes ACJAoNE. Heart rate variability indexes as a marker of chronic adaptation in athletes: a systematic review. 2015;20(2):108-18.

204. Selman AJA. The interaction between heart rate and respiration: Part 1-Experimental studies in man. 1982;4:131-9.

205. Zhang PZ, Tapp WN, Reisman SS, Natelson BHJItobe. Respiration response curve analysis of heart rate variability. 1997;44(4):321-5.

206. Chang Q, Liu R, Shen ZJIjoC. Effects of slow breathing rate on blood pressure and heart rate variabilities. 2013;169(1):e6-e8.

207. Li C, Chang Q, Zhang J, Chai W. Effects of slow breathing rate on heart rate variability and arterial baroreflex sensitivity in essential hypertension. 2018;97(18):e0639.

208. Heffernan KS, Kelly EE, Collier SR, Fernhall B. Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. Eur J Cardiovasc Prev Rehabil. 2006;13(1):80-6.

209. Kingsley JD, Panton LB, McMillan V, Figueroa A. Cardiovascular autonomic modulation after acute resistance exercise in women with fibromyalgia. Arch Phys Med Rehabil. 2009;90(9):1628-34.

210. Kingsley JD, Hochgesang S, Brewer A, Buxton E, Martinson M*, et al.* Autonomic Modulation in Resistance-Trained Individuals after Acute Resistance Exercise. Int J Sports Med. 2014;35(10):851-6.

211. Paz GA, Iglesias-Soler E, Willardson JM, de Freitas Maia M, Miranda H. Postexercise Hypotension and Heart Rate Variability Responses Subsequent to Traditional, Paired Set, and Superset Training Methods. J Strength Cond Res. 2017.

212. Kingsley J, Tai Y, Marshall E, Glasgow A, Oliveira R*, et al.* Autonomic modulation and baroreflex sensitivity after acute resistance exercise: responses between sexes. J Sports Med Phys Fitness. 2018.

213. Aranda C, De-la-Cruz-Torres B, Orellana JNJAdmddrdlFEdMdDydlCIdMdD. Effects of different automatic filters on the analysis of heart rate variability with Kubios HRV software. 2017;34(180):196-200.

214. Giles DA, Draper NJTJoS, Research C. Heart rate variability during exercise: a comparison of artefact correction methods. 2018;32(3):726-35.

215. Królak A, Wiktorski T, Bjørkavoll-Bergseth MF, Ørn SJS. Artifact Correction in Short-Term HRV during Strenuous Physical Exercise. 2020;20(21):6372.

216. Rezk C, Marrache R, Tinucci T, Mion D, Forjaz C. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. Eur J Appl Physiol. 2006;98(1):105-12.

217. Monteiro ER, Novaes JS, Fiuza AG, Portugal E, Triani FS*, et al.* Behavior of heart rate variability after 10 repetitions maximum load test for lower limbs. 2018;11(6):834.

218. Lima CD, Martinez PF, de Morais CS, Barbosa FSS, Ota GE*, et al.* Cardiovascular effects of a strength test (1RM) in prehypertensive subjects. Rev Bras Med Esporte. 2019;25(1):9-13.

219. Teixeira L, Ritti-Dias RM, Tinucci T, Mion D, Forjaz CLD. Post-concurrent exercise hemodynamics and cardiac autonomic modulation. Eur J Appl Physiol. 2011;111(9):2069-78.

220. Mayo X, Iglesias-Soler E, Carballeira-Fernandez E, Fernandez-Del-Olmo M. A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise. Eur J Sport Sci. 2016;16(8):996-1004.

221. de Freitas MC, Ricci-Vitor AL, Quizzini GH, de Oliveira J, Vanderlei LCM*, et al.* Postexercise hypotension and autonomic modulation response after full versus split body resistance exercise in trained men. J Exerc Rehabil. 2018;14(3):399-406.

222. Paz GA, Iglesias-Soler E, Willardson JM, de Freitas Maia M, Miranda HJTJoS*, et al.* Postexercise Hypotension and Heart Rate Variability Responses Subsequent to Traditional, Paired Set, and Superset Resistance Training Methods. 2019;33(9):2433-42.

223. Corrêa Neto VG, Benteso CM, Maranhão Neto GdA, Miranda H. Hypotension and heart rate variability after resistance exercise performed maximal and submaximal order. Motri. 2017;13(1):19-29.

224. Kliszczewicz BM, Esco MR, Quindry JC, Blessing DL, Oliver GD*, et al.* Autonomic Responses to an Acute Bout of High-Intensity Body Weight Resistance Exercise vs. Treadmill Running. J Strength Cond Res. 2016;30(4):1050-8.

225. Figueiredo T, Willardson JM, Miranda H, Bentes CM, Reis VM*, et al.* Influence of Rest Interval Length Between Sets on Blood Pressure and Heart Rate Variability After a Strength Training Session Performed By Prehypertensive Men. J Strength Cond Res. 2016;30(7):1813-24.

226. Iglesias-Soler E, Boullosa DA, Carballeira E, Sanchez-Otero T, Mayo X*, et al.* Effect of set configuration on hemodynamics and cardiac autonomic modulation after high-intensity squat exercise. Clin Physiol Funct Imaging. 2015;35(4):250-7.

227. Figueiredo T, Rhea MR, Peterson M, Miranda H, Bentes CM*, et al.* Influence of number of sets on blood pressure and heart rate variability after a strength training session. J Strength Cond Res. 2015;29(6):1556-63.

228. Saccomani MG, Figueiredo T, Kattenbraker MS, Simao R, de Moraes Forjaz CL*, et al.* Blood pressure and heart rate variability responses after isolated and combined sessions of aerobic and strength exercises. Int Sportmed J. 2014;15(2):178-87.

229. Okuno NM, Pedro RE, Leicht AS, de Paula Ramos S, Nakamura FY. Cardiac autonomic recovery after a single session of resistance exercise with and without vascular occlusion. J Strength Cond Res. 2014;28(4):1143-50.

230. Tibana RA, Boullosa DA, Leicht AS, Prestes J. Women with metabolic syndrome present different autonomic modulation and blood pressure response to an acute resistance exercise session compared with women without metabolic syndrome. Clin Physiol Funct Imaging. 2013;33(5):364-72.

231. Goessler KF, Doederlein Polito M. Effect of fixed and self-suggested rest intervals between sets of resistance exercise on post-exercise cardiovascular behavior. Brazilian Journal of Kineanthropometry and Human Performance. 2013;15(4):467-75.

232. Moher D, Liberati A, Tetzlaff J, Altman DG, Group P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. PLoS medicine. 2009;6(7):e1000097.

233. Higgins JP. Cochrane handbook for systematic reviews of interventions version 5.0. 1. The Cochrane Collaboration 2008 [Available from: http://www.cochrane-handbook.org.

234. Higgins JP, Altman DG, Gøtzsche PC, Jüni P, Moher D*, et al.* The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ. 2011;343:d5928.

235. National Heart L, and Blood Institute. Study Quality Assessment Tools [Available from: https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools.

236. Browning KN, Travagli RA. Functional organization of presynaptic metabotropic glutamate receptors in vagal Brainstem circuits. J Neurosci. 2007;27(34):8979-88.

237. Schoenfeld BJ. The mechanisms of muscle hypertrophy and their application 9 to resistance training. J STRENGTH COND RES. 2010;24(2857-2872):10.

238. USA. CfDCaP. Healthy weight - About adult BMI. 2017 [Available from: https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html.

239. Jidovtseff B, Harris NK, Crielaard J-M, Cronin JBJTJoS, Research C. Using the load-velocity relationship for 1RM prediction. 2011;25(1):267-70.

240. Loturco I, Pereira LA, Abad CCC, Gil S, Kitamura K*, et al.* Using bar velocity to predict maximum dynamic strength in the half-squat exercise. 2016;11(5):697-700.

241. Freitas TT, Calleja-González J, Alarcón F, Alcaraz PEJTJoS, Research C. Acute effects of two different resistance circuit training protocols on performance and perceived exertion in semiprofessional basketball players. 2016;30(2):407-14.

242. do Nascimento MA, Cyrino ES, Nakamura FY, Romanzini M, Pianca HJC*, et al.* Validation of the Brzycki equation for the estimation of 1-RM in the bench press. 2007;13(1):40e-2e.

243. Gilgen-Ammann R, Schweizer T, Wyss T. RR interval signal quality of a heart rate monitor and an ECG Holter at rest and during exercise. Eur J Appl Physiol. 2019;119(7):1525-32.

244. Penttilä J, Helminen A, Jartti T, Kuusela T, Huikuri HV*, et al.* Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: effects of various respiratory patterns. 2001;21(3):365-76.

245. Taylor K-L. Monitoring neuromuscular fatigue in high performance athletes. 2012.

246. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J*, et al.* Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. 2005;45(1):142-61.

247. Hermens HJ, Freriks B, Merletti R, Stegeman D, Blok J*, et al.* European recommendations for surface electromyography. 1999;8(2):13-54.

248. Gordon RJFH, Tillin NA, Tyler CJJAP, Nutrition,, Metabolism. The effect of head and neck per-cooling on neuromuscular fatigue following exercise in the heat. 2020;45(11):1238-46.

249. Kent-Braun JA, Le Blanc RJM, Medicine NOJotAAoE. Quantitation of central activation failure during maximal voluntary contractions in humans. 1996;19(7):861-9.

250. Bigland-Ritchie B, Jones D, Hosking G, Edwards RJCs, medicine m. Central and peripheral fatigue in sustained maximum voluntary contractions of human quadriceps muscle. 1978;54(6):609-14.

251. Kent-Braun JAJEjoap, physiology o. Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. 1999;80(1):57-63.

252. Chung LH, Callahan DM, Kent-Braun JAJJoAP. Age-related resistance to skeletal muscle fatigue is preserved during ischemia. 2007;103(5):1628-35.

253. Markofski MM, Braun WAJTJoS, Research C. Influence of menstrual cycle on indices of contraction-induced muscle damage. 2014;28(9):2649-56.

254. Kim J, Lee JJJoen, biochemistry. The relationship of creatine kinase variability with body composition and muscle damage markers following eccentric muscle contractions. 2015;19(2):123.

255. Koontz J. The Effects of Common Treatment Modalities on Delayed-Onset Muscle Soreness (DOMS). 2019.

256. Raees-Sadati J, Nakhostin-Roohi B, Siahkouhian M. The Effect of Grape Seed Extract Supplementation on Delayed Onset Muscle Soreness (DOMS) in Young Healthy Female Students. 2019.

257. Bijur PE, Silver W, Gallagher EJJAem. Reliability of the visual analog scale for measurement of acute pain. 2001;8(12):1153-7.

258. Boonstra AM, Preuper HRS, Reneman MF, Posthumus JB, Stewart REJIjorr. Reliability and validity of the visual analogue scale for disability in patients with chronic musculoskeletal pain. 2008;31(2):165-9.

259. Ferreira-Valente MA, Pais-Ribeiro JL, Jensen MPJP. Validity of four pain intensity rating scales. 2011;152(10):2399-404.

260. Day ML, McGuigan MR, Brice G, Foster CJTJoS, Research C. Monitoring exercise intensity during resistance training using the session RPE scale. 2004;18(2):353-8.

261. Grove JR, Prapavessis HJIJoSP. Preliminary evidence for the reliability and validity of an abbreviated profile of mood states. 1992.

262. Matthews MJ, Heron K, Todd S, Tomlinson A, Jones P*, et al.* Strength and endurance training reduces the loss of eccentric hamstring torque observed after soccer specific fatigue. 2017;25:39-46.

263. Williams JD, Abt G, Kilding AEJTJoS, Research C. Ball-sport endurance and sprint test (BEAST90): validity and reliability of a 90-minute soccer performance test. 2010;24(12):3209-18.

264. Isidoro NJ, Santana MDR, Valenti VE, Garner DM, de Abreu LC. Cardiac autonomic recovery after strength exercise in lower and upper limbs. Acta Cardiol. 2017;72(4):467-73.

265. Lima A, Forjaz CLD, Silva GQD, Meneses AL, Silva A*, et al.* Acute Effect of Resistance Exercise Intensity in Cardiac Autonomic Modulation After Exercise. Arq Bras Cardiol. 2011;96(6):498-503.

266. Buchheit M, Laursen P, Ahmaidi S. Parasympathetic reactivation after repeated sprint exercise. Am J Physiol Heart Circ Physiol. 2007;293:H133-41.

267. Beske SD, Alvarez GE, Ballard TP, Davy KP. Gender difference in cardiovagal baroreflex gain in humans. J Appl Physiol. 2001;91(5):2088-92.

268. Wehrwein EA, Joyner MJ. Regulation of blood pressure by the arterial baroreflex and autonomic nervous system. Handbook of clinical neurology. 117: Elsevier; 2013. p. 89-102.

269. Chourpiliadis C, Bhardwaj A. Physiology, Respiratory Rate: StatPearls Publishing, Treasure Island (FL); 2020 2020.

270. McCance KL, Huether SE. Pathophysiology-E-book: the biologic basis for disease in adults and children: Elsevier Health Sciences; 2018.

271. Strauß R, Ewig S, Richter K, König T, Heller G*, et al.* The prognostic significance of respiratory rate in patients with pneumonia: a retrospective analysis of data from 705,928 hospitalized patients in Germany from 2010-2012. Dtsch Arztebl Int. 2014;111(29-30):503-v.

272. Sasaki K, Maruyama RJTTjoem. Consciously controlled breathing decreases the high-frequency component of heart rate variability by inhibiting cardiac parasympathetic nerve activity. 2014;233(3):155-63.

273. Bernardi L, Wdowczyk-Szulc J, Valenti C, Castoldi S, Passino C*, et al.* Effects of controlled breathing, mental activity and mental stress with or without verbalization on heart rate variability. 2000;35(6):1462-9.

274. Patwardhan AR, Evans JM, Bruce EN, Eckberg DL, Knapp CFJJoAP. Voluntary control of breathing does not alter vagal modulation of heart rate. 1995;78(6):2087-94.

275. Secher N. Heart rate at the onset of static exercise in man with partial neuromuscular blockade. The Journal of physiology. 1985;368(1):481-90.

276. Lasevicius T, Ugrinowitsch C, Schoenfeld BJ, Roschel H, Tavares LD*, et al.* Effects of different intensities of resistance training with equated volume load on muscle strength and hypertrophy. Eur J Sport Sci. 2018;18(6):772-80.

277. Holm L, Reitelseder S, Pedersen TG, Doessing S, Petersen SG*, et al.* Changes in muscle size and MHC composition in response to resistance exercise with heavy and light loading intensity. J Appl Physiol. 2008;105(5):1454-61.

278. Mangine GT, Hoffman JR, Wang R, Gonzalez AM, Townsend JR*, et al.* Resistance training intensity and volume affect changes in rate of force

development in resistance-trained men. Eur J Appl Physiol. 2016;116(11-12):2367-74.

279. Thamm A, Freitag N, Figueiredo P, Doma K, Rottensteiner C*, et al.* Can heart rate variability determine recovery following distinct strength loadings? A randomized cross-over trial. 2019;16(22):4353.

280. Flatt AA, Globensky L, Bass E, Sapp BL, Riemann BLJS. Heart rate variability, neuromuscular and perceptual recovery following resistance training. 2019;7(10):225.

281. Schneider C, Wiewelhove T, Raeder C, Flatt AA, Hoos O*, et al.* Heart rate variability monitoring during strength and high-intensity interval training overload microcycles. 2019;10:582.

282. De Souza JC, Tibana RA, Cavaglieri CR, Vieira DCL, De Sousa NMF*, et al.* Resistance exercise leading to failure versus not to failure: effects on cardiovascular control. BMC Cardiovasc Disord. 2013;13.

283. Pareja-Blanco F, Rodriguez-Rosell D, Sanchez-Medina L, Ribas-Serna J, Lopez-Lopez C*, et al.* Acute and delayed response to resistance exercise leading or not leading to muscle failure. Clin Physiol Funct Imaging. 2017;37(6):630-9.

284. Cerqueira MS, Pereira R, Mesquita GNd, Rocha T, Moura Filho AGdJMRdEF. Rate of force development to evaluate the neuromuscular fatigue and recovery after an intermittent isometric handgrip task with different blood flow restriction conditions. 2019;25(1).

285. Ahtiainen JP, Pakarinen A, Kraemer WJ, Hakkinen KJCJoAP. Acute hormonal responses to heavy resistance exercise in strength athletes versus nonathletes. 2004;29(5):527-43.

286. Brownstein CG, Dent JP, Parker P, Hicks KM, Howatson G*, et al.* Etiology and recovery of neuromuscular fatigue following competitive soccer match-play. 2017;8:831.

287. Yu S, Katoh T, Makino H, Mimuno S, Sato SJRiSM. Age and heart rate variability after soccer games. 2010;18(4):263-9.

288. Heffernan KS, Kelly EE, Collier SR, Fernhall B. Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. Eur J Cardiovasc Prev Rehabil. 2006;13(1):80-6.

289. Nuuttila O-P, Kyröläinen H, Häkkinen K, Nummela AJIJoSM. Acute Physiological Responses to Four Running Sessions Performed at Different Intensity Zones. 2020.

290. Wiewelhove T, Fernandez-Fernandez J, Raeder C, Kappenstein J, Meyer T*, et al.* Acute responses and muscle damage in different high-intensity interval running protocols. 2015.

291. Leeder JD, van Someren KA, Gaze D, Jewell A, Deshmukh NI*, et al.* Recovery and adaptation from repeated intermittent-sprint exercise. 2014;9(3):489-96.

292. Gonzalez-Badillo JJ, Rodriguez-Rosell D, Sanchez-Medina L, Ribas J, Lopez-Lopez C*, et al.* Short-term Recovery Following Resistance Exercise Leading or not to Failure: How does manipulating the 'level of effort' impact post-exercise recovery? What are potential implications for optimizing athletic performance? Int J Sports Med. 2016;37(4):295-304.

293. Helland C, Midttun M, Saeland F, Haugvad L, Olstad DS*, et al.* A strength-oriented exercise session required more recovery time than a power-oriented exercise session with equal work. 2020;8:e10044.

294. Garrett J, Graham SR, Eston RG, Burgess DJ, Garrett LJ*, et al.* A Novel Method of Assessment for Monitoring Neuromuscular Fatigue in Australian Rules Football Players. 2019;14(5):598-605.

295. Markovic G, Dizdar D, Jukic I, Cardinale MJTJoS, Research C. Reliability and factorial validity of squat and countermovement jump tests. 2004;18(3):551-5.

296. Carlock JM, Smith SL, Hartman MJ, Morris RT, Ciroslan DA*, et al.* The relationship between vertical jump power estimates and weightlifting ability: a field-test approach. 2004;18(3):534-9.

297. Taylor K, Chapman D, Cronin J, Newton MJ, Gill NJJASC. Fatigue monitoring in high performance sport: a survey of current trends. 2012;20(1):12-23.

298. Heishman AD, Daub BD, Miller RM, Freitas ED, Frantz BA*, et al.* Countermovement jump reliability performed with and without an arm swing in NCAA division 1 intercollegiate basketball players. 2018;50:669.

299. Aoki MS, Ronda LT, Marcelino PR, Drago G, Carling C*, et al.* Monitoring training loads in professional basketball players engaged in a periodized training program. 2017;31(2):348-58.

300. Carroll TJ, Taylor JL, Gandevia SCJJoAP. Recovery of central and peripheral neuromuscular fatigue after exercise. 2017;122(5):1068-76.

301. Strojnik VJIJSM. Neuro-muscular fatigue and recovery dynamics following anaerobic interval workload. 2005;26:1-6.

302. Izquierdo M, Ibanez J, Calbet J, González-Izal M, Navarro-Amézqueta I*, et al.* Neuromuscular fatigue after resistance training. 2009;30(8):614.

303. Cheng AJ, Rice CLJJoAP. Fatigue and recovery of power and isometric torque following isotonic knee extensions. 2005;99(4):1446-52.

304. Fonseca FS, Costa BDdV, Ferreira MEC, Paes S, de Lima-Junior D*, et al.* Acute effects of equated volume-load resistance training leading to muscular failure versus non-failure on neuromuscular performance. 2020;18(2):94-100.

305. Gandevia SJAps. Neural control in human muscle fatigue: changes in muscle afferents, moto neurones and moto cortical drive. 1998;162(3):275-83.

306. Kent-Braun J, LeBlanc RJM, Sports Si, Exercise. Quantitating central activation failure during maximal voluntary isometric contractions. 452. 1995;27(5):S80.

307. Lagally KM, Robertson RJ, Gallagher KI, Goss FL, Jakicic JM*, et al.* Perceived exertion, electromyography, and blood lactate during acute bouts of resistance exercise. 2002;34(3):552-9.

308. Cheung K, Hume PA, Maxwell LJSm. Delayed onset muscle soreness. 2003;33(2):145-64.

309. Ide BN, Leme TC, Lopes CR, Moreira A, Dechechi CJ*, et al.* Time course of strength and power recovery after resistance training with different movement velocities. 2011;25(7):2025-33.

310. Tesch PA, Colliander EB, Kaiser PJEjoap, physiology o. Muscle metabolism during intense, heavy-resistance exercise. 1986;55(4):362-6.

311. Thornton MK, Potteiger JAJM, Sports Si, Exercise. Effects of resistance exercise bouts of different intensities but equal work on EPOC. 2002;34(4):715-22.

312. DaSilva SG, Goss FL, Metz KFJJoS, Research C. Perception of effort during resistance exercise. 1997;11(4):261-5.

313. Fragala MS, Kraemer WJ, Denegar CR, Maresh CM, Mastro AM*, et al.* Neuroendocrine-immune interactions and responses to exercise. 2011;41(8):621-39.

314. Furlan R, Piazza S, Dell'Orto S, Gentile E, Cerutti S*, et al.* Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. 1993;27(3):482-8.

315. Niewiadomski W, G siorowska A, Krauss B, Mroz A, Cybulski GJCP*, et al.* Suppression of heart rate variability after supramaximal exertion. 2007;27(5):309-19.

316. Dousset E, Avela J, Ishikawa M, Kallio J, Kuitunen S*, et al.* Bimodal recovery pattern in human skeletal muscle induced by exhaustive stretch-shortening cycle exercise. 2007;39(3):453.

317. McMurray RG, Soares J, Caspersen CJ, McCurdy T. Examining variations of resting metabolic rate of adults: a public health perspective. Med Sci Sports Exerc. 2014;46(7):1352-8.

318. Bray GA. Handbook of Obesity--Volume 1: Epidemiology, Etiology, and Physiopathology: CRC Press; 2014.

319. Dolezal BA. Muscle damage and resting metabolic rate after acute resistance exercise with an eccentric overload: University of Kansas; 1998.

320. Williamson DL, Kirwan JPJTJoGSABS, Sciences M. A single bout of concentric resistance exercise increases basal metabolic rate 48 hours after exercise in healthy 59–77-year-old men. 1997;52(6):M352-M5.

321. Burt DG, Lamb K, Nicholas C, Twist C. Effects of exercise-induced muscle damage on resting metabolic rate, sub-maximal running and post-exercise oxygen consumption. Eur J Sport Sci. 2014;14(4):337-44.

322. Paulsen G, Ramer Mikkelsen U, Raastad T, Peake JMJEir. Leucocytes, cytokines and satellite cells: what role do they play in muscle damage and regeneration following eccentric exercise? 2012;18.

323. Faulkner J, Opiteck J, Brooks SJIjosm. Injury to skeletal muscle during altitude training: induction and prevention. 1992;13(S 1):S160-S2.

324. Black CD, Elder CP, Gorgey A, Dudley GAJJoap. High specific torque is related to lengthening contraction-induced skeletal muscle injury. 2008;104(3):639-47.

325. González-Hernández JM, García-Ramos A, Castaño-Zambudio A, Capelo-Ramírez F, Marquez G*, et al.* Mechanical, metabolic, and perceptual acute responses to different set configurations in full squat. 2020;34(6):1581-90.

326. Bartolomei S, Sadres E, Church DD, Arroyo E, Iii JAG*, et al.* Comparison of the recovery response from high-intensity and high-volume resistance exercise in trained men. Eur J Appl Physiol. 2017;117(7):1287-98.

327. Paschalis V, Koutedakis Y, Jamurtas AZ, Mougios V, Baltzopoulos VJTJoS*, et al.* Equal volumes of high and low intensity of eccentric exercise in relation to muscle damage and performance. 2005;19(1):184-8.

328. Boullosa DA, Nakamura FY, Perandini LA, Leicht ASJMRdEF. Autonomic correlates of Yo-Yo performance in soccer referees. 2012;18(2):291-7.

329. Daanen HA, Lamberts RP, Kallen VL, Jin A, Van Meeteren NLJIjosp*, et al.* A systematic review on heart-rate recovery to monitor changes in training status in athletes. 2012;7(3):251-60.

330. Borresen J, Lambert MIJSm. Autonomic control of heart rate during and after exercise. 2008;38(8):633-46.

331. Lamberts RP, Swart J, Noakes TD, Lambert MIJEjoap. Changes in heart rate recovery after high-intensity training in well-trained cyclists. 2009;105(5):705-13.

332. Kamath MV, Fallen ELJCribe. Power spectral analysis of heart rate variability: a noninvasive signature of cardiac autonomic function. 1993;21(3):245-311.

333. Akselrod S, Gordon D, Ubel FA, Shannon DC, Berger A*, et al.* Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. 1981;213(4504):220-2.

334. Appel ML, Berger RD, Saul JP, Smith JM, Cohen RJJJotACoC. Beat to beat variability in cardiovascular variables: noise or music? 1989;14(5):1139-48.

335. Billman GEJFip. The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. 2013;4:26.

336. Yamamoto Y, Hughson RL, Peterson JCJJoap. Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis. 1991;71(3):1136-42.

337. Warren JH, Jaffe RS, Wraa CE, Stebbins CLJAJoP-R, Integrative, Physiology C. Effect of autonomic blockade on power spectrum of heart rate variability during exercise. 1997;273(2):R495-R502.

338. Houle MS, Billman GE. Low-frequency component of the heart rate variability spectrum: a poor marker of sympathetic activity. Am J Physiol. 1999;276(1):H215-23.

339. Houle MS, Billman GEJAJoP-H, Physiology C. Low-frequency component of the heart rate variability spectrum: a poor marker of sympathetic activity. 1999;276(1):H215-H23.

340. Martinmäki K, Häkkinen K, Mikkola J, Rusko HJEjoap. Effect of low-dose endurance training on heart rate variability at rest and during an incremental maximal exercise test. 2008;104(3):541-8.

341. Martinmäki K, Rusko HJEjoap. Time-frequency analysis of heart rate variability during immediate recovery from low and high intensity exercise. 2008;102(3):353-60.

342. Casties J-F, Mottet D, Le Gallais DJIJoSM. Non-linear analyses of heart rate variability during heavy exercise and recovery in cyclists. 2006;27(10):780-5.

343. Taylor JA, Myers CW, Halliwill JR, Seidel H, Eckberg DLJAJoP-H*, et al.* Sympathetic restraint of respiratory sinus arrhythmia: implications for vagal-cardiac tone assessment in humans. 2001;280(6):H2804-H14.

344. Malliani A, Julien C, Billman GE, Cerutti S, Piepoli MF*, et al.* Cardiovascular variability is/is not an index of autonomic control of circulation. 2006;101(2):684-8.

345. Pagani M, Lombardi F, Guzzetti S, Sandrone G, Rimoldi O*, et al.* Power spectral density of heart rate variability as an index of sympatho-vagal interaction in normal and hypertensive subjects. 1984;2(3):S383-5.

346. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R*, et al.* Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. 1986;59(2):178-93.

347. Eckberg DLJC. Sympathovagal balance: a critical appraisal. 1997;96(9):3224-32.

348. Kingwell BA, Thompson JM, Kaye DM, McPherson G, Jennings GL*, et al.* Heart rate spectral analysis, cardiac norepinephrine spillover, and muscle sympathetic nerve activity during human sympathetic nervous activation and failure. 1994;90(1):234-40.

349. Krieger JWJTJoS, Research C. Single vs. multiple sets of resistance exercise for muscle hypertrophy: a meta-analysis. 2010;24(4):1150-9.

350. Krieger JWJTJoS, Research C. Single versus multiple sets of resistance exercise: a meta-regression. 2009;23(6):1890-901.

351. Carpinelli RN, Otto RMJSm. Strength training. 1998;26(2):73-84.

352. Sammito S, Böckelmann I. [Options and limitations of heart rate measurement and analysis of heart rate variability by mobile devices: A systematic review]. Herzschrittmachertherapie & Elektrophysiologie. 2016;27(1):38-45.

353. Speer KE, Semple S, Naumovski N, McKune AJJEJoIiH, Psychology, Education. Measuring heart rate variability using commercially available devices in healthy children: A validity and reliability study. 2020;10(1):390-404.

XIII – APPENDIXES

XIII. APPENDIXES

This chapter provides supplementary information and documents related to this PhD study. Mainly this chapter divided into two main sections and first section provides the supplemental tables, figures and graphs related to the systematic reviews and meta-analysis. Second section provides the supplemental results, participant inform consent forms and questionnaires related to the experimental study.

