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Development and Validation of a Novel Training Load Calculator for Resistance Training

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Abstract – English

This thesis developed a training load calculator for resistance training and examined its criterion validity against selected biomarker responses. The calculator estimates internal training load from observable session variables by integrating volume, intensity, exertion, and exercise selection. Intensity is derived primarily from velocity-based measures, with perceived exertion as a fallback; exertion is estimated from velocity loss or perceived proximity to failure. The model provides three domain-specific indices for low, moderate, and high repetition loading schemes, as well as Total Training Load, calculated as their mean. These domains represent a continuum from more mechanical to more metabolically demanding loading schemes.

Validity was examined in a standardized laboratory protocol. Trained athletes completed one testing visit to determine one-repetition maximum load and six supervised barbell sessions involving the back squat, bench press, and conventional deadlift. Sessions differed in repetition range, percentage of maximum load, and rest interval duration. Lactate and salivary cortisol were collected at baseline and 10 minutes after exercise, while creatine kinase was measured at baseline and 24 hours after exercise. Biomarker responses were analyzed as absolute and within-participant relative changes.

The high-repetition domain was strongly associated with relative lactate change ($r = 0.856$, $p < 0.0001$), supporting its link to acute metabolic stress. The moderate-repetition domain correlated strongly with a rank-based creatine kinase, lactate, and cortisol composite ($r = 0.673$, $p < 0.0001$). The low-repetition domain showed weak or inverse associations. Overall, the findings provide proof-of-concept support and indicate a need for further validation against markers of mechanical loading.

Abstract – German

Diese Masterarbeit entwickelte ein Modell zur Quantifizierung der Trainingsbelastung im Krafttraining und untersuchte dessen Kriteriumsvalidität anhand ausgewählter Biomarkerreaktionen. Das Modell schätzt die interne Trainingsbelastung aus beobachtbaren Trainingsvariablen, indem Volumen, Intensität, Anstrengungsgrad und Übungsauswahl integriert werden. Die Intensität wird primär über geschwindigkeitsbasierte Messgrößen abgeleitet, mit dem subjektiven Belastungsempfinden als Ersatzweg. Der Anstrengungsgrad wird über Geschwindigkeitsverlust oder die subjektiv eingeschätzte Nähe zum Muskelversagen bestimmt. Das Modell liefert drei domänenspezifische Indizes für niedrige, moderate und hohe Wiederholungsbereiche sowie die Total Training Load als Mittelwert dieser Domänen. Konzeptionell sollen diese Domänen ein Kontinuum von stärker mechanisch geprägten bis stärker metabolisch anspruchsvollen Belastungsformen abbilden.

Die Validität wurde in einem standardisierten Laborprotokoll geprüft. Trainierte Athletinnen und Athleten absolvierten eine Testung zur Bestimmung der maximal einmal bewegbaren Last und sechs betreute Langhanteltrainingseinheiten mit Kniebeuge, Bankdrücken und konventionellem Kreuzheben. Die Einheiten unterschieden sich hinsichtlich Wiederholungsbereich, prozentualer Belastung bezogen auf die maximal einmal bewegbare Last und Pausendauer. Laktat und Speichelcortisol wurden zu Beginn und 10 Minuten nach Belastungsende erhoben, Kreatinkinase zu Beginn und 24 Stunden nach Belastungsende. Biomarkerreaktionen wurden als absolute und innerhalb der Teilnehmenden relative Veränderungen analysiert.

Die Domäne hoher Wiederholungszahlen war stark mit der relativen Laktatveränderung assoziiert ($r = 0,856$, $p < 0,0001$), was ihre Verbindung zu akutem metabolischem Stress unterstützt. Die Domäne moderater Wiederholungszahlen korrelierte stark mit einem rangbasierten Kreatinkinase-, Laktat- und Cortisol-Komposit ($r = 0,673$, $p < 0,0001$). Die Domäne niedriger Wiederholungszahlen zeigte schwache oder inverse Zusammenhänge. Insgesamt liefern die Ergebnisse einen Proof of Concept und zeigen weiteren Validierungsbedarf gegenüber Markern mechanischer Belastung.

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Preface

This thesis was completed within the framework of the validation study for the AIROW project. I would like to express my sincere gratitude to Prof. Nikolaus Hautsch and the AIROW Project for funding the study and for the opportunities they have provided that enabled me to pursue this work. The work could not have been realized without their support. I also thank the AIROW team for their cooperation and support throughout the study.

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Artificial intelligence tools, specifically ChatGPT by OpenAI, were used during the preparation of this thesis for language refinement and assistance with Python coding. All AI-assisted outputs were carefully reviewed, revised where necessary, and critically assessed by the author. No original scientific content, interpretations, or conclusions were generated by artificial intelligence. The author takes full ownership of, and complete responsibility for, all content presented in this thesis.

I feel a strong sense of gratitude for being able to pursue this work. What once seemed like a distant goal became a reality through the support of the individuals and institutions acknowledged above. I hope that the present thesis can contribute, in a small but meaningful way, to sport science and to the broader goal of improving health and performance through evidence-based practice.

1. Introduction

The German aphorism commonly rendered in Latin as *sola dosis facit venenum*, “only the dose makes the poison”, is widely attributed to Paracelsus (1538) and remains a foundational maxim in toxicology (Stumpf, 2006). Its core implication is that effects are dose-dependent and, therefore, cannot be evaluated without reference to magnitude and context. While frequently discussed in pharmacology, this principle extends to many domains in which exposure is deliberately manipulated to produce adaptation.

Resistance training is one such domain, with clear applications in both sport and medicine. In competitive sport, accurately quantifying training load is essential to balance a sufficient stimulus for continued performance adaptations with the need to manage fatigue, reduce injury risk, and avoid nonfunctional overreaching or overtraining (Bourdon et al., 2017; Meeusen et al., 2013; Schwellnus et al., 2016; Soligard et al., 2016). In clinical and public health contexts, resistance training is recommended in the World Health Organization’s physical activity guidelines, and is an integral component of contemporary prevention, treatment, and rehabilitation approaches across a range of conditions (Bull et al., 2020; Pedersen & Saltin, 2015). When prescribed therapeutically, training load must be planned, measured, and monitored to ensure that it stays within intended limits while still providing an adequate stimulus for the desired adaptations (Garber et al., 2011; Paluch et al., 2024; Wang et al., 2017).

Taken together, these applications highlight a common requirement: resistance training can only be optimized, and its risks appropriately managed, when the “dose” is quantified in a meaningful and reproducible way (Bourdon et al., 2017; Garber et al., 2011; Schwellnus et al., 2016; Soligard et al., 2016). Despite the well-established health and performance benefits of resistance training, its quantification remains insufficiently standardized (Scott et al., 2016). This is partly because the training stimulus is shaped by multiple interacting factors, including the amount of work performed, the relative intensity, the proximity to failure, and the overall structure of the session (Bourdon et al., 2017; Impellizzeri et al., 2019; Refalo et al., 2023; Schoenfeld, Grgic, et al., 2017; Schoenfeld, Ogborn, et al., 2017).

Several practical approaches exist to describe the external work completed, such as counting repetitions and sets, or calculating volume-load and total work (Scott et al., 2016). These metrics primarily quantify external load, meaning the work completed, described

independently of the athlete's internal characteristics. In contrast, internal load describes the acute psychophysiological response to this external work, and is influenced by factors such as relative intensity, exertion, and exercise selection (Halson, 2014; Impellizzeri et al., 2019; Power et al., 2022; Zourdos et al., 2016).

In this thesis, training load is used as an overarching concept that links the applied resistance training dose to the stress it elicits in the athlete. This definition integrates both external and internal load. External load refers to the work performed and can be described using practical metrics such as repetitions, sets, volume-load, or total work. These indicators are useful for characterizing what was completed, but they are not designed to fully represent the athlete's acute psychophysiological response. Internal load captures this response and is influenced by factors beyond the amount of work alone, including relative intensity, and exertion. Accordingly, the aim of the present investigation is to use observable training metrics to calculate estimates of internal training load and to validate these estimates against biomarker responses to resistance training. The overarching goal is to improve training load management and prescription in both athletic and medical settings by providing a more meaningful quantification of the resistance training dose.

1.1 Training load in resistance training

Generally, training load calculations consist of a volume factor and an intensity factor. The volume factor is weighted according to the intensity factor, resulting in a calculated training load. In endurance sports, models such as the Training Impulse (TRIMP) are commonly used. In TRIMP, volume is represented by training duration, while intensity is determined by heart rate. Accordingly, the time spent in specific heart rate zones is multiplied by the weighting factor of the respective zone, yielding an overall training load (Borresen & Lambert, 2009; Hayes & Quinn, 2009).

A key limitation of TRIMP as a training load measure is its inability to accurately quantify nonaerobic forms of exercise, such as resistance training. This limitation arises because heart rate responses during resistance exercise do not follow the same linear relationship with training intensity as they do in endurance activities. Heart rate increases disproportionately, making it unsuitable for direct application in resistance training load quantification (Borresen & Lambert, 2009; MacDougall et al., 1985; Scott et al., 2016). Attempts to modify the TRIMP model for resistance training have included substituting heart

rate reserve with percentage of one repetition maximum and replacing training duration with the total number of lifts performed (Busso et al., 1990). A further attempt to calculate training load in resistance training is session Rate of Perceived Exertion (sRPE). To calculate sRPE, the athlete provides a rating of perceived exertion on a scale ranging from 1 to 10 after the session, and this rating is multiplied by session duration in minutes, resulting in the sRPE load (Day et al., 2004; Foster et al., 2001; Scott et al., 2016).

A key objective of this master thesis is to move beyond such simplistic calculations and to employ a more sophisticated approach to calculating training load for resistance training.

Following the previously established framework for training load calculation, in which volume is multiplied by a weighting factor for intensity, volume in this context can be understood as training volume, that is the external training load, which is subsequently contextualized by training intensity. In this sense, volume may refer to any of the previously mentioned methods of measuring external training load, such as counting repetitions and sets or calculating volume-load and total work (McBride et al., 2009; Scott et al., 2016). Intensity in resistance training is commonly measured relative to the one repetition maximum 1RM) and expressed as a percentage of the 1RM (Kraemer & Ratamess, 2005). Given the fluctuation of the 1RM and the fact that it is unknown for many exercises, alternative methods such as velocity-based training and rate of perceived exertion (RPE) for individual sets are commonly used (Balsalobre-Fernández et al., 2021; Helms et al., 2017; Suchomel et al., 2021; Weakley et al., 2021; Zourdos et al., 2016). With velocity-based training, the force-velocity relationship is used, as higher force, that is, heavier load, corresponds to higher intensity and lower movement velocity (González-Badillo & Sánchez-Medina, 2010; Weakley et al., 2021). With RPE, the last repetition is typically rated in relation to repetitions in reserve. In resistance training, RPE is a subjective estimate of how hard a set is perceived, commonly anchored to the number of repetitions that could still be completed. Repetitions in reserve (RIR) denotes this estimated number of remaining repetitions. Thus, an RPE of 10 corresponds to 0 RIR, meaning no additional repetitions could be completed, whereas an RPE of 9 corresponds to approximately 1 RIR, an RPE of 8 to approximately 2 RIR, and progressively lower RPE values indicate that more repetitions were left in reserve (Helms et al., 2017; Zourdos et al., 2016).

When only volume and intensity are used as the sole parameters to calculate training load, an important limitation becomes evident. Given the typical set and repetition structure of

resistance training, the degree of exertion, or proximity to muscular failure within a set, becomes a key consideration (Helms et al., 2016; Pareja-Blanco et al., 2019; Scott et al., 2016; Suchomel et al., 2021). Motor unit recruitment increases over the course of a working set as lower threshold motor units fatigue and higher threshold motor units are recruited to maintain the required force (Cleary, 2025; Enoka & Duchateau, 2008; Mendell, 2005). Consequently, the closer a set is taken to muscular failure, the greater the training load it incurs (Sánchez-Medina & González-Badillo, 2011). This implies that a given amount of work performed at the same percentage of 1RM can impose different levels of training load depending on how it is distributed across sets, because set structure influences fatigue development and the proximity to muscular failure (Sánchez-Moreno et al., 2023). This exertion component within a set can be quantified via velocity loss, referring to the percentage difference between the fastest and last repetition within a set, or via an RPE rating according to the RPE and RIR scale (Helms et al., 2016; Sánchez-Medina & González-Badillo, 2011; Zourdos et al., 2016). Lastly, exercise selection plays a major role in the incurred training load, as multi-joint compound movements have consistently been shown to produce greater training load than single-joint exercises, performed in open kinetic chain (Barbosa et al., 2024; Kotikangas et al., 2025; Kraemer & Ratamess, 2005; Shaner et al., 2014). Therefore, four key variables for calculating training load in resistance exercise are volume, intensity, exertion, and exercise selection.

From a mechanistic perspective, resistance training adaptations are commonly discussed in relation to mechanical tension, muscle damage, and metabolic stress (Schoenfeld, 2010). Although these mechanisms overlap, mechanical tension and metabolic stress can each contribute to muscle damage through partially distinct physiological pathways. High forces and stretch can perturb myofibers and the extracellular matrix, particularly during eccentric actions where the stretching of elastic tissues contributes to the generation of resistive forces. In this context, nonuniform sarcomere lengthening can create shearing within myofibrils and deform membranes such as T-tubules, which may disrupt calcium homeostasis and initiate structural damage (Morgan & Proske, 2004; Proske & Morgan, 2001). The resulting myotrauma elicits an inflammatory response, including immune cell infiltration and cytokine and growth factor signaling, which supports debris clearance and contributes to satellite cell activation involved in repair and remodeling (Chargé & Rudnicki, 2004; Tidball, 2005). In contrast, metabolic stress arises from glycolytic metabolite accumulation and local hypoxia and is associated with cell swelling and increased reactive oxygen species activity (Häussinger, 1996; Powers & Jackson, 2008). These conditions may promote protein and membrane disruption and increase fiber degradation, thereby

providing an additional damage-related stimulus alongside mechanical loading (Schoenfeld, 2010).

Even though overlapping between these mechanisms is present, training sessions can be conceptualized as differing in the relative contribution of tension-related versus metabolite-related stress. Low-repetition training with heavy loads typically produces high mechanical tension and high neuromuscular demands, while generating comparatively less metabolic disturbance because set duration is short and rest periods are often longer. In contrast, higher repetition work with lighter loads and shorter rest intervals tends to increase local metabolic stress through prolonged time under tension and incomplete recovery, while mechanical tension per repetition is lower. Moderate-repetition schemes can be viewed as a mixed exposure in which both mechanical tension and metabolic stress contribute meaningfully, particularly when sets are performed with high exertion (Schoenfeld, 2010; Schoenfeld, Grgic, et al., 2017; Schoenfeld, Ogborn, et al., 2017).

These mechanistic differences also appear to have practical implications. More metabolically demanding resistance training sessions can impose a greater acute burden on subsequent endurance performance than less metabolically disruptive strength work, consistent with a larger contribution of peripheral fatigue and residual metabolic disturbance (Conceição et al., 2014). For example, in rowing, a bout of high intensity strength training increased markers of muscle damage and reduced rowing-specific maximal power, while 2,000 m ergometer performance remained unchanged 24 to 48 hours later (Gee et al., 2011). Such context effects underline that differences between low-, moderate-, and high-repetition resistance training are not only theoretical, but may translate into meaningful short-term changes in performance capacity and recovery demands (Fyfe et al., 2014).

In summary, accurately quantifying resistance training load requires consideration of four key variables: volume, intensity, exertion, and exercise selection. In addition, differentiating between low-, moderate-, and high-repetition training appears advisable, as these conditions impose training load through partly distinct physiological mechanisms.

1.2 Biomarker based monitoring of internal load in resistance training

This thesis aims to estimate internal resistance training load from observable training variables and to evaluate whether these estimates reflect the physiological stress elicited during a session. For this purpose, biomarkers were selected that capture complementary aspects of internal load. Blood lactate reflects the acute metabolic demands and glycolytic contribution of a session and responds within minutes. Cortisol reflects acute systemic endocrine stress. Creatine kinase reflects exercise induced muscle damage following mechanically disruptive loading. The following sections summarize the physiological basis, measurement considerations, and key determinants of each biomarker response, providing the conceptual foundation for the biomarker analysis.

1.2.1 Lactate

Blood lactate concentration is widely used as an internal marker of the acute metabolic demands imposed by exercise (Benítez-Muñoz & Cupeiro, 2025; Marston et al., 2017). In resistance training, lactate concentrations are typically interpreted as measure of the glycolytic contribution to ATP resynthesis. High concentrations indicate that lactate production was high relative to lactate removal and oxidation during and between sets (Brooks, 2018; Gladden, 2004). Importantly, contemporary work has reframed lactate as more than a metabolic end product (Gladden, 2004; Vavřička et al., 2024). Lactate is continuously produced under aerobic conditions, acts as a mobile substrate for oxidation, and participates in intercellular shuttling between tissues and within muscle, which supports its role as an intermediate in whole body metabolism rather than a waste product (Brooks, 2018; Gladden, 2004; Vavřička et al., 2024).

1.2.1.1 Time course and sampling considerations

A central methodological feature of lactate is its rapid response profile (Goodwin et al., 2007). In resistance training studies, blood lactate is commonly sampled immediately before a protocol and again shortly after completion to capture the acute accumulation associated with the session (Marston et al., 2017; Sánchez-Medina & González-Badillo, 2011; Sánchez-Moreno et al., 2023). This timing reflects that lactate responds on the time scale of minutes, which makes it suitable for characterizing within session metabolic stress but less suitable for monitoring delayed consequences of training such as structural muscle damage (Gladden, 2004; Goodwin et al., 2007; Vavřička et al., 2024).

1.2.1.2 Determinants and variability

Lactate concentrations reflect the balance of production and clearance, both of which are influenced by exercise intensity and the temporal density of work. During resistance training, lactate accumulation increases when the rate of energy demand is high and glycolytic flux rises faster than lactate can be removed or oxidized, which is more likely during repeated contractions with limited recovery. Lactate is produced even under aerobic conditions and can be used as a substrate for oxidation, meaning that an elevated blood lactate concentration primarily reflects a transient mismatch between production and clearance rather than a shift to an exclusively anaerobic state (Brooks, 2018; Gladden, 2004; Marston et al., 2017; Sánchez-Moreno et al., 2023; Vavřička et al., 2024).

Regarding sensitivity, lactate is not determined by external load alone (Marston et al., 2017). High relative loads can increase the instantaneous demand per repetition, but low to moderate loads performed for many repetitions with short rest intervals often produce higher lactate because total contraction time and work density are greater (Marston et al., 2017; Sánchez-Moreno et al., 2023). In practice, lactate is therefore typically more responsive to the combination of repetitions per set, rest interval duration, and proximity to failure than to load magnitude in isolation (Marston et al., 2017; Sánchez-Moreno et al., 2023; Schoenfeld, 2010).

Training status also affects lactate kinetics through adaptations that can increase the capacity to dispose of lactate and to sustain workloads at lower lactate concentrations when exercise intensity is considered relative to an individual's fitness (Baldwin et al., 2000; Messonnier et al., 2013). Consequently, lactate values should be interpreted relative to the task demands and the athlete's training background rather than as an absolute indicator of training stress in isolation (Vavřička et al., 2024).

1.2.1.3 Lactate responses across resistance training repetition ranges and programming variables

The theoretical framework by Schoenfeld (2010) proposes that low-, moderate-, and high-repetition resistance training can be characterized by distinct physiological emphases. Low-repetition training is typically performed with higher relative loads and longer rest intervals, thereby prioritizing mechanical tension. High-repetition training is commonly performed with

lower relative loads and shorter rest intervals, which increases metabolic stress through the accumulation of metabolites and reductions in intramuscular oxygen availability during repeated contractions (Schoenfeld, 2010).

This metabolic emphasis in high-repetition training provides a direct rationale for expecting larger lactate responses. As repetitions accumulate and rest periods are short, repeated contractions increase the rate of glycolytic energy contribution and can limit lactate clearance between sets. In this context, blood lactate functions as a practical marker of acute metabolic stress, but it should be interpreted as part of a broader lactate shuttle model in which lactate is continuously produced and can be oxidized and exchanged between tissues (Brooks, 2018; Fink et al., 2018; Gladden, 2004; Lawson et al., 2022; Vavřička et al., 2024).

Experimental data support that lactate is sensitive to repetition prescription and to the density of work, even when external volume is controlled. When resistance training protocols were equated for volume-load, protocols with more repetitions per set and shorter rest periods produced higher lactate concentrations than protocols with fewer repetitions per set and longer rest periods (Sánchez-Moreno et al., 2023). Similarly, when comparing a strength-oriented session with longer interset recovery to a hypertrophy-oriented session with shorter interset recovery, post exercise blood lactate was higher in the hypertrophy condition despite no difference in mechanical work (Marston et al., 2017). Together, these findings indicate that lactate is more sensitive to how work is distributed over time, including repetition structure and recovery duration, than to volume-load alone (Fink et al., 2018; Lawson et al., 2022; Marston et al., 2017; Sánchez-Moreno et al., 2023).

Beyond repetition range, lactate is also linked to acute fatigue expression. In strength-trained males, velocity loss accumulated across sets was strongly correlated with peak post exercise lactate, indicating that protocols producing larger decrements in repetition velocity also produced greater metabolic stress (Sánchez-Medina & González-Badillo, 2011). High intensity resistance circuit training elicited larger lactate increases than a traditional strength training format when workload was matched, and the higher lactate concentration was discussed as a potential contributor to greater peripheral fatigue (Márquez et al., 2017). In velocity loss-based resistance training, a larger velocity loss threshold produced greater acute lactate responses, and training reduced some protocol related differences, which is

consistent with training induced changes in the physiological response to a given loading strategy (Jukic et al., 2023; Walker et al., 2022).

At the same time, lactate does not fully explain perceived exertion or the subjective experience of effort. When intensity and number of sets were held constant while programming variables were manipulated, muscle activation and blood lactate did not differ between conditions, and lactate was not related to ratings of perceived exertion (Hiscock et al., 2016). This supports the interpretation that lactate is most informative about acute metabolic stress and work density, while other dimensions of training difficulty may be reflected by different markers.

1.2.1.4 Summary

Blood lactate concentration provides an internal marker of the acute metabolic demands of resistance training and responds rapidly on the time scale of minutes. Contemporary perspectives emphasize that lactate is not a metabolic waste product but a dynamic intermediate that is continuously produced and can be oxidized and shuttled between tissues, which supports interpreting elevated values as a transient imbalance between production and clearance (Gladden, 2004; Goodwin et al., 2007; Vavřička et al., 2024).

Within the repetition range framework described by Schoenfeld (2010), high repetition resistance training is expected to elicit comparatively larger lactate responses because the combination of repeated contractions and shorter rest intervals increases metabolic stress and limits clearance between sets (Schoenfeld, 2010). Empirical work indicates that lactate is sensitive to repetition structure and work density, including rest interval duration, and can differ between protocols even when volume-load is equated (Marston et al., 2017; Sánchez-Moreno et al., 2023).

Finally, pre-exercise carbohydrate availability and muscle glycogen may meaningfully modulate lactate accumulation; low glycogen states can blunt lactate responses at a given external workload (Benítez-Muñoz & Cupeiro, 2025; Hearris et al., 2018).

Overall, lactate appears more responsive to the temporal characteristics of the session, such as repetitions per set, rest intervals, and within set performance decline, than to load

magnitude in isolation, and training status modifies lactate kinetics and the magnitude of responses to a given protocol (Baldwin et al., 2000; Messonnier et al., 2013; Sánchez-Medina & González-Badillo, 2011).

1.2.2 Cortisol

Cortisol is a glucocorticoid hormone that is released from the adrenal glands when the body is exposed to stressors, including physical exercise. Its secretion is controlled by the hypothalamic–pituitary–adrenal axis, which coordinates the endocrine stress response. A primary function of cortisol is to help ensure that sufficient energy is available when demand increases. It supports this by promoting glucose availability, for example through increased glucose production by the liver, and by facilitating the mobilization of energy substrates such as amino acids and fatty acids that can be used for fuel or for maintaining blood glucose during and after exercise. In the context of exercise, cortisol is commonly interpreted as an endocrine marker reflecting the combined physiological stress of the session, including metabolic strain, the amount of muscle mass engaged, and the temporal density of work. Resistance exercise can elicit meaningful acute cortisol changes, but responses vary substantially with program variables and athlete characteristics (Athanasiou et al., 2023; Chang & Wang, 2026; Gotshalk et al., 1997; Häkkinen & Pakarinen, 1993; Kraemer & Ratamess, 2005; Lightman et al., 2020; Smilios et al., 2003).

In applied monitoring contexts, acute changes in cortisol are commonly considered alongside other endocrine and physiological measures to characterize the overall stress state elicited by a training session and to help interpret inter-individual variability in responses to similar external loads (B. T. Crewther et al., 2011, 2011, 2019; Halson, 2014; Springham et al., 2022).

1.2.2.1 Measurement and interpretation in blood and saliva

A key methodological issue in cortisol monitoring is that blood measurements typically reflect total cortisol unless a specific method is used to quantify the free fraction. In circulation, the majority of cortisol is bound to cortisol-binding globulin and albumin, while only a small fraction is unbound (El-Farhan et al., 2017). The unbound fraction is generally considered biologically active because it is available for tissue diffusion and receptor binding (Bikle, 2020; Levine et al., 2007). Consequently, total serum or plasma cortisol can change

due to shifts in binding proteins, independently of changes in the free fraction (Choi, 2022; Dichtel et al., 2019; El-Farhan et al., 2017).