13.1. STUDY 1

13.1.1. Results of the funnel plot asymmetry tests

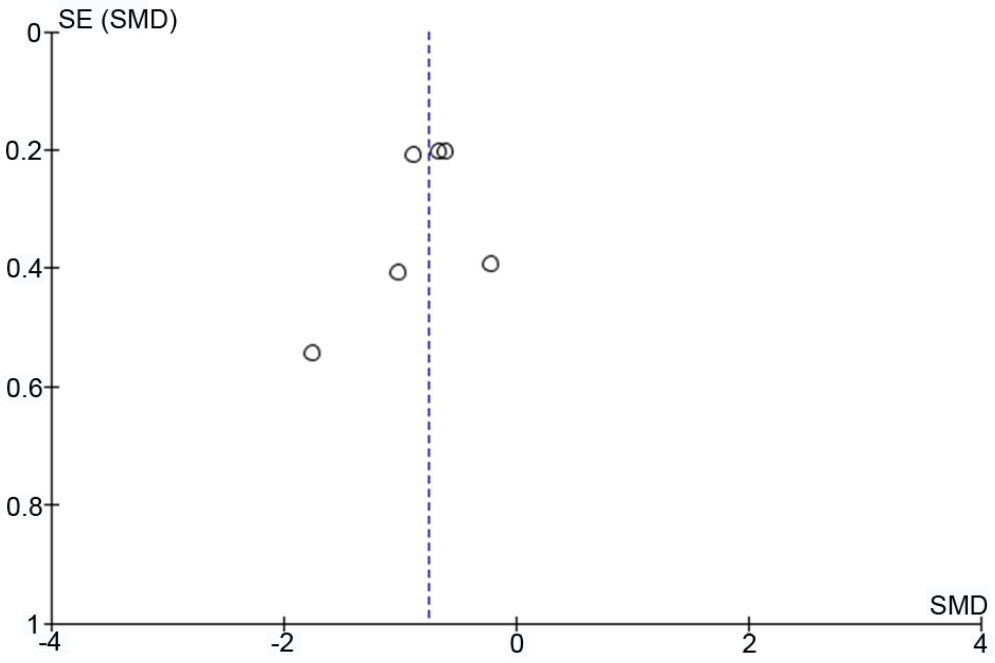


Figure 160. Funnel plot RMSSD control - treatment groups

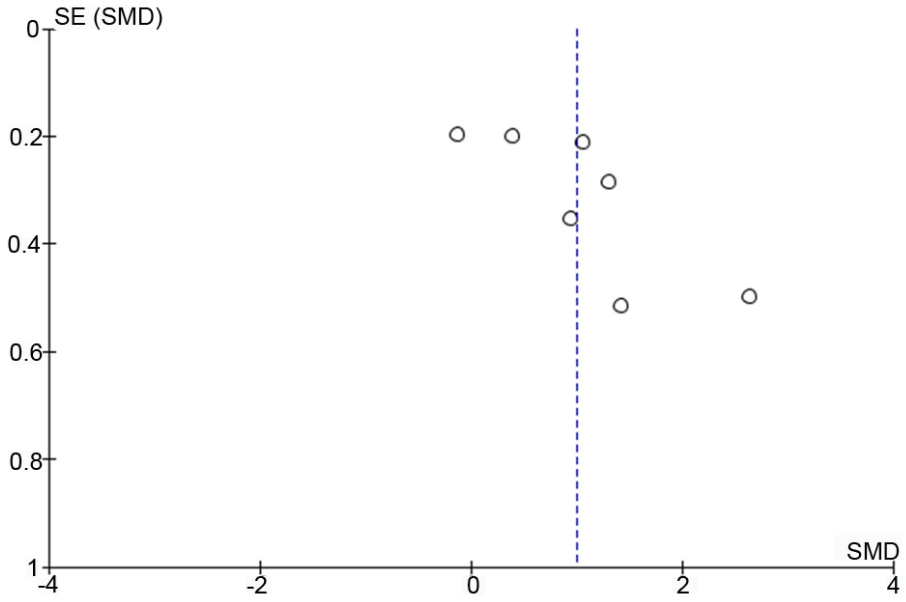


Figure 161: Funnel plot LF(nu) control - treatment groups

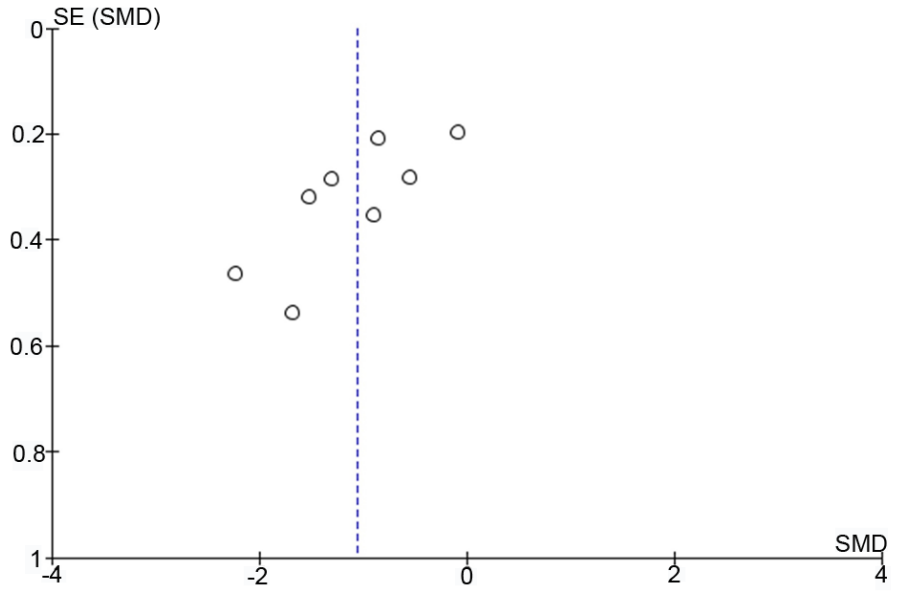


Figure 162: Funnel plot HF(nu) control - treatment groups

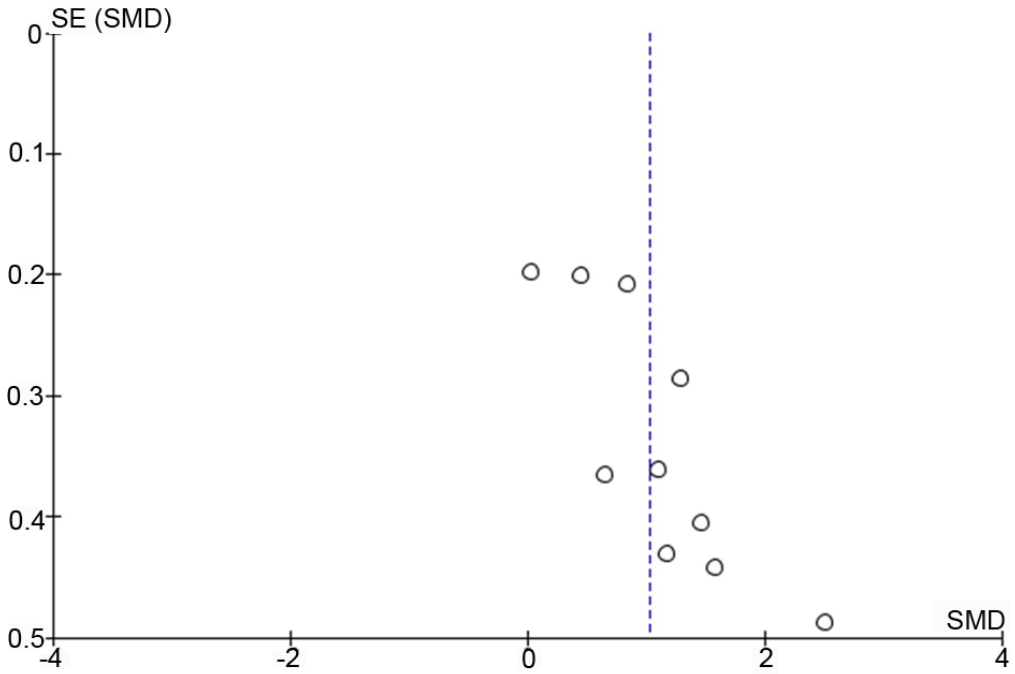


Figure 163: Funnel plot LF/HF ratio - control group vs. treatment group

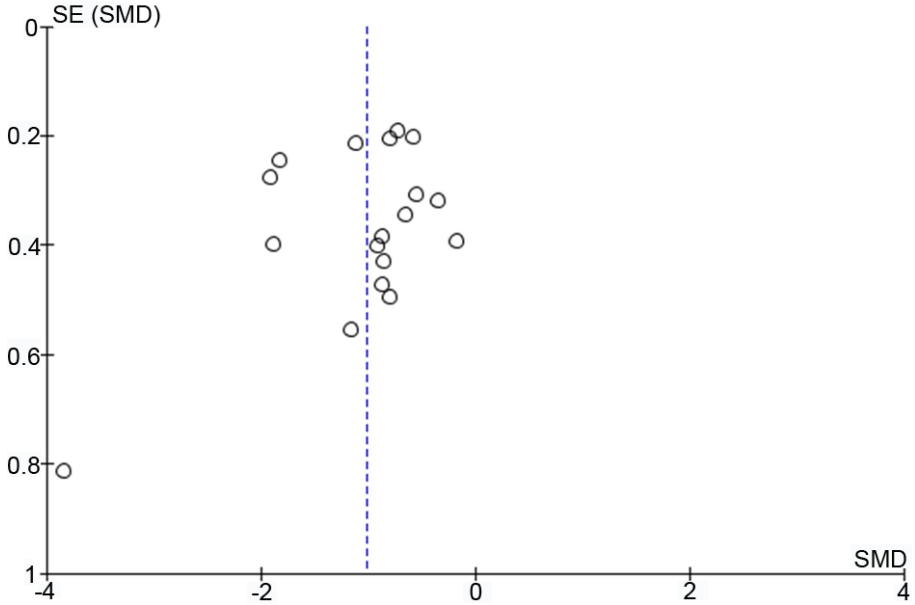


Figure 164: Funnel plot RMSSD - pre-post test

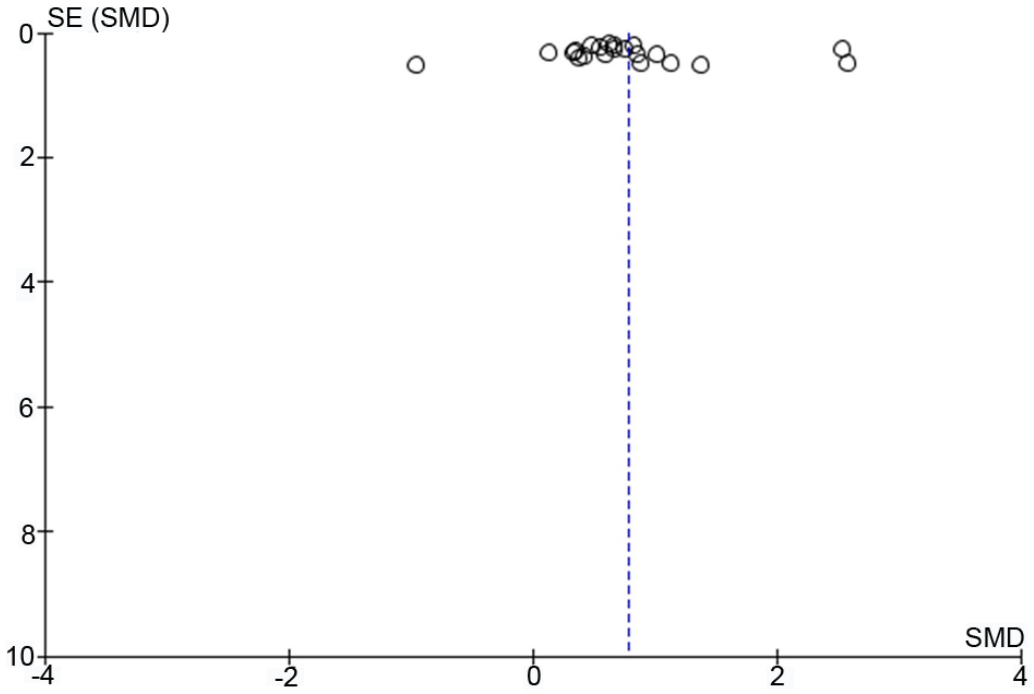


Figure 165: Funnel plot LF(nu) - pre-post test

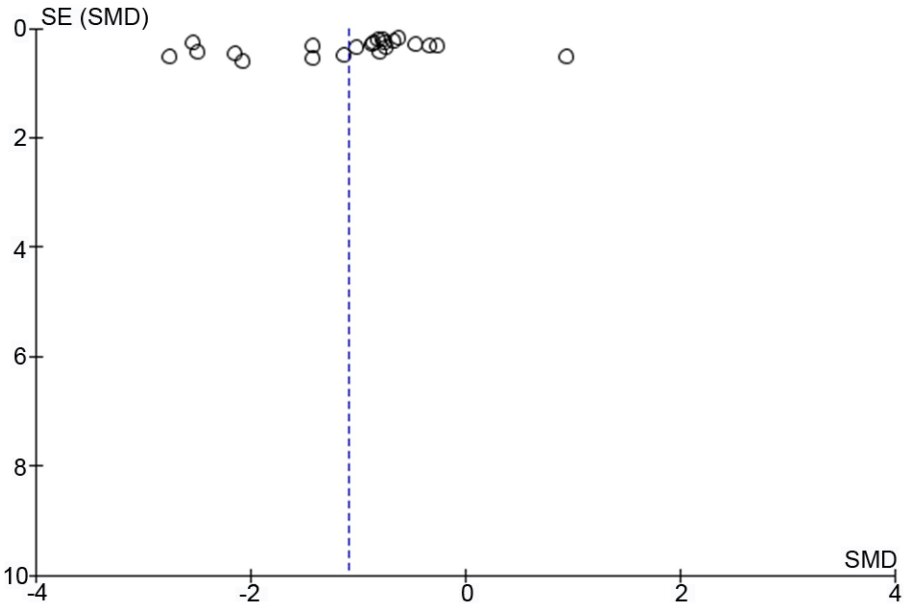


Figure 166: Funnel plot HF(nu) - pre-post test

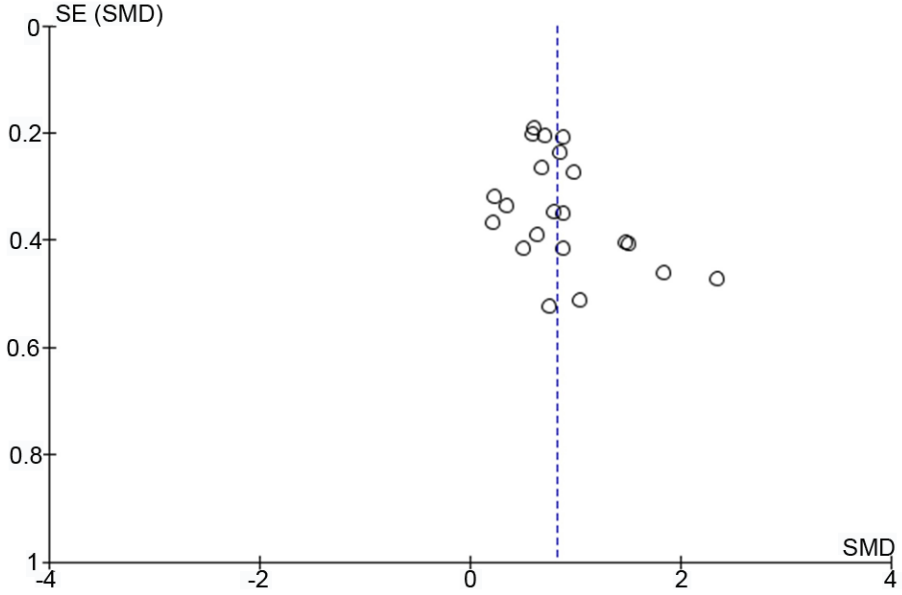


Figure 167: Funnel plot LF/HF ratio - pre-post test

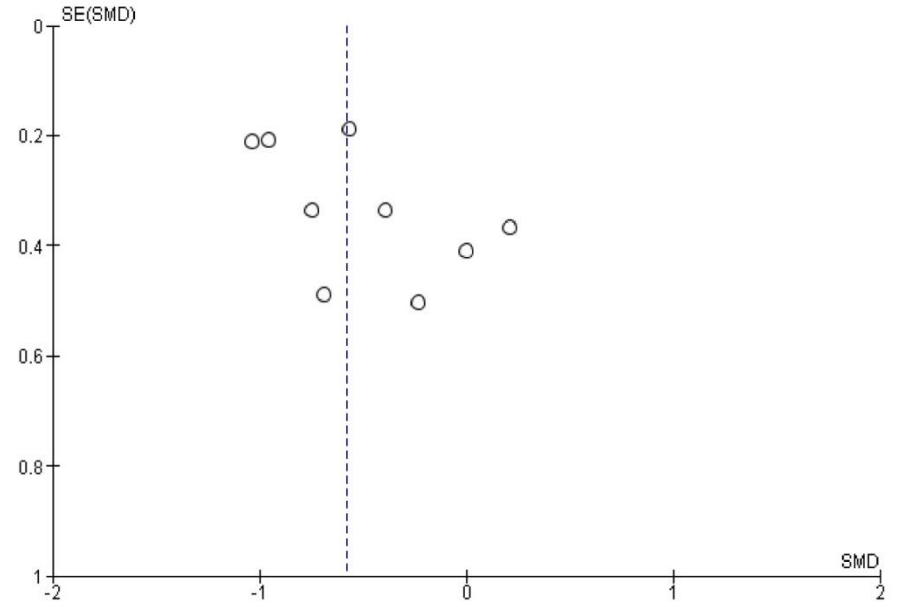


Figure 168: Funnel plot SDNN - pre-post test

13.1.2. Study quality assessment

Table 11. Quality Assessment of Controlled Intervention Studies - Study Quality Assessment Tool

Criteria	Kingsley et al., 2014	Mayo et al., 2016b	Saccomani et al., 2014	Kingsley et al., 2018	Lima et al., 2011	Rezk et al., 2006	Teixeira et al., 2011	Correa et al., 2017	Kingsley et al., 2016
1. Was the study described as randomized, a randomized trial, a randomized clinical trial, or an RCT?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🔴
2. Was the method of randomization adequate (i.e., use of randomly generated assignment)?	🟡	🟡	🟡	🟡	🟡	🟡	🟡	🟡	🔴
3. Was the treatment allocation concealed (so that assignments could not be predicted)?	🟡	🟢	🟢	🟡	🟡	🟢	🟢	🟡	🟡
4. Were study participants and providers blinded to treatment group assignment?	🔴	🟡	🟡	🟡	🟡	🟡	🟡	🟡	🟡
5. Were the people assessing the outcomes blinded to the participants' group assignments?	🟡	🟡	🟡	🟡	🟡	🟡	🟡	🟡	🟡
6. Were the groups similar at baseline on important characteristics that could affect outcomes (e.g., demographics, risk factors, co-morbid conditions)?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
7. Was the overall drop-out rate from the study at endpoint 20% or lower of the number allocated to treatment?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
8. Was the differential drop-out rate (between treatment groups) at endpoint 15 percentage points or lower?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
9. Was there high adherence to the intervention protocols for each treatment group?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
10. Were other interventions avoided or similar in the groups (e.g., similar background treatments)?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
11. Were outcomes assessed using valid and reliable measures, implemented consistently across all study participants?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
12. Did the authors report that the sample size was sufficiently large to be able to detect a difference in the main outcome between groups with at least 80% power?	🟢	🔴	🔴	🔴	🔴	🔴	🔴	🔴	🟢
13. Were outcomes reported or subgroups analyzed prespecified (i.e., identified before analyses were conducted)?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
14. Were all randomized participants analyzed in the group to which they were originally assigned, i.e., did they use an intention-to-treat analysis?	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢

Table 12. Quality Assessment Tool for Before-After (Pre-Post) Studies with No Control Group - Study Quality Assessment Tools

Criteria	Figueiredo et al. (2016)	Freitas et al. (2018)	Iglesias-Soler et al. (2014)	Kliszczewicz et al. (2016)	Lima et al. (2019)	Macêdo et al. (2019)	Monteiro et al. (2018)	Paz et al. (2017)	Thamm et al. (2019)	Tibana et al. (2013)	Isidoro et al. (2017)	Goessler et al. (2013)	Heffernan et al. (2006)	Kingsley et al. (2009)	Kingsley et al. (2010)	Okuno et al. (2014)	Mayo et al. (2016a)
1. Was the study question or objective clearly stated?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
2. Were eligibility/selection criteria for the study population prespecified and clearly described?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
3. Were the participants in the study representative of those who would be eligible for the test/service/intervention in the general or clinical population of interest?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
4. Were all eligible participants that met the prespecified entry criteria enrolled?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
5. Was the sample size sufficiently large to provide confidence in the findings?	G	Y	Y	Y	Y	Y	G	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
6. Was the test/service/intervention clearly described and delivered consistently across the study population?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
7. Were the outcome measures prespecified, clearly defined, valid, reliable, and assessed consistently across all study participants?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
8. Were the people assessing the outcomes blinded to the participants' exposures/interventions?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
9. Was the loss to follow-up after baseline 20% or less? Were those lost to follow-up accounted for in the analysis?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
10. Did the statistical methods examine changes in outcome measures from before to after the intervention? Were statistical tests done that provided p values for the pre-to-post changes?	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G	G
11. Were outcome measures of interest taken multiple times before the intervention and multiple times after the intervention (i.e., did they use an interrupted time-series design)?	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
12. If the intervention was conducted at a group level (e.g., a whole hospital, a community, *etc.*) did the statistical analysis take into account the use of individual-level data to determine effects at the group level?	Y	Y	Y	Y	Y	Y		Y	Y	Y	Y	Y	Y	Y	Y	Y	Y

13.1.3. Published scientific article (Study 1)

Reference:

Marasingha-Arachchige SU, Rubio-Arias JÁ, Alcaraz PE, Chung LH. Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis. Journal of Sport and Health Science. 2020. DOI: 10.1016/j.jshs.2020.11.008

ARTICLE IN PRESS

HOSTED BY

ELSEVIER

Available online at www.sciencedirect.com

ScienceDirect

Journal of Sport and Health Science 00 (2021) 1–17

www.jshs.org.cn

Review

Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis

Sajith U. Marasingha-Arachchige [a], Jacobo Á. Rubio-Arias [b], Pedro E. Alcaraz [a,c], Linda H. Chung [a,c,*]

[a] *UCAM Research Center for High Performance Sport, Catholic University of Murcia, Murcia 30107, Spain*

[b] *LFE Research Group, Department of Health and Human Performance, Faculty of Physical Activity and Sport Science-INEF, Universidad Politécnica de Madrid, Madrid 28040, Spain*

[c] *Faculty of Sport, Catholic University of Murcia, Murcia 30107, Spain*

Received 23 June 2020; revised 19 September 2020; accepted 28 September 2020

Abstract

Background: There is controversial evidence regarding the effect of acute resistance exercise (ARE) on heart rate variability (HRV) parameters, which indicates the activities of the cardiac autonomic nervous system. The aim of this study was to perform a systematic review and meta-analysis of the literature on the effect of ARE on HRV parameters and identify its possible moderating factors.

Methods: The PubMed–Medline, Web of Science, SPORTDiscus, and Cochrane databases were searched. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) declaration was followed, and the methodological quality of the studies was evaluated. The level of significance was set at $p \leq 0.05$. Twenty-six studies met the inclusion criteria. Main effects analyses between pre- and post-test interventions demonstrated an increase in normalized units low frequency ($p < 0.001$; standardized mean difference (SMD) = 0.78; 95% confidence interval (95%CI): 0.46–1.11) and low frequency/high frequency ratio ($p < 0.001$; SMD = 0.82; 95%CI: 0.64–0.99) and a decrease in standard deviation of the normal-to-normal (NN) interval ($p < 0.001$; SMD = −0.58; 95%CI: −0.85 to −0.30), root mean square of the successive differences ($p < 0.001$; SMD = −1.01; 95%CI: −1.29 to −0.74), and normalized units high frequency ($p < 0.001$; SMD: −1.08; 95%CI: −1.43 to −0.73) following ARE in healthy individuals (mean age (standard deviation) range: 15 ± 1 and 48 ± 2 years).

Results: There were differences between the subgroups in the number of sets used in an exercise ($p = 0.05$) for root mean square of the successive differences, as well as for exercise intensity ($p = 0.01$) and rest between sets ($p = 0.05$) for normalized units high frequency. Interestingly, there were differences between the subgroups in training volume for root mean square of the successive differences ($p = 0.01$), normalized units high frequency ($p = 0.003$) and normalized units low frequency ($p = 0.02$).

Conclusion: Overall, there was a withdrawal of cardiac parasympathetic and activation of cardiac sympathetic modulations following ARE, and these changes were greater with higher training volume ~30 min after ARE in healthy individuals. Furthermore, the number of sets, intensity, and rest between sets affected HRV parameters. However, gender, body mass index, and training status did not influence the changes in HRV parameters as a response to ARE.

Keywords: Cardiac; Parasympathetic; Sympathetic

1. Introduction

Resistance training plays an integral role in competitive athletes' training programs and is also widely used by recreationally active individuals to enhance their physical qualities (e.g., muscle strength, power output, and speed) and body composition (bone mass and muscle mass). More important, resistance training is used to reduce the risk of injury occurrence.[1,2] According to the National Strength and Conditioning Association (NSCA), resistance training entails a wide range of resistive loads and a variety of training modalities to optimize the effects of training and improve sports performance and overall health.[3] Physiological adaptation requires an adequate exercise stimulus to achieve training and performance goals. Furthermore, proper recovery from such training stress is necessary because the body may be exposed to continuous training-induced fatigue, which could lead to non-functional

Peer review under responsibility of Shanghai University of Sport.

*Corresponding author.

E-mail address: llchung@ucam.edu (L.H. Chung).

https://doi.org/10.1016/j.jshs.2020.11.008

overreaching or overtraining and, ultimately, to fatigue syndrome.[4] In a highly competitive environment, the most efficient and optimal recovery time is one of the major objectives of sports coaches and fitness trainers since it allows more time for improving an athlete's performance (i.e., more training sessions, better training adaptations and less risk of injuries). To address the recovery status, recent studies have shown that heart rate variability (HRV) parameters, such as root mean square of the successive differences (RMSSD), normalized units low frequency (LFnu), normalized units high frequency (HFnu), and low frequency/high frequency (LF/HF) ratio, could be used as a physiological monitoring tool for recovery[5–8] and standard deviation of the normal-to-normal (NN) interval (SDNN) to understand the overall autonomic modulation.[7]

HRV is the physiological variation in the time interval between heart beats.[9] The time between successive heartbeats is never constant and can vary slightly even when the heart rate appears stable.[10] Previous studies have reported that acute resistance exercise (ARE) increases the cardiac sympathetic modulation while decreasing the cardiac parasympathetic modulation.[11–16] During post-exercise recovery, the early phase of post-exercise is characterized by sympathetic predominance, and the cardiac parasympathetic stimulation is the predominant autonomic activity.[17] Changes in cardiac sympathetic and parasympathetic modulation can be monitored by examining HRV parameters.[18–20] Thus, adjusting the protocol of the ARE session according to the recovery status of the cardiac autonomic modulation (i.e., HRV) could be advantageous in optimizing the microcycle periodization, thereby increasing training adaptation and performance and, most importantly, avoiding injuries and overtraining.

The parameters of HRV are altered following an ARE session, and the magnitude of change may depend on the characteristics of the resistance training protocol, such as the number of repetitions, sets, rest time between sets, amount of exercise per workout, intensity (based on 1 repetition maximum (1RM)) and volume. In a review by Kingsley and Figueroa[21] that examined 10 studies published before 2014, cardiac parasympathetic modulation decreases (i.e., ↓HFnu) and cardiac sympathetic modulation increases (i.e., ↑LFnu and ↑LF/HF ratio) following a resistance training session in healthy young men and women. Since then, several experimental studies have examined the effect of ARE on HRV.[14,22–39] However, there are some discrepancies in the findings since some studies show the opposite effect on HRV parameters following an ARE session.[14,32,39,40] Furthermore, it is unclear what the magnitude of the ARE has on HRV parameters. Additionally, to our knowledge, no study has investigated (i.e., meta-analyses) the possible moderating factors of ARE that affect HRV parameters.

The overall goal of this systematic review and meta-analysis was to understand how an ARE session affects the HRV characteristics and identify the possible moderating factors that contribute to the cardiac autonomic activity during post-exercise recovery. Moreover, findings from this systematic review and meta-analysis may benefit the scientific community in better understanding how an ARE session affects the cardiac autonomic modulation, as well as in providing a monitoring tool for fitness trainers and coaches with regards to determining the athlete's recovery level. Therefore, the objectives of the present study were to (1) systematically review and conduct a meta-analysis of the studies that have investigated ARE on HRV parameters and (2) determine the factors that could affect the recovery process of cardiac autonomic modulation following an ARE session.

2. Methods

The recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) declaration[41] were followed during this methodological process.

2.1. Data sources

A comprehensive literature search was performed using PubMed—Medline, Web of Science, SPORTDiscus, and Cochrane Library electronic databases, from inception through November 30, 2019. The keywords and categorical searches were (1) "heart rate variability" OR "HRV" OR "vagal" OR "autonomic function" and (2) "resistance training" OR "strength training" OR "weight training" OR "power training" OR "weightlifting" OR "full body" OR "circuit*" OR "neuromuscular training" OR "bodyweight training". Second, the Boolean operator AND was used to combine categories (1) and (2). Additional records were identified while reviewing the reference lists of the books written in the relevant area.

2.2. Selection criteria

The eligibility criteria were pre-established by the authors. The inclusion criteria of articles included the following: (1) the study examined ARE on HRV after 1 training session, (2) study participants were healthy individuals or athletes (males or females), (3) the study gave a detailed explanation of the resistance training protocol, (4) the study provided information on outcomes both at baseline and following intervention, (5) the study reported data that was recorded between 8 and 30 min after the intervention, and (6) the study included at least 1 ARE training intervention group. Research studies were excluded for any of the following reasons: (1) the study had a sample population with pathologies, (2) the study was not an original investigation published in a peer-reviewed journal, (3) the study did not specify the test battery to be evaluated, (4) the study did not provide relevant data in the published article or if the corresponding author did not provide the data after being contacted, or (5) the study had methodological issues that may have led to potential risk of carryover effects due to inadequate recovery period ($\leq$24 h).

2.3. Study selection, data extraction, and outcomes

Two of the authors (SUMA and JARA) conducted the electronic database search and selection of included studies according to the previously established criteria. Any disagreements regarding the inclusion/exclusion of articles were discussed and resolved by consensus. The following data were extracted from the selected articles: authors, number of participants,

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

subject characteristics, exercise protocol, and outcomes of selected HRV parameters, including SDNN, RMSSD, HFnu, LFnu, and LF/HF ratio, since these are the most examined HRV parameters in other studies.[42–45] RMSSD and HFnu indicate the level of cardiac parasympathetic modulation,[42,45] while LFnu provides the degree of cardiac sympathetic modulation.[43,45] The LF/HF ratio presents the extent of sympathovagal balance, and SDNN represents overall autonomic modulation.[45] Thus, an increase in cardiac sympathetic modulation corresponds to an increase in LFnu and LF/HF ratio, while the dominance of cardiac parasympathetic modulation is shown by an increase in RMSSD and HFnu parameters.

2.4. Data synthesis

Data on the mean ± SD and sample size (n) were recorded from the included articles by one author (SUMA) and confirmed by a second author (JARA). The corresponding author of each included article was contacted if necessary data were not available in the published version. When studies reported two or more subgroups, the subgroups were combined into a single group in accordance with the Cochrane Handbook for Systematic Reviews of Interventions.[46] For studies that include pre- and post-intervention measurements, SD and n values were uploaded to the Review Manager software (RevMan 5.3; Cochrane Collaboration, Oxford, UK). In addition, mean, SD, and n values of the studies that included experimental and control groups were also uploaded. For each study, the mean difference (MD), change in SD and 95% confidence intervals (95%CIs) were calculated between pre- and post-intervention (i.e., differences within groups) and between experimental and control groups.

2.5. Meta-analyses

Meta-analyses were conducted on the changes in each outcome using Review Manager software. Since SDNN, RMSSD, LFnu, HFnu, and LF/HF ratio data were measured using different time durations (i.e., time period of collected data) or were presented using different units (e.g., natural logarithm or milliseconds squared), the MDs were standardized by dividing the values by their corresponding SDs and were weighted according to the inverse variance method. The standardized MD (SMD) in SDNN, RMSSD, LFnu, HFnu, and LF/HF ratio data for each study was pooled with a random-effects model.[46] The data analysis focused on the magnitude of the effects obtained.

2.6. Heterogeneity and risk of bias

The statistical heterogeneity between studies was evaluated using the Cochrane χ^2 test (I^2). I^2 values of <30%, 30%–60%, and >60% were considered as low, moderate and high levels of heterogeneity, respectively. A p value of ≤0.05 from the χ^2 test suggested the presence of heterogeneity,[47] which was likely due to the methodological diversity of the studies. Methodological quality was evaluated using the Study Quality Assessment Tools developed by the National Heart, Lung, and Blood Institute.[48] The tool for Quality Assessment of Controlled Intervention Studies was used for studies that included control groups, and the tool for Quality Assessment for Pre-Post Studies with No Control Group was used for studies that included only an experimental group. Publication bias was evaluated by analyzing the funnel plot asymmetry test.

2.7. Subgroup analyses

In our study, we decided to perform subgroup analyses using categorical variables and continuous variables without conducting meta-regression analysis for continuous variables. The reason for representing continuous variables as categorical variables for the subgroup analyses was to match the way these variables are presented by organizations like the NSCA. For example, the NSCA generally provides recommendations for training protocols (e.g., high intensity >85% 1RM, low intensity <65% 1RM). Thus, we believe that it is important to analyze the data in ways similar to practical scenarios in order to reduce the gap between the scientific evidence and practical application in resistance training sessions in the field or gym. Therefore, we performed subgroup analyses while considering the way resistance training sessions are practiced in the field, as well as the way they are presented in the NSCA guidelines.[1,49,50]

Subjects characteristics (gender, body mass index (BMI), and training status) and training characteristics (training intensity (% 1RM), number of repetitions, sets, rest between sets, amount of exercise per workout and training volume (number of repetitions × sets × exercises)) were assessed by subgroup analysis to examine their effect on selected HRV parameters. For BMI, ≤24.9 kg/m^2 (healthy weight) or >24.9 kg/m^2 (overweight) were considered as cut-off values, based on guidelines from the Centers for Disease Control and Prevention.[51] For gender, male and female were used for grouping trials. For resistance training variables, the cut-off values for grouping trials were determined by considering the way resistance training sessions are conducted in the field and the NSCA guidelines.[1,49,50] High (>85% 1RM), moderate (>65%–to 85% 1RM), and low (≤65% 1RM) values were used as cut-off points for training intensity.[1,49,50] Bodyweight as an intensity level was not included in the subgroup analysis. For the number of repetitions, <6, 6–10, and >10 repetitions were used as cut-off values. For the number of sets, cut-off values were set at <3, exactly 3, and >3 sets; and for the amount of exercise, <6, exactly 6, and >6 exercises per workout were used as cut-off values. For resting time between sets, <2 min, exactly 2 min, and >2 min were used as cut-off points. Regarding training volume (calculated as the number of repetitions × sets × exercises), cut-off points were set at <108 (low), 108 to <180 (medium), and ≥180 (high). Changes in possible moderating factors were expressed and analyzed as the difference between post- and pre-intervention values. Subgroup analyses were performed using Review Manager software.

3. Results

3.1. Study selection

From the initial electronic database search and other sources, 1449 records were identified. After removal of duplicates, 1076 titles and abstracts were evaluated, and

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

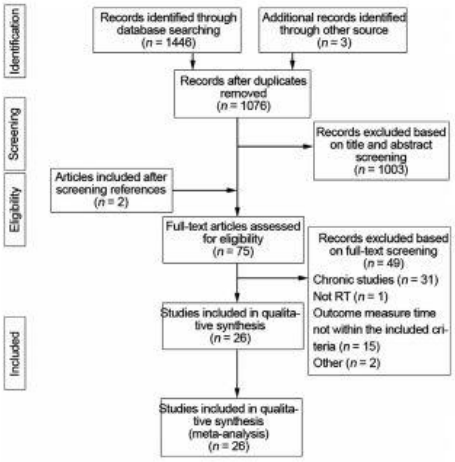


Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram regarding article selection for each stage of the systemic eligibility process. RT = resistance training.

1003 were excluded. Thus, the full text of 73 articles was assessed to determine eligibility for the inclusion of studies, and 2 additional studies were screened as a result of reviewing the reference lists. From these studies, 49 articles were excluded because they did not meet the inclusion criteria. After review, a total of 26 studies were included in the systematic review and meta-analysis.[14–16,22–31,34–40,52–57] All included articles were published between 2006 and 2019 (Fig. 1).

3.2. Characteristics of the interventions

Subjects were healthy and physically active, and the majority were resistance-exercise-trained individuals. Their age ranged (SD) between 15 ± 1 and 48 ± 2 years (mean ± SD). The samples included both males and females. BMI (SD) ranged from 20.0 ± 1.0 kg/m^2 to 27.5 ± 2.1 kg/m^2, although some studies did not report BMI values. The sample sizes in the included studies ranged from 8 to 34 subjects. Among the included studies, there were a total of 412 subjects for this systematic review and meta-analysis.

The amount of exercise performed during the resistance training sessions ranged from 1 to 8 exercises. The intensity of the resistance exercises performed ranged from bodyweight to 100% 1RM. Among these studies, 13 study groups performed at low intensity (≤65% 1RM), 25 performed at moderate intensity (>65% to 85% 1RM), and 3 performed at high intensity (>85%) (Table 1). With regards to measuring HRV parameters, most of the studies used Polar HR monitors and ECG monitors, with participants in a supine or seated position for 5–15 min. Additionally, most of the studies identified and corrected for or excluded the abnormalities (ectopic/artifacts) of beat-to-beat interval data before analyzing the HRV parameters. HRV measurement and data analyzing methods used in the included studies are presented in Supplementary Table 1.

3.3. Heterogeneity and risk of bias assessment

Except for SDNN (I^2 = 47%, p = 0.06), heterogeneity was present for changes in RMSSD (I^2 = 71%, $p < 0.001$), LFnu (I^2 = 83%, $p < 0.001$), HFnu (I^2 = 85%, $p < 0.001$), and LF/HF ratio (I^2 = 40%, p = 0.03) parameters among the pre-post intervention studies. Regarding control group interventions, heterogeneity was detected in LFnu (I^2 = 86%, $p < 0.001$), HFnu (I^2 = 80%, $p < 0.001$), and LF/HF ratio (I^2 = 78% $p <$ 0.001), but not in RMSSD (I^2 = 26%, p = 0.24).

The quality of the studies, according to the National Heart, Lung, and Blood Institute Study Quality Assessment Tools,[48] was high for the pre-post interventions (8.18 ± 0.53, out of a possible 12 points) and experimental-control interventions (9.56 ± 0.53, out of a possible 14 points) (see Supplementary Table 2, which illustrates the results of study quality). A funnel plot asymmetry test was used to determine publication bias. Visual interpretation of the funnel plot asymmetry tests (SMD values between pre-post tests and control-experimental tests) showed that SDNN, RMSSD, LFnu, HFnu and LF/HF ratio variables were asymmetrical, suggesting the presence of publication bias) (see Supplementary Figs. 1–9, which illustrate the results of the funnel plot asymmetry tests).

3.4. Main effects analysis

3.4.1. RMSSD

There were 18 effect size calculations from 15 studies (mean age = 23.5 years; 199 males, 42 females) that showed a decrease in RMSSD ($p < 0.001$; SMD = −1.01; 95%CI: −1.29 to −0.74) of ~30 min (8–30 min) after the ARE session compared to pre-test values. There were 6 effect size calculations from 4 studies (mean age = 22.3 years; 64 males, 58 females) that demonstrated a decrease in RMSSD ($p < 0.001$; SMD = −0.75; 95%CI: −1.01 to −0.49) post ~30 min (8–30 min) for ARE session compared to control groups (Fig. 2).

3.4.2. HFnu

There was a decrease in HFnu ($p < 0.001$; SMD = −1.08; 95%CI: −1.43 to −0.73) in 23 effect size calculations from 20 studies (mean age = 24.6 years; 251 males, 52 females) following ARE compared to baseline. When compared to a control group, the ARE group also decreased HFnu ($p < 0.001$; SMD = −1.06; 95%CI: −1.52 to −0.60) ~30 min (8–30 min) after the ARE session (Fig. 3) in 8 effect size calculations from 6 studies (mean age = 23.2 years; 74 males, 35 females).

3.4.3. LFnu

A total of 20 studies (mean age = 24.6 years; 250 males; 57 females), with 22 effect size calculations, showed an increase in LFnu ($p < 0.001$; SMD = 0.78; 95%CI: 0.46–1.11) after an ARE session compared to pre-intervention. Similarly, 6 studies (mean age = 23.2 years; 73 males, 40 females), with 7 effect size calculations, showed an increase in LFnu ($p < 0.001$;

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Table 1
Characteristics of the ARE sessions for each study.

Study	Participants	Control group	Training status	Age (year)	Protocol				
					Exercises (*n*)	Sets (*n*)	Repetitions (*n*)	Intensity (% 1RM)	Repetitions between sets
Macêdo et al. (2019)[19]	34 M: Healthy weight 19, Overweight 15	No	Adolescent	Healthy weight: 15 ± 1, Overweight: 16 ± 1	5	3	12	60	–
Thamm et al. (2019)[18]	10 M	No	RT	24 ± 3.8	1	5	10	70	120 s
					1	15	1	100	180 s
Lima et al. (2019)[22]	12 M	Yes (not healthy)	Normotensive	25.5 ± 5.7	1	1RM test			
Kingsley et al. (2018)[15]	27: 14 M, 13 F	Yes	RT	Men: 22 ± 3 Women: 23 ± 3	3	3	10	75	120 s
Monteiro et al. (2018)[14]	8 F	No	Recreationally RT	21.8 ± 2.2	4	1RM test			
de Freitas et al. (2018)[23]	16 M	No	Recreationally RT	24.9 ± 5.3	6	3	10	65	90 s
					3	6	10	65	90 s
					3	6	10	65	90 s
Paz et al. (2017)[24]	13 M	No	RT	26.2 ± 3.9	6	3	10	75	90 s
					6	3	10	75	90 s
					6	3	10	75	180 s
Neto et al. (2017)[25]	24 M	Yes	RT	20.5 ± 0.6 25 ± 4.1	1	4	UF	67	180 s
					1	4	8	67	180 s
Isidoro et al. (2017)[52]	29 M	No	Physically active	21.62 ± 2.63	3	3	12	70	–
Figueiredo et al. (2016)[30]	11 M (Pre-hypertensive)	No	RT	26.1 ± 3.6	8	3	8–10	70	120 s
					8	3	8–10	70	60 s
Kingsley et al. (2016)[29]	16: 11 M, 5 F	No	RT	23 ± 3	3	3	12	70	120 s
Kliszczewicz et al. (2016)[28]	10 M	No	Physically fit	26.4 ± 2.7	3	–	–	–	–
Mayo et al. (2016)[26]	13 M	Yes	RT	23 ± 3	1	5	34	75	720 s
					1	5	23.6	75	720 s
					1	5	32	75	720 s
					1	5	22.9	75	720 s
Mayo et al. (2016)[27]	17: 12 M, 5 F	Yes	RT	23 ± 3	1	5	8	80	180 s
					1	10	4	40	30 s
					1	40	1	10	18.5 s
Iglesias-Soler et al. (2015)[31]	10 M	No	RT	23 ± 4	1	3	UF	50	180 s
					1	3	–	50	–
Kingsley et al. (2014)[17]	34: Trained (9 F, 8 M), Untrained (7 F, 10 M)	Yes	Trained: whole-body RT for 6 ± 2 years, > 3 days a week. Untrained: had not participated in RT for ≥1 year	Trained: 22 ± 1 Untrained: 22 ± 2	4	3	10	75	120 s
Okuno et al. (2014)[33]	9 M	No	RT	24 ± 2.9	1	5	8 and last set UF	80	60 s
					1	5	16 and last set UF	40	60 s
Saccomani et al. (2014)[34]	10 M	Yes	RT	24.5 ± 1.1	8	3	12	60	120 s
Tibana et al. (2013)[16]	9 F	Yes (not healthy)	Sedentary	35.0 ± 6.7	6	3	10	60	120 s
Goessler et al. (2013)[17]	10 M	No	Physically active	23 ± 2	4	3	UF	75	120 s
					4	3	UF	75	120 s
					4	3	UF	75	Self-suggested
Teixeira et al. (2011)[54]	20: 10 M, 10 F	Yes	Healthy, normotensive	26 ± 1	6	3	20	50	45 s; 90 s btwn exer

(continued on next page)

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Table 1 (Continued)

Study	Participants	Control group	Training status	Age (year)	Protocol				
					Exercises (*n*)	Sets (*n*)	Repetitions (*n*)	Intensity (% 1RM)	Repetitions between sets
Lima et al. (2011)[35]	15 M	Yes	Healthy	22.2 ± 3.2	5	3	12,9,6	50	120 s
					5	3	12,9,6	70	120 s
Kingsley et al. (2010)[40]	15 M	Yes (not healthy)	Healthy	45 ± 5	1	5	10	75	90 s
Kingsley et al. (2009)[36]	9 M	Yes (not healthy)	Healthy	48 ± 2	10	1	12	60	–
Rezk et al. (2006)[16]	17: 8 M, 9 F	Yes	Healthy	23 ± 1	6	3	20	40	45 s; 90 s btwn exer
					6	3	10	80	45 s; 90 s btwn exer
Heffernan et al. (2006)[37]	14 M	No	Moderately active	25.3 ± 2.5	8	3	10	75	90 s

Abbreviations: 1RM = 1 repetition maximum; Btwn = between; exer = exercise; F = females; M = males; Reps = repetitions; RT = resistance-trained; UF = until failure.

SMD = 1.00; 95%CI: 0.43–1.56) in the ARE group compared to the control group (Fig. 4).

3.4.4. LF/HF ratio

In the 21 effect size calculations in 19 studies (mean age = 25.4 years; 235 males, 66 females), there was an increase in LF/HF ratio ($p < 0.001$; SMD = 0.82; 95%CI: 0.64–0.99) ~30 min (8–30 min) after ARE compared to baseline. A total of 10 effect size calculations from 8 studies (mean age = 22.9 years; 93 males, 53 females) also showed an increase in LF/HF ratio ($p < 0.001$; SMD = 1.02; 95%CI: 0.62–1.43) in the ARE group compared to the control group (Fig. 5).

3.4.5. SDNN

A total of 7 studies (mean age = 22.4 years; 103 males, 33 females), with 9 effect size calculations, showed a decrease in SDNN ($p < 0.001$; SMD = −0.58; 95%CI: −0.85 to −0.30) after an ARE session compared to pre-intervention (Fig. 6). However, the main effect analysis was not conducted for the ARE group compared with the control group due to the limited number of studies (only 1 study).

3.5. Subgroup analysis

3.5.1. RMSSD

For the subject characteristics, there was no difference in effect between subgroups based on gender ($p = 0.12$), BMI ($p = 0.44$), or training status ($p = 0.48$). With respect to resistance training variables, the number of sets ($p = 0.05$) and training volume ($p = 0.01$) showed a difference in effect between subgroups. Moreover, the SMD data showed that 3 sets and higher training volume had the greatest effect on RMSSD, whereas <3 sets and lower training volume had the least effect when comparing subgroups following resistance exercises. However, no other variables (exercises ($p = 0.07$), intensity ($p = 0.41$), repetitions ($p = 0.39$), and rest ($p = 0.31$)) indicated a difference in effect between subgroups (Table 2).

3.5.2. HFnu

For the subject characteristics, there was no difference in effect between subgroups for gender ($p = 0.75$), BMI ($p = 0.74$), or training status ($p = 0.15$). Regarding resistance training variables, intensity ($p = 0.01$), rest between sets ($p = 0.05$), and training volume ($p = 0.003$) showed a difference in effect between subgroups. Furthermore, SMD data revealed that low intensity, <2 min of rest and higher training volume had the greatest effect on HFnu, whereas high intensity, 2 min of rest and lower training volume had the least effect compared to subgroups following ARE. However, there was no difference in effect between subgroups for all the other variables (repetitions ($p = 0.10$), sets ($p = 0.93$), and exercises ($p = 0.37$)) (Table 2).

3.5.3. LFnu

Regarding the subject characteristics, there was no difference in effect between subgroups for gender ($p = 0.63$), BMI ($p = 0.37$), and training status ($p = 0.45$). Except for training volume ($p = 0.02$), all the other resistance training variables

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

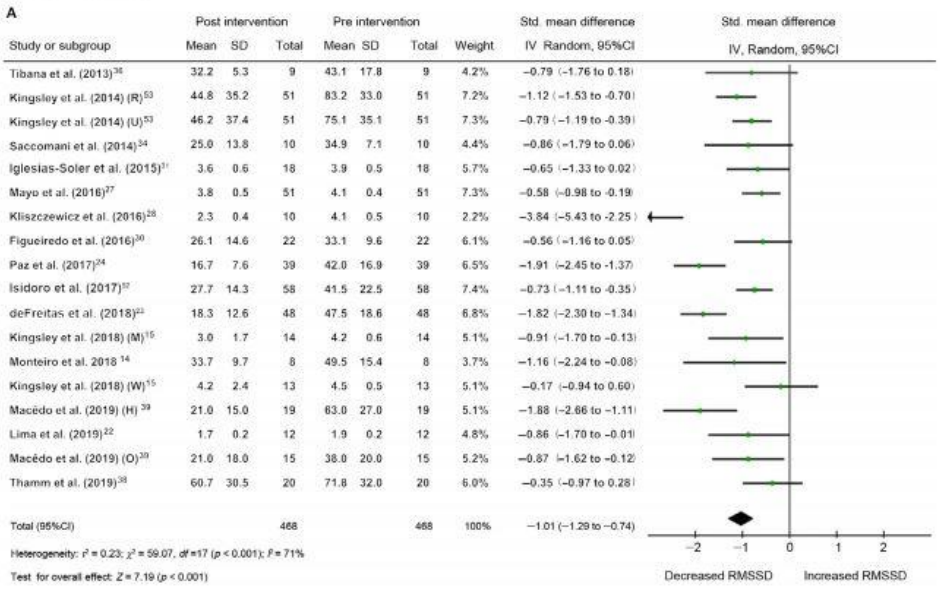


Fig. 2. Forest plots for the acute effects of RT on RMSSD. (A) Acute effects of RT sessions on RMSSD pre- *vs.* post-intervention. (B) Acute effects of RT sessions on RMSSD control group *vs.* treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; *df* = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained; RMSSD = root mean square of the successive differences; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

(intensity ($p = 0.15$), sets ($p = 0.90$), exercises ($p = 0.17$), repetitions ($p = 0.46$), and rest ($p = 0.41$)) show no difference in effect between subgroups following resistance exercises. SMD data for training volume showed that a higher training volume had a greater effect and that a lower training volume had a lesser effect on LFnu compared to other subgroups following resistance exercises (Table 2).

3.5.4. LF/HF ratio

Concerning the subject characteristics (gender ($p = 0.65$), BMI ($p = 0.77$), and training status ($p = 0.55$)) and resistance training variables (intensity ($p = 0.24$), repetitions ($p = 0.82$), sets ($p = 0.56$), exercises ($p = 0.51$), rest ($p = 0.99$), and volume ($p = 0.62$)), there was no difference in effect between subgroups (Table 2).

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

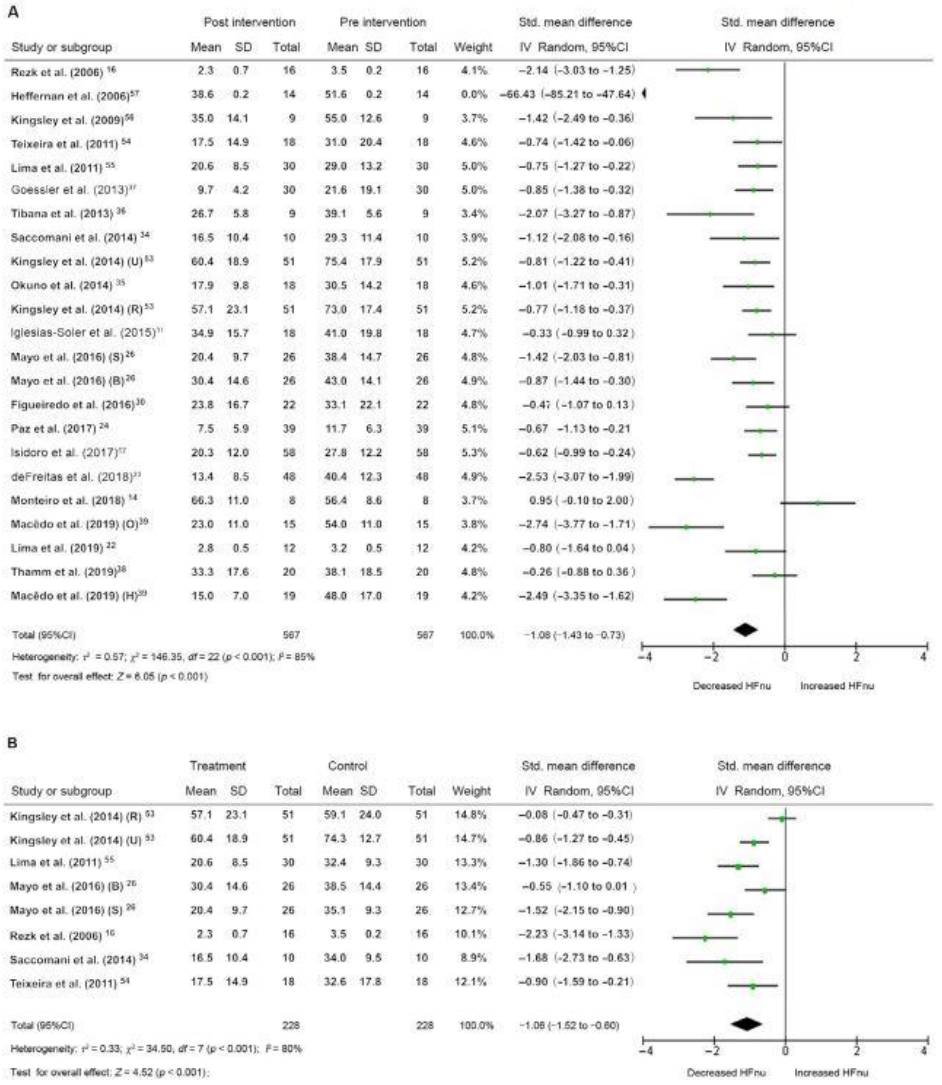
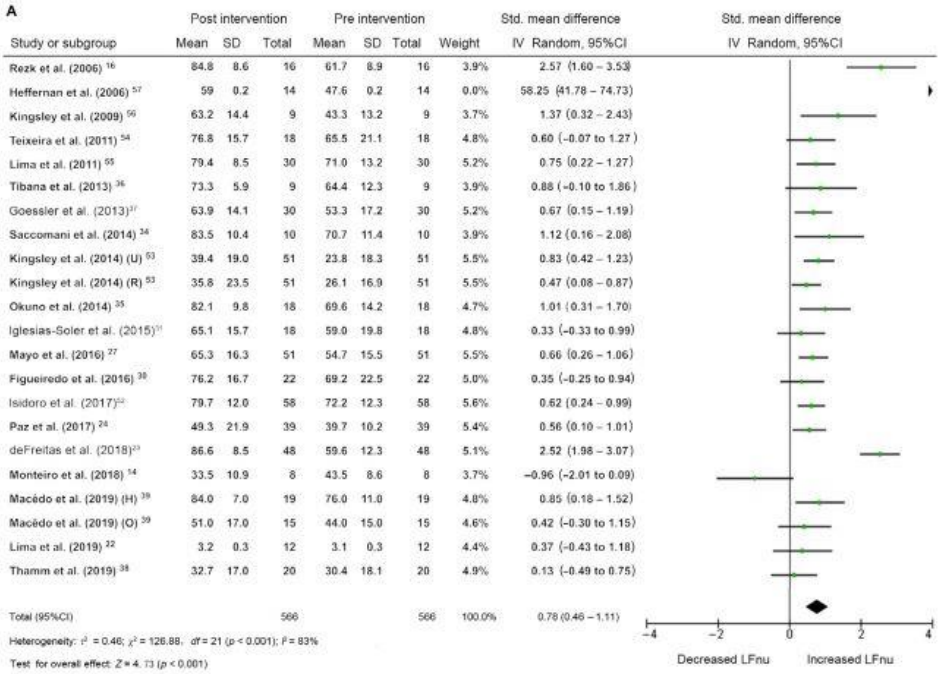


Fig. 3. Forest plots for the acute effects of RT on HFnu. (A) Acute effects of RT sessions on HFnu pre- *vs.* post-intervention. (B) Acute effects of RT sessions on HFnu control group *vs.* treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. B = bench press; CI = confidence interval; *df* = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained; S = parallel squat; HFnu = normalized units high frequency; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008



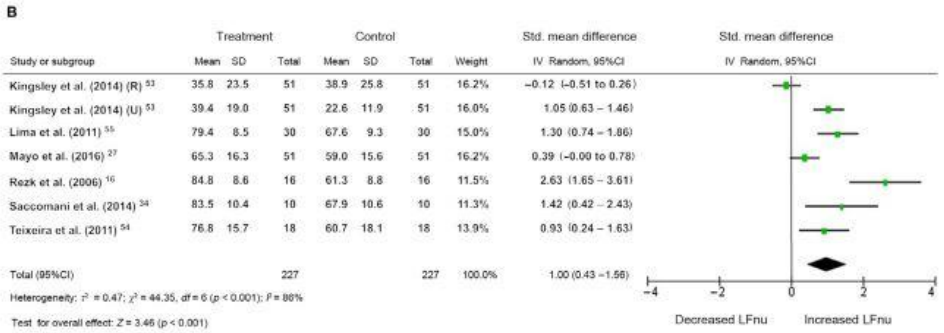


Fig. 4. Forest plots for the acute effects of RT on LFnu. (A) Acute effects of RT sessions on LFnu pre- *vs.* post-intervention. (B) Acute effects of RT sessions on LFnu control group *vs.* treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; *df* = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; O = overweight; R = resistance trained; LFnu = normalized units low frequency; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

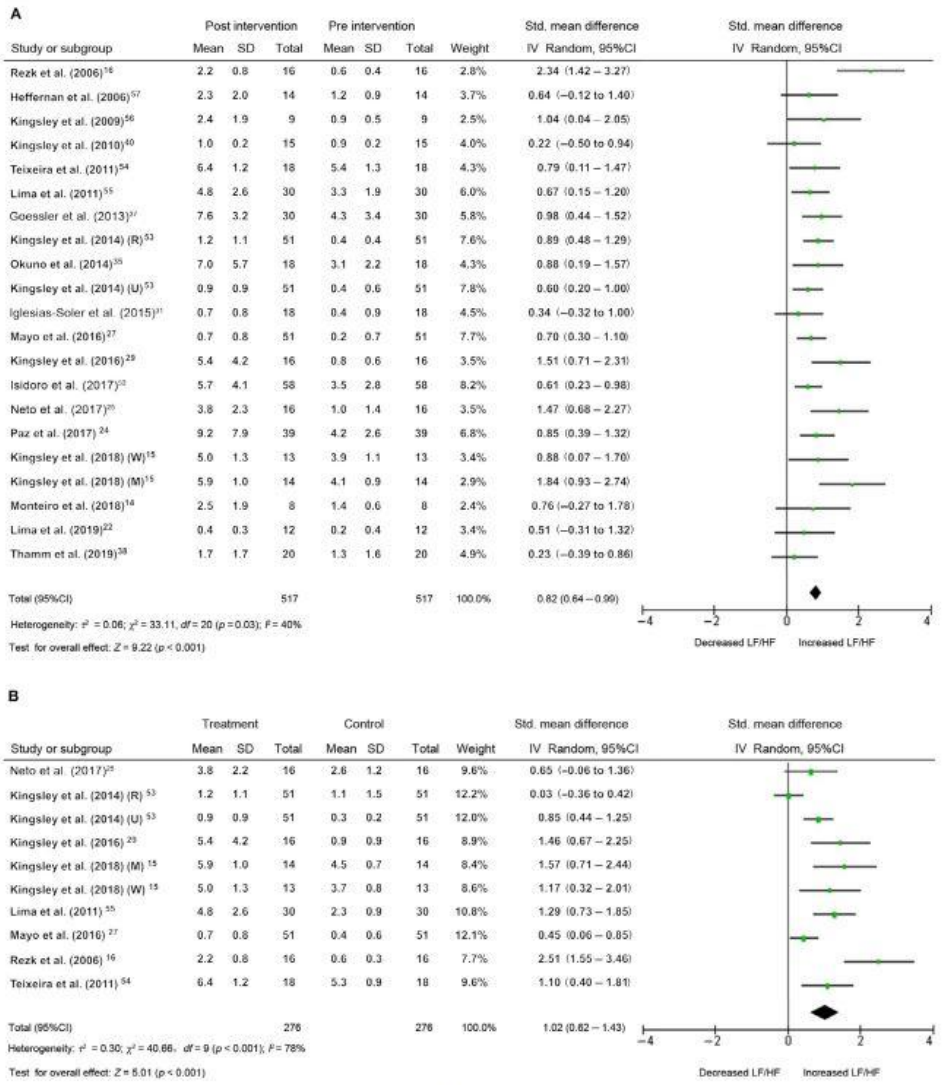


Fig. 5. Forest plots for the acute effects of RT on LF/HF ratio. (A) Acute effects of RT sessions on LF/HF ratio pre *vs.* post-intervention. (B) Acute effects of RT sessions on LF/HF ratio control group *vs.* treatment group. Squares represent the SMD for each trial. Diamonds represent the pooled across trials. CI = confidence interval; *df* = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; R = resistance trained; LF/HF = low frequency/high frequency; RT = resistance training; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

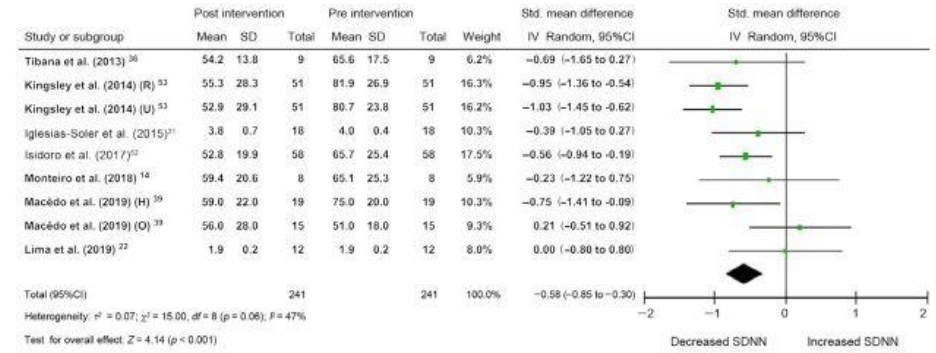


Fig. 6. Forest plots for the acute effects of RT on SDNN. Acute effects of RT sessions on SDNN pre *vs.* post-intervention. Squares represent the SMD for each trial. Diamonds represent the pooled SMD across trials. CI = confidence interval; *df* = degrees of freedom; H = healthy weight; IV = inverse variance; M = men; R = resistance trained; RT = resistance training; SDNN = standard deviation of the NN interval; SMD = standardized mean difference; Std. = standard; U = untrained; W = women.

4. Discussion

The main aim of this meta-analysis was to provide essential information regarding the recovery status of cardiac autonomic activity following an ARE session, particularly identifying the moderating factors that affect HRV parameters. The principal findings demonstrated a significant decrease in cardiac parasympathetic modulation and an increase in cardiac sympathetic modulation following an ARE session (~30 min). Moreover, overall autonomic modulation showed a significant decrease after an ARE session. The reduction of RMSSD and HFnu parameters indicates a withdrawal of cardiac parasympathetic modulation,[42,44,45] and the increase in the LFnu parameter suggests the domination of cardiac sympathetic modulation[44,45] after an ARE session. Furthermore, an increase in the LF/HF ratio suggests a shift in sympathovagal balance towards sympathetic domination,[42,44] and a reduction in the SDNN value indicates a decrease in overall autonomic modulation.[45] Along these lines, our findings are in accordance with the review article conducted by Kingsley and Figueroa,[21] which examined the ARE on HRV parameters.[21] It is important to acknowledge that the interpretation of the LF parameter and the LF/HF ratio has been controversial. Some authors consider the LF parameter to be a cardiac sympathetic modulation marker, while others believe that it reflects both sympathetic and parasympathetic modulation. With regards to the LF/HF ratio, some authors interpret this variable as a cardiac sympathetic modulation marker, while others suggest that it is a reflection of sympathovagal balance.[45] Overall, our meta-analysis suggests that the early recovery phase is still predominated by cardiac sympathetic activity.

A physiological explanation for this phenomenon during the recovery phase of resistance training may be that there is a decrease in plasma volume as a result of an acute cardiovascular imbalance.[16] This imbalance may be a result of the blood entering into the interstitial cellular space,[55] which changes the sensitivity of the arterial baroreflex in order to maintain the blood pressure changes caused by a decrease in stroke volume (a consequence of an increase in heart rate after resistance exercise).[16] This creates a greater activation of metaboreceptors and mechanoreceptors, thus providing adequate blood flow in order to meet the metabolic demands of the active muscles.[16,32,33] Also, there may be an increase in peripheral vascular resistance in arterial vessels supplying visceral organs, where redistributed blood flows to the active muscles during the recovery process.[16,32,33] Moreover, Buchheit et al.[58] have suggested that the levels of fast-twitch muscle fiber recruitment, catecholamine release and accumulation of lactate, hydrogen ions and inorganic phosphate may play a role in decreasing cardiac parasympathetic modulation, thereby increasing cardiac sympathetic modulation. Thus, evaluating HRV variables can be useful in determining cardiac autonomic stress, which may be beneficial for fitness trainers or coaches to use as a monitoring tool for measuring the effect of the training load following an ARE session on the cardiac autonomic system.

Our subgroup analyses revealed that training volume is an important moderating factor for RMSSD, LFnu, and HFnu parameters. The number of sets is a moderating factor for RMSSD parameter, while exercise intensity and rest between sets are moderating factors for HFnu parameter. These aforementioned moderating factors affect the recovery process of cardiac autonomic modulation following a resistance training session. Therefore, fitness trainers and coaches could monitor and adjust the training load by measuring the changes in cardiac autonomic modulation using HRV variables such as RMSSD (training volume, number of sets per exercise) and HFnu (number of exercises, reset between sets). The following provides a more detailed discussion of each of the subgroup analyses.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Table 2
Subgroup analyses assessing potential moderating factors for heart rate variability parameters in studies included in the meta-analysis.

	Studies			ARE			
Methodological factors	n^a	References	SMD (95%CI)	I^2	I_p	p	p_{diff}
RMSSD							
Gender							
Male	12	15, 22–24, 28, 30, 31, 34, 38, 39, 52	−1.16 (−1.56 to −0.76)	77	<0.001	<0.001	0.12
Female	3	14, 15, 36	−0.61 (−1.19 to −0.03)	16	0.30	0.04	
BMI (kg/m²)							
≤24.9	8	15, 22, 24, 27, 34, 36, 39, 52	−0.98 (−1.41 to −0.54)	73	<0.001	<0.001	0.44
>24.9	3	15, 30, 39	−0.74 (−1.14 to −0.34)	0	0.72	<0.001	
Training status							
Resistance trained	11	14, 15, 23, 24, 27, 30, 31, 34, 38, 53	−0.94 (−1.30 to −0.57)	74	<0.001	<0.001	0.48
Not trained	7	22, 28, 36, 39, 52, 53	−1.15 (−1.62 to −0.67)	70	0.002	<0.001	
Exercise intensity (%RM)							
High (>85)	2	31, 38	−0.52 (−1.06 to 0.01)	0	0.53	0.06	0.41
Moderate (>65–85)	9	15, 24, 30, 38, 52, 53	−0.89 (−1.20 to −0.58)	63	0.006	<0.001	
Low (≤65)	7	23, 27, 34, 36, 39	−1.01 (−1.56 to −0.46)	75	<0.001	<0.001	
Number of repetitions							
<6	2	27, 38	−0.49 (−1.03 to −0.06)	0	0.59	0.08	0.39
6–10	8	15, 23, 27, 36, 38, 53	−0.94 (−1.31 to −0.58)	64	0.008	<0.001	
>10	5	27, 34, 39, 52	−0.86 (−1.37 to −0.35)	66	0.02	0.001	
Number of sets							
<3	1	27	−0.10 (−0.78 to −0.57)	—	—	0.76	0.05
3	13	15, 23, 24, 30, 31, 34, 36, 39, 52, 53	−1.02 (−1.31 to −0.73)	65	<0.001	<0.001	
>3	5	23, 27, 38	−0.99 (−1.50 to −0.49)	58	0.05	<0.001	
Number of exercises							
<6	14	14, 15, 22, 23, 27, 31, 38, 39, 52, 53	−0.89 (−1.11 to −0.67)	44	0.04	<0.001	0.07
6	3	23, 24, 36	−1.69 (−2.42 to −0.96)	61	0.08	<0.001	
>6	2	30, 34	−0.65 (−1.15 to −0.14)	0	0.59	0.01	
Rest between sets (min)							
<2	9	23, 24, 27, 30, 36, 39	−1.16 (−1.63 to −0.70)	70	<0.001	<0.001	0.31
2	7	15, 30, 34, 38, 53	−0.77 (−1.02 to −0.51)	13	0.33	<0.001	
>2	4	24, 27, 31, 38	−1.01 (−1.68 to −0.33)	58	0.07	0.003	
Training volume							
Low (<108)	6	15, 27, 38, 53	−0.63 (−0.85 to −0.41)	0	0.44	<0.001	0.01
Moderate (108–<180)	4	24, 52, 53	−1.29 (−1.88 to −0.70)	76	0.006	<0.001	
High (≥180)	5	23, 34, 36, 39	−1.32 (−1.83 to −0.81)	56	0.06	<0.001	
HFnu							
Gender							
Male	16	22–24, 26, 30, 31, 34, 35, 37–39, 52, 55, 57	−1.14 (−1.59 to −0.68)	88	<0.001	<0.001	0.75
Female	3	14, 36, 56	−0.84 (−2.65 to 0.98)	88	<0.001	0.37	
BMI (kg/m²)							
≤24.9	12	16, 22, 24, 26, 34, 36, 39, 52, 54, 55, 57	−1.25 (−1.78 to −0.71)	86	<0.001	<0.001	0.74
>24.9	3	30, 39, 56	−1.50 (−2.87 to −0.13)	86	<0.001	0.03	
Training status							
Resistance trained	10	14, 23, 24, 26, 31, 34, 35, 38, 53	−0.85 (−1.33 to −0.36)	84	<0.001	<0.001	0.15
Not trained	12	16, 22, 36, 37, 39, 52, 53, 54, 55, 56, 57	−1.40 (−1.97 to −0.83)	87	<0.001	<0.001	
Exercise intensity (%RM)							
High (>85)	2	31, 38	−0.34 (−0.87 to 0.19)	0	0.97	0.20	0.01
Moderate (>65–85)	13	16, 24, 26, 30, 35, 37, 38, 52, 53, 55, 57	−0.93 (−1.32 to −0.53)	81	<0.001	<0.001	
Low (≤65)	10	16, 23, 34–36, 39, 54–56	−1.58 (−2.19 to −0.96)	80	<0.001	<0.001	
Number of repetitions							
<6	1	38	−0.36 (−1.24 to −0.53)	—	—	0.43	0.10
6–10	7	16, 23, 36, 38, 53, 57	−1.58 (−2.64 to −0.53)	93	<0.001	0.003	
>10	9	16, 26, 34, 39, 52, 54, 56	−1.39 (−1.87 to −0.91)	74	<0.001	<0.001	
Number of sets							
<3	1	56	−1.42 (−2.49 to −0.36)	—	—	0.009	0.93
3	16	16, 23, 24, 30, 31, 34, 36, 37, 39, 52–55, 57	−1.21 (−1.63 to −0.79)	85	<0.001	<0.001	
>3	5	23, 26, 35, 38	−1.22 (−1.94 to −0.49)	85	<0.001	0.001	
Number of exercises							
<6	15	14, 22, 23, 26, 31, 35, 37–39, 52, 53, 55	−1.00 (−1.35 to −0.65)	81	<0.001	<0.001	0.37
6	5	16, 23, 24, 36, 54	−1.51 (−2.26 to −0.75)	79	<0.001	<0.001	
>6	4	30, 34, 56, 57	−2.04 (−4.48 to 0.39)	94	<0.001	0.10	

(continued on next page)

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Table 2 (Continued)