In contrast, cortisol measured in saliva is widely considered a valid and reliable reflection of unbound blood cortisol. This is because unbound cortisol diffuses from blood into saliva, while protein-bound cortisol does not enter saliva to the same extent. For applied monitoring, salivary sampling also offers practical advantages, including low participant burden, avoidance of venipuncture-related stress, and feasibility for repeated sampling around training and competition (El-Farhan et al., 2017, 2024; Kirschbaum & Hellhammer, 1994).

These differences have direct ramifications for measurement choice. Total blood cortisol is informative but also more vulnerable to confounding by binding-protein dynamics and the sampling procedure itself, particularly when repeated measures are required in training environments. Salivary cortisol provides a non-invasive estimate of the biologically active fraction and can, therefore, be preferable when the primary goal is to monitor acute hypothalamic–pituitary–adrenal axis responses to resistance training with minimal disruption to the athlete (Choi, 2022; El-Farhan et al., 2017; Kirschbaum & Hellhammer, 1994; Weckesser et al., 2014).

At the same time, salivary cortisol requires standardization. Cortisol exhibits a pronounced diurnal rhythm and is sensitive to timing, recent food and drink intake, oral contamination, and pre-analytic handling. Therefore, sampling should be conducted at consistent times of day, with standardized pre-sampling conditions, and with clearly defined post-exercise sampling windows aligned to the expected time course of the cortisol response (Gröschl et al., 2001; Hansen et al., 2008; Kirschbaum & Hellhammer, 1994; Shirtcliff et al., 2001; Stalder et al., 2016).

1.2.2.2 Acute cortisol responses to resistance training and repetition ranges

Building on the repetition-range framework described by Schoenfeld (2010), cortisol is expected to be more responsive to protocols that combine large amounts of work, limited recovery, and substantial glycolytic and systemic stress, which are characteristics more commonly associated with moderate- to high-repetition training configurations than with

low-repetition strength-oriented configurations (Häkkinen & Pakarinen, 1993; Kraemer et al., 1993; Kraemer & Ratamess, 2005; Schoenfeld, 2010; Smilios et al., 2003).

Acute cortisol responses to resistance exercise depend strongly on how volume, intensity, rest intervals, and exercise selection are combined within the session. Even when the primary exercise is held constant, different loading schemes and resistance exercise formats have been shown to produce distinct salivary cortisol responses, indicating that session structure meaningfully modifies endocrine stress (Beaven et al., 2008; B. Crewther et al., 2008). Exercise selection also matters, as sessions involving larger amounts of active muscle mass can produce more pronounced hormonal changes than exercises engaging less total muscle mass (Geisler et al., 2019). Isolated manipulation of intensity can increase cortisol under specific conditions, but evidence suggests that intensity should be interpreted together with the overall workload and recovery structure (McGuigan et al., 2004). In particular, comparisons of high-volume and high-intensity resistance training indicate larger cortisol responses under higher-volume conditions, with attenuation across repeated exposure, which is consistent with cortisol being sensitive to the systemic and metabolic stress imposed by session density and total work (Gotshalk et al., 1997; Mangine et al., 2015).

1.2.2.3 Summary

Cortisol provides an internal marker of the acute endocrine stress associated with resistance exercise and is sensitive to how repetitions, rest intervals, total work, and exercise selection are combined within a session (Athanasiou et al., 2023; Kraemer & Ratamess, 2005; Smilios et al., 2003). This supports its use as a complementary indicator when sessions differ in metabolic and systemic demands despite similar external loads (Helms et al., 2020; Kraemer & Ratamess, 2005).

Salivary cortisol offers a non-invasive estimate of the biologically active cortisol fraction and is, therefore, well suited for repeated sampling around training sessions with minimal disruption, particularly when the aim is to capture acute session responses (El-Farhan et al., 2017, 2024; Kirschbaum & Hellhammer, 1994).

1.2.3 Creatine kinase

Creatine kinase (CK) is a commonly used blood-based marker to quantify exercise-induced muscle damage following strenuous physical activity (Baird et al., 2012; Brancaccio et al., 2007). Despite its widespread use, the utility of CK as a monitoring variable for training load from resistance training is debated. CK responses show pronounced biological variability and, across resistance-exercise studies, associations with common external load metrics (e.g., volume-load) are typically only weak to moderate. Accordingly, CK is generally recommended as a supportive indicator within a multi-method monitoring framework rather than as a stand-alone proxy for training load (Bourdon et al., 2017; Haller et al., 2023; Halson, 2014; Koch et al., 2014; Machado et al., 2012). In resistance training, increases in CK are generally interpreted as an indirect sign that muscle fibers have experienced structural disruption and that intracellular enzymes have escaped into the blood. This leakage is most likely when exercise places high mechanical strain on the muscle, particularly during eccentric muscle actions, which can disturb the sarcolemma and the contractile apparatus, including structures around the Z-disks. When this occurs, membrane permeability increases and CK can move from the inside of the muscle fiber into the interstitial space (Clarkson & Hubal, 2002; Hody et al., 2019; Proske & Morgan, 2001). From there, CK reaches the circulation via the lymphatic system (Koch et al., 2014). Importantly, this process does not occur to the same extent after all resistance exercise, because the loading must exceed what the muscle is accustomed to (Clarkson & Hubal, 2002; Proske & Morgan, 2001; Stožer et al., 2020). Consequently, an intensity threshold is assumed, such that substantial CK increases are expected primarily when the stimulus is sufficiently demanding relative to the athlete's prior exposure (Koch et al., 2014).

1.2.3.1 Time course and sampling considerations

A key characteristic of CK is its delayed response relative to many acute markers of internal load. Peak CK levels after an exercise bout have been reported to occur between 24 and 96 hours post-exercise (Baird et al., 2012; Koch et al., 2014; Sayers et al., 2000). In the context of resistance training, the time course can be extended, with peak activity reported to occur several days after the bout, depending on training status and the nature of the stimulus (Schoenfeld, 2012).

1.2.3.2 Determinants of CK responses in resistance training

The magnitude of CK responses is influenced by multiple factors, and the relationship between CK and the amount of work performed is not straightforward. Volume-load,

typically expressed as the product of load, repetitions, and sets, summarizes total tonnage lifted. Although a larger volume-load might be expected to produce higher levels of muscle trauma and thus greater CK activity, available evidence suggests a more complex relationship (Koch et al., 2014). Indeed, the literature reports that CK–workload associations are often tenuous; correlations between CK change and volume-load are generally mild and experimental manipulations that increase total volume do not necessarily yield proportionally larger CK responses. This inconsistency is a central reason CK-based monitoring is frequently described as controversial for training load quantification (Koch et al., 2014; Machado et al., 2012). These findings are consistent with the view that CK does not scale linearly with volume-load and that other characteristics of the stimulus may be more influential.

Among the most relevant determinants are eccentric loading, the novelty of the stimulus, proximity to failure, and training status. Eccentric contractions are consistently associated with more pronounced exercise-induced muscle damage, and muscle damage severity depends on exercise type, intensity, and duration (Schoenfeld, 2012). In addition, resistance exercise performed to muscular failure has been shown to induce higher CK concentrations compared with non-failure protocols, indicating greater muscle damage under failure conditions (González-Hernández et al., 2021; Vieira et al., 2022). Training status further moderates CK responses. Trained individuals can experience substantial delayed-onset muscle soreness without showing the same magnitude of CK elevations typically observed in untrained individuals, suggesting that soreness and CK do not necessarily track proportionally (Vincent & Vincent, 1997). This aligns with the repeated-bout effect described in the exercise-induced muscle damage literature, whereby repeated exposure to a similar loading stimulus attenuates subsequent damage responses, potentially in part via extracellular matrix (intramuscular connective tissue) remodeling that increases tissue resilience and reduces excessive fiber strain during eccentric loading (Hyldahl et al., 2017; Mackey et al., 2011).

Inter-individual variability in CK is very high. This variability is large enough that between-athlete comparisons are of limited value, and even within-athlete interpretation benefits from individualized baselines and change thresholds. Test–retest work suggests that CK reliability can be only moderate (Reichel et al., 2020). CK concentrations are influenced by age, sex, race, muscle mass, physical activity, and environmental conditions, and elevated values can occur in healthy individuals without indicating pathology; however, persistently

high resting CK in athletes should be interpreted in clinical context and may warrant evaluation, particularly when accompanied by relevant symptoms (Brancaccio et al., 2007; George et al., 2016; Kyriakides et al., 2010; Moghadam-Kia et al., 2016; Mougios, 2007).

1.2.3.3 CK responses across repetition ranges

Building on the repetition-range framework described by Schoenfeld (2010), CK responses are expected to be most pronounced when the loading conditions impose high mechanical strain on muscle fibers, particularly during unaccustomed eccentric actions, rather than being determined by an intensity threshold alone. In this context, contraction mode, eccentric contraction velocity, and muscle–tendon unit behavior, such as fascicle lengthening or strain and operating length, are likely to be key modifiers of the magnitude of CK leakage after a bout (Hicks et al., 2017; Nogueira et al., 2013; Proske & Morgan, 2001; Schoenfeld, 2010). Protocols characterized by lower relative loads may therefore elicit smaller CK responses on average, even when perceived exertion is high, because the resulting strain stimulus may less consistently disrupt membrane integrity, especially when eccentric stress, contraction velocity, and operating lengths are controlled or familiar (Koch et al., 2014; Nogueira et al., 2013; Proske & Morgan, 2001). Consistent with this interpretation, lower CK activity was reported following an eight-week low-load, high-repetition regimen in elite weightlifters compared with high-load and combined regimens (Yeom et al., 2023).

Conversely, low-repetition, high-load training is more likely to exceed the threshold for mechanical disruption, particularly when eccentric stress is substantial or when the stimulus is unaccustomed, which supports the expectation of comparatively larger CK responses (Clarkson & Hubal, 2002; Koch et al., 2014; Proske & Morgan, 2001; Schoenfeld, 2012; Stožer et al., 2020). Moderate repetition training may also be associated with pronounced CK elevations, especially when sets are performed close to muscular failure. Failure protocols have been shown to elicit higher CK concentrations than non-failure protocols, indicating greater muscle damage under failure conditions (González-Hernández et al., 2021; Morán-Navarro et al., 2017; Vieira et al., 2022). In summary, CK responses are expected to be more pronounced following low- and moderate-repetition resistance training than following high-repetition training, depending on load magnitude, eccentric stress, novelty, and proximity to failure (Koch et al., 2014; Schoenfeld, 2010, 2012).

1.2.3.4 Relationship between CK, muscle damage, and training adaptation

The interpretation of CK as a monitoring variable should be considered independently from assumptions about the functional role of muscle damage in adaptation. Importantly, CK elevations are often poorly related to functional outcomes such as soreness and strength loss, indicating that high CK does not automatically imply impaired performance capacity or insufficient recovery. This further limits its suitability as a stand-alone 'readiness' or training load marker (Koch et al., 2014; Nosaka et al., 2002; Schoenfeld, 2012). A mechanistic rationale exists for exercise-induced muscle damage contributing to hypertrophy via inflammatory signaling, satellite cell activity, and remodeling processes, yet the evidence does not support a simple dose response in which greater damage necessarily produces greater hypertrophy. The review literature further suggests that excessive damage can impair force production and reduce training quality, potentially limiting the accumulation of effective training volume and frequency over time (Schoenfeld, 2012). Accordingly, CK is best considered a marker of the magnitude of disruption and the likely recovery demand, rather than a direct marker of beneficial adaptation.

From an applied perspective, pronounced CK elevations can occur in healthy individuals following eccentric exercise without renal impairment (Clarkson et al., 2006; Rawson et al., 2017), while persistently elevated resting CK in athletes may indicate the need for clinical evaluation in specific cases (Brancaccio et al., 2007; Brewster et al., 2007; Kyriakides et al., 2010; Moghadam-Kia et al., 2016; Mougios, 2007). Protocol- and modality-specific effects further underscore the need for contextual interpretation. Therefore, CK should be interpreted within the broader training context, including contraction type, novelty, proximity to failure, and athlete training status (Baird et al., 2012).

1.2.3.5 Summary

Creatine kinase provides a delayed and context-dependent marker of exercise-induced muscle damage that is primarily sensitive to mechanical disruption and threshold effects rather than to volume-load alone. When interpreted using the repetition range framework described by Schoenfeld (2010), CK responses are expected to be comparatively smaller following low-load, high-repetition resistance training and more pronounced following higher-load low- and moderate-repetition training, particularly when eccentric stress is substantial or sets are performed close to muscular failure. Overall, CK should be interpreted as contextual information about muscle disruption and recovery demand, not as a direct surrogate of resistance training training load. Best practice recommendations

emphasize combining biomarkers with performance/neuromuscular and subjective monitoring rather than relying on any single marker (Bourdon et al., 2017; Halson, 2014).