Methodological factors	Studies			ARE			
	n^a	References	SMD (95%CI)	I^2	I_p	p	p_{diff}
Rest between sets (min)							
<2	11	16, 23, 24, 30, 35–37, 39, 54, 57	−1.72 (−2.49 to −0.95)	89	<0.001	<0.001	0.05
2	7	30, 34, 37, 38, 53, 55	−0.72 (−0.94 to −0.51)	0	0.57	<0.001	
>2	5	24, 26, 31, 38	−0.85 (−1.23 to −0.47)	25	0.25	<0.001	
Training volume							
Low (<108)	4	37, 38, 53	−0.56 (−0.82 to −0.29)	0	0.52	<0.001	0.003
Moderate (108–<180)	8	24, 26, 52, 53, 55, 56	−1.02 (−1.33 to −0.70)	55	0.03	<0.001	
High (≥180)	8	16, 23, 34, 36, 39, 54, 57	−2.17 (−3.22 to −1.12)	90	<0.001	<0.001	
LFnu							
Gender							
Male	14	22–24, 26, 30, 31, 34, 35, 37–39, 52, 55, 57	0.79 (0.33–1.26)	87	<0.001	<0.001	0.63
Female	3	14, 36, 56	0.43 (−0.93 to 1.80)	81	0.005	0.53	
BMI (kg/m^2)							
≤24.9	18	16, 22, 24, 27, 34, 36, 39, 52, 54, 55, 57	0.91 (0.41–1.41)	84	<0.001	<0.001	0.37
>24.9	4	30, 39, 56	0.58 (0.06–1.11)	31	0.24	0.03	
Training status							
Resistance trained	10	14, 23, 24, 27, 30, 31, 34, 35, 38, 53	0.65 (0.18–1.13)	86	<0.001	0.007	0.45
Not trained	12	16, 22, 36, 37, 39, 52, 53, 54, 55, 56, 57	0.91 (0.44–1.38)	83	<0.001	<0.001	
Exercise intensity (%RM)							
High (>85)	2	31, 38	0.32 (−0.21 to 0.85)	0	0.94	0.24	0.15
Moderate (>65–85)	12	16, 24, 27, 30, 35, 37, 38, 52, 53, 55, 57	0.81 (0.38–1.24)	82	<0.001	<0.001	
Low (≤65)	11	16, 23, 27, 34–36, 39, 54–56	1.02 (0.53–1.52)	78	<0.001	<0.001	
Number of repetitions							
<6	2	27, 38	0.59 (0.04–1.14)	0	0.40	0.04	0.46
6–10	8	16, 23, 27, 36, 38, 53, 57	1.27 (0.35–2.19)	93	<0.001	0.007	
>10	8	16, 27, 34, 39, 52, 54, 56	0.77 (0.45–1.08)	34	0.16	<0.001	
Number of sets							
<3	2	27, 56	0.78 (−0.21 to 1.76)	61	0.11	0.12	0.90
3	16	16, 23, 24, 30, 31, 34, 36, 37, 39, 52–55, 57	0.86 (0.50–1.22)	81	<0.001	<0.001	
>3	5	23, 27, 35, 38	1.05 (0.24–1.86)	86	<0.001	0.01	
Number of exercises							
<6	14	14, 22, 23, 27, 31, 35, 37–39, 52, 53, 55	0.66 (0.38–0.95)	73	<0.001	<0.001	0.17
6	5	16, 23, 24, 36, 54	1.33 (0.52–2.15)	83	<0.001	0.001	
>6	4	30, 34, 56, 57	2.11 (−0.32 to 4.54)	94	<0.001	0.09	
Rest between sets (min)							
<2	12	16, 23, 24, 27, 30, 35–37, 39, 54, 57	1.08 (0.43–1.74)	89	<0.001	0.001	0.41
2	7	30, 34, 37, 38, 53, 55	0.62 (0.39–0.85)	6	0.38	<0.001	
>2	4	24, 27, 31, 38	0.63 (0.22–1.04)	0	0.85	0.002	
Training volume							
Low (<108)	5	27, 37, 38, 53	0.46 (0.25–0.68)	0	0.41	<0.001	0.02
Moderate (108–<180)	6	24, 52, 53, 55, 56	0.97 (0.57–1.37)	62	0.02	<0.001	
High (≥180)	8	16, 23, 34, 36, 39, 54, 57	1.51 (0.45–2.56)	92	<0.001	0.005	
Low frequency/high frequency ratio							
Gender							
Male	11	15, 22, 24, 25, 31, 35, 37, 38, 52, 55, 57	0.77 (0.54 – 0.99)	33	0.13	<0.001	0.65
Female	4	14, 15, 40, 56	0.65 (0.22 – 1.08)	0	0.51	0.003	
BMI (kg/m^2)							
≤24.9	10	15, 16, 22, 24, 25, 27, 52, 54, 55, 57	0.85 (0.60 – 1.10)	43	0.07	<0.001	0.77
>24.9	3	15, 40, 56	1.00 (0.02 – 1.98)	74	0.02	0.05	
Training status							
Resistance trained	11	14, 15, 24, 25, 27, 29, 31, 35, 38, 53	0.87 (0.63–1.12)	38	0.09	<0.001	0.55
Not trained	10	16, 22, 37, 40, 52, 53, 54, 55, 56, 57	0.76 (0.51–1.02)	44	0.07	<0.001	
Exercise intensity (%RM)							
High (>85)	2	31, 38	0.42 (−0.11 to 0.95)	0	0.70	0.12	0.24
Moderate (>65–85)	16	15, 16, 24, 25, 27, 29, 35, 37, 38, 40, 52, 53, 55, 57	0.89 (0.69–1.09)	37	0.07	<0.001	
Low (≤65)	6	16, 27, 35, 54–56	0.73 (0.35–1.11)	30	0.21	<0.001	
Number of repetitions							
<6	2	27, 38	0.71 (0.16–1.27)	0	0.67	0.01	0.82
6–10	11	15, 16, 25, 27, 29, 40, 53, 57	0.89 (0.59–1.19)	48	0.04	<0.001	
>10	5	16, 27, 52, 54, 56	0.78 (0.39–1.17)	35	0.19	<0.001	

(continued on next page)

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Table 2 (Continued)

Methodological factors	Studies		SMD (95%CI)	ARE			
	n^a	References		I^2	I_p	p	p_{diff}
Number of sets							
<3	2	27, 56	0.62 (0.04–1.19)	3	0.31	0.03	0.56
3	13	15, 16, 24, 29, 31, 37, 52-55, 57	0.90 (0.65–1.15)	53	0.01	<0.001	
>3	5	25, 27, 35, 38, 40	0.71 (0.30–1.12)	50	0.09	<0.001	
Number of exercises							
<6	16	14, 15, 22, 25, 27, 29, 31, 35, 37, 38, 40, 52, 53, 55	0.76 (0.58–0.93)	30	0.12	<0.001	0.51
6	3	16, 24, 54	1.25 (0.44–2.06)	77	0.01	0.003	
>6	2	56, 57	0.79 (0.18–1.40)	0	0.53	0.01	
Rest between sets (min)							
<2	8	16, 24, 27, 35, 37, 40, 54, 57	0.87 (0.52–1.23)	51	0.04	<0.001	0.99
2	8	15, 29, 37, 38, 53, 55	0.91 (0.60–1.22)	45	0.08	<0.001	
>2	5	24, 25, 27, 31, 38	0.90 (0.54–1.27)	0	0.60	<0.001	
Training volume							
Low (<108)	10	15, 25, 27, 29, 37, 38, 40, 53	0.79 (0.51–1.07)	49	0.04	<0.001	0.62
Moderate (108–<180)	6	24, 52, 53, 55, 56	0.93 (0.61–1.26)	44	0.11	<0.001	
High (≥180)	3	16, 54, 57	1.22 (0.25–2.18)	78	0.01	0.01	

Note: I^2 = heterogeneity; I_p = p values for heterogeneity; n^a = number of acute resistance exercise-trained groups within the selected studies; p = test for overall effect; p_{diff} = test for subgroup differences.
Abbreviations: ARE = acute resistance exercise; BMI = body mass index; HFnu = normalized units high frequency; MD = mean difference; %RM = Percentage of 1 repetition maximum; RT = resistance training; SMD = standardized mean difference.

4.1. Subjects characteristics

Subgroup analyses did not show a significant difference between males and females (gender) for RMSSD, HFnu, LFnu, and LF/HF ratio parameters. These findings are in line with the findings of Kingsley et al.,[15] who concluded that changes in HRV parameters in response to an ARE were not influenced by gender differences. The BMI subgroup analyses also demonstrated no significant effect on RMSSD, HFnu, LFnu, and LF/HF ratio parameters. Similarly, Macêdo et al.[39] reported that changes in HRV parameters in response to ARE were not affected by body weight. However, Beske et al.[59] reported that lower cardiovagal baroreflex gain was marginally related to higher body fat percentage. When cardiovagal baroreflex sensitivity is lower, there is a weaker response to the changes in systolic blood pressure, and this does not effectively change the heart rate.[60] Therefore, higher body fat mass may have a minimal effect on cardiac sympathetic modulation and thus may only trigger a minimal change in heart rate. Likewise, the analyses of the training status subgroup demonstrated no significant effect on RMSSD, HFnu, LFnu, and LF/HF ratio variables. These findings are again in line with the findings of Kingsley et al.,[53] who concluded that changes in HRV parameters in response to the ARE were not influenced by training status. Thus, our study showed that gender, BMI, and training status do not play a role in cardiac autonomic modulation changes following an ARE sessions. Therefore, trainers and coaches may not need to specialize a resistance training session based on an individual's gender, BMI level or training status. However, we believe that further investigations on the relationship between BMI and HRV parameters related to an ARE session are needed.

4.2. Training characteristics

4.2.1. Number of repetitions, sets, and exercises per workout

There were no significant differences among the number of repetitions subgroups for RMSSD, LFnu, HFnu, and LF/HF ratio parameters. Interestingly, a significant difference was demonstrated between subgroups for the RMSSD parameter and the number of sets and number of exercises, but this significant difference was not demonstrated for the LFnu and HFnu parameters. Additionally, SMD results showed that the RMSSD parameter was affected greatly by an ARE session that included exactly 3 sets per exercise but was not affected greatly when there were <3 sets per exercise. Our findings agree with the findings of Figueiredo et al.,[33] who reported a reduced cardiac sympathetic modulation response with a lower number of sets of resistance training compared to a higher number sets. Therefore, performing ≥3 sets per exercise generates a higher sympathetic stress and may delay the recovery process compared to performing <3 sets per exercise.

SMD data also demonstrated that the RMSSD parameter may be affected by the number of exercises, with a higher effect shown for exactly 6 exercises, although this did not reach statistical significance ($p_{diff} = 0.07$). Thus, performing 3 sets per exercise, and possibly 6 exercises per session, generates a greater withdrawal of cardiac parasympathetic modulation after an ARE session. It remains to be determined whether the number of exercises truly has an effect on RMSSD.

4.2.2. Rest between sets

The rest period only had an effect on the HFnu parameter. SMD data showed that HFnu was greatly affected by an ARE session that included <2 min of rest between sets but was less affected when there was exactly 2 min or >2 min of rest between sets. In line with our findings, Goessler et al.[37] suggested that at least 2 min of rest between sets reduces the post-exercise cardiac sympathetic modulation following ARE. Therefore, having <2 min of resting time between sets generates greater withdrawal of cardiac parasympathetic modulation, and 2 min or more minutes of rest between sets creates lesser withdrawal of cardiac parasympathetic modulation,

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

independent of the other variables of resistance training. These results indicate that having <2 min of rest between sets delays the recovery process following an ARE session compared to ≥2 min of rest between sets.

4.2.3. Exercise intensity

Based on our subgroup analysis, the exercise intensity (low, moderate, or high) of an ARE session is not a moderating factor for RMSSD, LFnu, or LF/HF ratio. Figueiredo et al.[32] showed no differences between the level of intensity of a training session (60% 1RM, 70% 1RM, and 80% of 1RM) and RMSSD. Additionally, Rezk et al.[16] demonstrated no difference in LFnu or HFnu when comparing 40% 1RM and 80% 1RM training sessions. This is an interesting finding given that, although ARE has an effect on cardiac sympathetic and cardiac parasympathetic modulation, the different intensity levels did not make a significant contribution to cardiac autonomic modulation; thus, they worked independently of other covariables related to resistance training. However, in our study, only the HFnu parameter showed a significant difference between exercise intensity subgroups. Surprisingly, our SMD results showed that low exercise intensity had the greatest effect and high exercise intensity had the least effect on the HFnu parameter. One possible explanation for the difference shown between the HFnu and RMSSD parameters (both of which represent cardiac parasympathetic modulation) is that the included studies in each subgroup was different but the tendency of the findings was the same: a lower intensity had a higher effect. This may be a consequence of having a longer training duration of lower intensity. Another explanation may be that respiration control influences HFnu during HRV measurements.[21,42] Also, in our review, included studies in which lower exercise intensities were performed used a higher training volume and included studies in which higher exercise intensities were performed used a lower training volume. All these factors should be taken into consideration when interpreting the outcomes of our meta-analysis. Furthermore, our results indicated that there is a direct relationship between higher training volume and greater cardiac sympathetic activation and withdrawal of cardiac parasympathetic modulation.

4.2.4. Training volume

There was a significant difference between subgroups based on training volumes in the RMSSD, LFnu, and HFnu parameters. Our finding is consistent with the findings of Figueiredo et al.,[33] who suggested that higher training volume increases the recruitment of additional motor units, thus minimizing the likelihood of muscular failure during the concentric phase of lifting (concentric failure) and triggering a progressive activation of the cardiac sympathetic modulation.[61] Moreover, our SMD results revealed that higher training volume had a greater effect and lower training volume had the lesser effect on RMSSD, HFnu, and LFnu parameters.

Our results indicated that higher training volume produces a greater activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation. In other words, when the human body experiences a higher level of resistance training volume, the magnitude of activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation is higher than it is with lower training volume. On the other hand, previous studies have reported that a low volume of high-intensity resistance training greatly improves strength, muscle size,[62,63] force production and rate of force development[64] compared to a high volume of moderate- or low-intensity resistance training. Thus, our meta-analysis suggests that a low training volume of high-intensity ARE would enable athletes to have the optimal training load or stimulus without creating a large change in cardiac autonomic modulation, thus allowing for an early recovery without ultimately sacrificing training adaptation and performance.

We acknowledge that there are a few limitations in our meta-analysis. First, only a limited number of studies were included due to the lack of research on ARE interventions that had HRV parameters as an outcome variable. In fact, only 26 studies (with a total of 412 participants) met the inclusion criteria for our systematic review and meta-analysis, and of these studies, only 21 reported on the LFnu and HFnu parameters, only 19 included results for the LF/HF ratio parameter, only 15 reported on the RMSSD parameter and only 7 reported SDNN parameter results. Second, some of the included studies had a small sample size (range: 8–34), which may have negatively impacted our findings. Third, there was a presence of heterogeneity in several moderating factors, and this should be taken into consideration when interpreting the outcomes of our meta-analysis. Fourth, different methodological procedures, equipment and software were used to measure HRV parameters in the different studies.

5. Conclusion

This systematic review and meta-analysis demonstrated that there is a decrease of overall autonomic modulation, withdrawal of cardiac parasympathetic modulation and activation of cardiac sympathetic modulation following an ARE session (after ~30 min) in healthy individuals. Importantly, there is a greater effect of training volume on the activation of cardiac sympathetic modulation and withdrawal of cardiac parasympathetic modulation ~30 min after resistance exercises in healthy individuals. Furthermore, the number of sets, the intensity of exercise, and amount of rest between sets play an important role in the ARE on HRV parameters. However, gender, BMI and training status do not significantly influence the changes in HRV parameters as a response to ARE. With regards to the practical application of our findings, we would recommend that fitness trainers and coaches consider HRV evaluations as aids to programming their athletes' training sessions, depending on the stress goals they want to apply. Based on our meta-analysis, the variables related to resistance training modify the stress applied to the cardiac autonomic system. Thus, ARE sessions that comprise ≥3 sets, <2 min of rest between sets, and 6 exercises at low intensity would lead to greater stress on the cardiac sympathetic system. This information can help coaches and trainers optimize training sessions and improve the recovery process.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

Acknowledgment

We would like to thank all authors of included studies who responded to our requests for additional information for our meta-analysis.

Authors' contributions

All authors participated in the study design and provided critical input regarding the manuscript draft; SUMA performed the methodology, analyzed and interpreted the findings and drafted the manuscript; JARA contributed to article selection, helped with the risk-of-bias assessment, confirmed the extracted data, and helped with the interpretation of the findings. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Competing interests

The authors declare that they have no competing interests.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jshs.2020.11.008.

References

1. Haff GG, Triplett NT. *Essentials of strength training and conditioning*. 4th ed. Champaign, IL: Human Kinetics; 2015.
2. Brown LE. *Strength training*. Champaign, IL: Human Kinetics; 2007.
3. Faigenbaum AD, Kraemer WJ, Blimkie CJ, et al. Youth resistance training: updated position statement paper from the National Strength and Conditioning Association. *J Strength Cond Res* 2009;**23**(Suppl. 5):S60–79.
4. Bellenger CR, Fuller JT, Thomson RL, Davison K, Robertson EY, Buckley JD. Monitoring athletic training status through autonomic heart rate regulation: a systematic review and meta-analysis. *Sports Med* 2016;**46**:1461–86.
5. Makivić B, Djordjević MN, Willis MS. Heart Rate Variability (HRV) as a tool for diagnostic and monitoring performance in sport and physical activities. *J Exerc Physiol Online* 2013;**16**:103–31.
6. Dong JG. The role of heart rate variability in sports physiology. *Exp Ther Med* 2016;**11**:1531–6.
7. Ferreira LL, Vanderlei LC, Guida HL, et al. Response of cardiac autonomic modulation after a single exposure to musical auditory stimulation. *Noise Health* 2015;**75**:108–15.
8. Plews DJ, Laursen PB, Stanley J, Kilding AE, Buchheit M. Training adaptation and heart rate variability in elite endurance athletes: opening the door to effective monitoring. *Sports Med* 2013;**43**:773–81.
9. ChuDuc H, NguyenPhan K, NguyenViet D. A review of heart rate variability and its applications. *Int J Sci Res* 2013;**7**:80–5.
10. Achten J, Jeukendrup AE. Heart rate monitoring: applications and limitations. *Sports Med* 2003;**33**:517–38.
11. Martinez CAG, Quintana AO, Vila XA, et al. *Heart rate variability analysis with the R package RHRV*. New York, NY: Springer; 2017.
12. Gaskell E, Rostron C. *Therapeutics and human physiology: how drugs work*. Oxford: Oxford University Press; 2013.
13. Balestra C, Kot J, Efrati S, Guerrero F, Blatteau JE, Besnard S. *Coping with extreme environments: a physiological/psychological approach*. Lausanne: Frontiers Media SA; 2019.
14. Monteiro ER, Novaes JS, Fiuza AG, et al. Behavior of heart rate variability after 10 repetitions maximum load test for lower limbs. *Int J Exerc Sci* 2018;**11**:834–43.
15. Kingsley JD, Tai YL, Marshall EM, et al. Autonomic modulation and baroreflex sensitivity after acute resistance exercise: responses between sexes. *J Sports Med Phys Fitness* 2019;**59**:1036–44.
16. Rezk CC, Marrache RC, Tinucci T, Mion Jr D, Forjaz CL. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. *Eur J Appl Physiol* 2006;**98**:105–12.
17. Chen JL, Yeh DP, Lee JP, et al. Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. *J Strength Cond Res* 2011;**25**:1546–52.
18. Risk M, Bril V, Broadbridge C, Cohen A. Heart rate variability measurement in diabetic neuropathy: review of methods. *Diabetes Technol Ther* 2001;**3**:63–76.
19. Boullosa DA, Tuimil JL, Leicht AS, Crespo-Salgado JJ. Parasympathetic modulation and running performance in distance runners. *J Strength Cond Res* 2009;**23**:626–31.
20. Kryzhanovsky B, Dunin-Barkowski W, Redko V. Advances in neural computation, machine learning, and cognitive research. *Neuroinformatics* 2017;**736**:173–9.
21. Kingsley JD, Figueroa A. Acute and training effects of resistance exercise on heart rate variability. *Clin Physiol Funct Imaging* 2016;**36**:179–87.
22. Lima CD, Martinez PF, Morais CS, Barbosa FSS, Ota GE, Junior SAD. Cardiovascular effects of a strength test (1RM) in prehypertensive subjects. *Rev Bras Med Esporte* 2019;**25**:9–13.
23. deFreitas MC, Ricci-Vitor AL, Quizzini GH, et al. Postexercise hypotension and autonomic modulation response after full versus split body resistance exercise in trained men. *J Exerc Rehabil* 2018;**14**:399–406.
24. Paz GA, Iglesias-Soler E, Willardson JM, Maia MF, Miranda H. Postexercise hypotension and heart rate variability responses subsequent to traditional, paired set, and superset training methods. *J Strength Cond Res* 2019;**33**:2433–42.
25. Neto VGC, Benteso CM, Neto GDAM, Miranda H. Hypotension and heart rate variability after resistance exercise performed maximal and submaximal order. *Motri* 2017;**13**:19–29.
26. Mayo X, Iglesias-Soler E, Fariñas-Rodríguez J, Fernández-Del-Olmo M, Kingsley JD. Exercise type affects cardiac vagal autonomic recovery after a resistance training session. *J Strength Cond Res* 2016;**30**:2565–73.
27. Mayo X, Iglesias-Soler E, Carballeira-Fernández E, Fernández-Del-Olmo M. A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise. *Eur J Sport Sci* 2016;**16**:996–1004.
28. Kliszczewicz BM, Esco MR, Quindry JC, et al. Autonomic responses to an acute bout of high-intensity body weight resistance exercise *vs.* treadmill running. *J Strength Cond Res* 2016;**30**:1050–8.
29. Kingsley JD, Mayo X, Tai YL, Fennell C. Arterial stiffness and autonomic modulation after free-weight resistance exercises in resistance trained individuals. *J Strength Cond Res* 2016;**30**:3373–80.
30. Figueiredo T, Willardson JM, Miranda H, et al. Influence of rest interval length between sets on blood pressure and heart rate variability after a strength training session performed by prehypertensive men. *J Strength Cond Res* 2016;**30**:1813–24.
31. Iglesias-Soler E, Boullosa DA, Carballeira E, et al. Effect of set configuration on hemodynamics and cardiac autonomic modulation after high-intensity squat exercise. *Clin Physiol Funct Imaging* 2015;**35**:250–7.
32. Figueiredo T, Willardson JM, Miranda H, Bentes CM, Reis VM, Simão R. Influence of load intensity on postexercise hypotension and heart rate variability after a strength training session. *J Strength Cond Res* 2015;**29**:2941–8.
33. Figueiredo T, Rhea MR, Peterson M, et al. Influence of number of sets on blood pressure and heart rate variability after a strength training session. *J Strength Cond Res* 2015;**29**:1556–63.
34. Saccomani MG, Figueiredo T, Kattenbraker MS, Simao R, Forjaz CLDM, Polito MD. Blood pressure and heart rate variability responses after isolated and combined sessions of aerobic and strength exercises. *Int Sportmed J* 2014;**15**:178–87.
35. Okuno NM, Pedro RE, Leicht AS, de Paula Ramos S, Nakamura FY. Cardiac autonomic recovery after a single session of resistance exercise with and without vascular occlusion. *J Strength Cond Res* 2014;**28**:1143–50.
36. Tibana RA, Boullosa DA, Leicht AS, Prestes J. Women with metabolic syndrome present different autonomic modulation and blood pressure

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

response to an acute resistance exercise session compared with women without metabolic syndrome. *Clin Physiol Funct Imaging* 2013;**33**:364–72.
37. Goessler KF, Polito MD. Effect of fixed and self-suggested rest intervals between sets of resistance exercise on post-exercise cardiovascular behavior. *Rev Bras Cineantropom Desempenho Hum* 2013;**15**:467–75.
38. Thamm A, Freitag N, Figueiredo P, et al. Can heart rate variability determine recovery following distinct strength loadings? A randomized crossover trial. *Int J Environ Res Public Health* 2019;**16**:4353. doi:10.3390/ijerph16224353.
39. Macêdo RF, Miranda JM, Maçal AC, Queiroz JCC, Bastos AA, Wichi RB. Overweight not influence the cardiac autonomic and cardiovascular response after resistance exercise in sedentary adolescents. *Motri* 2019;**15**:3–15.
40. Kingsley JD, McMillan V, Figueroa A. The effects of 12 weeks of resistance exercise training on disease severity and autonomic modulation at rest and after acute leg resistance exercise in women with fibromyalgia. *Arch Phys Med Rehabil* 2010;**91**:1551–7.
41. Moher D, Liberati A, Tetzlaff J, Altman DG, PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med* 2009;**6**: e1000097. doi:10.1371/journal.pmed.1000097.
42. Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. *Front Public Health* 2017;**5**:258. doi:10.3389/fpubh.2017.00258.
43. Ernst G. Hidden signals—the history and methods of heart rate variability. *Front Public Health* 2017;**5**:265. doi:10.3389/fpubh.2017.00265.
44. Ernst G. Heart-rate variability—more than heart beats? *Front Public Health* 2017;**5**:240. doi:10.3389/fpubh.2017.00240.
45. Task-Force. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation* 1996;**93**:1043–65.
46. Higgins JP. *Cochrane handbook for systematic reviews of interventions version 5.0. 1. The Cochrane Collaboration*. Available at: http://www.cochrane-handbook.org. [accessed 25.01.2019].
47. Higgins JP, Altman DG, Gøtzsche PC, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ* 2011;**343**:d5928. doi:10.1136/bmj.d5928.
48. National Heart, Lung, and Blood Institute. *Study Quality Assessment Tools*. Available at: https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools. [accessed 10.01.2020].
49. Schoenfeld BJ. The mechanisms of muscle hypertrophy and their application to resistance training. *J Strength Cond Res* 2010;**24**:2857–72.
50. Browning KN, Travagli RA. Functional organization of presynaptic metabotropic glutamate receptors in vagal Brainstem circuits. *J Neurosci* 2007;**27**:8979–88.
51. Centers for Disease Control and Prevention, USA. *Healthy weight - About adult BMI*. 2017. Available at: https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html. [accessed 22.09.2019].
52. Isidoro NJ, Santana MDR, Valenti VE, Garner DM, deAbreu LC. Cardiac autonomic recovery after strength exercise in lower and upper limbs. *Acta Cardiol* 2017;**72**:467–73.
53. Kingsley JD, Hochgesang S, Brewer A, Buxton E, Martinson M, Heidner G. Autonomic modulation in resistance-trained individuals after acute resistance exercise. *Int J Sports Med* 2014;**35**:851–6.
54. Teixeira L, Ritti-Dias RM, Tinucci T, Mion Júnior D, Forjaz CL. Post-concurrent exercise hemodynamics and cardiac autonomic modulation. *Eur J Appl Physiol* 2011;**111**:2069–78.
55. Lima A, Forjaz CL, Silva GQ, Menêses AL, Silva AJ, Ritti-Dias RM. Acute effect of resistance exercise intensity in cardiac autonomic modulation after exercise. *Arq Bras Cardiol* 2011;**96**:498–503.
56. Kingsley JD, Panton LB, McMillan V, Figueroa A. Cardiovascular autonomic modulation after acute resistance exercise in women with fibromyalgia. *Arch Phys Med Rehabil* 2009;**90**:1628–34.
57. Heffernan KS, Kelly EE, Collier SR, Fernhall B. Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. *Eur J Cardiovasc Prev Rehabil* 2006;**13**:80–6.
58. Buchheit M, Laursen PB, Ahmaidi S. Parasympathetic reactivation after repeated sprint exercise. *Am J Physiol Heart Circ Physiol* 2007;**293**:H133–41.
59. Beske SD, Alvarez GE, Ballard TP, Davy KP. Gender difference in cardiovagal baroreflex gain in humans. *J Appl Physiol* 2001;**91**:2088–92.
60. Wehrwein EA, Joyner MJ. Regulation of blood pressure by the arterial baroreflex and autonomic nervous system. *Handb Clin Neurol* 2013;**117**:89–102.
61. Secher NH. Heart rate at the onset of static exercise in man with partial neuromuscular blockade. *J Physiol* 1985;**368**:481–90.
62. Lasevicius T, Ugrinowitsch C, Schoenfeld BJ, et al. Effects of different intensities of resistance training with equated volume load on muscle strength and hypertrophy. *Eur J Sport Sci* 2018;**18**:772–80.
63. Holm L, Reitelseder S, Pedersen TG, et al. Changes in muscle size and MHC composition in response to resistance exercise with heavy and light loading intensity. *J Appl Physiol* 2008;**105**:1454–61.
64. Mangine GT, Hoffman JR, Wang R, et al. Resistance training intensity and volume affect changes in rate of force development in resistance-trained men. *Eur J Appl Physiol* 2016;**116**:2367–74.

Please cite this article as: Sajith U. Marasingha-Arachchige et al., Factors that affect heart rate variability following acute resistance exercise: A systematic review and meta-analysis, Journal of Sport and Health Science (2021), https://doi.org/10.1016/j.jshs.2020.11.008

13.2. STUDY 2

13.2.1. Supplementary results

13.2.1.1. Heart rate variability

13.2.1.1.1. Strength 100 versus Power 100 training

SampEn

There was no overall treatment effect on SampEn (p = 0.403). However, there was an overall time effect on SampEn (p = 0.013). No significant group x time interaction for SampEn was observed (p = 0.264; Figure 169).

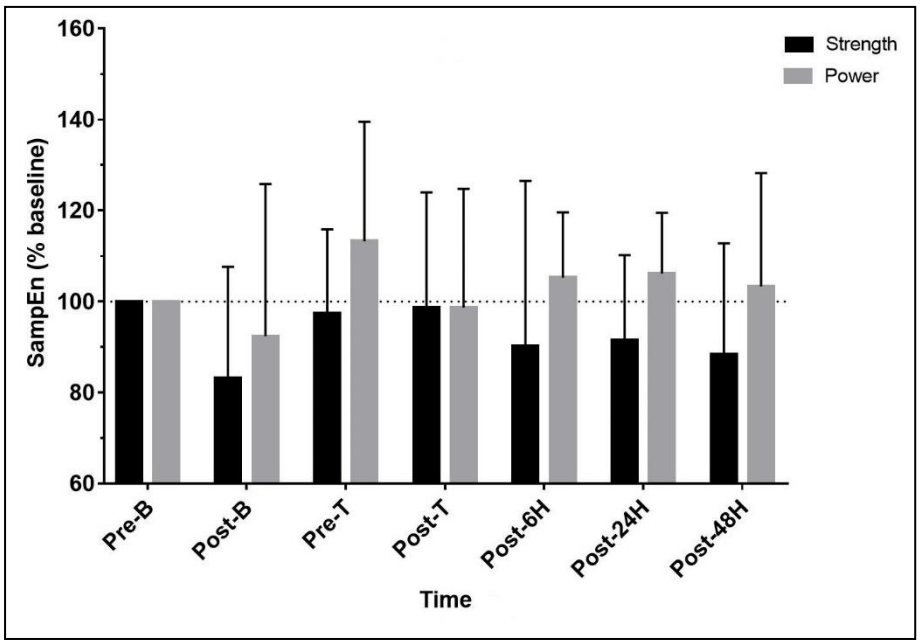


Figure 169. Comparison between S100 and P100 on SampEn values (n = 10)

Simple main effects over time revealed that SampEn differed significantly between time points in S100 (P = 0.036), but not for P100 (P = 0.123) trial. In S100, (Post-B (p = 0.427, ES = -1.12), Pre-T (p = 1.000, ES = -0.07), Post-T (p = 1.000, ES = -0.08), Post-6H (p = 1.000, ES = -0.23), Post-24H (p = 1.000, ES = -0.45) and Post-48H

(p = 1.000, ES = -0.58)) and P100 trial (Post-B (p = 1.000, ES = -0.43), Pre-T (p = 0.520, ES = 0.92), Post-T (p = 1.000, ES = -0.21), Post-6H (p = 1.000, ES = 0.39), Post-24H (p = 1.000, ES = 0.42) and Post-48H (p = 1.000, ES = 0.11)) showed no significant difference at all the time points compared to Pre-B value.

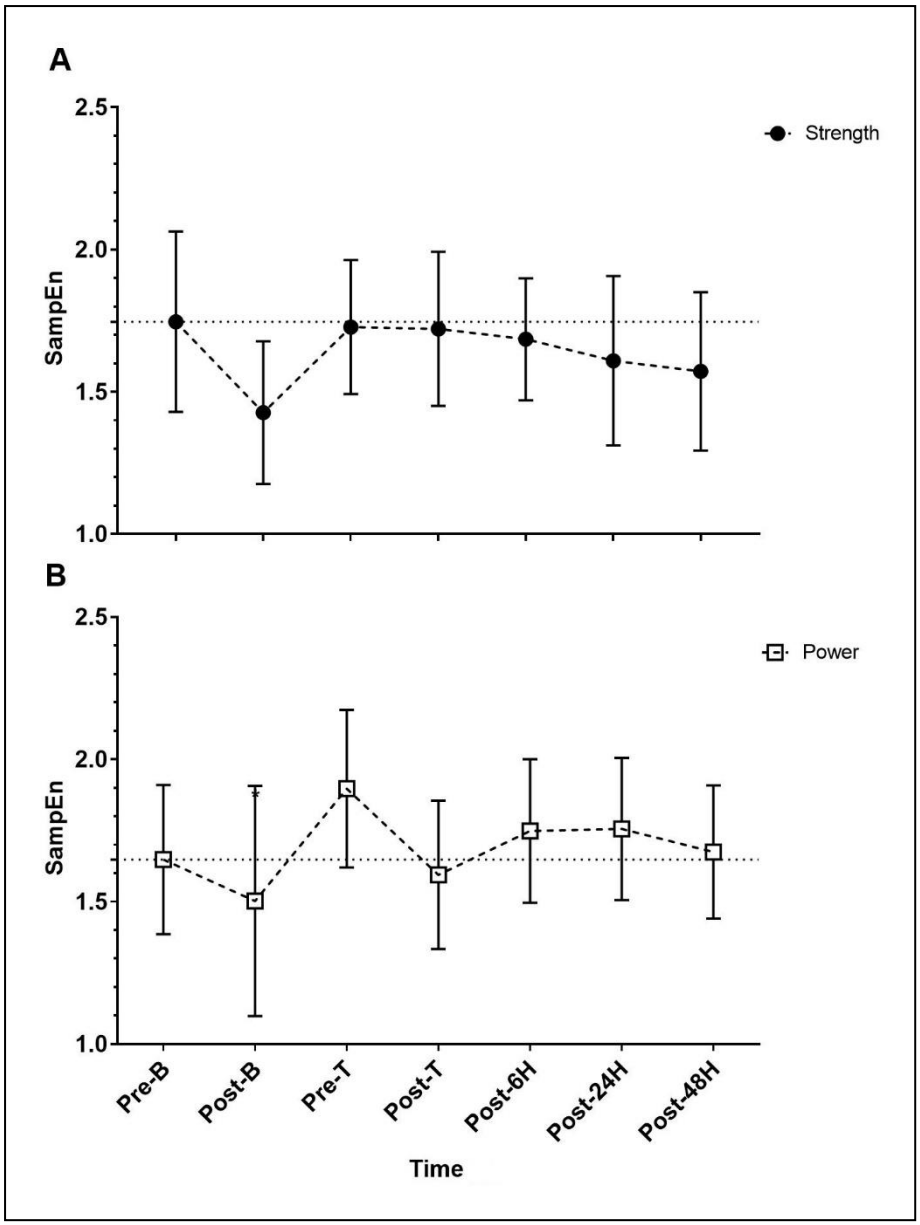


Figure 170. Changes in mean SampEn values in (A) Strength 100 and (B) Power 100 protocols (n = 10).

These results revealed that both protocols did not significantly influence the heart rate complexity throughout the trial compared to Pre-B value. However, SampEn decreased following the M-Beast protocol and ARE protocols for in both protocols, and only P100 trail gradually returned to Pre-B value. According to the ES results, SampEn recovered to baseline (Pre-B) at Post-6H for P100, whereas S100 did not recover at Post-48H (Figure 170).

SD1/SD2 ratio

There was an overall treatment effect ($p = 0.027$) and an overall time effect ($p < 0.001$) on SD1/SD2 ratio. However, no significant group x time interaction for SD1/SD2 ratio was observed ($p = 0.453$). simple main effects for treatment showed that SD1/SD2 ratio was significantly lower in the strength modality at Post-48H ($p = 0.001$; Figure 171) compared to the power modality.

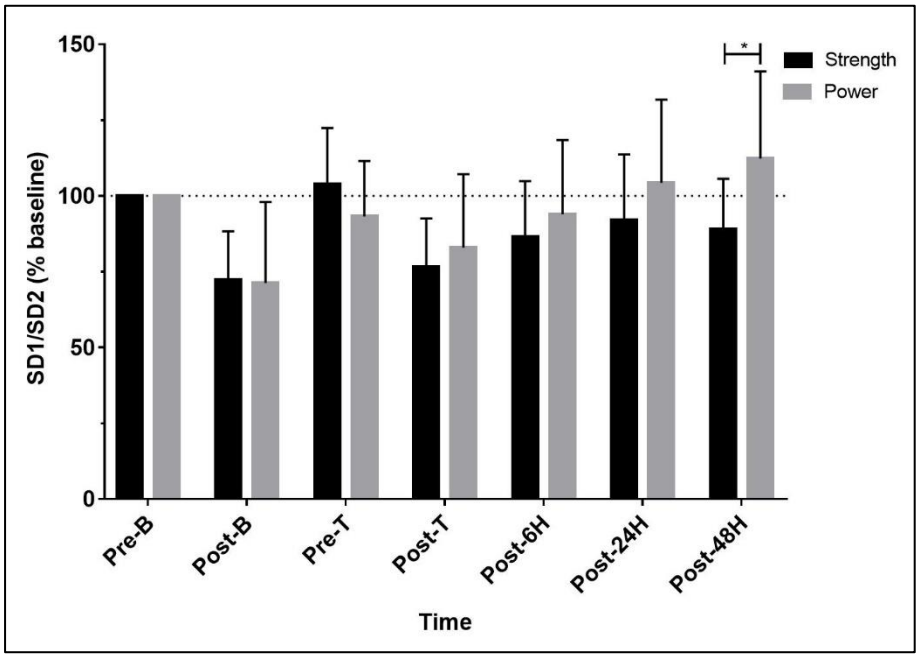


Figure 171. Comparison between S100 and P100 on SD1/SD2 ratio values (n = 11). * Significant pairwise comparison differences between strength and power modalities ($p \leq 0.05$).

Simple main effects over time revealed that SD1/SD2 ratio differed significantly between time points in S100 ($P < 0.001$) and P100 ($P = 0.001$) trials. In S100, no significant time differences were observed at Pre-T ($P = 1.000$, ES = 0.16), Post-6H ($P = 0.633$, ES = -0.67), Post-24H ($P = 1.000$, ES = -0.33), and Post-48H ($P = 0.602$, ES = -0.60), except at Post-B ($P = 0.005$, ES = -1.30) and Post-T ($P = 0.016$, ES = -1.04), compared to Pre-B value.

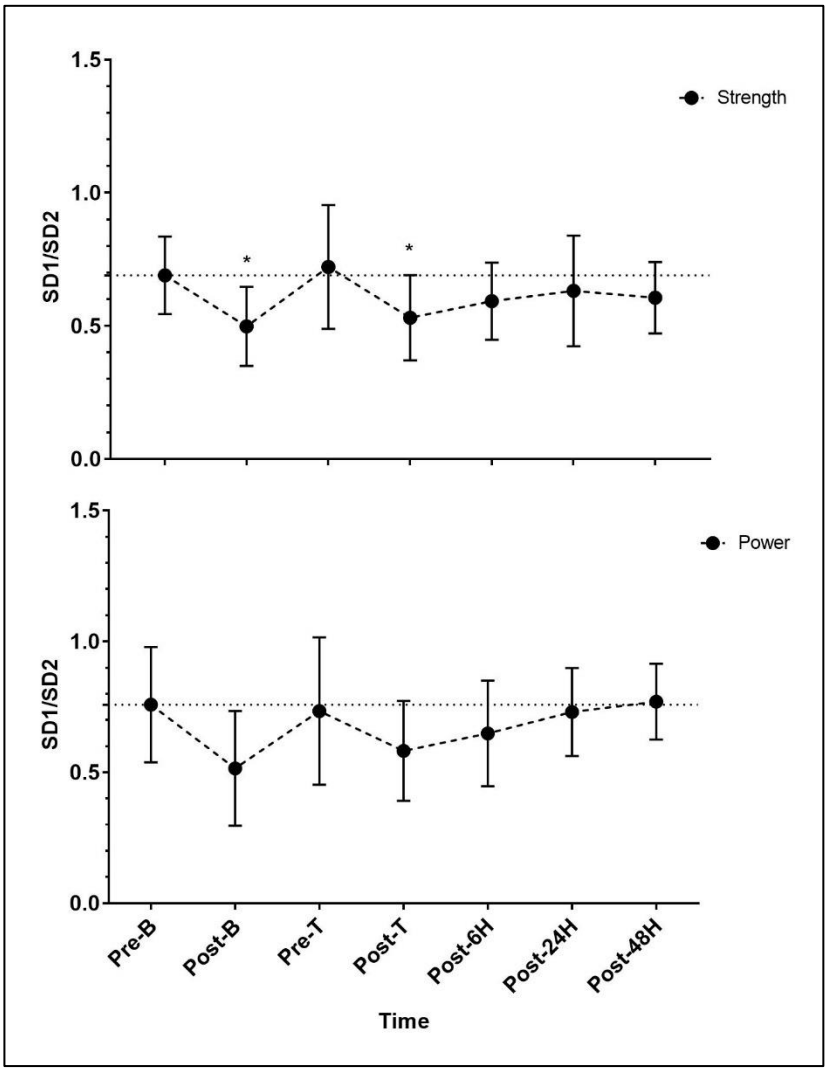


Figure 172. Changes in SD1/SD2 ratio value parameter in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

In P100, no significant time differences were shown at (Post-B (p = 0.150, ES = -1.11), Pre-T (p = 1.000, ES = -0.10), Post-T (p = 0.778, ES = -0.86), Post-6H (p = 1.000, ES = -0.52), Post-24H (p = 1.000, ES = -0.14) and Post-48H (p = 1.000, ES = -0.06)) all the time points. These results revealed that SD1/SD2 ratio decreased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values. According to the ES results, SD1/SD2 ratio recovered to baseline (Pre-B) at Post-48H for P100, whereas S100 did not recover at Post-48H. (Figure 172).

Systolic blood pressure

There was neither an overall treatment effect (p = 0.489) nor an overall time effect (p = 0.328) on Systolic blood pressure. No significant group x time interaction for Systolic blood pressure was observed (p = 0.874; Figure 173).

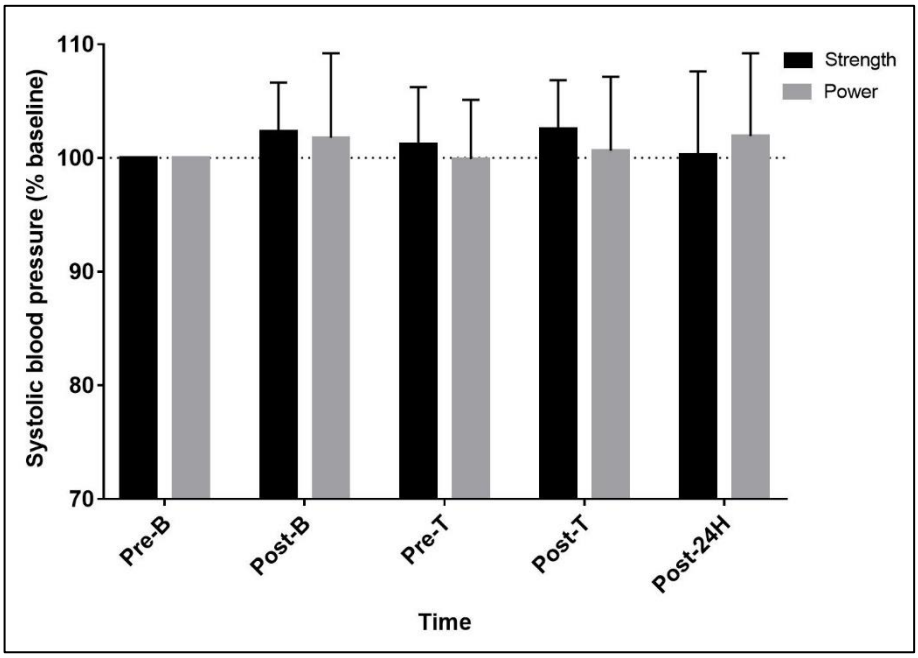


Figure 173. Comparison between S100 and P100 on Systolic blood pressure values (n = 11)

Diastolic blood pressure

There was neither an overall treatment effect (p = 0.533) nor an overall time effect (p = 0.381) on Diastolic blood pressure. No significant group x time interaction for Diastolic blood pressure was observed (p = 0.330; Figure 174).

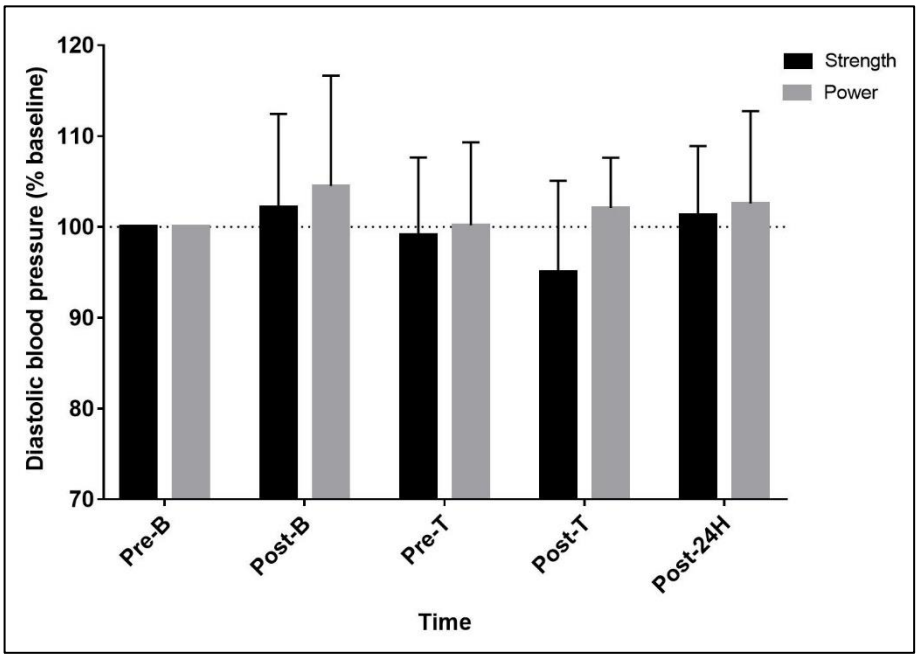


Figure 174. Comparison between S100 and P100 on Diastolic blood pressure values (n = 11)

Resting Heart rate

There was no overall treatment effect on resting HR (p = 0.244). However, there was an overall time effect on resting HR (p < 0.001). No significant group x time interaction for resting HR was observed (p = 0.131; Figure 175).

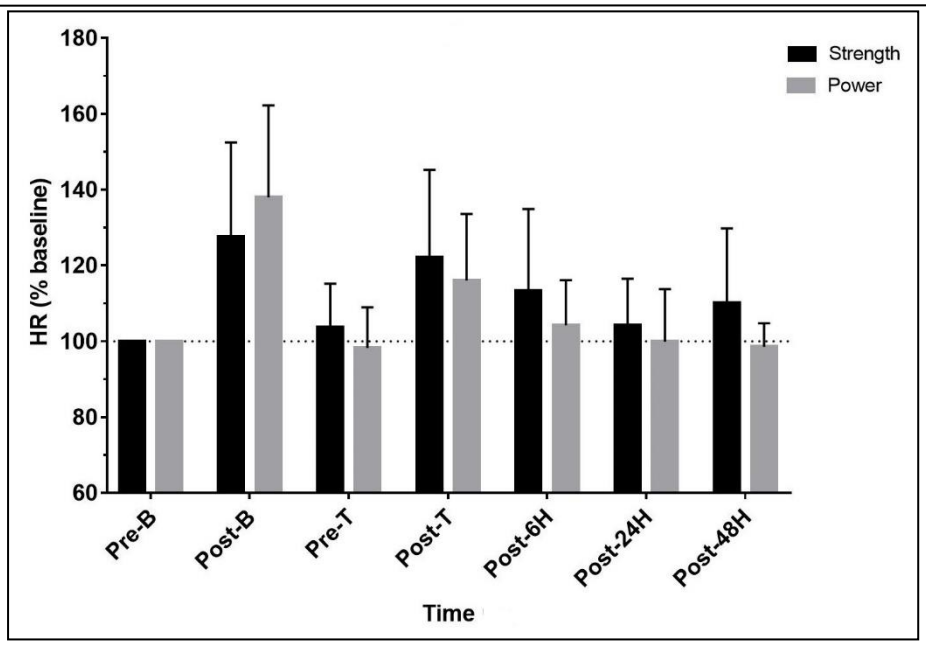


Figure 175. Comparison between S100 and P100 on resting HR values (n = 11)

Simple main effects over time that resting HR differed significantly between time points in S100 ($P < 0.001$) and P100 ($P < 0.001$) trials. Compared to Pre-B, significant time differences were observed at Post-B (S100: $p = 0.046$, ES = 0.93; P100: $p = 0.009$, ES = 1.48) in both protocols, but not in other time points (Pre-T (S100: $p = 1.000$, ES = 0.11; P100: $p = 1.000$, ES = -0.11), Post-T (S100: $p = 0.199$, ES = 0.71; P100: $p = 0.293$, ES = 0.79), Post-6H (S100: $p = 1.000$, ES = 0.50; P100: $p = 1.000$, ES = 0.25), Post-24H (S100: $p = 1.000$, ES = 0.13; P100: $p = 1.000$, ES = -0.08) and Post-48H (S100: $p = 1.000$, ES = 0.37; P100: $p = 1.000$, ES = -0.13)) in both training protocols. These results revealed that resting HR increased following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B values.

According to the ES results, resting HR recovered to baseline (Pre-B) at Post-24H for P100, whereas S100 did not recover at Post-48H. (Figure 176).

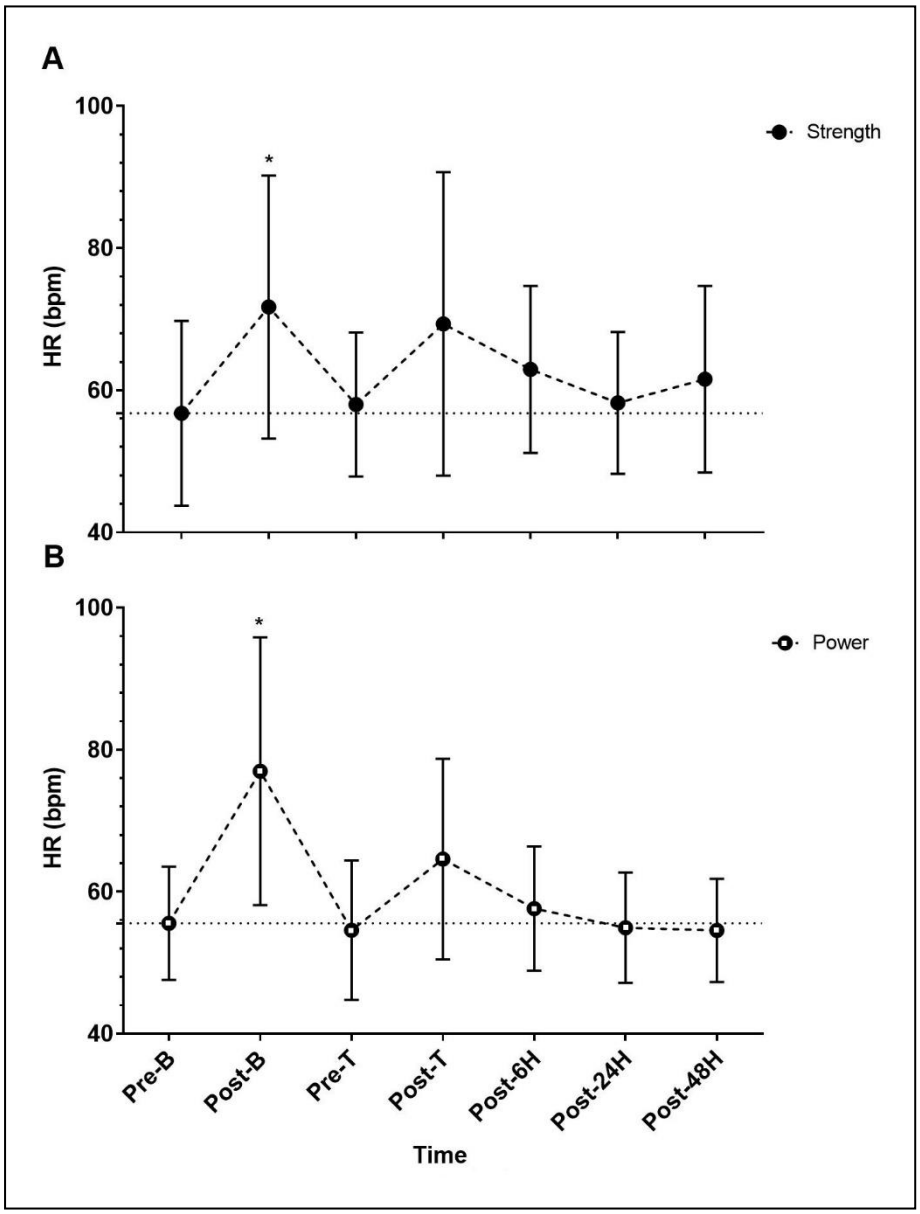


Figure 176. Changes in resting HR values in (A) S100 and (B) P100 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

13.2.1.1.2. Strength 100 versus Strength 75 versus Strength 50 training

SampEn

There was an overall treatment effect and time effect on SampEn ($p = 0.045$). However, there was an overall time effect on SampEn ($p < 0.001$). However, no significant group x time interaction for SampEn was observed ($p = 0.857$). Yet, Simple main effects for treatment showed that SampEn was not significantly different between treatments (S100 vs S75 vs S50) at all the time points (Figure 177).

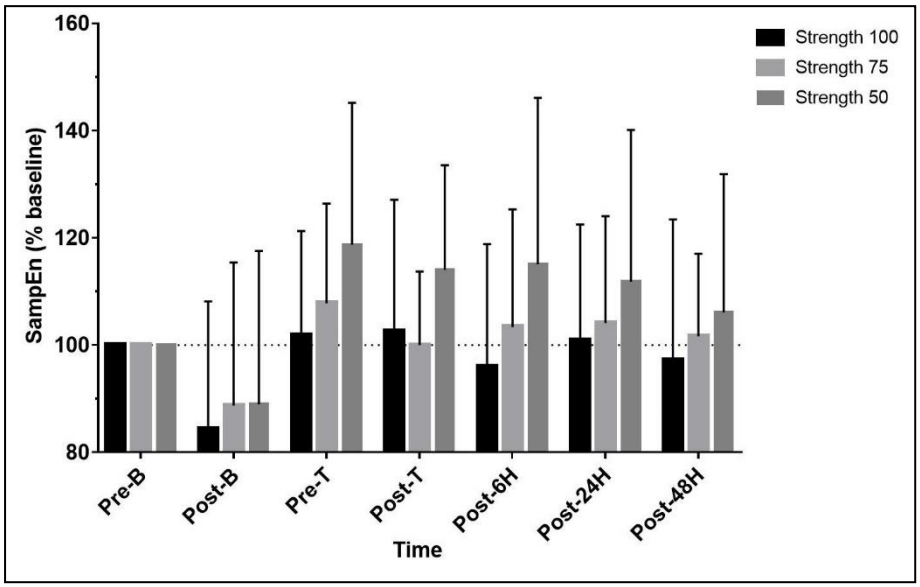


Figure 177. Comparison of S100, S75 and S50 on SampEn values (n = 11).

Simple main effects over time revealed that SampEn differed significantly between time points in S100 ($P = 0.015$), S75 ($P = 0.006$) and S50 ($P = 0.009$) trial. In S100, no significant time differences were observed at all the time points (Post-B ($p = 0.109$, ES = -0.96), Pre-T ($p = 1.000$, ES = -0.10), Post-T ($p = 1.000$, ES = -0.03), Post-6H ($p = 1.000$, ES = -0.36), Post-24H ($p = 1.000$, ES = -0.13) and Post-48H ($p = 1.000$, ES = -0.36)) compared to Pre-B. Similarly, P75 also showed no significant time differences at all the time points (Post-B ($p = 0.811$, ES = -0.82), Pre-T ($p = 1.000$, ES = 0.52), Post-T ($p = 1.000$, ES = -0.12), Post-6H ($p = 1.000$, ES = 0.11), Post-24H ($p =$

1.000, ES = 0.22) and Post-48H (p = 0.621, ES = 0.07)) compared to Pre-B. In the same way, there was no significant time differences were observed at all the time points in S50 (Post-B (p = 1.000, ES = -0.60), Pre-T (p = 1.000, ES = 0.81), Post-T (p = 0.451, ES = 0.70), Post-6H (p = 1.000, ES = 0.59), Post-24H (p = 1.000, ES = 0.45) and Post-48H (p = 1.000, ES = 0.15) compared to Pre-B (Figure 178). These results revealed that SampEn decreased following the M-Beast protocol in all 3 trials. However, SampEn decreased following the ARE protocol only in S75 and S50 trials. Even though there was a no significant difference between time points, ES results showed that SampEn was not recovered to the respected Pre-B value even at Post-48H time point for S100 trial. However, SampEn was recovered to the Pre-B level at the Post-6H time point for S75 trial and SampEn level was maintained higher than Pre-B level throughout the trial in S50 trial.

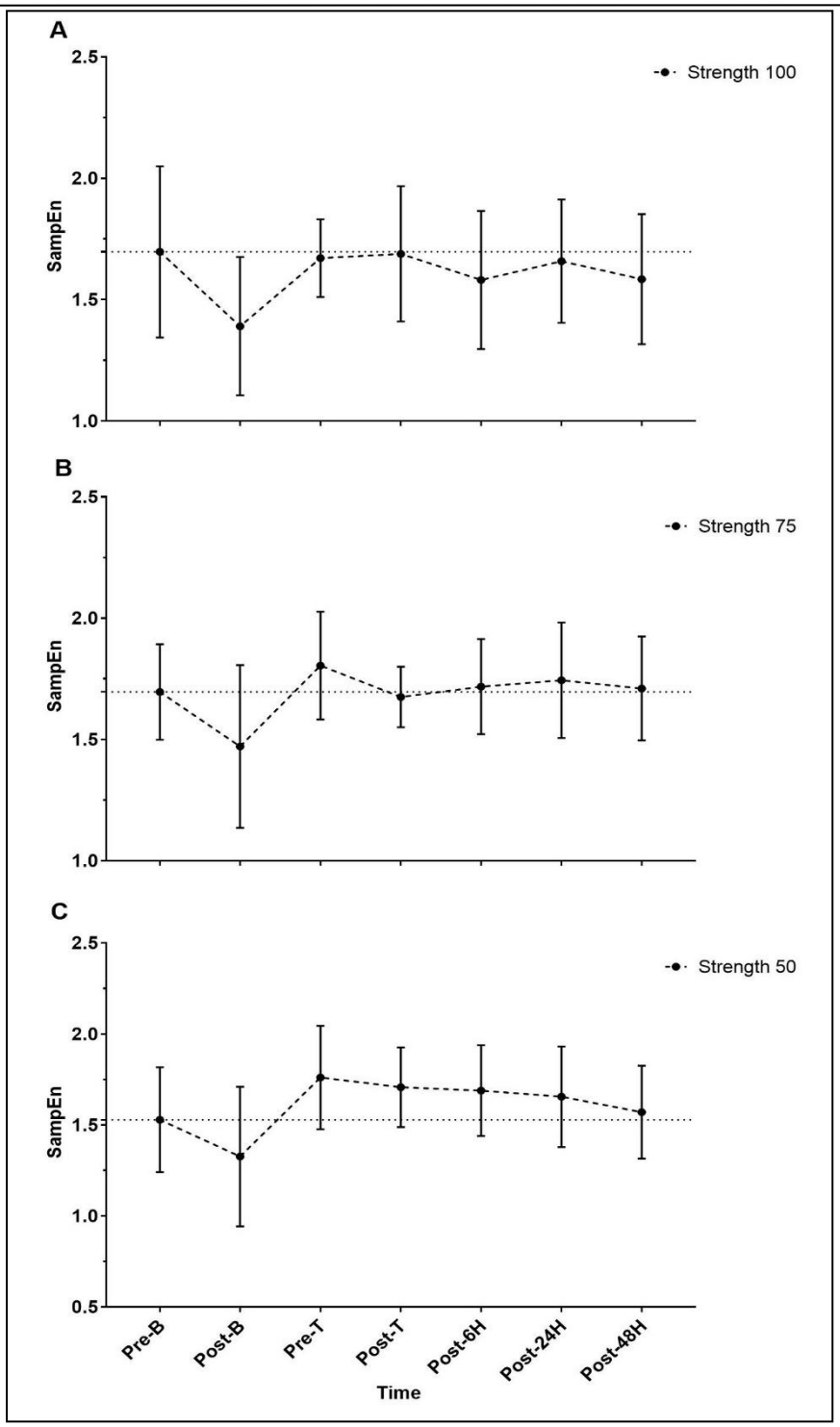


Figure 178. Changes in SampEn parameter in (A) S100, (B) S75 and (C) S50 protocols (n = 12).

SD1/SD2 ratio

There was no overall treatment effect on SD1/SD2 ratio ($p = 0.083$). However, there was an overall time effect on SD1/SD2 ratio ($p < 0.001$), and group x time interaction for SD1/SD2 ratio was observed ($p = 0.040$; Figure 179). simple main effects for treatment showed that SD1/SD2 ratio was significantly different between treatments (S100 vs S75 vs S50) at Post-T ($p = 0.002$, (S100 vs S75: $p = 1.000$; S100 vs S50: $p = 0.006$)).

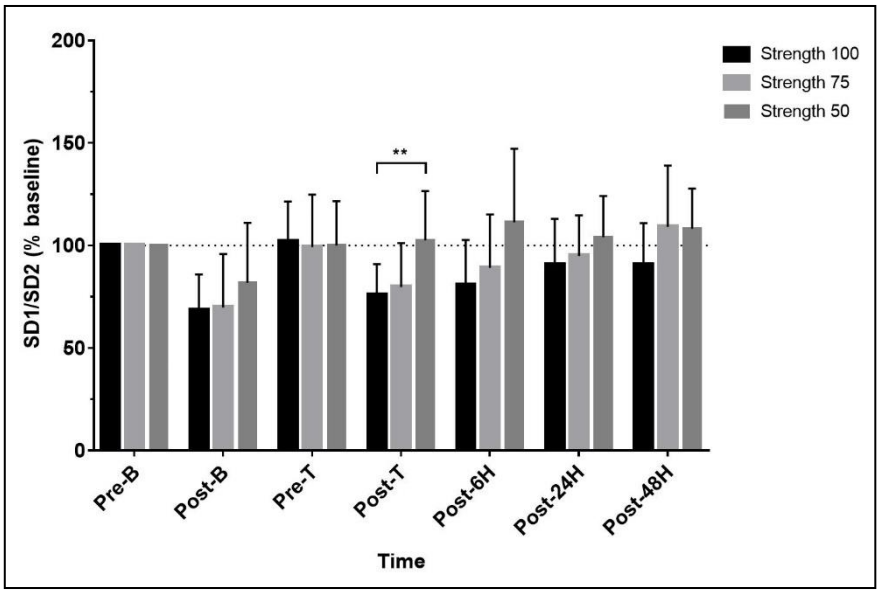


Figure 179. Comparison between S100, S75 and S50 on SD1/SD2 ratio values (n = 13). ** Significant pairwise comparison differences in S50 compared to S100 ($p \leq 0.05$).

Simple main effects over time revealed that SD1/SD2 ratio differed significantly between time points in S100 ($P < 0.001$) and S75 ($P < 0.001$), except in S50 ($P = 0.109$) trial. In S100, no significant time differences were observed at Pre-T ($p = 1.000$, ES = 0.07), Post-6H ($p = 0.227$, ES = -1.03), Post-24H ($p = 1.000$, ES = -0.38) and Post-48H ($p = 1.000$, ES = -0.38), except at Post-B ($p = 0.003$, ES = -1.58) and Post-T ($p = 0.002$, ES = -1.08) compared to Pre-B. Similarly, P75 also showed no significant time differences at Pre-T ($p = 1.000$, ES = -0.13), Post-T ($p = 0.222$, ES = -1.11), Post-6H ($p = 1.000$, ES = -0.67), Post-24H ($p = 1.000$, ES = -0.30) and Post-48H

(p = 1.000, ES = 0.25), except at Post-B (p = 0.031, ES = -1.21) compared to Pre-B. However, there was no significant time differences were observed at all the time points in S50 (Post-B (p = 1.000, ES = -0.50), Pre-T (p = 1.000, ES = -0.12), Post-T (p = 1.000, ES = -0.08), Post-6H (p = 1.000, ES = 0.17), Post-24H (p = 1.000, ES = 0.07) and Post-48H (p = 1.000, ES = 0.23), compared to Pre-B. These results revealed that SD1/SD2 ratio decreased following the M-Beast protocol in all 3 trials. However, SD1/SD2 ratio decreased following the ARE protocol only in S100 and S75 trials gradually returned to Pre-B values. According to the ES results, SD1/SD2 ratio of S50 recovered to baseline (Pre-B) at Post-6H, whereas S75 needed longer time (Post-48H) to recover. Interestingly, S100's level did not recover at Post-48H (Figure 180).

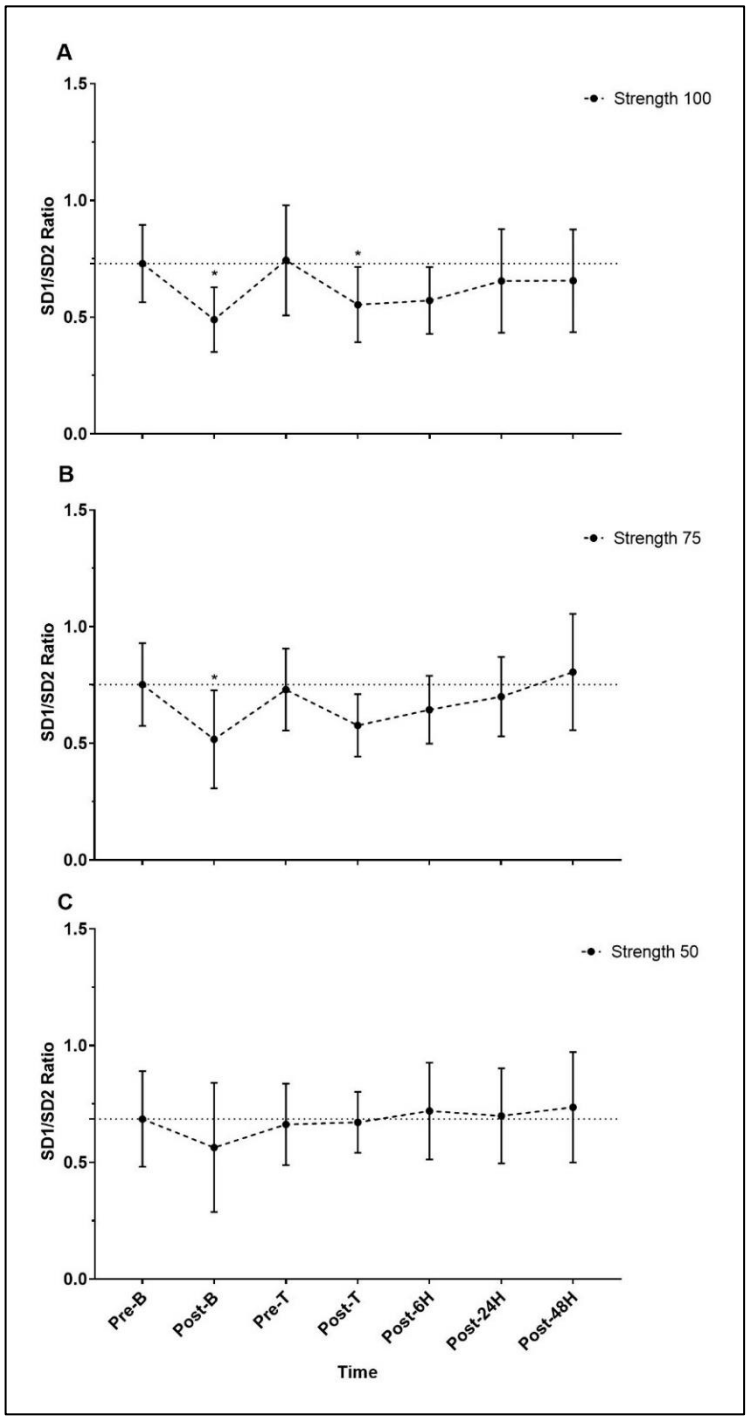


Figure 180. Changes in SD1/SD2 ratio values in (A) S100, (B) S75 and (C) S50 protocols (n = 13). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis

Systolic blood pressure

There was neither an overall treatment effect (p = 0.705) nor an overall time effect (p = 0.090) on Systolic blood pressure. No significant group x time interaction for Systolic blood pressure was observed (p = 0.980; Figure 181).

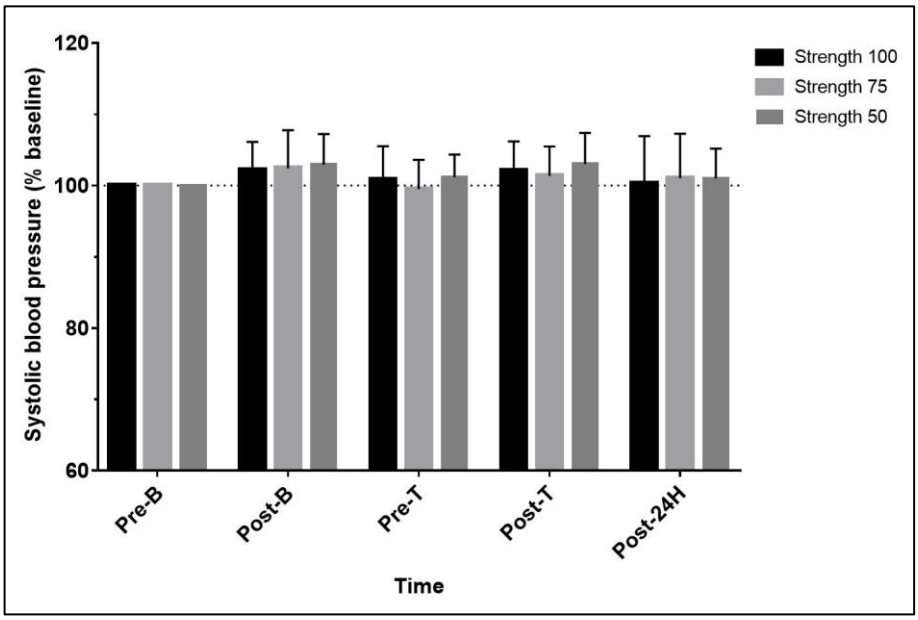


Figure 181. Comparison between S100, S75 and S50 on Systolic blood pressure values (n = 13).

Diastolic blood pressure

There was no overall treatment effect on Diastolic blood pressure (p = 0.915). However, there was an overall time effect on Diastolic blood pressure (p = 0.020). No significant group x time interaction for Diastolic blood pressure was observed (p = 0.776; Figure 182). Furthermore, Simple main effects over time revealed that Diastolic blood pressure not significantly different between time points in S100 (P = 0.155), S75 (P = 0.725) and S50 (P = 0.237) trials.