2. Methods

The methods chapter is divided into two parts. First, the development of the training load calculation model is described, including its conceptual basis, the observable inputs used for computation, and how domain specific and total training load (TL) outputs are derived. Second, the validation study is outlined, including participant procedures, standardized resistance training sessions, blood biomarker collection and analysis, and the statistical analysis used to examine whether the calculated training load estimates reflect acute and delayed biomarker responses.

2.1 Training load calculation model

The training load calculation model was developed to estimate internal resistance training training load from observable training variables. In line with the conceptual framework introduced in Chapter 1, the model integrates four determinants that are particularly relevant for resistance training: volume, intensity, exertion, and exercise selection. The implementation was designed to support automated processing of sensor-derived data, with a primary emphasis on velocity-based inputs. The model was operationalized using custom Python scripts for data handling and computation.

2.1.1 Rationale and core inputs

The model was conceptualized to rely on variables that, in principle, can be collected with minimal manual input given sufficient automation. Training volume is represented through the performed set and repetition structure together with the performed exercise, which provides an objective description of the completed external work. Training intensity is classified primarily using mean concentric velocity, leveraging the inverse relationship between concentric movement velocity and relative loading.

Accordingly, the primary data inputs for the velocity-based pathway are the exercise performed, the number of repetitions completed, and repetition-level velocity data. In

addition to mean concentric velocity as the main intensity descriptor, the decline in velocity within a set is used to quantify exertion. When velocity data are unavailable, the model supports a substitution pathway using a manually recorded set-level RPE value, expressed on a 1–10 scale.

2.1.2 Set-level calculation sequence

All indices are calculated at the set level and then aggregated across the session. Although the three domain-specific indices differ in how they weight repetitions and intensity, they follow the same overarching calculation sequence.

1. **Repetition coefficients (volume contribution):** Each repetition within a set is assigned a repetition coefficient. The coefficient generally decreases as the number of repetitions within the set increases. This reflects that, within longer sets, the incremental contribution of a single additional repetition is reduced.

2. **Intensity coefficient (velocity-based):** Mean concentric velocity is mapped to an intensity coefficient using predefined coefficient mappings. Lower velocities correspond to larger coefficients, consistent with the force–velocity relationship.

3. **Base set load:** For the set, the repetition coefficients are multiplied by the mapped intensity coefficient. These repetition-level values are summed to yield a base set load.

4. **Exertion coefficient (velocity loss):** Exertion is incorporated using a velocity loss coefficient. Velocity loss is defined as the percentage difference between the fastest and the final repetition velocities within a set. Larger velocity loss corresponds to higher exertion and increases the set-level load estimate.

5. **Exercise coefficient (exercise selection):** Finally, the set load is scaled by an exercise-specific coefficient. Exercises involving higher external loads and a larger contribution of multiple body regions and muscle groups receive higher coefficients.

This step operationalizes exercise selection as a determinant of load and allows sets to be contextualized by exercise characteristics.

2.1.3 RPE-based substitution pathway

When velocity data are not available for a set, the model substitutes the velocity-based intensity and exertion information using RPE. In this pathway, an RPE coefficient is selected from a predefined mapping and applied in place of the velocity-derived intensity coefficient. The RPE coefficient is chosen such that it accounts for the combined effect of intensity and proximity to failure implied by the RPE and RIR anchored interpretation.

In practice, repetition coefficients are multiplied by the mapped RPE coefficient and summed to yield a base set value. Where velocity loss information is unavailable, the RPE mapping and the associated repetition rules are defined to produce comparable estimates to the velocity-based pathway for equivalent sets. The exercise coefficient is then applied in the same manner as in the velocity-based pathway.

2.1.4 Domain-specific load variables

To reflect meaningful differences between low-, moderate-, and high-repetition resistance training, three domain-specific load variables are computed separately. These are referred to as the low,moderate, and high repetition load domain. Each domain uses its own repetition-coefficient profiles, intensity-coefficient mappings, and repetition-based scaling rules to prioritize the type of stress that the respective domain is intended to capture.

Low repetition load domain (LL): This domain is intended to emphasize high mechanical tension and high-intensity work. Repetition coefficients place greater weight on early repetitions, and the intensity mapping is designed to be highly sensitive to changes in velocity and thus relative intensity. To prevent overestimation when repetition counts extend beyond the low-repetition range, additional scaling rules are applied so that the low-repetition estimate decreases as repetitions rise beyond the domain’s target range.

Moderate repetition load domain (ML): This domain represents a mixed condition between low- and high-repetition work. Repetition coefficients are structured to emphasize

moderate repetition ranges, while intensity sensitivity is lower than in the low-repetition domain but higher than in the high-repetition domain. Scaling rules are applied when repetition counts extend into high-repetition ranges.

High repetition load domain (HL): This domain is intended to emphasize accumulated metabolic work. Repetition coefficients are structured such that the domain becomes increasingly relevant as repetition counts increase. The intensity mapping is comparatively stable, such that velocity or RPE have a smaller influence on the output than in the low- and moderate-repetition domains. Because metabolic demand within a set generally increases with additional repetitions, this domain does not apply downscaling rules with increasing repetition counts.

2.1.5 Aggregation and outputs

For each session, set-level values are summed within each domain to obtain three session-level domain loads. TL is then computed as the arithmetic mean of the three domain-specific load variables.

The calculator outputs the three domain-specific indices and TL for each set and for the full session.

2.1.6 Computational structure and illustrative example

For each domain $d \in \{LL, ML, HL\}$, the load of a single set is computed according to the following general formulation:

$$Load_d = \left(\sum_{r=1}^{n} RF_{d(r)} \cdot IF_{d(x_r)} \right) \cdot S_{d(n)} \cdot E \cdot C_{ex}$$

Where n denotes the total number of repetitions performed in the set and r represents the repetition index $(\mathrm{r} = 1, \ldots, \mathrm{n})$. The term $\mathrm{RF_d(r)}$ denotes the domain-specific repetition coefficient assigned to repetition r, and $\mathrm{IF_d(x_r)}$ represents the corresponding domain-specific intensity coefficient. The intensity coefficient is derived either from repetition-level

mean concentric velocity ($\mathrm{x_r} = \mathrm{v_r}$) in the velocity-based pathway or from set-level RPE ($\mathrm{x_r} = \mathrm{RPE}$) in the substitution pathway.

The factor $\mathrm{S_d(n)}$ denotes a domain-specific repetition-range scaling term. In the LL and ML domains, this term downscales contributions when repetition counts exceed the intended repetition range, whereas the HL domain does not apply repetition-range downscaling. The coefficient E represents an exertion adjustment derived from within-set velocity loss in the velocity-based pathway. Finally, $\mathrm{C_{ex}}$ denotes an exercise-specific coefficient.

Together, these components yield a deterministic set-level load value for the respective domain. Session-level domain loads are subsequently obtained by summing set-level values within each domain. TL is then calculated as the arithmetic mean of the three domain-specific session loads.

Illustrative example

The following example illustrates the computational sequence using simplified coefficients. Numerical values are provided solely to demonstrate the calculation logic.

Example set

Exercise: squat.

Repetitions: $n = 5$.

Mean concentric velocities ($\mathrm{m \cdot s^{-1}}$): $0.52, 0.50, 0.47, 0.45, 0.41$.

The calculation below demonstrates the procedure for the Low repetition load domain (LL). The ML and HL domain loads are computed analogously using their respective repetition-coefficient profiles, intensity mappings, and repetition-range scaling rules. For a given domain, each repetition r is assigned a repetition coefficient $RF_d(r)$, and each repetition velocity v_r is mapped to an intensity coefficient $IF_d(v_r)$. The base set value is obtained by summing repetition-wise products $RF_d(r) \cdot IF_d(v_r)$. This base value is then adjusted by (i) a repetition-range scaling term $S_d(n)$, where applicable, (ii) an exertion coefficient E based on within-set velocity loss, and (iii) the exercise coefficient C_{ex}.

Step 1: Repetition coefficients

Repetitions 1–5: $2.0, 1.2, 0.7, 0.4, 0.2$.

Step 2: Intensity coefficients

Velocity-mapped values: $1.05, 1.10, 1.20, 1.25, 1.40$.

Step 3: Base set load

$$Base_{LL} = (2.0 \cdot 1.05) + (1.2 \cdot 1.10) + (0.7 \cdot 1.20) + (0.4 \cdot 1.25) + (0.2 \cdot 1.40)$$

$$Base_{LL} = 2.10 + 1.32 + 0.84 + 0.50 + 0.28 = 5.04$$

Step 4: Repetition-range scaling

$$S_{LL(5)} = 0.21$$

$$Scaled_{LL} = Base_{LL} \cdot S_{LL(5)} = 5.04 \cdot 0.21 = 1.058$$

Step 5: Velocity-loss adjustment

$$VL = \frac{0.52 - 0.41}{0.52} \cdot 100 = 21\%$$

A velocity-loss coefficient E is then obtained from a predefined mapping $E = f(VL)$ that increases monotonically with greater velocity loss. For illustration, assume:

$$E = 1.126$$

$$Load_{LL} = Scaled_{LL} \cdot E = 1.058 \cdot 1.126 = 1.19$$

Step 6: Exercise coefficient

If the exercise coefficient for the squat is:

$$C_{ex} = 1.0$$

$$FinalSetLoad_{LL} = Load_{LL} \cdot C_{ex} = 1.19$$

ML and HL domain loads are derived using the same computational sequence but with domain-specific repetition coefficients, intensity mappings, and repetition-range scaling rules.

2.2 Validation study

The training load calculator was intended to estimate internal resistance training training load from observable training variables. The present study evaluated whether the calculator outputs were meaningfully associated with biomarker responses to standardized resistance training sessions. Validity was assessed by relating the three domain-specific training load variables (LL, ML, HL) and TL to session specific biomarker changes across multiple controlled conditions. Subjects were briefed on the procedure and purpose of the trial before signing a written informed consent form. The methodological protocol was conducted following the Declaration of Helsinki and approved by a local ethics committee (reference number 01324).

2.2.1 Participants

A total sample of 20 trained athletes was recruited (10 male and 10 female), representing a sample size comparable to prior resistance training investigations examining biomarker responses and acute physiological effects under standardized protocols (Ammar et al., 2018; Callegari et al., 2017; Geisler et al., 2019; González-Hernández et al., 2021; Machado et al., 2012; Marston et al., 2017; Pareja-Blanco et al., 2019; Sánchez-Moreno et al., 2023).

Eligibility criteria were defined to reduce variability attributable to insufficient training experience and to ensure safe and consistent execution of the protocol. Participants were required to be 18 to 45 years of age, to have at least two years of structured resistance training experience, and to demonstrate technical proficiency in the back squat, bench press, and conventional deadlift. Training history is considered relevant because physiological responses to comparable external loads are moderated by prior exposure and

adaptation (Baldwin et al., 2000; Brancaccio et al., 2007; Clarkson & Hubal, 2002; Messonnier et al., 2013; Schoenfeld, 2012; Walker et al., 2022).

Participants were excluded if they reported recent injury limiting training performance, relevant metabolic or endocrine disorders, or other conditions likely to confound biomarker responses. Pre-participation screening was conducted using the Physical Activity Readiness Questionnaire (PAR-Q) (S. Thomas et al., 1992). Participants were instructed to maintain habitual nutritional and hydration practices across the study period, although these factors were not formally assessed. Participants were also instructed to keep a consistent sleep schedule and to manage life stress as consistently as possible across visits, as changes in these factors may influence biomarker levels and their interpretability (Hansen et al., 2008; Jardine et al., 2023; Maughan & Burke, 2011; D. T. Thomas et al., 2016).

All 20 participants completed the whole study protocol. Participant characteristics are summarized in Table 1.

Variable	**Mean ± SD (Min–Max)**
Age (y)	25.4 ± 3.4 (20.0–32.0)
Body mass (kg)	77.0 ± 12.6 (55.9–101.1)
Height (cm)	174.8 ± 9.4 (160–191)
Relative squat 1RM (kg·kg_BM^-1)	1.42 ± 0.32 (0.98–2.00)
Relative bench press 1RM (kg·kg_BM^-1)	1.01 ± 0.27 (0.63–1.51)
Relative conventional deadlift 1RM (kg·kg_BM^-1)	1.71 ± 0.42 (1.19–2.47)

Table 1. Participant characteristics.

2.2.2 Study design, schedule, and session order

The protocol comprised seven laboratory visits scheduled at weekly intervals; depending on pauses, the overall study duration could extend beyond seven weeks. The first visit was dedicated to one repetition maximum testing in the back squat, bench press, and conventional deadlift to individualize subsequent loading prescriptions. Participants were extensively briefed on the study purpose and protocol before signing a written consent form. Basic characteristics including sex, age, height, and body mass were recorded. Health

eligibility was assessed using the PAR-Q. Testing followed a general warm-up followed by lift-specific warm-ups with incremental load increases until the maximum successful attempt was reached. The general warm-up consisted of 3–5 minutes of cycling on a bicycle ergometer at a self-selected submaximal (non-quantified) intensity, followed by dynamic movements including leg swings and arm circles.