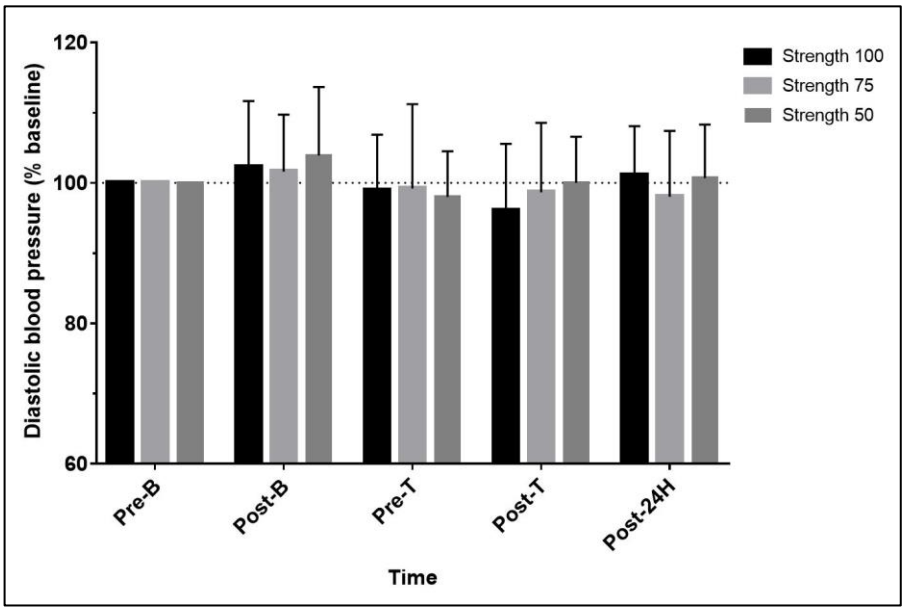


Figure 182. Comparison between S100, S75 and S50 on Diastolic blood pressure values (n = 13).

Resting Heart rate

There was no overall treatment effect on resting HR ($p = 0.276$). However, there was an overall time effect on resting HR ($p < 0.001$), and group x time interaction for resting HR was observed ($p = 0.054$; Figure 183).

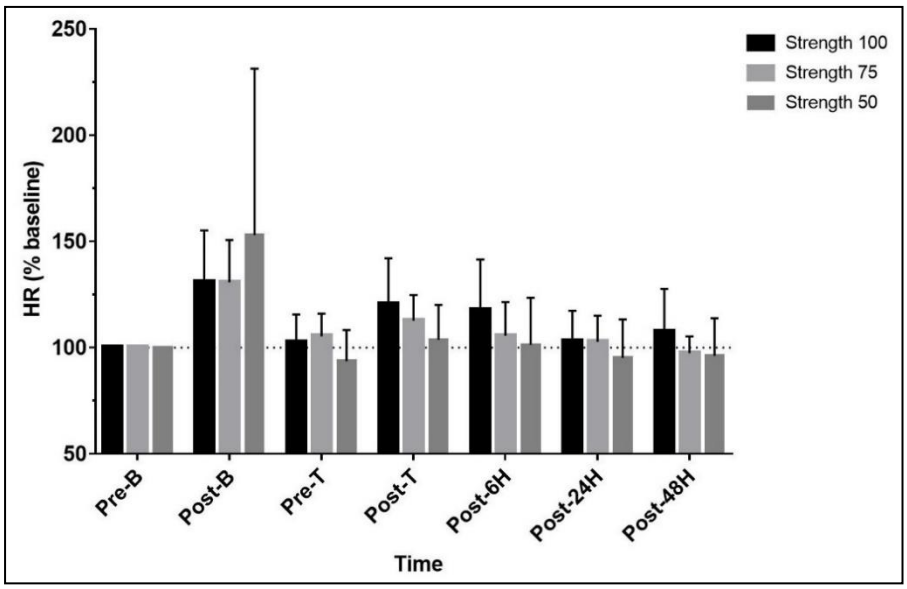


Figure 183. Comparison of S100, S75 and S50 on resting HR values (n = 13)

Simple main effects over time revealed that resting HR different significantly between time points in S100 ($P < 0.001$), S75 ($P < 0.001$) and S50 ($P = 0.005$) trials. In S100, no significant time differences were observed at Pre-T ($p = 1.000$, ES = 0.07), Post-T ($p = 0.107$, ES = 0.70), Post-6H ($p = 0.324$, ES = 0.71), Post-24H ($p = 1.000$, ES = 0.10) and Post-48H ($p = 1.000$, ES = 0.27) except at Post-B ($p = 0.006$, ES = 1.12) compared to Pre-B. In S75, no significant time differences were observed at Pre-T ($p = 1.000$, ES = 0.34), Post-6H ($p = 1.000$, ES = 0.32), Post-24H ($p = 1.000$, ES = 0.17) and Post-48H ($p = 1.000$, ES = -0.17) except Post-B ($p = 0.002$, ES = 1.61) and Post-T ($p = 0.039$, ES = 0.76) compared to Pre-B. In S50, no significant time differences were observed at all the time points (Post-B: $p = 0.340$, ES = 1.07; Pre-T: $p = 1.000$, ES = -0.37; Post-T: $p = 1.000$, ES = 0.11; Post-6H: $p = 1.000$, ES = -0.06; Post-24H: $p = 1.000$, ES = -0.31 and Post-48H: $p = 1.000$, ES = -0.32) compared to Pre-B. These results revealed that resting HR increased following the M-Beast protocol and ARE protocols for all 3 training loads, and it gradually returned to Pre-B values. According to the ES results, resting HR recovered to baseline (Pre-B) at Post-6H for S50 and at Post-48H for S75, whereas S100 did not recover at Post-48H. (Figure 184).

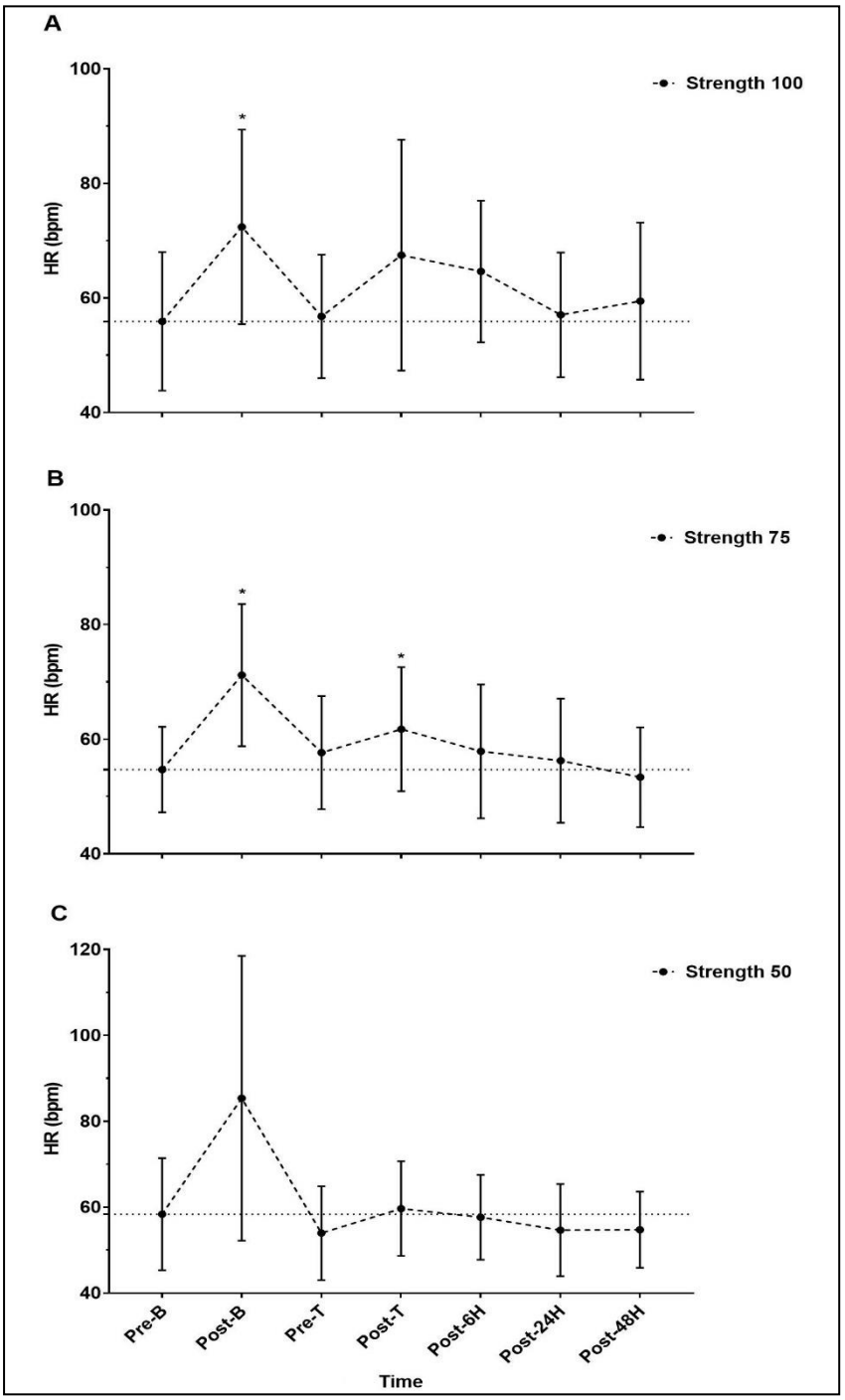


Figure 184. Changes in resting HR values in (A) S100, (B) S75 and (C) S50 protocols (n = 13).
* Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

13.2.1.1.3. Power 100 versus Power 75 versus Power 50 training

SampEn

There was no overall treatment effect on SampEn (p = 0.734). However, there was an overall time effect on SampEn (p = 0.052). No significant group x time interaction for SampEn was observed (p = 0.546; Figure 185).

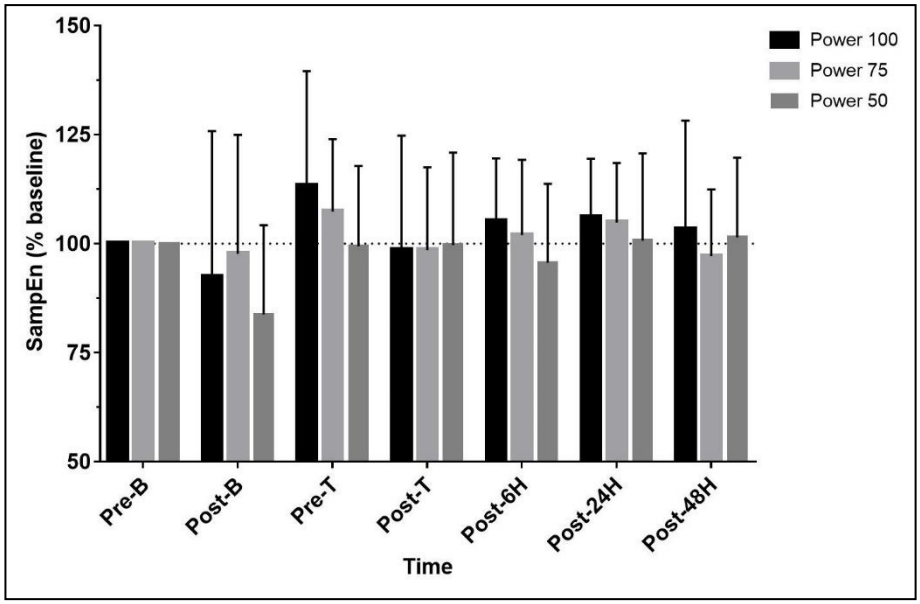


Figure 185. Comparison of P100, P75 and P50 on SampEn values (n = 11)

Simple main effects over time revealed that SampEn not differed significantly between time points in P100 (P = 0.112) and P75 (P = 0.490) except for P50 (P = 0.046) trial. All 3 training loads ((P100: Post-B (p = 1.000, ES = -0.58), Pre-T (p = 1.000, ES = 0.63), Post-T (p = 1.000, ES = -0.31), Post-6H (p = 1.000, ES = 0.27), Post-24H (p = 1.000, ES = 0.34) and Post-48H (p = 1.000, ES = 0.02)), (P75:Post-B (p = 1.000, ES = -0.22), Pre-T (p = 1.000, ES = 0.52), Post-T (p = 1.000, ES = -0.26), Post-6H (p = 1.000, ES = 0.05), Post-24H (p = 1.000, ES = 0.34) and Post-48H (p = 1.000, ES = -0.31)) and (P50: Post-B (p = 0.450, ES = -1.14), Pre-T (p = 1.000, ES = -0.14), Post-T (p = 1.000, ES = -0.10), Post-6H (p = 1.000, ES = -0.48), Post-24H (p = 1.000,

ES = -0.09) and Post-48H (p = 1.000, ES = 0.01))) showed no significant time differences between time points compared to respective Pre-B. Even though all 3 training loads showed no significant difference compared to their respected Pre-B values, SampEn decreased following the M-Beast protocol in all 3 training loads and ARE protocols for P100 and P75 training loads, and it gradually returned to Pre-B values. Interestingly, P50 did not decrease the SampEn following the ARE protocol. According to the ES results, SampEn of P100 and P50 were recovered to the baseline (Pre-B) at Post-6H and Post-48H respectively, whereas P75's did not recover at Post-48H (Figure 186).

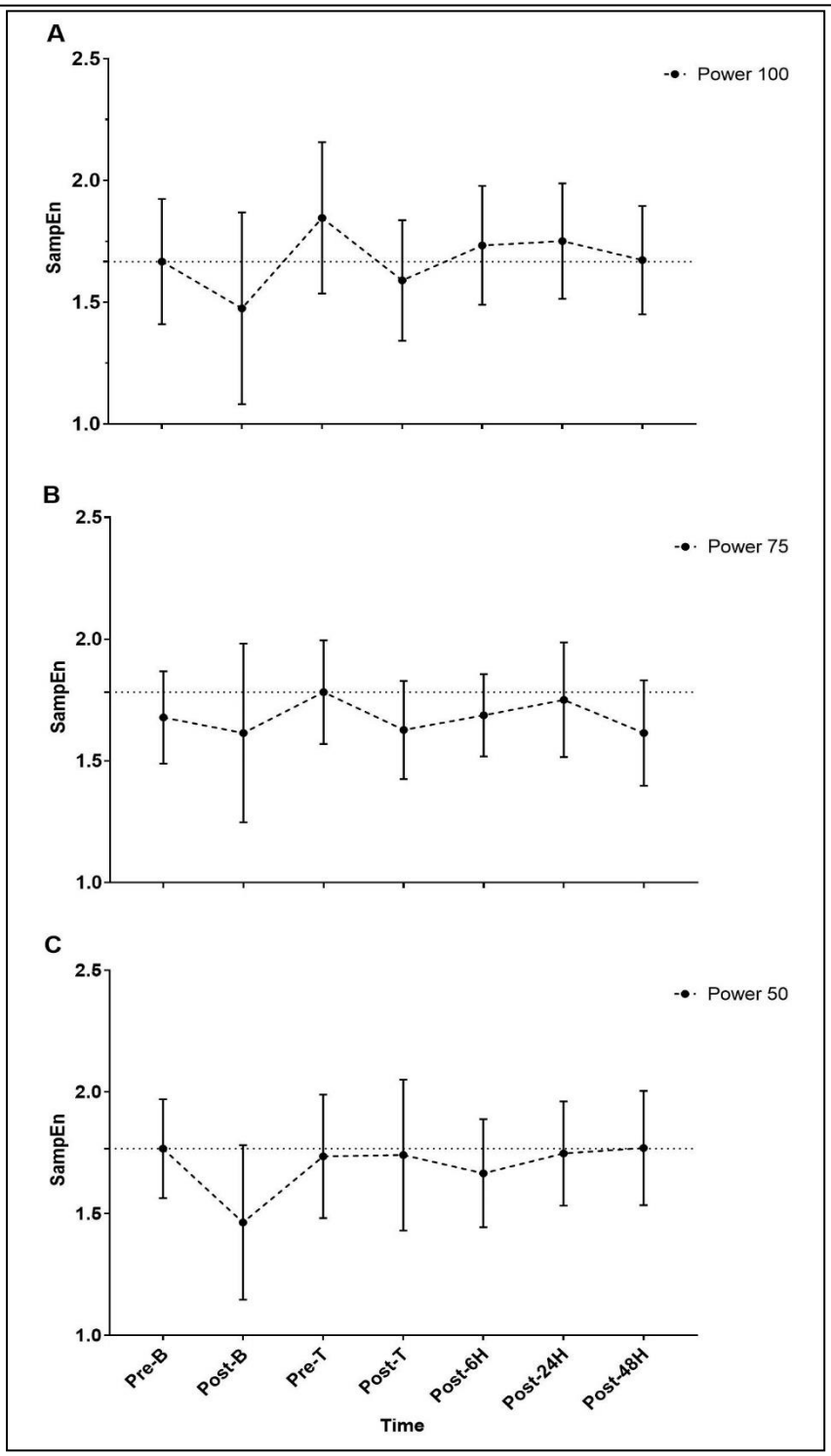


Figure 186. Changes in mean SampEn values in (A) P100, (B) P75 and (C) P50 protocols (n = 11).

SD1/SD2 ratio

There was no overall treatment effect on SD1/SD2 ratio ($p = 0.530$). However, there was an overall time effect on SD1/SD2 ratio ($p < 0.001$). No significant group x time interaction for SD1/SD2 ratio was observed ($p = 0.833$; Figure 187)

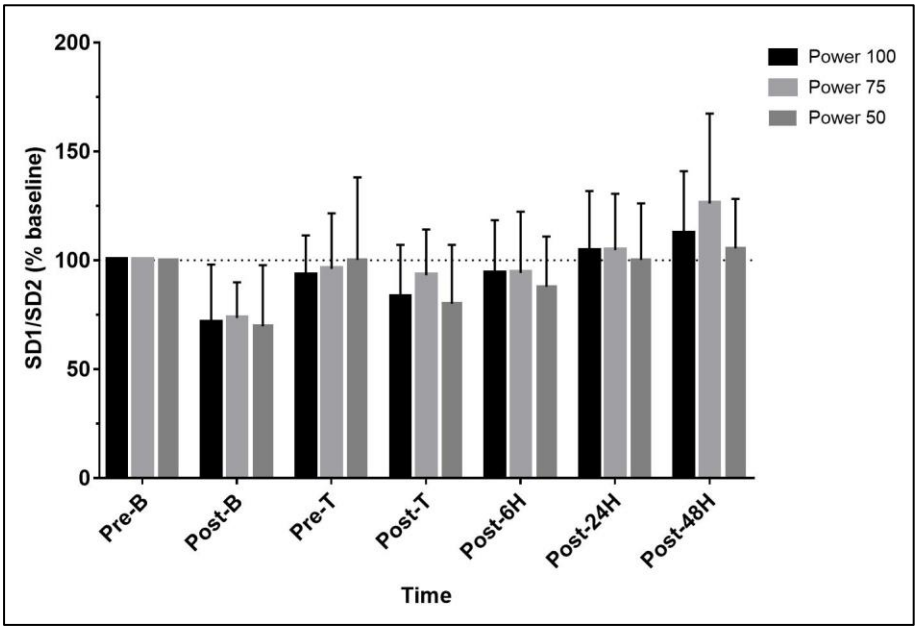


Figure 187. Comparison of P100, P75 and P50 on SD1/SD2 ratio values (n = 11).

Simple main effects over time revealed that SD1/SD2 ratio differed significantly between time points in P100 ($P < 0.001$) and P75 ($P = 0.001$) and P50 ($P = 0.001$) trials. In P100, no significant time differences were observed at all the time points (Post-B ($p = 0.181$, ES = -1.32), Pre-T ($p = 1.000$, ES = -0.41), Post-T ($p = 1.000$, ES = -0.83), Post-6H ($p = 1.000$, ES = -0.36), Post-24H ($p = 1.000$, ES = 0.10) and Post-48H ($p = 1.000$, ES = 0.42)) compared to Pre-B. Similarly, P50 also showed no significant time differences at all the time points (Post-B ($p = 0.150$, ES = -1.11), Pre-T ($p = 1.000$, ES = -0.10), Post-T ($p = 0.778$, ES = -0.86), Post-6H ($p = 1.000$, ES = -0.52), Post-24H ($p = 1.000$, ES = -0.14) and Post-48H ($p = 0.621$, ES = -0.10)) compared to Pre-B. In P75, no significant time differences were observed at Pre-T ($p = 1.000$, ES = -0.30), Post-T ($p = 1.000$, ES = -0.14), Post-6H ($p = 1.000$, ES = -0.32), Post-24H

($p = 1.000$, ES = 0.07) and Post-48H ($p = 1.000$, ES = 0.54), except at Post-B ($p = 0.051$, ES = -1.02), compared to Pre-B (Figure 188). These results revealed that SD1/SD2 ratio decreased following the M-Beast protocol in all 3 trials. However, SD1/SD2 ratio decreased following the ARE protocol only in P100 and P50 trials , and it gradually returned to Pre-B values. Interestingly, P75 did not decrease the SD1/SD2 ratio following the ARE protocol. According to the ES results, SD1/SD2 ratio of P100 and P75 were recovered to the baseline (Pre-B) at Post-24H, whereas P50's did recover at Post-48H.

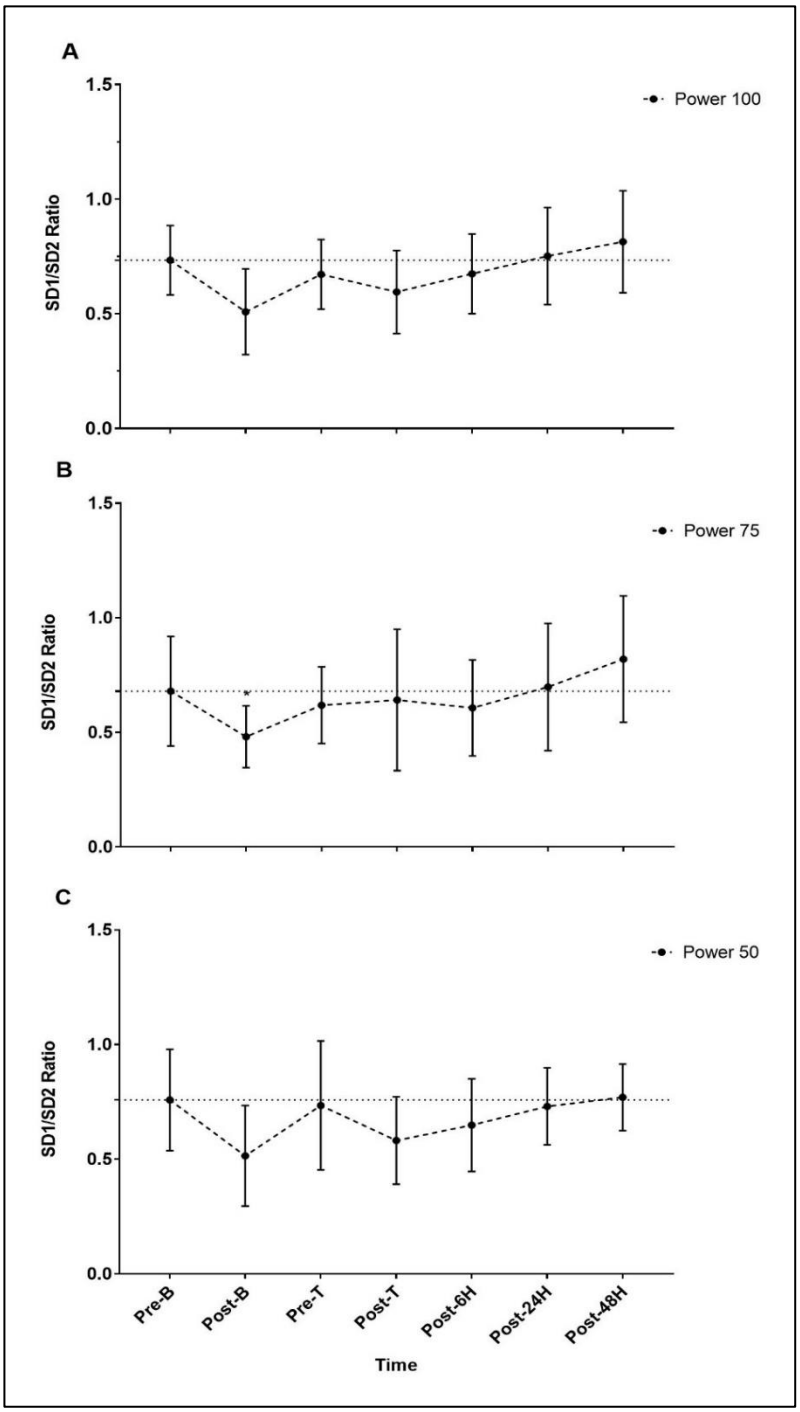


Figure 188. Changes in mean SD1/SD2 ratio values in (A) P100, (B) P75 and (C) P50 protocols (n = 11).). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

Systolic blood pressure

There was neither an overall treatment effect (p = 0.648) nor an overall time effect (p = 0.408) on Systolic blood pressure. No significant group x time interaction for Systolic blood pressure was observed (p = 0.746; Figure 189).

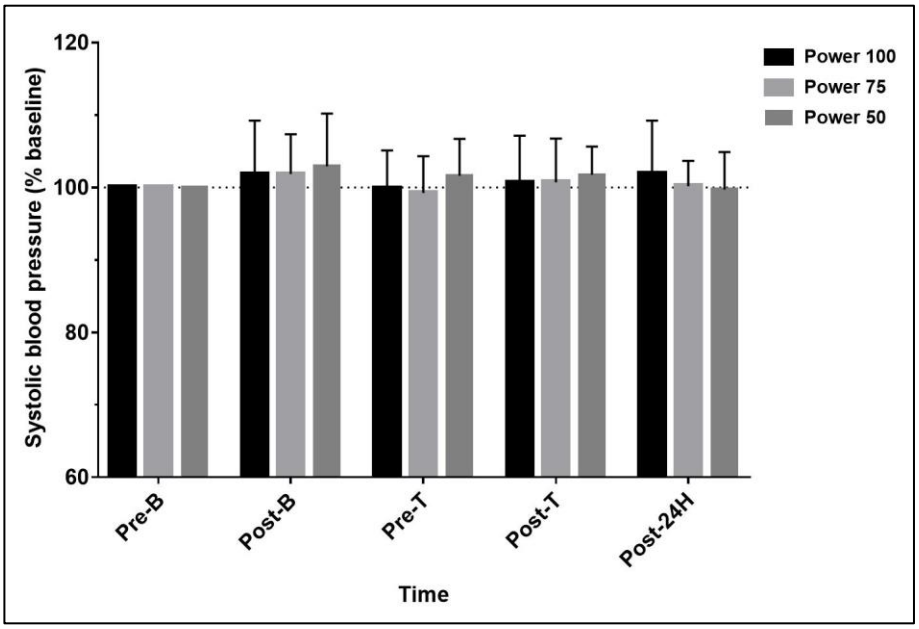


Figure 189. Comparison of P100, P75 and P50 on Systolic blood pressure values (n = 11).

Diastolic blood pressure

There was neither an overall treatment effect (p = 0.663) nor an overall time effect (p = 0.232) on Diastolic blood pressure. No significant group x time interaction for Diastolic blood pressure was observed (p = 0.852; Figure 190)

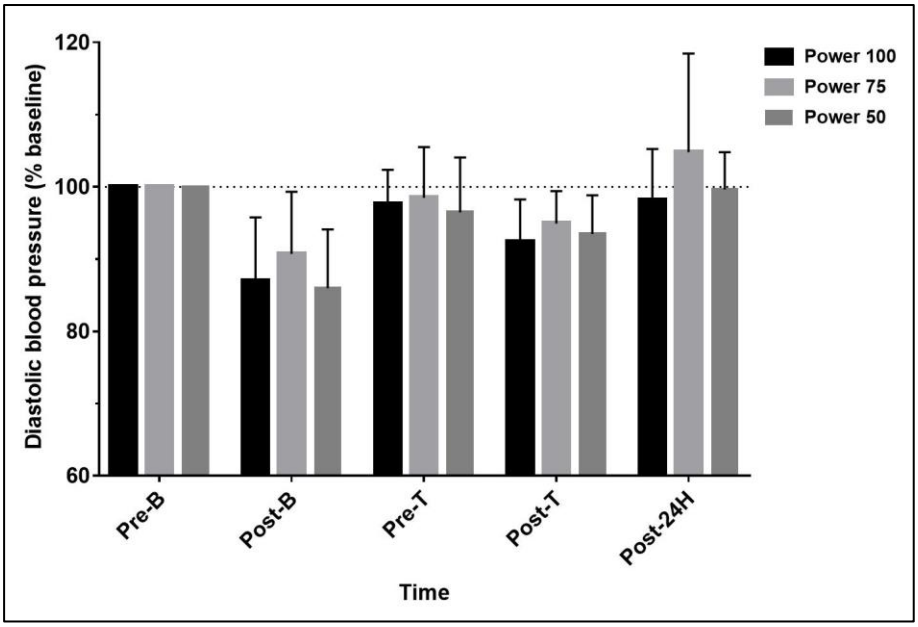


Figure 190. Comparison of P100, P75 and P50 on Diastolic blood pressure values (n = 11).

Resting Heart rate

There was no overall treatment effect on resting HR ($p = 0.583$). However, there was an overall time effect on resting HR ($p < 0.001$). No significant group x time interaction for resting HR was observed ($p = 0.277$; Figure 191).

Simple main effects over time revealed that resting HR different significantly between time points in P100 ($P < 0.001$), P75 ($P < 0.001$) and P50 ($P = 0.001$) trial. In P100, no significant time differences were observed at Pre-T ($p = 1.000$, ES = -0.11), Post-T ($p = 0.293$, ES = 0.79), Post-6H ($p = 1.000$, ES = 0.25), Post-24H ($p = 1.000$, ES = -0.08), Post-48H ($p = 1.000$, ES = -0.13) except at Post-B ($p = 0.005$, ES = 1.48) compared to Pre-B. Similarly, P75 also showed no significant time differences at Pre-T ($p = 1.000$, ES = 0.47), Post-T ($p = 1.000$, ES = 0.68), Post-6H ($p = 1.000$, ES = 0.62), Post-24H ($p = 1.000$, ES = 0.53), Post-48H ($p = 1.000$, ES = 0.10) except at Post-B ($p < 0.001$, ES = 1.56) compared to Pre-B. In P50, no significant time differences

were observed at all the time points (Post-B (p = 0.248, ES = 0.92), Pre-T (p = 1.000, ES = 0.01), Post-T (p = 1.000, ES = 0.06), Post-6H (p = 1.000, ES = 0.10), Post-24H (p = 1.000, ES = -0.10) and Post-48H (p = 1.000, ES = -0.15)) compared to Pre-B.

These results revealed that resting HR increased following the M-Beast protocol (significantly increased in P100 and P75) and ARE protocols in all 3 training loads, and it gradually returned to Pre-B values. According to the ES results, resting HR of P100 and P50 were recovered at Post-24H, whereas P75's level did not recover at Post-48H (Figure 192).

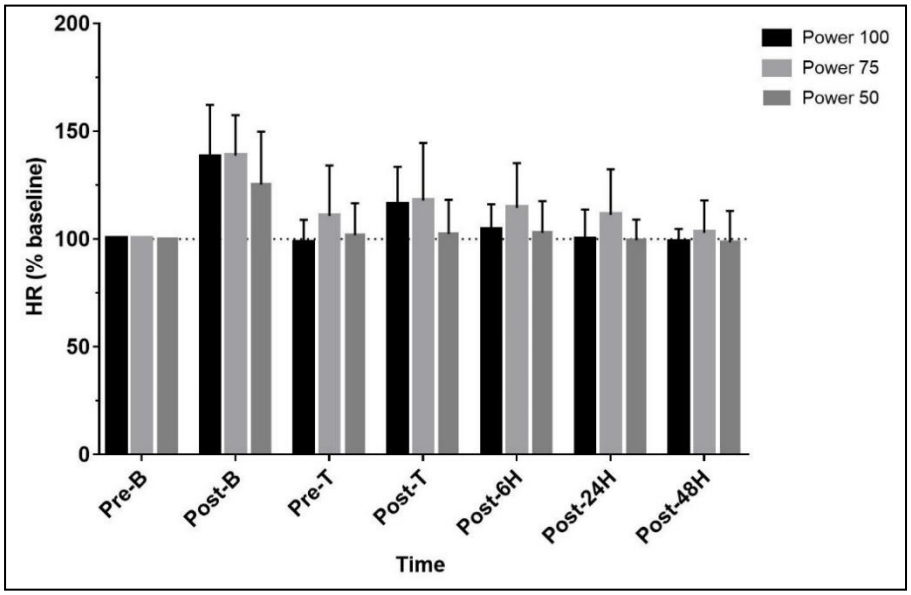


Figure 191. Comparison of P100, P75 and P50 on resting HR values (n = 11)

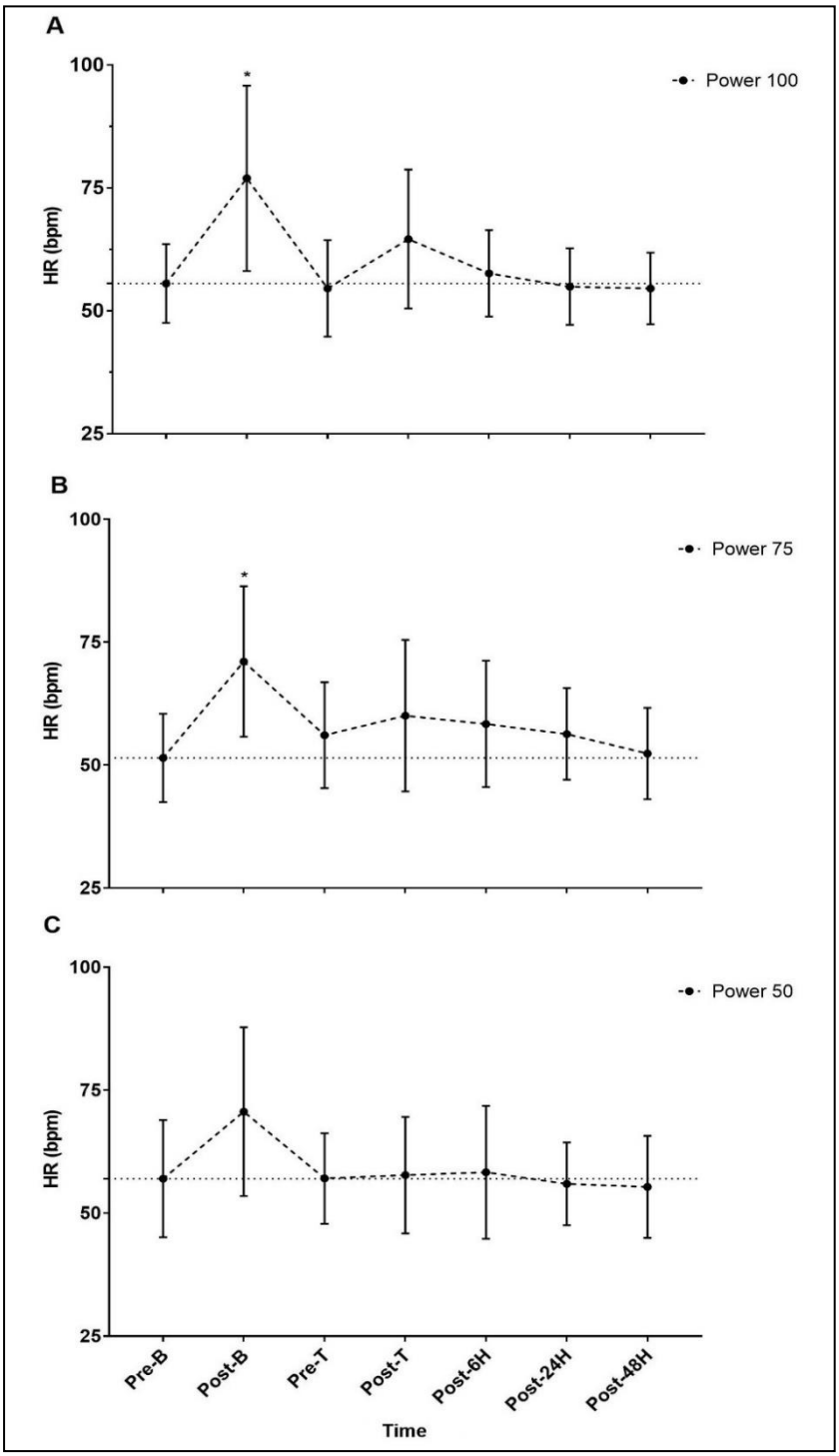


Figure 192. Changes in resting HR values in (A) P100, (B) P75 and (C) P50 protocols (n = 11). * Significant time difference compared to Pre-B ($p \leq 0.05$) from post-hoc Bonferroni analysis.