In weeks 2 to 7, participants completed six standardized training sessions designed to create systematic variation in the calculator outputs. Each participant performed one session per week under supervised laboratory conditions. Wherever possible, visits were scheduled on the same day of the week, and the same time of day was always ensured to account for diurnal variation in cortisol. In cases of illness or time constraints, the protocol was paused and could be extended by up to a maximum of two weeks.

To reduce confounding from uncontrolled training stress, participants were instructed to refrain from strenuous physical activity for 48 hours before each laboratory visit and for 24 hours after each visit (until the CK measurement on the day after the session).

The order of the six sessions was as follows. High and low condition refer to the higher and lower training load variant within the respective session type.

1. Week 2: High condition of the LL session
2. Week 3: Low condition of the LL session
3. Week 4: High condition of the ML session
4. Week 5: Low condition of the ML session
5. Week 6: High condition of the HL session
6. Week 7: Low condition of the HL session

2.2.3 Training sessions and standardization

All sessions consisted exclusively of barbell-based, multi-joint compound movements, specifically back squat, bench press, and conventional deadlift. Each session included all three lifts.

Sessions were constructed to emphasize the primary stressor that the respective training load domain is intended to capture. For the LL sessions, the key variable is mechanical tension. Therefore, the sole difference between the high and low condition was the prescribed relative intensity, while the number of sets and repetitions was kept identical. For the ML sessions, both intensity and volume were reduced in the low condition, and rest intervals were shorter than in the LL sessions to introduce additional metabolic stress in accordance with the repetition range framework. For the HL sessions, relative intensity was kept identical between conditions and only sets and repetitions were reduced in the low condition, resulting in the primary difference being total work and metabolic stress. Rest intervals were further reduced compared with the ML sessions to further increase metabolic stress (Schoenfeld, 2010).

Training prescriptions were defined using sets, repetitions, and relative intensity expressed as percent of one repetition maximum, based on the individual testing performed in week 1. For each exercise (back squat, bench press, and conventional deadlift), participants completed the sets and repetitions specified below at the indicated percentage of 1RM.

1. LL session, high condition: 3 × 3 at 90%1RM.
2. LL session, low condition: 3 × 3 at 75%1RM.
3. ML session, high condition: 4 × 10 at 70%1RM.
4. ML session, low condition: 3 × 10 at 55%1RM.
5. HL session, high condition: 3 × 30 at 40%1RM.
6. HL session, low condition: 2 × 20 at 40%1RM.

Inter-set rest periods were standardized within each session type and progressively reduced across domains to manipulate work density, with 2 to 3 minutes in the LL sessions, 60 to 90 seconds in the ML sessions, and 30 to 60 seconds in the HL sessions (Athanasiou et al., 2023; Schoenfeld, 2010).

Mean concentric velocity was recorded for every repetition using the RepOne linear position transducer (RepOne Strength, New York, NY, USA), which has previously been validated for barbell velocity assessment (Renner et al., 2024). Velocity data were used as the primary sensor-derived input for the training load calculator.

2.2.4 Session workflow

To standardize salivary cortisol assessment and reduce pre-analytic variability, participants were instructed to refrain from food intake for 2 h before each laboratory session and consume only water during this period (Gröschl et al., 2001; Kirschbaum & Hellhammer, 1994). Participants were also instructed to avoid dental cleaning and tooth brushing within the 2 h before sampling to minimize oral contamination and potential blood leakage (Gröschl et al., 2001). Pre-analytic conditions were standardized across visits, including consistent sampling timing and uniform post-collection handling procedures (Kirschbaum & Hellhammer, 1994; Stalder et al., 2016).

Upon arrival, participants rinsed their mouths with water and then remained seated for 10 minutes. During this period, baseline capillary blood sampling for lactate and creatine kinase was performed. After the 10-minute seated period, a baseline saliva sample was collected using cortisol salivettes.

The warm-up then commenced and consisted of a general warm-up followed by a specific warm-up for each lift. The specific warm-up was performed using incremental loading in the back squat, bench press, and conventional deadlift until the first working set load was reached. Working sets were performed to muscular failure, which was commonly reached in the high condition sessions but not in the low condition sessions. During the training session, only water consumption was permitted.

After completion of the final repetition, participants rested for 10 minutes, after which lactate and salivary cortisol samples were collected. During this 10-minute period, no further water consumption was permitted to avoid dilution of the saliva sample.

In addition to the primary biomarker sampling described above, dried blood spot (DBS) samples were collected for a separate, ancillary investigation examining metabolome trajectories in response to standardized resistance training sessions. One DBS was obtained at each of three time points: together with the baseline capillary sampling, immediately after the completion of the last repetition, and together with the post-exercise lactate sampling. These DBS samples were collected via earlobe sampling and stored for

subsequent metabolomics analyses under a separate research question and analysis plan; they were not part of the outcomes or statistical analyses of the present validation study.

2.2.5 Biomarker analysis

Biomarker sampling was performed at standardized time points relative to each training session. Lactate and creatine kinase were measured at baseline during the 10-minute seated period prior to the warm-up, and salivary cortisol was sampled at baseline immediately after the 10-minute seated period. Lactate and salivary cortisol were then measured 10 minutes after completion of the final repetition of the final exercise. Creatine kinase was measured 24 hours after the training session. The timing of all samples was kept consistent across conditions within each participant.

Creatine kinase was analyzed using a cartridge-based point-of-care analyzer (SimplexTAS™ 101; TASCOM Co., Ltd., Anyang-si, Gyeonggi-do, Republic of Korea) operated with single-use CK reagent cartridges (SimplexTAS™ 101 CK reagent cartridges; TASCOM Co., Ltd., Anyang-si, Gyeonggi-do, Republic of Korea). For each planned CK time point, capillary whole blood was obtained via earlobe sampling, loaded onto the CK cartridge, and analyzed immediately on the SimplexTAS™ 101 device. In accordance with the manufacturer's instructions, reagent cartridges were stored at approximately 5 °C and allowed to reach room temperature before use. CK concentrations (IU/L) displayed by the analyzer were recorded. The device reported CK concentrations up to 1200 IU/L; values above this threshold were displayed as ">1200" and recorded as 1200 IU/L.

Blood lactate concentration was assessed from capillary blood using an enzymatic bench-top analyzer (BIOSEN C-Line; EKF-diagnostic GmbH, Barleben, Germany). For each lactate time point, a capillary sample was collected via earlobe sampling into 20 µL sodium-heparinized end-to-end plastic capillaries and immediately transferred into the manufacturer's glucose/lactate hemolyzing solution (micro test tube; EKF-diagnostic GmbH, Barleben, Germany). The prepared sample was then loaded into the BIOSEN C-Line for automated analysis and the lactate concentration (mmol/L) was recorded. The analyzer reported lactate concentrations down to 0.5 mmol/L; values below this threshold were displayed as "<0.5" and recorded as 0.5 mmol/L. The analyzer was calibrated before each measurement session in accordance with the manufacturer's calibration procedure.

Salivary cortisol was collected using Salivette Cortisol collection devices (SARSTEDT AG & Co. KG, Nümbrecht, Germany). Participants placed the absorbent swab in the mouth for 2 min, positioned between the cheek and teeth, to obtain a standardized saliva sample. After collection, the swab was returned to the salivette tube and centrifuged to separate saliva from the swab, and the saliva fraction was recovered in the lower part of the tube. Samples were then frozen at −20°C and stored until shipment to an external laboratory, where salivary cortisol concentration (ng/mL) was determined.

2.2.6 Data processing and statistical analysis

For each laboratory session, training load outputs were calculated, and pre- and post-exercise biomarker concentrations were recorded and compiled in a spreadsheet file. The spreadsheet was subsequently exported as a comma-separated values file for further processing and statistical analysis using custom Python scripts (Renner, 2026). Descriptive statistics were computed for participant characteristics.

Subsequently, biomarker responses were quantified as absolute change scores, defined as the post-exercise minus pre-exercise concentration, yielding positive values for increases and negative values for decreases. In addition, relative change scores were computed within each participant and biomarker to facilitate within-subject comparison across sessions. Relative change was expressed on a 0–100 scale and calculated using within-participant min–max scaling. The minimum observed change (Δ; representing either the largest decrease or the smallest increase) was mapped to 0, and the maximum observed change (Δ; largest increase) was mapped to 100, and session-specific relative change scores were computed as 100 × (Δ − Δ_min) divided by (Δ_max − Δ_min). All values were stored in a cleaned dataset which was used for subsequent statistical analysis.

Training load outputs for LL, ML, and HL as well as TL were summarized descriptively across sessions and visualized by displaying the session-wise mean and standard deviation.

Biomarker data were summarized descriptively across sessions by reporting pre- and post-exercise concentrations and by summarizing session-specific responses using absolute change scores. Scores reflecting changes in absolute biomarker levels were visualized by displaying the mean and standard deviation of the absolute change per session.

Given the substantial inter-individual variability in biomarker response magnitude reported in the literature and observed in the present dataset, associations between training load outputs and biomarker responses were evaluated using relative biomarker change scores (Koch et al., 2014; Kudielka et al., 2009; Nosaka & Clarkson, 1996; Pacitti et al., 2025; Preobrazenski et al., 2019). Singular load–biomarker relationships were examined pairwise for four load variables (LL, ML, HL, TL) and three biomarkers (creatine kinase, lactate, and cortisol), yielding 12 tests. To isolate within-participant covariation (repeated-measures effects) rather than between-participant differences in overall level, both the session-level load value and the session-level biomarker relative delta were demeaned within participant by subtracting each participant's mean across available sessions for the respective variable; Pearson's correlation coefficient was computed on these demeaned values (r_within) and the association was additionally summarized by an ordinary least squares slope on demeaned values (slope_within), equivalent to a regression through the origin in deviation-score space. Statistical significance was evaluated using a within-participant permutation procedure (10,000 permutations per test) in which demeaned load deviations were randomly reassigned within each participant while demeaned biomarker deviations were held fixed to generate a participant-structure–preserving null distribution; two-sided permutation p-values were calculated as the proportion of permuted correlations with absolute magnitude greater than or equal to the observed absolute correlation (with a +1 correction in the numerator and denominator). Biomarker distributional asymmetry was summarized as the skewness of the raw (non-demeaned) biomarker relative change scores used in each test.

To summarize mixed physiological demands, an additional analysis used a rank-based composite biomarker index (mean rank) for the ML. Within each participant, relative biomarker change scores (Δrel; 0–100) were converted to within-participant percentile ranks across the six sessions. For each session, percentile ranks were averaged across creatine kinase, lactate, and salivary cortisol and expressed on a centered −100 to 100 scale as 200 × (mean percentile rank) − 100. The resulting mean-rank composite was correlated with the ML using the same within-participant demeaning approach and permutation-testing framework described above.

The complete dataset including all Python scripts used for analysis can be found in an online repository (Renner, 2026).

3. Results

All 20 participants (10 male, 10 female) completed the 1RM visit and all six training sessions (120 observations). Training load outputs and pre–post biomarker pairs (lactate, creatine kinase, and salivary cortisol) were complete for all sessions.

3.1 Training load calculations

Session-level training load outputs for LL, ML, HL, and TL are presented in Figure 1 as mean ± SD across participants. All 20 participants were included in the analysis.

Overall, Figure 1 illustrates a clear domain-specific pattern across the six sessions. The LL output was highest in the first two sessions and decreased sharply thereafter, whereas the ML peaked in session 3. In contrast, the HL output was minimal in sessions 1 and 2 and reached its maximum in session 5. TL, computed as the arithmetic mean of the three domain-specific variables, reflected these shifts in relative domain contribution across sessions.

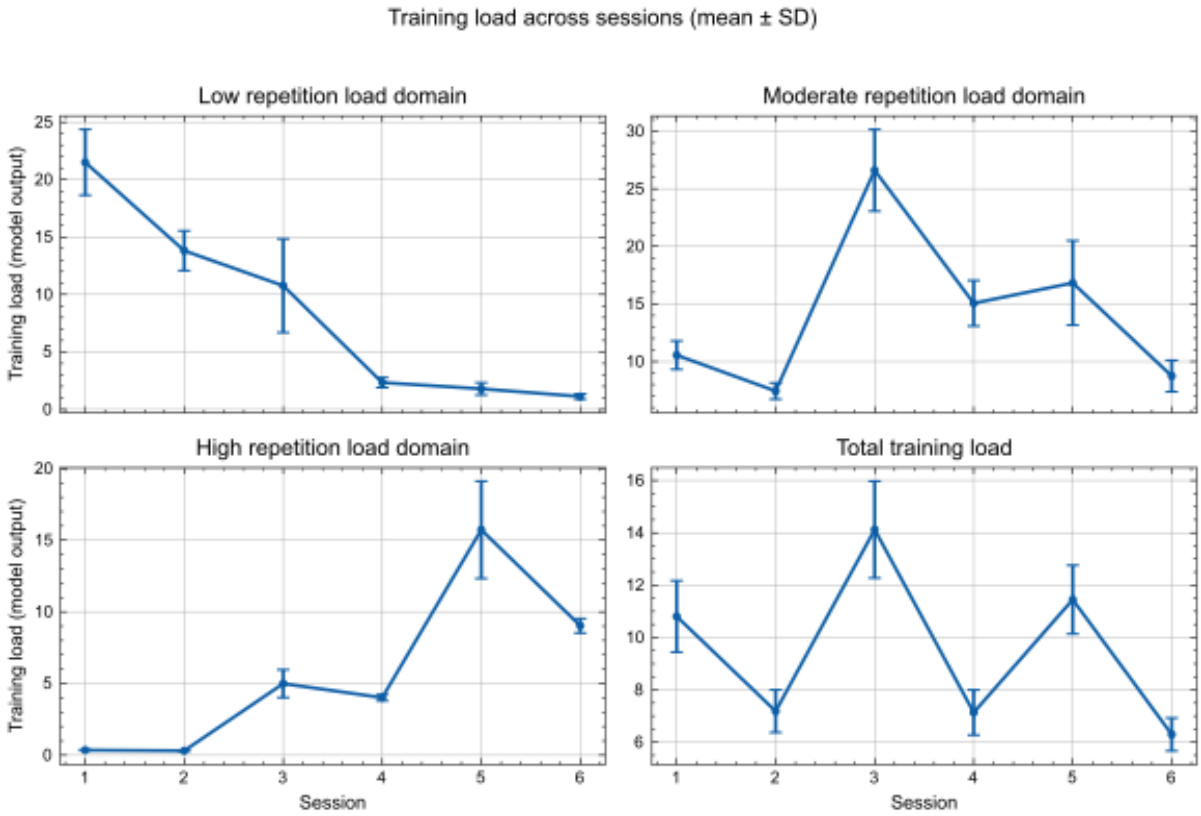

Figure 1. Session-wise mean ± SD training load outputs for LL, ML, HL, and TL across the six laboratory sessions.

For LL the highest output was observed in session 1 with a mean of 21.51 ± 2.86, followed by session 2 with 13.81 ± 1.73. Output remained moderate in session 3 at 10.76 ± 4.07 and then decreased markedly in later sessions, reaching 2.33 ± 0.44 in session 4, 1.79 ± 0.53 in session 5, and 1.12 ± 0.23 in session 6. This pattern indicates that session-to-session variation in the LL output was dominated by a pronounced decline from early to later sessions, with comparatively small variability once outputs approached the lower range.

For ML the largest mean output was observed in session 3 at 26.61 ± 3.55, representing the global maximum of this domain across the protocol. The remaining sessions showed lower values, including 10.56 ± 1.24 in session 1 and 7.44 ± 0.70 in session 2. In the later sessions, moderate-domain outputs were 15.06 ± 1.98 in session 4, 16.83 ± 3.67 in session 5, and 8.74 ± 1.36 in session 6. Variability within this domain was greatest in sessions with higher mean outputs, particularly sessions 3 and 5, as reflected by larger standard deviations.

For HL outputs were minimal in the first two sessions, with 0.36 ± 0.03 in session 1 and 0.31 ± 0.01 in session 2. Outputs increased substantially in session 3 to 4.99 ± 0.97 and remained in a similar range in session 4 at 4.02 ± 0.22. The highest high-domain output occurred in session 5, with 15.73 ± 3.39, followed by session 6 with 9.03 ± 0.50. Thus, the HL demonstrated a strong contrast between early sessions with near-zero values and later sessions with markedly higher outputs, with the largest dispersion observed at the session showing the highest mean.

For TL the highest mean output was observed in session 3 with 14.12 ± 1.85. TL was also comparatively elevated in session 5 at 11.45 ± 1.31 and session 1 at 10.81 ± 1.36. Lower values were observed in session 2 at 7.19 ± 0.81 and session 4 at 7.14 ± 0.86, with the lowest mean in session 6 at 6.30 ± 0.63. Across sessions, the TL trajectory mirrored the combined changes in the three domain outputs, with its peak in session 3 occurring alongside the maximum ML output.

Taken together, Figure 1 demonstrates that the calculator produced systematic differences in domain-specific outputs across the six sessions. The relative contribution of each domain varied substantially by session configuration, and TL reflected these shifts as an aggregate summary of the three domain-specific load variables.

3.2 Biomarker responses

Biomarker responses are summarized across the six laboratory sessions using absolute change scores, defined as post-exercise minus pre-exercise concentration for lactate and salivary cortisol, and 24 h post-exercise minus baseline for creatine kinase. Session-wise mean ± SD of these absolute change scores is shown in Figure 2. Pre- and post-exercise concentrations are reported descriptively alongside the change scores.

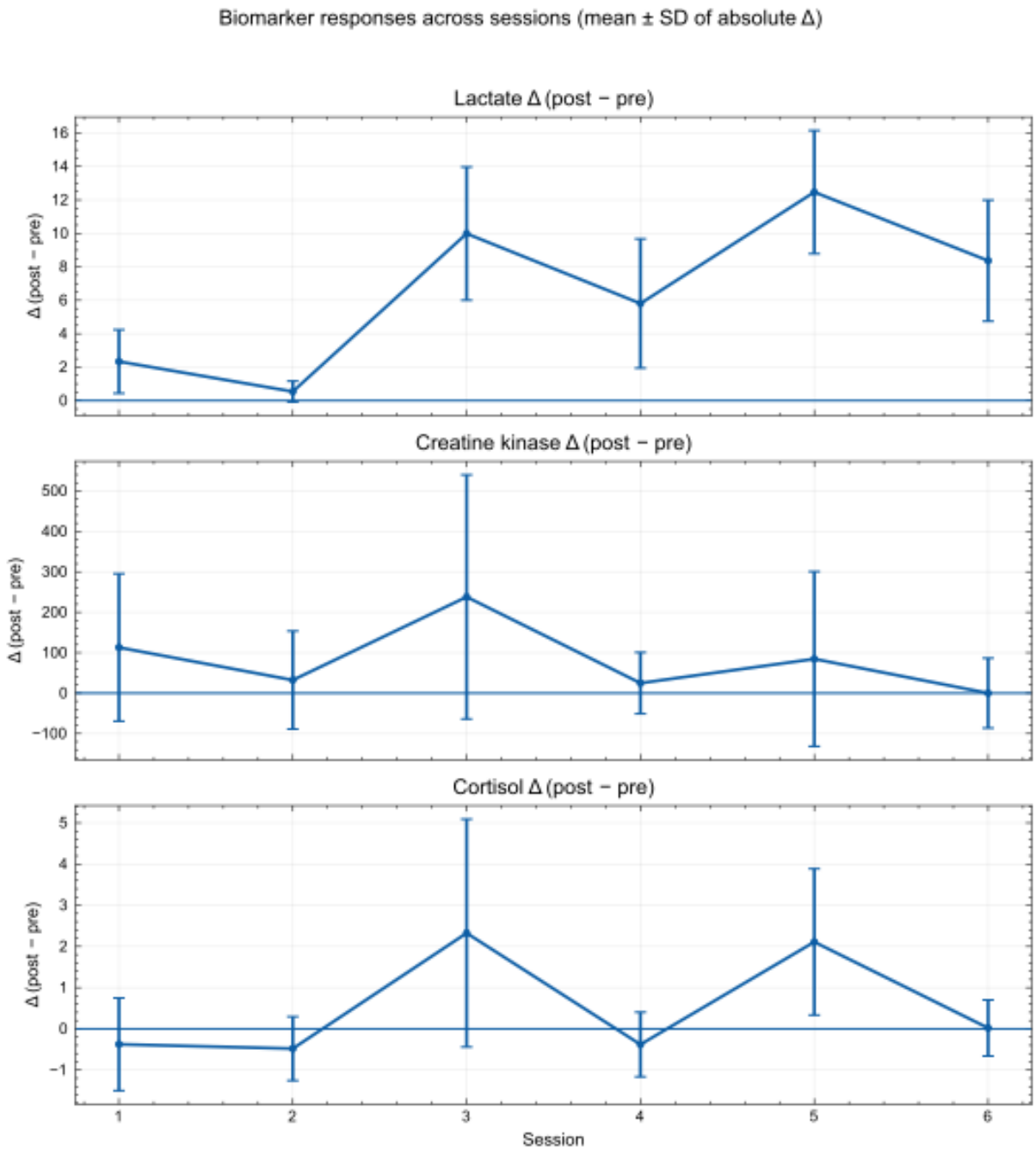

Figure 2. Session-wise mean ± SD absolute biomarker change scores (Δ post–pre) for blood lactate, creatine kinase, and salivary cortisol across the six laboratory sessions.

3.2.1 Lactate

Baseline blood lactate concentration, pooled across all sessions, was 0.62 ± 0.25 mmol/L. Post-exercise lactate concentrations differed markedly by session (Figure 2), with the lowest mean in session 2 (1.25 ± 0.80 mmol/L) and progressively higher values in the later sessions, peaking in session 5 (13.02 ± 3.66 mmol/L). The remaining post-exercise means

were 2.90 ± 1.94 mmol/L (session 1), 10.63 ± 4.03 mmol/L (session 3), 6.42 ± 3.92 mmol/L (session 4), and 9.03 ± 3.60 mmol/L (session 6).

Consistent with these post-exercise patterns, lactate increased on average in every session. Mean absolute lactate change (Δ) was smallest in session 2 (Δ 0.54 ± 0.62 mmol/L) and session 1 (Δ 2.34 ± 1.90 mmol/L), and substantially larger in the later sessions: Δ 9.99 ± 3.98 mmol/L (session 3), Δ 5.81 ± 3.86 mmol/L (session 4), Δ 12.47 ± 3.68 mmol/L(session 5), and Δ 8.37 ± 3.62 mmol/L (session 6).

3.2.2 Creatine kinase

Baseline creatine kinase (CK), pooled across all sessions, was 201.95 ± 151.70 IU/L. At 24 h post-exercise, CK concentrations varied by session (Figure 2), with the highest mean observed in session 3 (436.25 ± 355.18 IU/L). Post-exercise means in the remaining sessions were 331.50 ± 264.83 IU/L (session 1), 221.75 ± 145.95 IU/L (session 2), 239.45 ± 178.13 IU/L (session 4), 267.30 ± 262.85 IU/L (session 5), and 208.00 ± 127.84 IU/L (session 6).

Absolute CK change scores (Δ) showed pronounced dispersion across sessions (Figure 2). The largest mean CK increase occurred in session 3 (Δ 237.90 ± 301.82 IU/L). Smaller mean increases were observed in session 1 (Δ 113.00 ± 182.27 IU/L), session 5 (Δ 84.55 ± 215.95 IU/L), session 2 (Δ 32.30 ± 121.14 IU/L), and session 4 (Δ 24.80 ± 75.83 IU/L). In session 6, the mean change was Δ 0.00 ± 86.38 IU/L, indicating no net change on average, alongside substantial inter-individual variability in CK responses.

3.2.3 Cortisol

Baseline salivary cortisol, pooled across all sessions, was 2.25 ± 1.31 ng/mL. Post-exercise cortisol concentrations varied by session (Figure 2), with the lowest mean observed in session 2 (1.58 ± 0.87 ng/mL) and the highest means in sessions 3 (4.48 ± 2.94 ng/mL) and 5 (4.35 ± 2.52 ng/mL). The remaining post-exercise means were 2.29 ± 1.21 ng/mL (session 1), 1.77 ± 1.00 ng/mL (session 4), and 2.26 ± 1.36 ng/mL (session 6).

Absolute cortisol change scores (Δ) were small relative to lactate and CK and varied in direction across sessions (Figure 2). Mean decreases were observed in session 1 (Δ −0.38 ± 1.12 ng/mL), session 2 (Δ −0.48 ± 0.78 ng/mL), and session 4 (Δ −0.38 ± 0.78 ng/mL). Mean increases were observed in session 3 (Δ 2.33 ± 2.77 ng/mL) and session 5 (Δ 2.11 ± 1.78 ng/mL). Session 6 showed a near-zero mean change (Δ 0.02 ± 0.68 ng/mL), with directionally mixed individual responses.

3.3 Load–biomarker associations

Associations between training load outputs and biomarker responses were evaluated using within-participant correlation analyses based on the relative biomarker change scores described in Section 2.2.6. Results are presented separately for individual biomarkers and for composite biomarker indices.

Within-participant correlations between the four load variables (LL, ML, HL, TL) and relative biomarker change scores (Δrel; 0–100) are shown in Figure 3.