13.2.1.2. Central fatigue

13.2.1.2.1. Strength 100 versus Power 100 training.

Ratio of MVC force to tetanic force

There was no overall treatment effect on MVC/Tetanic force ratio (p = 0.614). However, there was an overall time effect trend on MVC/Tetanic force ratio (p = 0.064). No significant group x time interaction for MVC/Tetanic force ratio was observed (p = 0.614; Figure 193).

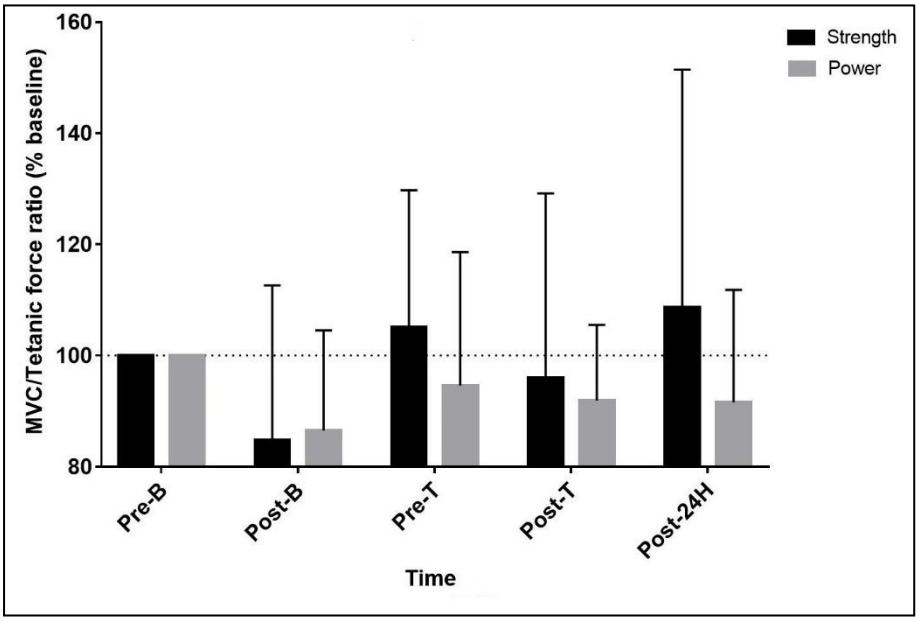


Figure 193. Comparison between S100 and P100 on MVC/Tetanic force ratio values (n = 9)

Simple main effects over time revealed that MVC/Tetanic force ratio did not significantly differ between time points in S100 (P = 0.192) and P100 (P = 0.255) trial. In S100, no significant time differences were observed at Post-B (P = 0.936, ES = -0.88), Pre-T (P = 1.000, ES = 0.03), Post-T (P = 1.000, ES = -0.40), and Post-24H (P = 1.000, ES = 0.12), compared to Pre-B value. In P100, no significant time differences were shown at Post-B (P = 0.506, ES = -0.51), Pre-T (p = 1.000, ES = 0.15), Post-T (p = 1.000, ES = -0.05), and Post-24H (p = 1.000, ES = -0.09), compared to Pre-B. These

results revealed that MVC/Tetanic force ratio decreased (no significant difference) following the M-Beast protocol and ARE protocols for both training modalities, and it gradually returned to Pre-B. ES results also showed that, MVC/Tetanic force ratio of S100 recovered at Post-24H, whereas P100's level did not yet recover at Post-24H (Figure 194).

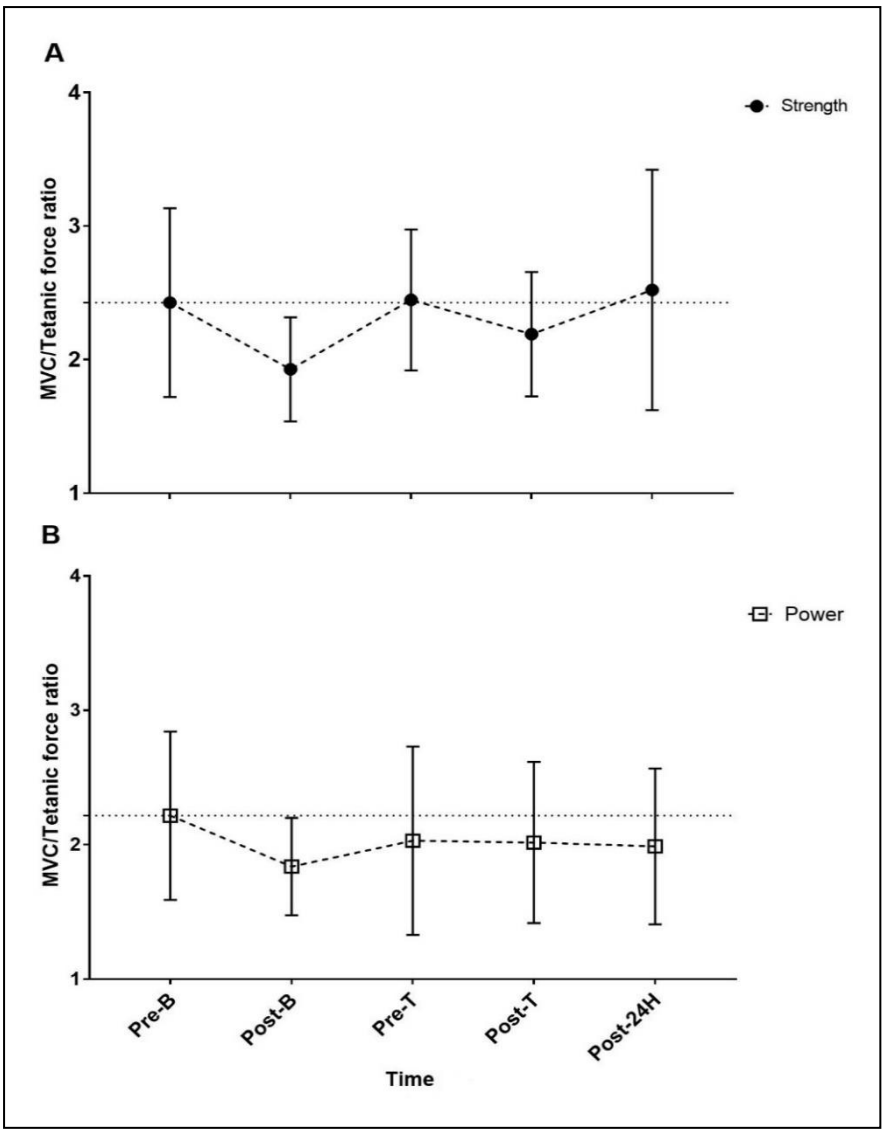


Figure 194. Changes in mean MVC/Tetanic force ratio values in (A) S100 and (B) P100 protocols (n = 09).

13.2.1.2.2. Strength 100 versus Strength 75 versus Strength 50 training

Ratio of MVC force to tetanic force

There was neither an overall treatment effect (p = 0.601) nor an overall time effect (p = 0.124) on MVC/Tetanic force ratio. No significant group x time interaction for MVC/Tetanic force ratio was observed (p = 0.090; Figure 195). These results showed that S100, S75 and S50 trials did not significantly effect the MVC/Tetanic force ratio.

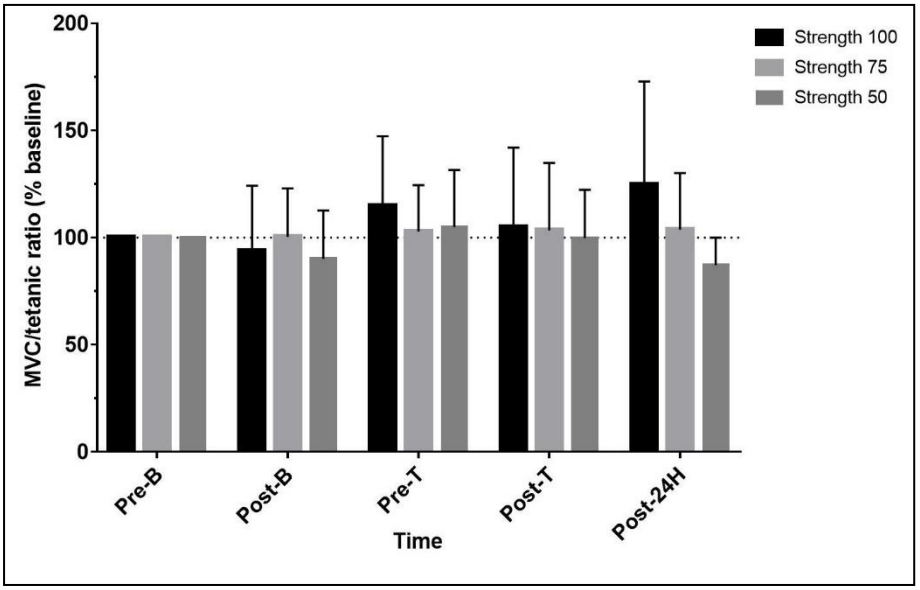


Figure 195. Comparison of S100, S75 and S50 on MVC/tetanic ratio values (n = 11)

13.2.1.2.3. Power 100 versus Power 75 versus Power 50 training

Ratio of MVC force to tetanic force

There was an overall treatment effect (p = 0.052) and an overall time effect (p = 0.045) on MVC/tetanic ratio. However, there was no significant treatment x time interaction for MVC/tetanic ratio (p = 0.460). Simple main effects for treatment showed that MVC/tetanic ratio was significantly different between treatments

(P100 vs P75 vs P50) at Post-24H (p = 0.025, (P100 vs P75: p = 0.142 ; P100 vs P50: p = 1.000)) (Figure 196).

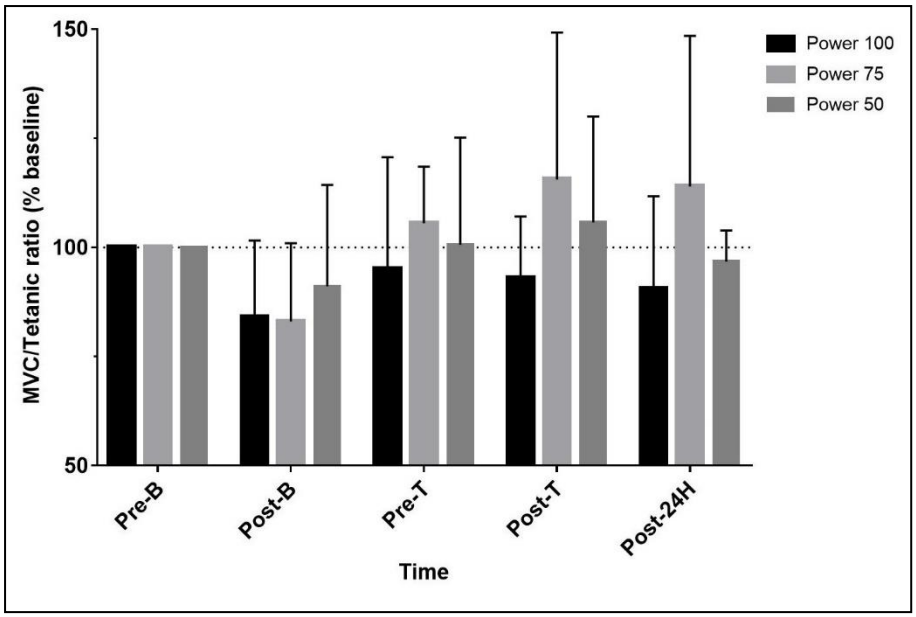


Figure 196. Comparison of P100, P75 and P50 on MVC/tetanic ratio values (n = 08)

Simple main effects over time revealed that MVC/tetanic ratio not different significantly between time points in P100 (P = 0.304) and P50 (P = 0.457), except in S75 (P = 0.037). In P100, no significant time differences were observed at all the time points (Post-B (p = 0.418, ES = -0.86), Pre-T (p = 1.000, ES = -0.26), Post-T (p = 1.000, ES = -0.27) and Post-24H (p = 1.000, ES = -0.41)) compared to Pre-B. Similarly, P75 also showed no significant time differences at all the time points (Post-B (p = 0.312, ES = -0.74), Pre-T (p = 1.000, ES = 0.17), Post-T (p = 1.000, ES = 0.41) and Post-24H (p = 1.000, ES = 0.34)) compared to Pre-B. In the same way, P50 also showed no significant time differences at all the time points (Post-B (p = 1.000, ES = -0.48), Pre-T (p = 1.000, ES = -0.01), Post-T (p = 1.000, ES = 0.26) and Post-24H (p = 1.000, ES = -0.25) compared to Pre-B. These results show that MVC/tetanic ratio decreased following the M-Beast protocol in all 3 training loads and ARE protocols of P100 training load. According to the ES results, MVC/tetanic ratio of P75 remained

recovered from Pre-T, whereas P100's and P50's level did not yet recover at Post-24H. (Figure 197).

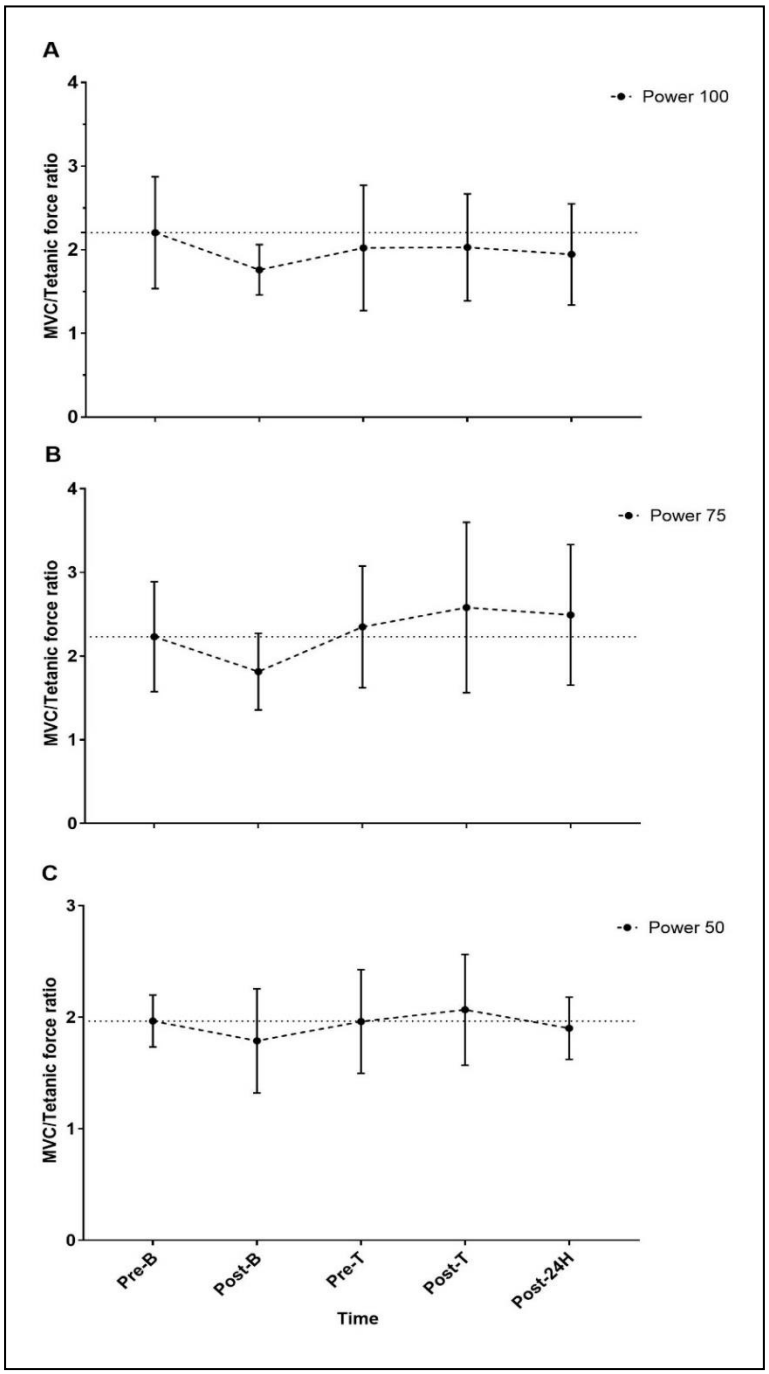


Figure 197. Changes in MVC/tetanic ratio values in (A) P100, (B) P75 and (C) P50 protocols (n = 08).

13.2.2. Effect size results

13.2.2.1. Heart rate variability parameters

Table 13. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of HRV parameters

Trials	Post-B	Pre-T	Post-T	Post-6H	Post-24H	Post-48H
			pNN50			
S100 vs P100						
S100	-1.76	-0.63	-1.98	-1.47	-0.92	-0.80
P100	-1.55	-0.43	-1.14	-0.72	-0.37	0.03
S100 vs S75 vs S50						
S100	-1.98	-0.63	-1.79	-1.67	-0.86	-0.69
S75	-1.91	-0.51	-1.15	-0.97	-0.50	-0.02
S50	-1.26	-0.12	-0.40	-0.25	0.08	0.28
P100 vs P75 vs P50						
P100	-1.50	-0.39	-1.08	-0.72	-0.34	0.03
P75	-2.20	-0.66	-0.83	-0.78	-0.09	-0.44
P50	-2.07	-0.39	-0.89	-0.79	-0.15	-0.11
			SDNN			
S100 vs P100						
S100	-1.27	-0.57	-1.38	-1.09	-0.50	-0.21
P100	-1.16	-0.36	-0.83	-0.59	-0.23	-0.22
S100 vs S75 vs S50						
S100	-1.40	-0.62	-1.41	-1.13	-0.56	-0.27
S75	-1.30	-0.40	-0.72	-0.66	-0.31	-0.03
S50	-1.17	-0.32	-0.68	-0.54	-0.09	0.09
P100 vs P75 vs P50						
P100	-1.16	-0.36	-0.83	-0.59	-0.23	-0.22
P75	-1.33	-0.45	-0.82	-0.37	0.01	-0.08
P50	-1.04	-0.32	-0.18	-0.34	0.09	0.09
			Ln RMSSD			
S100 vs P100						
S100	-1.41	-0.50	-1.42	-1.27	-0.82	-0.41
P100	-1.60	-0.42	-1.06	-0.65	-0.20	-0.01
S100 vs S75 vs S50						
S100	-1.65	-0.54	-1.45	-1.39	-0.84	-0.42

S75	-1.62	-0.35	-0.99	-0.78	-0.30	0.06
S50	-1.27	-0.28	-0.57	-0.33	-0.02	0.15
P100 vs P75 vs P50						
P100	-1.60	-0.42	-1.06	-0.65	-0.20	-0.01
P75	-1.48	-0.48	-0.85	-0.50	-0.03	0.11
P50	-1.21	-0.30	-0.60	-0.38	0.07	0.15
LF(nu)						
S100 vs P100						
S100	0.64	-0.35	0.20	0.21	0.30	0.13
P100	1.08	0.82	0.89	0.88	0.99	0.62
S100 vs S75 vs S50						
S100	0.62	-0.30	0.17	0.32	0.32	0.29
S75	0.76	0.13	0.90	0.57	0.49	0.30
S50	0.56	-0.05	-0.55	-0.25	-0.24	-0.42
P100 vs P75 vs P50						
P100	1.08	0.82	0.89	0.88	0.99	0.62
P75	0.57	0.08	-0.27	-0.13	-0.22	-0.39
P50	0.84	0.43	0.97	0.62	0.09	-0.01
HF(nu)						
S100 vs P100						
S100	-0.37	0.53	-0.16	0.27	-0.04	0.02
P100	-1.17	-0.75	-0.97	-0.84	-1.01	-0.59
S100 vs S75 vs S50						
S100	-0.41	0.41	-0.13	-0.05	-0.16	-0.16
S75	-0.85	-0.16	-1.29	-0.95	-0.61	-0.18
S50	-0.63	0.16	0.38	0.10	0.02	0.35
P100 vs P75 vs P50						
P100	-1.17	-0.75	-0.97	-0.84	-1.01	-0.59
P75	-0.55	-0.43	0.16	-0.33	0.15	0.09
P50	-0.70	-0.14	-0.79	-0.46	0.03	0.09
LF/HF ratio						
S100 vs P100						
S100	0.64	-0.35	0.20	0.21	0.30	0.13
P100	1.08	0.82	0.89	0.88	0.99	0.62
S100 vs S75 vs S50						
S100	0.62	-0.30	0.17	0.32	0.32	0.29
S75	0.76	0.13	0.90	0.57	0.49	0.30
S50	0.56	-0.05	-0.55	-0.25	-0.24	-0.42

P100 vs P75 vs P50						
P100	1.08	0.82	0.89	0.88	0.99	0.62
P75	0.57	0.08	-0.27	-0.13	-0.22	-0.39
P50	0.84	0.43	0.97	0.62	0.09	-0.01
Total power						
S100 vs P100						
S100	-1.00	-0.47	-1.20	-0.86	-0.35	-0.15
P100	-0.90	-0.35	-0.65	-0.61	-0.10	-0.38
S100 vs S75 vs S50						
S100	-1.10	-0.58	-1.23	-0.93	-0.44	-0.26
S75	-1.05	-0.42	-0.68	-0.65	-0.30	-0.27
S50	-1.02	-0.53	-0.57	-0.60	-0.22	-0.07
P100 vs P75 vs P50						
P100	-0.90	-0.35	-0.65	-0.61	-0.10	-0.38
P75	-0.88	-0.37	-0.46	-0.28	-0.26	-0.38
P50	-0.96	-0.51	-0.38	-0.61	-0.06	-0.09
SampEn						
S100 vs P100						
S100	-1.12	-0.07	-0.08	-0.23	-0.45	-0.58
P100	-0.43	0.92	-0.21	0.39	0.42	0.11
S100 vs S75 vs S50						
S100	-0.96	-0.10	-0.03	-0.36	-0.13	-0.36
S75	-0.82	0.52	-0.12	0.11	0.22	0.07
S50	-0.60	0.81	0.70	0.59	0.45	0.15
P100 vs P75 vs P50						
P100	-0.58	0.63	-0.31	0.27	0.34	0.02
P75	-0.22	0.52	-0.26	0.05	0.34	-0.31
P50	-1.14	-0.14	-0.10	-0.48	-0.09	0.01
SD1						
S100 vs P100						
S100	-1.30	-0.42	-1.42	-1.14	-0.72	-0.33
P100	-1.35	-0.38	-0.94	-0.57	-0.21	-0.04
S100 vs S75 vs S50						
S100	-1.46	-0.48	-1.45	-1.25	-0.75	-0.35
S75	-1.41	-0.35	-0.99	-0.78	-0.32	0.03
S50	-1.25	-0.27	-0.58	-0.38	-0.03	0.15
P100 vs P75 vs P50						
P100	-1.35	-0.38	-0.94	-0.57	-0.21	-0.04

P75	-1.46	-0.41	-0.78	-0.43	-0.02	0.08
P50	-1.31	-0.35	-0.66	-0.47	0.04	0.13
			SD2			
S100 vs P100						
S100	-1.21	-0.66	-1.30	-1.04	-0.40	-0.14
P100	-1.03	-0.35	-0.74	-0.59	-0.25	-0.39
S100 vs S75 vs S50						
S100	-1.32	-0.69	-1.33	-1.02	-0.46	-0.21
S75	-1.19	-0.43	-0.54	-0.56	-0.29	-0.10
S50	-1.07	-0.33	-0.69	-0.60	-0.12	0.04
P100 vs P75 vs P50						
P100	-1.03	-0.35	-0.74	-0.59	-0.25	-0.39
P75	-1.14	-0.41	-0.77	-0.29	0.01	-0.16
P50	-0.83	-0.30	0.01	-0.25	0.13	0.06
			SD2/SD1			
S100 vs P100						
S100	1.20	-0.05	0.89	0.59	0.50	0.58
P100	1.50	0.48	0.98	0.48	0.01	-0.24
S100 vs S75 vs S50						
S100	1.42	0.03	0.88	0.86	0.54	0.52
S75	1.46	0.17	1.22	0.71	0.34	-0.14
S50	0.71	0.05	-0.10	-0.17	-0.06	-0.16
P100 vs P75 vs P50						
P100	1.50	0.48	0.98	0.48	0.01	-0.24
P75	1.01	0.20	0.34	0.31	-0.01	-0.51
P50	1.22	0.29	0.94	0.60	0.09	-0.21
			SD1/SD2			
S100 vs P100						
S100	-1.30	0.16	-1.04	-0.67	-0.33	-0.60
P100	-1.11	-0.10	-0.86	-0.52	-0.14	0.06
S100 vs S75 vs S50						
S100	-1.58	0.07	-1.08	-1.03	-0.38	-0.38
S75	-1.21	-0.13	-1.11	-0.67	-0.30	0.25
S50	-0.50	-0.12	-0.08	0.17	0.07	0.23
P100 vs P75 vs P50						
P100	-1.32	-0.41	-0.83	-0.36	0.10	0.42
P75	-1.02	-0.30	-0.14	-0.32	0.07	0.54
P50	-1.11	-0.10	-0.86	-0.52	-0.14	0.06

Stress Score index						
S100 vs P100						
S100	0.99	0.83	0.98	1.34	0.90	0.25
P100	1.26	0.27	0.85	0.60	0.24	0.14
S100 vs S75 vs S50						
S100	1.15	0.80	0.99	1.27	0.88	0.26
S75	1.15	0.29	0.52	0.56	0.10	0.01
S50	0.88	0.30	0.71	0.51	0.02	-0.11
P100 vs P75 vs P50						
P100	1.26	0.27	0.85	0.60	0.24	0.14
P75	1.15	0.47	0.98	0.48	0.12	0.44
P50	0.79	0.19	0.19	-0.01	-0.18	-0.15
Systolic blood pressure						
S100 vs P100						
S100	0.32	0.15	0.34	-	0.04	-
P100	0.37	0.28	0.29	-	-0.06	-
S100 vs S75 vs S50						
S100	0.33	0.10	0.30	-	0.04	-
S75	0.37	-0.08	0.23	-	0.15	-
S50	0.47	0.21	0.61	-	0.16	-
P100 vs P75 vs P50						
P100	0.27	-0.04	0.09	-	0.32	-
P75	0.29	-0.09	0.11	-	0.04	-
P50	0.37	0.28	0.29	-	-0.06	-
Diastolic blood pressure						
S100 vs P100						
S100	0.21	-0.15	-0.61	-	0.15	-
P100	0.42	0.09	0.22	-	0.03	-
S100 vs S75 vs S50						
S100	0.25	-0.19	-0.52	-	0.14	-
S75	0.18	-0.12	-0.25	-	-0.27	-
S50	0.42	-0.26	-0.05	-	0.05	-
P100 vs P75 vs P50						
P100	0.47	-0.03	0.21	-	0.25	-
P75	0.31	-0.13	-0.15	-	-0.29	-
P50	0.42	0.09	0.22	-	0.03	-
Resting HR						
S100 vs P100						

S100	0.93	0.11	0.71	0.50	0.13	0.37
P100	1.48	-0.11	0.79	0.25	-0.08	-0.13
S100 vs S75 vs S50						
S100	1.12	0.07	0.70	0.71	0.10	0.27
S75	1.61	0.34	0.76	0.32	0.17	-0.17
S50	1.07	-0.37	0.11	-0.06	-0.31	-0.32
P100 vs P75 vs P50						
P100	1.48	-0.11	0.79	0.25	-0.08	-0.13
P75	1.56	0.47	0.68	0.62	0.53	0.10
P50	0.92	0.01	0.06	0.10	-0.10	-0.15

Abbreviations: P = Power training modality; S = Strength training modality

Table 14. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of HRV parameters

Trials	Post-T	Post-6H	Post-24H	Post-48H
		pNN50		
S100 vs P100				
S100	-1.09	-0.71	-0.28	-0.15
P100	-0.72	-0.28	0.08	0.46
S100 vs S75 vs S50				
S100	-0.99	-0.87	-0.26	-0.07
S75	-0.57	-0.43	0.03	0.49
S50	-0.28	-0.13	0.20	0.42
P100 vs P75 vs P50				
P100	-0.69	-0.34	0.06	0.43
P75	-0.26	-0.15	0.59	0.17
P50	-0.46	-0.34	0.22	0.27
		SDNN		
S100 vs P100				
S100	-0.87	-0.57	-0.02	0.33
P100	-0.50	-0.24	0.12	0.19
S100 vs S75 vs S50				
S100	-0.90	-0.59	-0.04	0.34
S75	-0.36	-0.30	0.12	0.41
S50	-0.37	-0.20	0.24	0.42

P100 vs P75 vs P50				
P100	-0.50	-0.24	0.12	0.19
P75	-0.39	0.05	0.40	0.30
P50	0.08	0.01	0.43	0.44
Ln RMSSD				
S100 vs P100				
S100	-1.05	-0.80	-0.33	0.08
P100	-0.67	-0.24	0.24	0.43
S100 vs S75 vs S50				
S100	-1.04	-0.88	-0.31	0.11
S75	-0.63	-0.43	0.06	0.42
S50	-0.30	-0.04	0.26	0.43
P100 vs P75 vs P50				
P100	-0.67	-0.24	0.24	0.43
P75	-0.41	-0.03	0.45	0.59
P50	-0.31	-0.07	0.38	0.46
LF(nu)				
S100 vs P100				
S100	0.61	0.14	0.42	0.44
P100	0.15	0.02	0.15	-0.22
S100 vs S75 vs S50				
S100	0.51	0.38	0.49	0.51
S75	1.00	0.69	0.42	0.04
S50	-0.22	0.07	0.16	-0.20
P100 vs P75 vs P50				
P100	0.15	0.02	0.15	-0.22
P75	-0.64	-0.18	-0.69	-0.66
P50	0.52	0.26	-0.16	-0.21
HF(nu)				
S100 vs P100				
S100	-0.61	-0.14	-0.42	-0.44
P100	-0.15	-0.02	-0.15	0.21
S100 vs S75 vs S50				
S100	-0.50	-0.39	-0.50	-0.51
S75	-1.00	-0.69	-0.42	-0.04
S50	0.21	-0.08	-0.16	0.20
P100 vs P75 vs P50				
P100	-0.15	-0.02	-0.15	0.21

P75	0.64	0.16	0.69	0.66
P50	-0.52	-0.26	0.16	0.21
		LF/HF ratio		
S100 vs P100				
S100	0.56	0.41	0.62	0.45
P100	0.06	-0.15	-0.08	-0.38
S100 vs S75 vs S50				
S100	0.49	0.50	0.61	0.57
S75	0.78	0.43	0.36	0.18
S50	-0.37	-0.15	-0.14	-0.29
P100 vs P75 vs P50				
P100	0.06	-0.15	-0.08	-0.38
P75	-0.44	-0.30	-0.35	-0.63
P50	0.26	0.10	-0.35	-0.42
		Total power		
S100 vs P100				
S100	-0.94	-0.49	0.04	0.32
P100	-0.36	-0.30	0.20	-0.01
S100 vs S75 vs S50				
S100	-0.93	-0.48	0.06	0.35
S75	-0.36	-0.32	0.14	0.23
S50	-0.09	-0.06	0.30	0.43
P100 vs P75 vs P50				
P100	-0.36	-0.30	0.20	-0.01
P75	-0.16	0.07	0.19	0.00
P50	0.08	-0.06	0.43	0.47
		SampEn		
S100 vs P100				
S100	-0.02	-0.19	-0.44	-0.60
P100	-1.13	-0.56	-0.54	-0.87
S100 vs S75 vs S50				
S100	0.08	-0.39	-0.06	-0.39
S75	-0.72	-0.41	-0.26	-0.43
S50	-0.21	-0.27	-0.38	-0.71
P100 vs P75 vs P50				
P100	-0.91	-0.40	-0.35	-0.64
P75	-0.75	-0.50	-0.14	-0.78
P50	0.02	-0.29	0.05	0.14

		SD1		
S100 vs P100				
S100	-1.03	-0.73	-0.29	0.08
P100	-0.57	-0.20	0.18	0.35
S100 vs S75 vs S50				
S100	-1.04	-0.82	-0.28	0.12
S75	-0.67	-0.44	0.04	0.40
S50	-0.31	-0.09	0.25	0.42
P100 vs P75 vs P50				
P100	-0.57	-0.20	0.18	0.35
P75	-0.35	-0.02	0.39	0.50
P50	-0.33	-0.11	0.41	0.50
		SD2		
S100 vs P100				
S100	-0.72	-0.45	0.08	0.47
P100	-0.45	-0.27	0.07	0.01
S100 vs S75 vs S50				
S100	-0.75	-0.42	0.07	0.46
S75	-0.16	-0.19	0.19	0.35
S50	-0.38	-0.26	0.23	0.37
P100 vs P75 vs P50				
P100	-0.45	-0.27	0.07	0.01
P75	-0.42	0.08	0.34	0.16
P50	0.22	0.10	0.44	0.39
		SD2/SD1		
S100 vs P100				
S100	0.90	0.60	0.52	0.59
P100	0.65	0.06	-0.40	-0.61
S100 vs S75 vs S50				
S100	0.85	0.81	0.50	0.47
S75	0.94	0.46	0.14	-0.29
S50	-0.17	-0.23	-0.11	-0.22
P100 vs P75 vs P50				
P100	0.65	0.06	-0.40	-0.61
P75	0.19	0.11	-0.20	-0.74
P50	0.58	0.23	-0.22	-0.46
		SD1/SD2		
S100 vs P100				

S100	-0.96	-0.66	-0.41	-0.61
P100	-0.63	-0.35	-0.02	0.16
S100 vs S75 vs S50				
S100	-0.94	-0.88	-0.39	-0.38
S75	-0.98	-0.53	-0.17	0.35
S50	0.06	0.30	0.19	0.35
P100 vs P75 vs P50				
P100	-0.46	0.01	0.43	0.75
P75	0.09	-0.06	0.35	0.88
P50	-0.63	-0.35	-0.02	0.16
Stress Score index				
S100 vs P100				
S100	0.77	0.79	0.23	-0.54
P100	0.67	0.37	-0.04	-0.16
S100 vs S75 vs S50				
S100	0.76	0.75	0.22	-0.55
S75	0.28	0.32	-0.22	-0.30
S50	0.50	0.24	-0.29	-0.44
P100 vs P75 vs P50				
P100	0.67	0.37	-0.04	-0.16
P75	0.51	0.01	-0.37	-0.10
P50	0.02	-0.22	-0.39	-0.35
Systolic blood pressure				
S100 vs P100				
S100	0.24	-	-0.06	-
P100	0.05	-	-0.35	-
S100 vs S75 vs S50				
S100	0.24	-	-0.03	-
S75	0.27	-	0.19	-
S50	0.43	-	0.01	-
P100 vs P75 vs P50				
P100	0.12	-	0.37	-
P75	0.16	-	0.11	-
P50	0.05	-	-0.35	-
Diastolic blood pressure				
S100 vs P100				
S100	-0.45	-	0.28	-
P100	0.13	-	-0.05	-

S100 vs S75 vs S50				
S100	-0.34	-	0.30	-
S75	-0.07	-	-0.11	-
S50	0.25	-	0.33	-
P100 vs P75 vs P50				
P100	0.23	-	0.28	-
P75	-0.01	-	-0.13	-
P50	0.13	-	-0.05	-
Resting HR				
S100 vs P100				
S100	0.68	0.45	0.02	0.30
P100	0.82	0.33	0.04	0.00
S100 vs S75 vs S50				
S100	0.66	0.68	0.03	0.22
S75	0.39	0.02	-0.14	-0.46
S50	0.52	0.35	0.06	0.08
P100 vs P75 vs P50				
P100	0.82	0.33	0.04	0.00
P75	0.30	0.19	0.02	-0.37
P50	0.06	0.11	-0.12	-0.18

Abbreviations: P = Power training modality; S = Strength training modality

13.2.2.2. Performance variables

Table 15. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of performance variables.

Trial	Post-B	Pre-T	Post-T	Post-24H
Bench Press relative peak power				
S100 vs P100				
S100	-0.26	-0.14	-0.34	-0.19
P100	-0.18	-0.05	-0.10	0.04
S100 vs S75 vs S50				
S100	-0.29	-0.17	-0.38	-0.22
S75	-0.24	-0.18	-0.26	-0.12
S50	-0.23	-0.07	-0.14	-0.03
P100 vs P75 vs P50				
P100	-0.18	-0.05	-0.10	0.04

P75	-0.24	-0.07	-0.14	0.03
P50	-0.31	-0.05	-0.12	0.01
Countermovement jump height				
S100 vs P100				
S100	-0.43	-0.30	-0.46	-0.24
P100	-0.31	-0.20	-0.21	-0.02
S100 vs S75 vs S50				
S100	-0.39	-0.24	-0.46	-0.23
S75	-0.32	-0.08	-0.26	-0.16
S50	-0.20	-0.09	-0.14	0.04
P100 vs P75 vs P50				
P100	-0.31	-0.20	-0.21	-0.02
P75	-0.20	-0.13	-0.16	0.13
P50	-0.27	-0.10	-0.23	0.03
Countermovement jumps relative peak power				
S100 vs P100				
S100	-0.93	-0.74	-0.97	-0.72
P100	-0.53	-0.34	-0.52	-0.07
S100 vs S75 vs S50				
S100	-0.87	-0.67	-0.96	-0.68
S75	-0.30	-0.18	-0.40	-0.32
S50	-0.36	-0.15	-0.30	0.03
P100 vs P75 vs P50				
P100	-0.53	-0.34	-0.52	-0.07
P75	-0.10	0.10	0.00	0.29
P50	-0.28	-0.09	-0.29	0.07

Abbreviations: P = Power training modality; S = Strength training modality

Table 16. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of performance variables

Trial	Post-T	Post-24H
Bench Press relative peak power		
S100 vs P100		
S100	-0.19	-0.05
P100	-0.05	0.09
S100 vs S75 vs S50		

S100	-0.21	-0.05
S75	-0.08	0.06
S50	-0.07	0.04
P100 vs P75 vs P50		
P100	-0.05	0.09
P75	-0.07	0.09
P50	-0.07	0.06
Countermovement jumps height		
S100 vs P100		
S100	-0.18	0.04
P100	-0.03	0.17
S100 vs S75 vs S50		
S100	-0.21	0.01
S75	-0.17	-0.08
S50	-0.05	0.13
P100 vs P75 vs P50		
P100	-0.03	0.17
P75	-0.03	0.26
P50	-0.12	0.13
Countermovement jumps relative peak power		
S100 vs P100		
S100	-0.21	0.04
P100	-0.19	0.26
S100 vs S75 vs S50		
S100	-0.26	0.01
S75	-0.23	-0.16
S50	-0.16	0.19
P100 vs P75 vs P50		
P100	-0.19	0.26
P75	-0.08	0.19
P50	-0.19	0.16

Abbreviations: P = Power training modality; S = Strength training modality

13.2.2.3. Neuromuscular fatigue

Table 17. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of neuromuscular fatigue indicators.

Trial	Post-B	Pre-T	Post-T	Post-24H
Maximal voluntary contractions peak force				
S100 vs P100				
S100	-0.33	-0.26	-0.76	-0.43
P100	-0.38	-0.27	-0.45	-0.26
S100 vs S75 vs S50				
S100	-0.35	-0.25	-0.66	-0.39
S75	-0.30	-0.10	-0.30	-0.22
S50	-0.24	-0.12	-0.23	-0.09
P100 vs P75 vs P50				
P100	-0.34	-0.25	-0.42	-0.29
P75	-0.40	-0.15	-0.16	0.13
P50	-0.34	-0.24	-0.23	0.03
Rate of force development (0–200 ms) in maximal voluntary contraction				
S100 vs P100				
S100	-0.19	-0.34	-0.48	-0.63
P100	-0.82	-0.67	-0.86	-0.17
S100 vs S75 vs S50				
S100	-0.51	-0.22	-0.72	-0.50
S75	-0.33	0.01	-0.23	-0.05
S50	-0.39	-0.26	-0.50	-0.06
P100 vs P75 vs P50				
P100	-0.58	-0.54	-0.74	-0.11
P75	-0.78	-0.41	-0.50	0.00
P50	-0.68	-0.20	-0.04	0.11

Abbreviations: P = Power training modality; S = Strength training modality

Table 18. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of neuromuscular fatigue indicators.

Trial	Post-T	Post-24H
Maximal voluntary contractions peak force		
S100 vs P100		
S100	-0.53	-0.20
P100	-0.20	-0.02
S100 vs S75 vs S50		
S100	-0.48	-0.16
S75	-0.20	-0.12
S50	-0.11	0.03
P100 vs P75 vs P50		
P100	-0.18	-0.07
P75	-0.02	0.29
P50	0.01	0.27
Rate of force development (0–200 ms) in maximal voluntary contraction		
S100 vs P100		
S100	-0.08	-0.26
P100	-0.23	0.47
S100 vs S75 vs S50		
S100	-0.37	-0.24
S75	-0.24	-0.06
S50	-0.27	0.26
P100 vs P75 vs P50		
P100	-0.24	0.41
P75	-0.09	0.38
P50	0.15	0.28

Abbreviations: P = Power training modality; S = Strength training modality

13.2.2.4. Central fatigue

Table 19. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of central fatigue indicators.

Trial	Post-B	Pre-T	Post-T	Post-24H
Central activation ratio				
S100 vs P100				
S100	-2.54	-2.31	-2.10	-2.12
P100	-2.55	-1.34	-2.07	-1.03
S100 vs S75 vs S50				
S100	-2.53	-1.81	-2.24	-2.34
S75	-4.18	-1.47	-1.93	-2.03
S50	-1.83	-1.72	-1.50	-0.85
P100 vs P75 vs P50				
P100	-2.37	-1.34	-0.93	-0.96
P75	-1.82	-1.17	-2.47	-0.56
P50	-1.71	-1.95	-1.88	-0.49
Ratio of MVC force to tetanic force				
S100 vs P100				
S100	-0.88	0.03	-0.40	0.12
P100	-0.51	0.15	-0.05	-0.09
S100 vs S75 vs S50				
S100	-0.53	0.30	-0.11	0.51
S75	-0.01	0.03	0.11	0.06
S50	-0.44	0.11	-0.07	-0.61
P100 vs P75 vs P50				
P100	-0.86	-0.26	-0.27	-0.41
P75	-0.74	0.17	0.41	0.34
P50	-0.48	-0.01	0.26	-0.25

Abbreviations: P = Power training modality; S = Strength training modality

Table 20. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of central fatigue indicators.

Trial	Post-T	Post-24H
Central activation ratio		
S100 vs P100		
S100	-0.63	-0.77
P100	-0.60	-0.05
S100 vs S75 vs S50		
S100	-0.33	0.31
S75	-0.82	-0.32
S50	0.19	0.31
P100 vs P75 vs P50		
P100	-0.60	0.02
P75	-0.36	0.41
P50	-0.29	1.31
Ratio of MVC force to tetanic force		
S100 vs P100		
S100	-0.52	0.10
P100	-0.02	-0.07
S100 vs S75 vs S50		
S100	-0.54	0.30
S75	0.09	0.03
S50	-0.17	-0.67
P100 vs P75 vs P50		
P100	0.01	-0.11
P75	0.26	0.18
P50	0.22	-0.16

Abbreviations: P = Power training modality; S = Strength training modality

13.2.2.5. Peripheral fatigue

Table 21. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of peripheral fatigue indicators.

Trial	Post-B	Pre-T	Post-T	Post-24H
Tetanic force				
S100 vs P100				
S100	-0.10	-0.23	-0.33	-0.25
P100	-0.29	0.02	-0.19	-0.06
S100 vs S75 vs S50				
S100	-0.24	-0.48	-0.48	-0.55
S75	-0.28	-0.19	-0.40	-0.32
S50	-0.10	-0.19	-0.21	0.19
P100 vs P75 vs P50				
P100	-0.26	0.00	-0.23	-0.08
P75	-0.30	-0.15	-0.52	-0.04
P50	-0.18	-0.21	-0.70	0.04
Maximum rate of force development on Tetanic contraction				
S100 vs P100				
S100	-0.23	-0.05	-0.24	-0.25
P100	-0.28	-0.14	-0.41	-0.51
S100 vs S75 vs S50				
S100	-0.16	-0.06	-0.22	-0.39
S75	-0.13	-0.08	-0.27	-0.07
S50	-0.63	-0.38	0.22	-0.24
P100 vs P75 vs P50				
P100	-0.20	-0.07	-0.31	-0.47
P75	-0.59	-0.22	-0.44	0.05
P50	-0.11	-0.37	-0.07	0.03
Maximum rate of force relaxation on Tetanic contraction				
S100 vs P100				
S100	0.27	0.18	0.27	0.06
P100	-0.39	-0.07	-0.24	-0.08
S100 vs S75 vs S50				
S100	0.17	0.20	0.17	0.07

S75	0.03	0.38	0.22	0.04
S50	0.11	0.21	-0.10	0.16
P100 vs P75 vs P50				
P100	-0.25	-0.04	-0.14	-0.02
P75	-0.07	0.55	0.26	-0.04
P50	-0.08	-0.05	0.20	0.01
Twitch force				
S100 vs P100				
S100	-0.16	-0.03	-0.31	-0.04
P100	-0.08	0.16	-0.10	0.08
S100 vs S75 vs S50				
S100	-0.25	-0.21	-0.40	-0.20
S75	-0.13	-0.03	-0.29	-0.17
S50	-0.29	-0.18	-0.06	-0.03
P100 vs P75 vs P50				
P100	-0.09	0.15	-0.13	0.03
P75	-0.35	-0.29	-0.34	0.07
P50	-0.29	-0.03	-0.14	0.00
$T_{1/2}$				
S100 vs P100				
S100	0.80	0.68	0.74	0.68
P100	0.14	0.17	0.32	0.31
S100 vs S75 vs S50				
S100	0.58	0.49	0.63	0.36
S75	0.41	-0.06	0.00	0.23
S50	0.82	0.25	0.48	0.15
P100 vs P75 vs P50				
P100	0.15	-0.04	0.21	0.30
P75	-0.30	0.14	0.35	0.62
P50	0.28	0.06	-0.36	-0.36
Twitch-to-tetanus ratios				
S100 vs P100				
S100	-0.33	0.16	-0.11	0.34
P100	0.12	0.21	0.04	0.07
S100 vs S75 vs S50				
S100	-0.22	0.22	0.02	0.45
S75	0.35	0.37	0.23	0.36
S50	-0.21	-0.07	0.19	-0.17

P100 vs P75 vs P50				
P100	0.06	0.22	0.06	0.03
P75	-0.17	0.05	-0.13	0.24
P50	-0.15	0.09	0.48	-0.13

Abbreviations: P = Power training modality; S = Strength training modality

Table 22. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of peripheral fatigue indicators.

Trial	Post-T	Post-24H
Tetanic force		
S100 vs P100		
S100	-0.12	-0.05
P100	-0.20	-0.08
S100 vs S75 vs S50		
S100	0.00	-0.14
S75	-0.19	-0.12
S50	-0.02	0.38
P100 vs P75 vs P50		
P100	-0.23	-0.08
P75	-0.38	0.08
P50	-0.48	0.25
Maximum rate of force development on Tetanic contraction		
S100 vs P100		
S100	-0.20	-0.21
P100	-0.37	-0.54
S100 vs S75 vs S50		
S100	-0.17	-0.34
S75	-0.20	0.00
S50	0.65	0.13
P100 vs P75 vs P50		
P100	-0.34	-0.60
P75	-0.30	0.27
P50	0.28	0.42

Maximum rate of force relaxation on Tetanic contraction		
S100 vs P100		
S100	0.10	-0.14
P100	-0.09	0.00
S100 vs S75 vs S50		
S100	-0.02	-0.13
S75	-0.16	-0.32
S50	-0.27	-0.03
P100 vs P75 vs P50		
P100	-0.04	0.02
P75	-0.21	-0.58
P50	0.22	0.06
Twitch force		
S100 vs P100		
S100	-0.25	-0.01
P100	-0.25	-0.09
S100 vs S75 vs S50		
S100	-0.16	0.00
S75	-0.27	-0.14
S50	0.13	0.15
P100 vs P75 vs P50		
P100	-0.26	-0.13
P75	-0.11	0.37
P50	-0.09	0.02
$T_{1/2}$		
S100 vs P100		
S100	0.40	-0.16
P100	0.22	0.21
S100 vs S75 vs S50		
S100	0.37	-0.18
S75	0.06	0.28
S50	0.20	-0.08
P100 vs P75 vs P50		
P100	0.27	0.39
P75	0.18	0.42
P50	-0.51	-0.53
Twitch-to-tetanus ratios		
S100 vs P100		

S100	-0.32	0.16
P100	-0.16	-0.16
S100 vs S75 vs S50		
S100	-0.22	0.19
S75	-0.13	-0.04
S50	0.27	-0.11
P100 vs P75 vs P50		
P100	-0.15	-0.23
P75	-0.20	0.22
P50	0.35	-0.20

Abbreviations: P = Power training modality; S = Strength training modality

13.2.2.6. Perceptual responses

Table 23. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-B value of perceptual responses.

Trials	Post-B	Pre-T	Post-T	Post-6H	Post-24H
			DOMS		
S100 vs P100					
S100	3.68	3.78	4.61	6.14	10.05
P100	4.63	4.12	3.74	5.32	5.35
S100 vs S75 vs S50					
S100	4.02	4.06	4.97	5.59	10.33
S75	4.23	3.95	8.51	6.66	9.14
S50	3.13	3.65	5.40	6.39	5.56
P100 vs P75 vs P50					
P100	4.63	4.12	3.74	5.32	5.35
P75	3.68	4.43	5.42	5.88	5.43
P50	5.25	5.94	5.61	7.36	5.91

Abbreviations: P = Power training modality; S = Strength training modality

Trials	Day 2	Day 2 6H	Day 3
		POMS	
S100 vs P100			
S100	-0.27	-0.38	-0.43
P100	0.27	0.39	-0.11
S100 vs S75 vs S50			
S100	-0.21	-0.24	-0.36
S75	0.54	0.38	0.36
S50	0.02	0.04	-0.45
P100 vs P75 vs P50			
P100	0.27	0.39	-0.11
P75	0.24	0.24	0.18
P50	0.13	0.32	-0.01

Abbreviations: P = Power training modality; S = Strength training modality

Table 24. Effect sizes (ES) between S100 vs P100, S100 vs S75 vs S50 and P100 vs P75 vs P50 for each time point in relation to the corresponding Pre-T value of perceptual responses.

Trials	Post-T	Post-6H	Post-24H
		DOMS	
S100 vs P100			
S100	1.17	2.50	4.16
P100	0.57	0.97	1.85
S100 vs S75 vs S50			
S100	1.26	2.27	4.25
S75	0.72	2.50	3.95
S50	0.32	1.12	1.89
P100 vs P75 vs P50			
P100	0.57	0.97	1.85
P75	0.43	1.12	2.02
P50	1.22	1.88	2.41

Abbreviations: P = Power training modality; S = Strength training modality

Trials	Day 2 6H	Day 3
	POMS	
S100 vs P100		
S100	-0.10	-0.16
P100	0.08	-0.47
S100 vs S75 vs S50		
S100	-0.01	-0.15
S75	-0.24	-0.22
S50	0.01	-0.53
P100 vs P75 vs P50		
P100	0.08	-0.47
P75	0.01	-0.04
P50	0.18	-0.16

Abbreviations: P = Power training modality; S = Strength training modality

13.2.3. Associations

13.2.3.1. Strength 100

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in CMJ RPP (Post-T, p = 0.024) marker in S100 trial. Correlation coefficients (r) related to S100 trial are presented in Table 25.

Table 25. Correlation coefficients in S100 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = 0.10	r = 0.20	r = 0.23	r = 0.29
Δ in CMJ height	r = 0.28	r = -0.42	r = 0.14	r = 0.24
Δ in CMJ RPP	r = 0.40	r = -0.17	r = 0.62*	r = 0.39
Neuromuscular fatigue				
Δ in MVC peak force	r = 0.25	r = -0.16	r = 0.16	r = 0.12
Δ in RFD^{200MVC}	r = 0.34	r = -0.02	r = -0.22	r = -0.28

Central fatigue				
Δ in CAR	r = 0.12	r = 0.62	r = 0.50	r = 0.06
Δ in MVC/Tet ratio	r = -0.01	r = 0.43	r = 0.35	r = 0.29
Peripheral fatigue				
Δ in tet force	r = 0.01	r = -0.18	r = -0.18	r = -0.17
Δ in RFD^{tet}	r = -0.43	r = 0.29	r = -0.32	r = -0.28
Δ in RFR^{tet}	r = 0.37	r = 0.01	r = 0.15	r = 0.34
Δ in twitch force	r = -0.04	r = 0.32	r = 0.20	r = 0.35
Δ in $T_{1/2}$	r = 0.01	r = -0.17	r = 0.18	r = 0.05
Δ in twitch/tet ratio	r = 0.18	r = 0.37	r = 0.48	r = 0.50
Perceptual markers				
Δ in DOMS	r = 0.01	r = -0.19	r = 0.15	r = 0.35

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.3.2. Strength 75

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in CMJ RPP (Post-T, p = 0.035), MVC/Tet ratio (Pre-T, p = 0.013 and Post-T, p = 0.047) and Tetanic force (Post-T, 0.011) markers in S75 trial. Correlation coefficients (r) related to S75 trial are presented in Table 26.

Table 26. Correlation coefficients in S75 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = -0.37	r = -0.17	r = -0.15	r = -0.42
Δ in CMJ height	r = -0.04	r = 0.15	r = 0.40	r = 0.15
Δ in CMJ RPP	r = 0.12	r = 0.40	r = 0.59*	r = -0.08

Neuromuscular fatigue				
Δ in MVC peak force	r = 0.60	r = 0.40	r = 0.08	r = 0.44
Δ in RFD^{200MVC}	r = 0.38	r = 0.27	r = -0.11	r = 0.37
Central fatigue				
Δ in CAR	r = 0.09	r = 0.00	r = 0.31	r = -0.42
Δ in MVC/Tet ratio	r = 0.16	r = 0.72*	r = 0.61*	r = 0.09
Peripheral fatigue				
Δ in tet force	r = -0.28	r = -0.51	r = -0.73*	r = 0.20
Δ in RFD^{tet}	r = -0.23	r = -0.08	r = -0.36	r = -0.16
Δ in RFR^{tet}	r = 0.26	r = -0.19	r = 0.47	r = -0.24
Δ in twitch force	r = -0.28	r = -0.46	r = -0.40	r = 0.36
Δ in $T_{1/2}$	r = 0.34	r = -0.56	r = -0.28	r = -0.44
Δ in twitch/tet ratio	r = -0.06	r = 0.24	r = 0.47	r = 0.03
Perceptual markers				
Δ in DOMS	r = 0.12	r = -0.12	r = -0.15	r = 0.07

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.3.3. Strength 50

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in CMJ height (Pre-T, p = 0.011 and Post-T, p = 0.46), and RFD^{tet} (Post-24H, p = 0.015) markers in S50 trial. Correlation coefficients (r) related to S50 trial are presented in Table 27.

Table 27. Correlation coefficients in S50 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = -0.09	r = 0.41	r = -0.05	r = -0.14
Δ in CMJ height	r = 0.18	r = -0.68*	r = -0.56*	r = -0.18
Δ in CMJ RPP	r = 0.15	r = 0.37	r = 0.34	r = -0.09
Neuromuscular fatigue				
Δ in MVC peak force	r = 0.00	r = -0.18	r = 0.01	r = -0.04
Δ in RFD^{200MVC}	r = 0.01	r = -0.23	r = -0.53	r = -0.16
Central fatigue				
Δ in CAR	r = 0.32	r = -0.46	r = 0.04	r = -0.19
Δ in MVC/Tet ratio	r = 0.12	r = -0.25	r = 0.29	r = -0.15
Peripheral fatigue				
Δ in tet force	r = -0.10	r = 0.40	r = -0.36	r = 0.48
Δ in RFD^{tet}	r = 0.14	r = 0.40	r = 0.01	r = 0.71*
Δ in RFR^{tet}	r = 0.26	r = 0.32	r = 0.31	r = -0.43
Δ in twitch force	r = -0.27	r = 0.06	r = -0.15	r = 0.36
Δ in $T_{1/2}$	r = 0.37	r = 0.07	r = -0.53	r = -0.56
Δ in twitch/tet ratio	r = -0.41	r = -0.02	r = 0.00	r = 0.29
Perceptual markers				
Δ in DOMS	r = -0.13	r = -0.47	r = -0.29	r = -0.26

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.3.4. Power 100

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in DOMS (Post-T, p = 0.050) marker in P100 trial. Correlation coefficients (r) related to P100 trial are presented in Table 28.

Table 28. Correlation coefficients in P100 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = -0.37	r = -0.06	r = 0.45	r = -0.11
Δ in CMJ height	r = 0.11	r = 0.11	r = 0.22	r = -0.47
Δ in CMJ RPP	r = -0.18	r = 0.12	r = -0.03	r = -0.30
Neuromuscular fatigue				
Δ in MVC peak force	r = 0.30	r = 0.54	r = -0.30	r = 0.22
Δ in RFD^{200MVC}	r = -0.14	r = 0.41	r = -0.03	r = 0.03
Central fatigue				
Δ in CAR	r = 0.44	r = 0.19	r = -0.39	r = -0.25
Δ in MVC/Tet ratio	r = 0.10	r = 0.01	r = -0.47	r = 0.22
Peripheral fatigue				
Δ in tet force	r = -0.46	r = -0.20	r = -0.25	r = 0.02
Δ in RFD^{tet}	r = -0.09	r = -0.03	r = -0.48	r = -0.04
Δ in RFR^{tet}	r = 0.58	r = 0.37	r = 0.46	r = -0.52
Δ in twitch force	r = -0.16	r = -0.51	r = -0.04	r = 0.21
Δ in $T_{1/2}$	r = 0.16	r = 0.14	r = -0.46	r = -0.28
Δ in twitch/tet ratio	r = 0.45	r = 0.03	r = 0.05	r = 0.08
Perceptual markers				
Δ in DOMS	r = 0.28	r = 0.46	r = 0.60*	r = -0.19

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.3.5. Power 75

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in CMJ RPP (Post-B, p = 0.036) and DOMS (Pre-T, p = 0.022) marker in P75 trial. Correlation coefficients (r) related to P75 trial presented in Table 29.

Table 29. Correlation coefficients in P75 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = -0.23	r = 0.25	r = -0.14	r = -0.10
Δ in CMJ height	r = -0.42	r = 0.34	r = 0.37	r = 0.01
Δ in CMJ RPP	r = -0.63*	r = 0.00	r = 0.02	r = 0.45
Neuromuscular fatigue				
Δ in MVC peak force	r = -0.17	r = 0.38	r = -0.41	r = 0.31
Δ in RFD^{200MVC}	r = -0.02	r = -0.21	r = 0.57	r = 0.12
Central fatigue				
Δ in CAR	r = 0.05	r = 0.12	r = 0.14	r = 0.02
Δ in MVC/Tet ratio	r = 0.19	r = 0.19	r = -0.62	r = 0.19
Peripheral fatigue				
Δ in tet force	r = -0.43	r = 0.33	r = 0.62	r = 0.07
Δ in RFD^{tet}	r = 0.41	r = -0.10	r = 0.21	r = 0.10
Δ in RFR^{tet}	r = 0.69	r = -0.19	r = -0.29	r = -0.14
Δ in twitch force	r = -0.38	r = -0.02	r = 0.67	r = 0.19
Δ in $T_{1/2}$	r = 0.46	r = -0.17	r = 0.23	r = 0.29
Δ in twitch/tet ratio	r = -0.14	r = 0.19	r = -0.33	r = -0.10
Perceptual markers				
Δ in DOMS	r = 0.18	r = 0.68*	r = 0.13	r = 0.01

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.3.6. Power 50

No significant associations were observed between changes in variables with the changes in Ln RMSSD, except for changes in CMJ height (Post-B, p = 0.004), RFD^{200MVC} (Post-T, p = 0.037) and CAR (Post-24H, p = 0.021) markers in P50 trial. Correlation coefficients (r) related to P50 trail presented in Table 30.

Table 30. Correlation coefficients in P50 trial between the changes in the Ln RMSSD and other objective and subjective markers relative to Pre-B values.

Marker	Δ in Ln RMSSD			
	Post-B	Pre-T	Post-T	Post-24H
Performance markers				
Δ in BP RPP	r = -0.22	r = -0.14	r = -0.25	r = 0.09
Δ in CMJ height	r = 0.78*	r = 0.09	r = 0.08	r = -0.17
Δ in CMJ RPP	r = 0.25	r = -0.36	r = -0.12	r = -0.53
Neuromuscular fatigue				
Δ in MVC peak force	r = 0.36	r = 0.55	r = -0.10	r = 0.55
Δ in RFD^{200MVC}	r = 0.36	r = -0.46	r = -0.74*	r = -0.41
Central fatigue				
Δ in CAR	r = 0.26	r = 0.58	r = 0.38	r = 0.79*
Δ in MVC/Tet ratio	r = 0.52	r = -0.20	r = 0.19	r = 0.02
Peripheral fatigue				
Δ in tet force	r = -0.48	r = 0.27	r = -0.26	r = 0.41
Δ in RFD^{tet}	r = -0.38	r = 0.23	r = 0.00	r = 0.24
Δ in RFR^{tet}	r = 0.50	r = -0.54	r = -0.07	r = -0.29
Δ in twitch force	r = 0.05	r = -0.06	r = -0.43	r = -0.21
Δ in $T_{1/2}$	r = 0.04	r = 0.13	r = 0.55	r = 0.29
Δ in twitch/tet ratio	r = 0.36	r = -0.29	r = -0.17	r = -0.10
Perceptual markers				
Δ in DOMS	r = 0.04	r = -0.60	r = -0.42	r = 0.03

Abbreviations: BP = Bench press; CAR = Central activation ratio; CMJ = Countermovement jump; DOMS = Delayed on-set muscle soreness; Ln RMSSD = natural logarithm of the root mean square of successive differences; MVC = maximal voluntary isometric contraction; RFD = Rate of force development; RFR = Rate of force relaxation; RPP = Relative peak power; $T_{1/2}$ = half-time of force relaxation; Tet = Tetanic; Δ = Changes; r = Correlation coefficient value; * = significant association.

13.2.4. Informed consent form

CONSENTIMIENTO INFORMADO / INFORMED CONSENT

Yo, ..., con DNI/NIE:.....

DECLARO:

Haber sido informado/a del estudio y procedimientos de la investigación del Proyecto titulado: Identifying the optimal training load for adequate recovery based on heart rate variability response following an acute bout of hypertrophy and power resistance training. *(Identificación de la carga de entrenamiento óptima para una recuperación adecuada basada en la respuesta de variabilidad de la frecuencia cardiaca después de una sesión de Fuerza maxima y entrenamiento de resistencia de potencia.)*

Los investigadores que van a acceder a mis datos personales y a los resultados de las pruebas son: Marasingha Arachchige Sajith Udayanga, Dr. Pedro Alcaraz Ramón, Dra. Linda Chung, Dra. Elena Marín Cascales, Dr. Cristian Marín Pagán, Francisco Javier Martínez Noguera, Jorge Carlos Vivas, Tomás Trindade de Freitas, Andrés Pérez Hernández, Laura Campoy de Haro y Dr. Salvador Monreal Sánchez.

Asimismo, he podido hacer preguntas del estudio, comprendiendo que me presto de forma voluntaria al mismo y que en cualquier momento puedo abandonarlo sin que me suponga perjuicio de ningún tipo.

CONSIENTO:

1.-) Someterme a las siguientes pruebas exploratorias (en su caso): Anexo V.

2.-) El uso de los datos obtenidos según lo indicado en el párrafo siguiente:

En cumplimiento del Reglamento (UE) 2016/679 del Parlamento Europeo y del Consejo, de 27 de abril de 2016, Real Decreto-Ley 5/2018, de 27 de julio y Ley Orgánica 15/1999, de 13 de diciembre, de Protección de Datos de Carácter Personal, le comunicamos que la información que ha facilitado y la obtenida como consecuencia de las exploraciones a las que se va a someter pasará a formar parte del fichero automatizado INVESOCIAL, cuyo titular es la FUNDACIÓN UNIVERSITARIA SAN ANTONIO, con la finalidad de INVESTIGACIÓN Y DOCENCIA EN LAS ÁREAS DE CONOCIMIENTO CIENCIAS SOCIALES, JURÍDICAS, DE LA EMPRESA Y DE LA COMUNICACIÓN. Tiene derecho a acceder a esta información y cancelarla o rectificarla, dirigiéndose al domicilio de la entidad, en Avda. de los Jerónimos de Guadalupe 30107 (Murcia). Esta entidad le garantiza la adopción de las medidas oportunas para asegurar el tratamiento confidencial de dichos datos.

En Guadalupe (Murcia) a ..... de ..... de 20

El investigador,

Fdo:..... Fdo:.....

13.2.5. Physical activity readiness questionnaire

Physical Activity Readiness Questionnaire

Name: ……..

Please answer to all the questions.

No	Question	Yes	No
01.	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?	☐	☐
02.	Do you feel pain in your chest when you do physical activity?	☐	☐
03.	In the past month, have you had chest pain when you were not doing physical activity?	☐	☐
04.	Do you lose your balance because of dizziness or do you ever lose consciousness?	☐	☐
05.	Do you have a bone or joint problem that could be made worse by a change in your physical activity?	☐	☐
06.	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?	☐	☐
07.	Do you know of any other reason why you should not do physical activity?	☐	☐

……

Signature

Thank you.

13.2.6. Delayed on set muscle soreness

Delayed onset muscle soreness (DOMS)

Name: …….. No: ……. Group: …….

Training method: Strength / Power **Training load: 100% / 75% / 50%**

Testing session : Pre Beast protocol / Post Beast protocol / Pre training / Post training / Post 6h / Post 24h / Post 48h

Please mark your pain level

No pain Unbearable pain

Thank you.

13.2.7. BORG scale of perceived exertion

Identifying the optimal training load for adequate recovery based on heart rate variability response following an acute bout of maximal strength and power resistance training

Borg Scale of Perceived Exertion

Name: …….. **No: …….**

Training method: Strength / Power **Training load: 100% / 75% / 50%**

Testing session : Post Beast protocol / Post training

How was your workout? (Please circle your rating)

Rating	Description
0	NOTHING AT ALL
0.5	VERY, VERY LIGHT
1	VERY LIGHT
2	FAIRLY LIGHT
3	MODERATE
4	SOMEWHAT HARD
5	HARD
6	
7	VERY HARD
8	
9	
10	VERY VERY HARD (MAXIMAL)

Thank you.

13.2.8. Profile of mood states questionnaire

Profile of mood states questionnaire

Name: …….. **No: …….**

Training method: Strength / Power **Training load: 100% / 75% / 50%**

Testing session : Pre Beast protocol / Post Beast protocol / Pre training / Post training / Post 6h / Post 24h

Please **CIRCLE THE NUMBER THAT BEST DESCRIBES HOW YOU FEEL RIGHT NOW.**

Item	Not At All	A Little	Moderately	Quite a lot	Extremely
Tense	0	1	2	3	4
Angry	0	1	2	3	4
Worn Out	0	1	2	3	4
Unhappy	0	1	2	3	4
Proud	0	1	2	3	4
Lively	0	1	2	3	4
Confused	0	1	2	3	4
Sad	0	1	2	3	4
Active	0	1	2	3	4
On-edge	0	1	2	3	4
Grouchy	0	1	2	3	4
Ashamed	0	1	2	3	4
Energetic	0	1	2	3	4
Hopeless	0	1	2	3	4
Uneasy	0	1	2	3	4
Restless	0	1	2	3	4
Unable to concentrate	0	1	2	3	4
Fatigued	0	1	2	3	4
Competent	0	1	2	3	4
Annoyed	0	1	2	3	4
Discouraged	0	1	2	3	4
Resentful	0	1	2	3	4
Nervous	0	1	2	3	4
Miserable	0	1	2	3	4
Confident	0	1	2	3	4

PLEASE TURN TO THE NEXT PAGE

Profile of mood states questionnaire (Continued).

Item	Not At All	A Little	Moderately	Quite a lot	Extremely
Bitter	0	1	2	3	4
Exhausted	0	1	2	3	4
Anxious	0	1	2	3	4
Helpless	0	1	2	3	4
Weary	0	1	2	3	4
Satisfied	0	1	2	3	4
Bewildered	0	1	2	3	4
Furious	0	1	2	3	4
Full of Pep	0	1	2	3	4
Worthless	0	1	2	3	4
Forgetful	0	1	2	3	4
Vigorous	0	1	2	3	4
Uncertain about things	0	1	2	3	4
Bushed	0	1	2	3	4
Embarrassed	0	1	2	3	4

THANK YOU FOR YOUR COOPERATION

PLEASE BE SURE YOU HAVE ANSWERED EVERY ITEM