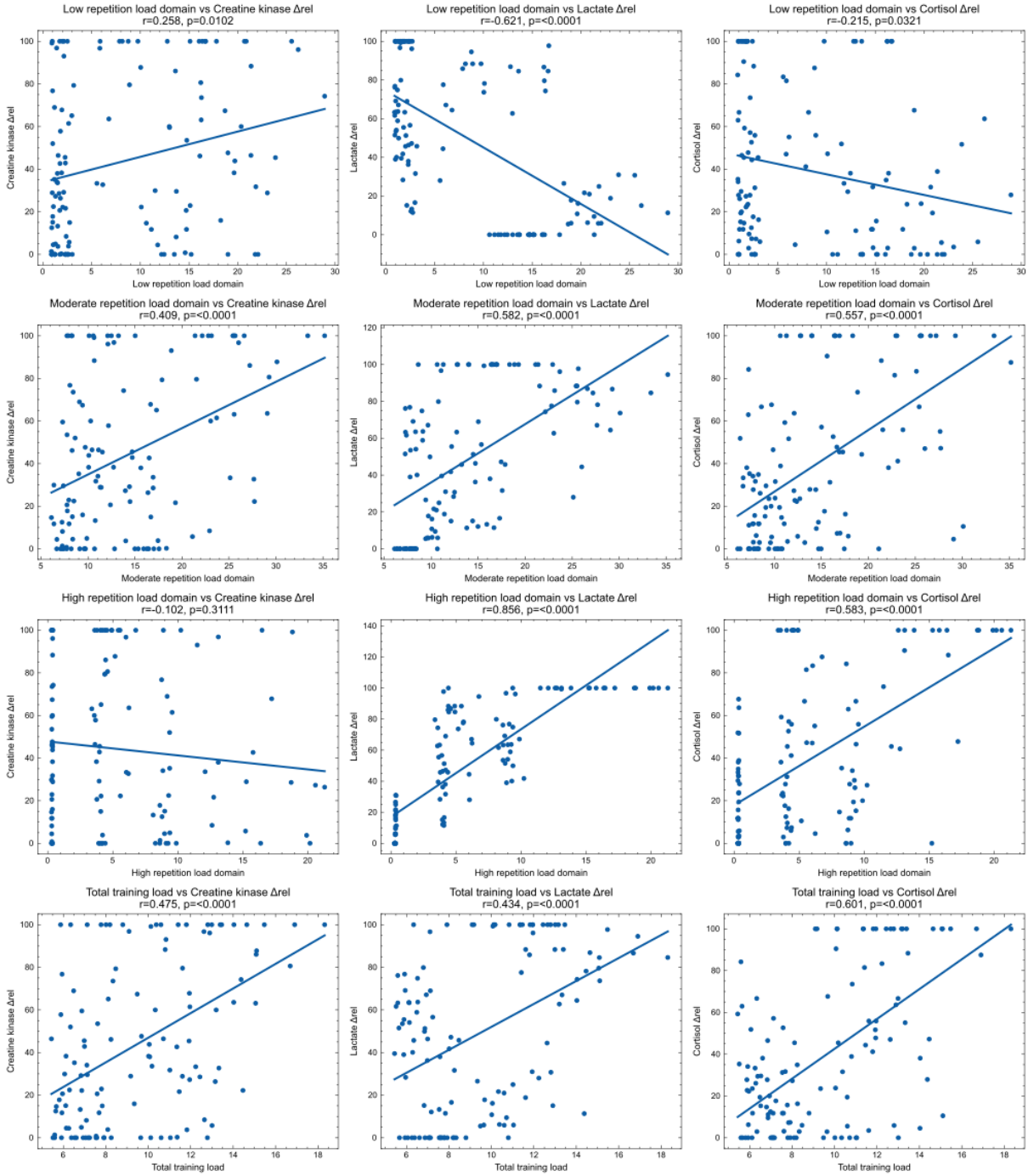

Figure 3. Within-participant correlations between session-level training load outputs (LL, ML, HL, TL) and relative biomarker change scores (CK, lactate, cortisol; Δrel 0–100). Each panel shows pooled session observations with a fitted regression line; r and permutation-derived p-values are reported in-panel.

For CK Δrel, positive associations were observed for LL (r = 0.258, p = 0.0102), ML (r = 0.409, p < 0.0001), and TL (r = 0.475, p < 0.0001). In contrast, HL was not associated with CK Δrel (r = −0.102, p = 0.3111).

For lactate Δrel, LL was negatively associated with lactate responses (r = −0.621, p < 0.0001). Positive associations were observed for ML (r = 0.582, p < 0.0001), HL (r = 0.856, p < 0.0001), and TL (r = 0.434, p < 0.0001).

For cortisol Δrel, LL was negatively associated with cortisol responses (r = −0.215, p = 0.0321). Positive associations were observed for ML (r = 0.557, p < 0.0001), HL (r = 0.583, p < 0.0001), and TL (r = 0.601, p < 0.0001).

The within-participant correlation between ML and a rank-based composite biomarker index (rank-mean; CK, lactate, cortisol; −100 to 100) is shown in Figure 4 (r = 0.673, p < 0.0001).

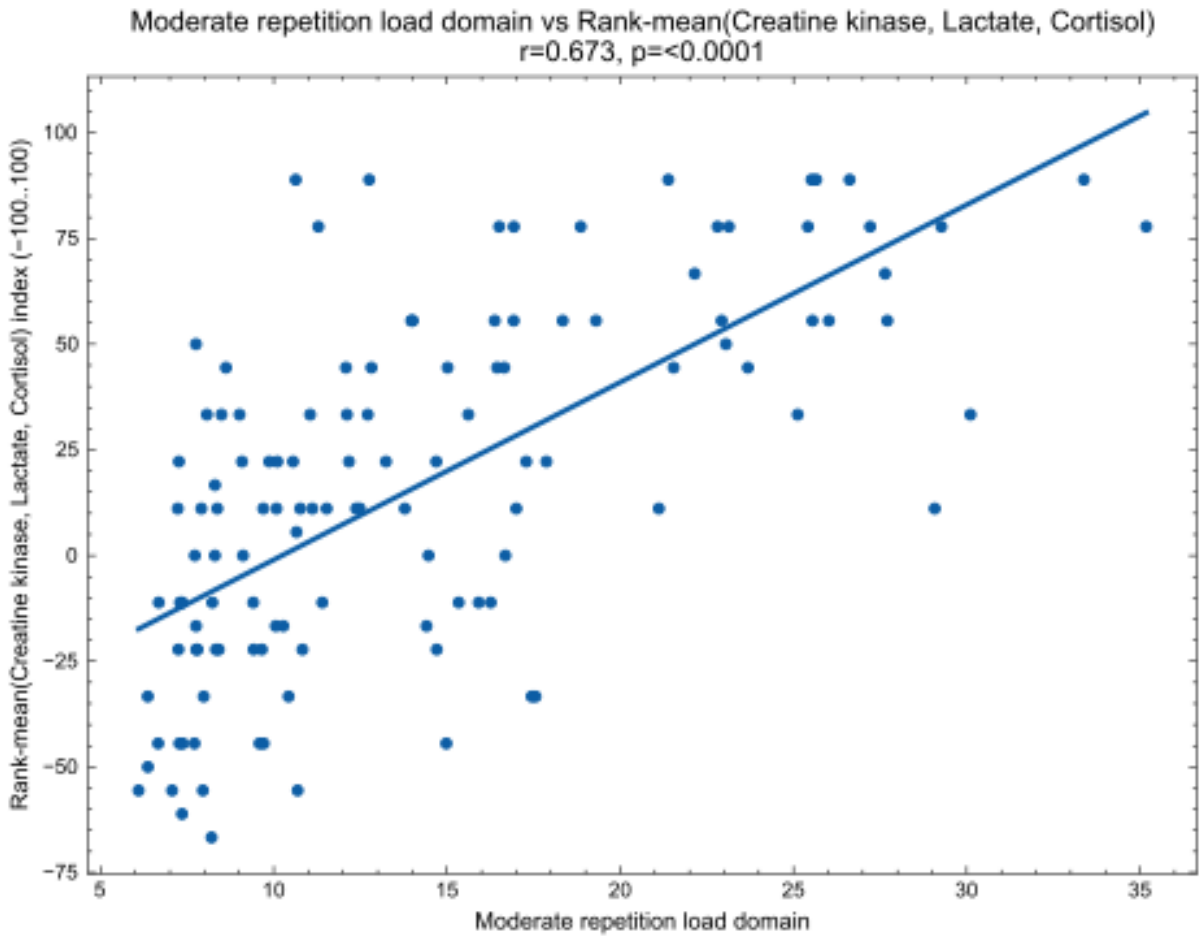

Figure 4. Within-participant correlation between the ML and rank-mean (CK, lactate, cortisol) (−100 to 100). Session observations are shown with a fitted regression line; r and permutation-derived p-values are reported in-panel.

4. Discussion

This discussion integrates the conceptual rationale and the empirical findings to evaluate whether the training load calculator yields meaningful estimates of internal stress under the present standardized resistance training protocol. The section first summarizes domain-specific validity patterns and their physiological plausibility and then discusses practical implications and study limitations.

Correlations are interpreted as associations and not as method agreement; accordingly, emphasis is placed on the pattern and physiological plausibility of the observed coefficients rather than on qualitative labels (Akoglu, 2018; Mukaka, 2012; Schober et al., 2018).

4.1 Overview and rationale

The present study evaluated the validity of a novel resistance training load calculator by examining whether its domain-specific outputs were meaningfully associated with biomarker responses to standardized resistance training sessions. Overall, the observed pattern of associations provides promising proof-of-concept support that the proposed calculation framework can yield meaningful training load estimates under the present standardized conditions. This interpretation, however, depends on the extent to which the selected biomarkers align with the dominant physiological stimulus that each load domain was intended to represent. Accordingly, validity is most informatively discussed not only in terms of coefficient magnitude, but also in terms of (i) why specific domain–biomarker associations were expected, (ii) why other relationships were inherently challenging to detect with the available criteria, and (iii) why composite external criteria were required for domains that reflect multiple, partially distinct stress pathways.

4.2 Interpretation of domain-specific validity

4.2.1 High repetition load domain

Within-participant analyses showed that HL was most closely associated with lactate Δrel ($r = 0.856$, $p < 0.0001$) and also positively associated with cortisol Δrel ($r = 0.583$, $p < 0.0001$). This pattern is physiologically plausible because high-repetition resistance exercise performed with short rest intervals and prolonged time under tension increases glycolytic flux and metabolite accumulation, for which blood lactate provides a practical

acute marker (Marston et al., 2017; Sánchez-Moreno et al., 2023; Schoenfeld, 2010). From a construct-validity perspective, the strong lactate association supports that the calculator can estimate session-level metabolic demand from observable training variables when the dominant stimulus is predominantly metabolic.

At the same time, lactate has limitations as a single criterion for metabolically highly demanding sessions. In the present analysis, lactate was evaluated using within-participant min–max scaled relative change scores (0–100), which imposes an upper bound; if a participant expressed similarly high lactate change scores in more than one session, observations may cluster near the upper limit and reduce differentiation among the most demanding sessions. More generally, lactate reflects a time-dependent balance of production and clearance, such that incremental increases in metabolic demand may not translate proportionally into higher measured concentrations at a fixed post-exercise sampling time point (Gladden, 2004; Goodwin et al., 2007).

4.2.2 Moderate repetition load domain

Associations between ML and single biomarkers were more complex, which is consistent with moderate-repetition resistance training reflecting a mixed stimulus in which both mechanical tension and metabolic stress contribute meaningfully. In the within-participant analyses, ML was positively associated with lactate responses ($r = 0.582$, $p < 0.0001$), creatine kinase responses ($r = 0.409$, $p < 0.0001$), and salivary cortisol responses ($r = 0.557$, $p < 0.0001$).

Within the repetition-range framework described by Schoenfeld (2010), this pattern is plausible because ML should not be expected to map cleanly onto a single physiological pathway. Instead, it represents a blended stimulus combining substantial external loading with meaningful accumulated work. Creatine kinase may capture part of the resulting muscle disruption or recovery demand, but it remains an indirect and highly variable marker; therefore, very large creatine kinase correlations are not necessarily expected, and moderate associations can still be informative within a multi-marker validity framework (Bourdon et al., 2017; Koch et al., 2014; Machado et al., 2012; Schoenfeld, 2010).

To better align the criterion with this mixed-domain construct, validity for ML was additionally evaluated using a rank-based composite biomarker index integrating creatine kinase,

lactate, and salivary cortisol. This composite showed a higher association with ML ($r = 0.673$, $p < 0.0001$) than any single biomarker alone. Conceptually, this makes sense because the moderate domain is intended to reflect concurrent contributions from metabolite accumulation (captured pragmatically by lactate), muscle damage or recovery demand (captured indirectly by creatine kinase), and a broader systemic stress response (captured by cortisol). Taken together, the pattern supports interpreting ML output as tracking a blended stress profile more effectively when the external criterion reflects multiple relevant pathways rather than relying on a single biomarker.

4.2.3 Low repetition load domain

LL showed the weakest and least consistent associations across the examined validity relationships. In the within-participant analyses, LL was negatively associated with lactate responses ($r = -0.621$, $p < 0.0001$) and salivary cortisol responses ($r = -0.215$, $p = 0.0321$), and it showed only a small positive association with creatine kinase responses ($r = 0.258$, $p = 0.0102$). Importantly, the negative associations did not indicate that tension dominant loading caused decreases in lactate or cortisol. Rather, they reflected the session contrast in this protocol, such that sessions with comparatively high LL values were the sessions in which acute lactate responses were comparatively small. Consequently, decreases in lactate are not a meaningful criterion for higher LL output, and lactate-based criteria primarily index metabolic perturbation rather than mechanical tension.

A key interpretive point is that this domain was intended to reflect high mechanical tension with comparatively low metabolic stress, as exemplified by the low repetition prescriptions with longer rest intervals. Under such conditions, a limited lactate response is expected relative to metabolically dense sessions, and the observed inverse association largely reflects the trade off in domain contribution across sessions. For cortisol, acute responses appear to be strongly influenced by training volume and metabolic density, with high volume and short rest protocols producing larger cortisol elevations, whereas strength type, low volume protocols often show minimal changes and can show decreases over the post exercise window, partly reflecting non-bout determinants and diurnal decline even under standardized sampling procedures (Ahtiainen et al., 2003; Scudese et al., 2016; Smilios et al., 2003). Creatine kinase is also an indirect and highly variable marker that reflects disruption and recovery demand rather than mechanical tension *per se* and pronounced creatine kinase responses are not consistently observed after all heavy load sessions,

particularly in trained individuals and when the stimulus is not novel (Machado et al., 2012; Nosaka & Clarkson, 1996).

Taken together, these findings suggest a criterion mismatch for LL, because the available biomarkers were not designed to directly index tension dominant internal load. The inverse lactate association therefore supports the intended construct only in a relative sense, namely that high LL sessions were not the sessions with high metabolic perturbation. The findings should not be interpreted in the sense that lactate can quantify tension dominant load. Future work should prioritize outcome measures that more directly reflect neuromuscular strain and mechanical tension-related stress, enabling a more appropriate validity test for the LL output.

4.2.4 Total training load

The rationale for computing differentiated training load domains was that resistance training is not a single stimulus. Common programming choices (repetition range, relative intensity, and rest interval structure) can produce meaningfully different acute physiological states, with implications for recovery, subsequent training quality, and longer-term adaptation (Grgic et al., 2018; Kraemer & Ratamess, 2005; Pareja-Blanco et al., 2019; Sánchez-Medina & González-Badillo, 2011). Accordingly, validity in this study is most informatively evaluated at the domain level using biomarkers that plausibly reflect domain-specific response profiles, rather than expecting a single global index to align strongly with any single physiological marker.

In the within-participant analyses, TL showed positive associations with all three biomarkers, including creatine kinase ($r = 0.475$, $p < 0.0001$), lactate ($r = 0.434$, $p < 0.0001$), and salivary cortisol ($r = 0.601$, $p < 0.0001$). However, because TL is defined as the mean of LL, ML, and HL, its correlations with single biomarkers are structurally constrained when biomarkers are domain-selective or show opposing directions across domains. In this dataset, lactate and cortisol were inversely associated with LL, and creatine kinase showed no meaningful association with HL. As a result, aggregation across domains can attenuate the observable association between TL and any single biomarker, even when the domain-specific outputs track their intended constructs well. Within this context, TL is best interpreted as a pragmatic session-level summary of overall internal demand, while domain-

specific validity patterns provide the more informative test of whether the calculator captures distinct physiological response profiles across session configurations.

4.3 Practical implications

The present results suggest that the training load calculator can provide practically relevant information for monitoring and session planning when direct physiological measurements are not feasible. However, implications should be interpreted within the constraints of a proof-of-concept validation under tightly standardized laboratory conditions and within the biomarker set used here.

The most promising validity evidence was observed for HL and ML. HL showed a close correspondence with lactate ($r = 0.856$) and a positive association with cortisol ($r = 0.583$), indicating that the calculator can differentiate sessions with a strong metabolic emphasis when work density and time under tension are high. Similarly, ML showed a convergent pattern across biomarkers, with positive associations for lactate ($r = 0.582$), creatine kinase ($r = 0.409$), and cortisol ($r = 0.557$). This combined evidence supports interpreting the moderate domain output as reflecting a mixed stress profile. Consistent with this interpretation, when the external criterion integrated all three biomarkers via a rank-based composite, the association with ML increased to $r = 0.673$, $p < 0.0001$, providing the strongest single validity statistic for this domain in the present study. In contrast, LL showed weak and partly inverse relationships with the available biomarkers (lactate $r = -0.621$, cortisol $r = -0.215$, creatine kinase $r = 0.258$). This pattern is most consistent with criterion mismatch rather than a definitive failure of the calculator, because the biomarker set does not directly index tension-dominant internal load. This limitation also constrains interpretation of TL, which is computed as the mean of the three domains. Although TL was positively associated with creatine kinase ($r = 0.475$), lactate ($r = 0.434$), and cortisol ($r = 0.601$), aggregation across domains can attenuate correlations with single biomarkers when biomarkers are domain selective or show opposing directions across domains.

Practically, this supports prioritizing domain-specific outputs for interpretation and using TL as a broad summary metric whose validity is necessarily bounded by the weakest criterion match among its component domains. Future work should therefore focus on identifying and validating external criteria that more directly reflect tension-dominant loading to enable a more complete evaluation of LL and, by extension, the TL construct.

4.4 Limitations

While the present investigation yielded promising proof-of-concept results for a novel framework to quantify resistance training training load, several limitations should be considered. Accordingly, the findings should be interpreted as preliminary validity evidence, and the current version of the training load calculator should be regarded as requiring further refinement.

First, inter-set rest duration was not considered in the current calculation model. This is a relevant omission because shorter rest intervals can substantially increase metabolic stress, such that a session performed in a low- or moderate-repetition range may elicit a markedly higher metabolic demand and a different overall stress profile than would be expected based on sets, repetitions, and relative load alone (de Salles et al., 2009; Fink et al., 2018; Marston et al., 2017; Sánchez-Moreno et al., 2023; Schoenfeld, 2010). In the present investigation, this limitation was of limited practical relevance because rest intervals were standardized and controlled across sessions. However, in applied training environments, rest intervals are often neither standardized nor routinely measured, which may become a major limitation when applying the training load calculations in real-world settings (Ibbott et al., 2019). Future versions of the training load calculator should therefore incorporate rest duration, or an equivalent proxy of work density, as an explicit input variable.

Second, several coefficients implemented in the current version of the calculator were based primarily on expert judgement rather than empirical derivation. Although the present validation suggests that the overall output produced plausible estimates of training load within the investigated protocol, the specific parameter values and weighting factors cannot be considered evidence-based at this stage. Future research should aim to systematically derive, test, and iteratively refine these coefficients, for example by calibrating them against external criteria and evaluating their robustness across exercises, loading schemes, and athlete populations. A pragmatic sequence would be to first establish evidence-based exercise coefficients, followed by exertion-related coefficients, and finally to evaluate repetition- and velocity-related coefficients in conjunction. Such stepwise evidence-based parameterization is likely to improve the validity and generalizability of the model outputs.

Third, the current model output represents an internal, model-specific scale and is therefore primarily comparable to outputs generated by the same model version. At present, direct comparability and transferability to other training load quantification approaches are limited, because no calibration or mapping to external metrics has been established. Future work should therefore evaluate how the model output relates to commonly used external load and internal load measures and, if warranted, develop conversion or normalization procedures (Bourdon et al., 2017).

Fourth, the biomarker criteria themselves impose important constraints on validity inference and likely contributed to the domain-specific pattern observed. Salivary cortisol and creatine kinase are influenced by multiple non-training determinants, and even under standardized sampling procedures can show substantial variability; moreover, acute cortisol responses to resistance exercise are strongly shaped by work volume and density (e.g., set volume, rest intervals) and can be minimal or directionally negative after low-volume, strength-type protocols when metabolic demand is low and the diurnal decline dominates the post-exercise window (Brancaccio et al., 2007; Gröschl et al., 2001; Hansen et al., 2008; Kirschbaum & Hellhammer, 1994; Kudielka et al., 2009; Mougios, 2007; Stalder et al., 2016). Creatine kinase should likewise be interpreted cautiously as an indirect marker of disruption and recovery demand, with large inter-individual variability and delayed kinetics; because only a single 24 h post-exercise value was obtained, the present CK change scores reflect early post-session changes rather than peak responses, which can occur later depending on bout characteristics and training status (Baird et al., 2012; Koch et al., 2014; Sayers et al., 2000; Schoenfeld, 2012). Moreover, although participants were instructed to refrain from strenuous physical activity for 48 h before each laboratory visit, this restriction may not have been sufficient to fully control for residual CK elevation from prior training, as CK can remain elevated for several days after hard exercise, particularly following eccentric or unaccustomed loading (Baird et al., 2012). Consequently, a portion of the observed 24 h CK change may have reflected carryover from antecedent training stress rather than the isolated effect of the standardized laboratory bout, adding noise to the CK criterion and potentially attenuating associations with the calculator outputs. In addition, analytic censoring introduced minor ceiling and floor effects: CK values above 1200 IU/L were recorded as 1200 IU/L (three observations), and lactate values below 0.5 mmol/L were recorded as 0.5 mmol/L. Together with the within-participant min–max scaling used to compute Δrel, these limits may have compressed variability at the extremes and attenuated associations for the affected sessions. These constraints are consistent with the criterion-mismatch interpretation for the LL and, by extension, help explain why TL

correlations with single biomarkers can be attenuated despite promising domain-specific evidence, including the stronger moderate domain association when the external criterion integrated all three biomarkers via rank-mean.

Fifth, although the frequent attainment of muscular failure in the high-condition sessions can be considered a methodological strength because proximity to failure materially influences the training stimulus and any training load quantification approach should be capable of reflecting this, it also introduced a limitation in the present protocol. Specifically, the prescribed target repetitions were frequently not achieved in the high-condition configurations, such that the realized repetition ranges deviated from the intended session design. This deviation resulted in higher LL estimates in the ML high-condition configuration and higher LL and ML outputs in the HL high-condition configuration than originally intended. Consequently, control over the applied stimulus was reduced and interpretation of domain-specific contrasts is partly constrained because the performed work did not always correspond to the planned repetition structure. At the same time, these deviations may be viewed as an ecologically valid feature of resistance training, and a valid and reliable training load calculator should be able to accommodate incomplete repetition prescriptions due to failure while still producing meaningful estimates.

4.5 Conclusion

This thesis developed a novel resistance training training load calculator designed to estimate internal training stress from observable session variables. The model integrates volume, intensity (via a velocity-based pathway with an RPE substitution option), exertion (via velocity loss or RPE-derived proximity to failure), and exercise selection, and provides three domain-specific indices (LL, ML, HL) plus TL reflecting their mean. By structuring outputs along a continuum from tension-dominant to metabolically demanding work, the calculator aims to reflect that resistance training is not a single uniform stimulus and that common programming choices can meaningfully shift the dominant stress profile within a session.

Criterion validity was evaluated in a standardized laboratory protocol using barbell compound exercises and systematic manipulation of repetition range, %1RM, and rest intervals. Within-participant analyses indicated a coherent and physiologically plausible pattern of associations between calculated load outputs and biomarker responses. HL

aligned closely with lactate and was positively associated with salivary cortisol, supporting its intended role as an index of metabolic stress under dense, high-repetition loading. ML showed convergent positive associations with lactate, cortisol, and creatine kinase, consistent with a mixed stress profile. Importantly, validity evidence for this mixed-domain construct strengthened further when a rank-based composite biomarker criterion was used, highlighting that multi-pathway constructs are more appropriately evaluated against integrated external criteria than against single biomarkers alone.

In contrast, LL demonstrated weaker and partly inverse associations with lactate and cortisol and only a small positive association with creatine kinase. This pattern is best interpreted as a limitation of the external criteria rather than as a definitive failure of the low-repetition construct, because the selected biomarkers primarily reflect metabolic disturbance, endocrine stress, and delayed muscle disruption and therefore provide only an indirect and potentially mismatched criterion for tension-dominant loading. Consequently, the present results support the broader approach of domain-specific quantification, while also showing that a complete validity assessment requires outcome measures that more directly capture neuromuscular strain and tension-related stress.

Overall, the findings provide proof-of-concept validity for the calculator under controlled conditions and support continued development toward applied monitoring use. Priorities for refinement include incorporating work-density information (e.g., rest duration or equivalent proxies), empirically calibrating coefficients and scaling rules, and extending validation to broader training contexts, exercises, and populations. Future validation work should combine biomarker criteria with more construct-matched external outcomes for tension-dominant loading to enable a more complete evaluation of LL and, by extension, to strengthen interpretability of TL as an aggregated session-level summary.

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