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From Variability to Precision: A Conceptual Framework, Methodological Considerations and Analytical Approaches in Personalised Resistance Training Prescription

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ABSTRACT

The advancement of personalised resistance training is hindered by a pervasive methodological limitation, namely the conflation of external exposure (nominal prescriptions such as sets, repetitions, and load) with the latent internal biological dose. Inspired by established frameworks in medicine and epidemiology, this article introduces a conceptual framework to resolve this limitation by redefining resistance training dose as an internal biological stimulus that can be inferred using mechanistically relevant mechanicaland physiological proxies. Building upon this framework, the multiple sources of individual level variability in delivered dose and observed response—including between-individual dispersion, individual-by-treatment interaction (treatment-contrast heterogeneity), and day-to-day fluctuations—are systematically mapped. Using real-world data, different trial designs are evaluated for their ability to identify and estimate these variance components via mixed-effects modelling. Consistent with the statistical structure of unreplicated crossover designs, single crossover trials are methodologically limited for evaluating individualised responses to training. The illustrative analyses make this limitation explicit in applied data, showing that such designs cannot fully separate true between-individual differences in treatment contrasts from measurement error and day-to-day noise. Consequently, estimating stable individual-by-treatment differences requires replication within individuals and treatments, with replicate crossover designs and statistical partial pooling providing a stronger basis for distinguishing meaningful treatment-contrast heterogeneity from noisy individual estimates. Finally, after distinguishing the estimands and variance components relevant to personalised prescription—including average treatment effects, stable individual-by-treatment differences, and within-individual fluctuations—autoregulatory paradigms are identified as a candidate vehicle for delivering more consistent internal stimuli across time by dynamically aligning prescribed exposure with individuals' fluctuating biological state. Ultimately,

advancing precision resistance training requires abandoning the assumption that a uniform prescription delivers a uniform dose. Instead, the field must embrace both between-individual heterogeneity and within-individual fluctuation as fundamental biological realities, systematically investigating these sources of variance through more robust experimental designs and analytical methods.

1. INTRODUCTION AND SCOPE

Major professional and public health organisations endorse resistance training as an effective exercise modality for enhancing physical fitness and athletic performance, and for providing health benefits across the lifespan in both healthy and clinical populations [1–3]. Advancing how resistance training is prescribed has led to a growing interest in *precision-oriented* (e.g., stratified or subgroup-based) and *personalised* (e.g., individual specific) approaches. Traditionally, resistance training prescription has relied on static frameworks, operationalised through fixed percentages of one-repetition maximum (1RM) and predetermined volume schemes, which fail to account for daily fluctuations in physical capacity. In response, autoregulatory methods—including rating of perceived exertion (RPE), repetitions in reserve (RIR), and velocity-based training (VBT)—have gained prominence. These approaches represent a critical step forward by dynamically adjusting training loads to accommodate fluctuations in physical capacity.

However, to advance current autoregulatory models further and to conceptualise more capacity-responsive personalisation, it is necessary to situate resistance training within a broader conceptual landscape, drawing on frameworks from both medicine and epidemiology. A core element of this perspective requires delineating the constructs of *treatment*, *exposure*, *dose*, and *response*. In resistance training, the *treatment* is typically the prescribed programme or rule (e.g., a fixed number of sets and repetitions, the instruction

to terminate a set at a 20% velocity loss, or at 2RIR). The causal pathway to outcomes, however, runs through what is actually performed—*the exposure* (e.g., the absolute load lifted on a given day)—and the resulting internal stimulus experienced by the individual—*the dose* (e.g., the mechanical or metabolic stress). This dose ultimately accumulates into longer-term adaptation, or *the response*. Viewed through this lens, while autoregulation successfully individualises the daily exposure, it often assumes that the prescribed treatment target (e.g., the specific velocity loss or RIR target) provides the optimal dose across an entire cohort. More complete personalisation requires moving beyond solely adjusting daily exposures to meet a uniform benchmark; it necessitates determining whether the optimal target itself differs systematically between individuals and within the same individual over time.

Delineating this pathway has two immediate implications for research on personalisation. First, acute studies are uniquely valuable for isolating the treatment → exposure → dose segment of this pathway, allowing dose expression and execution variability to be examined without the additional complexity of accumulated adaptation. Second, because real-world training is inherently variable, evaluating personalisation requires not only estimating average treatment differences, but also decomposing the between- and within-individual sources of variation in delivered dose, dose proxies, or dose-related outcomes. This decomposition determines whether individual deviations are meaningful and repeatable, or merely transient noise.

Consequently, this article explores how different research designs—particularly replicate crossover structures—can identify these sources of variance and support inference at multiple levels. In replicate crossover designs, repeated exposures to each resistance training programme enable estimation of (i) population-average differences in achieved dose (fixed effects), (ii) systematic effect modification consistent with precision-oriented approaches (moderators/strata), and (iii) individual-specific deviations (individual-level

random effects). Crucially, these designs can help separate these factors from within-individual instability across repeated exposures and residual fluctuations across multiple units of work (e.g., set-to-set, repetition-to-repetition), provided there is sufficient replication at the relevant level. In this way, the proposed modelling strategy is intentionally multi-level: it conceptualises *personalisation* not as a single estimand, but as an inference built from evidence about average effects, structured heterogeneity, individual deviations, and repeatability of those deviations. Ultimately, we outline future directions for integrating these principles into resistance training research and practice to strengthen claims about personalised prescription.

2. PERSONALISATION IN PERSPECTIVE: LESSONS FROM MEDICINE

The conceptual rationale underpinning personalised medicine offers a useful framework for understanding developments in resistance training prescription. Personalised medicine is commonly defined as the tailoring of medical care to optimise therapeutic benefit for specific patients or patient groups, typically leveraging genetic, molecular, or physiological information [4]. In practice, this approach involves matching treatments to the characteristics of each patient by using validated biomarkers—measurable indicators of biological states or conditions [5,6]. Knowledge of a disease's underlying molecular pathology informs therapy selection, with therapies often combined to address an individual patient's specific profile [7,8]. The patient's response is then monitored, typically through surrogate measures, allowing treatment to be continually adapted to optimise outcomes [9]. However, more complete individualisation requires both a mechanistic understanding of relevant biomarkers and the practical capacity to monitor and act upon them in real time. In many clinical contexts, such information is either incomplete, too costly to obtain, or not actionable at the individual level. When individual-level tailoring is unfeasible, precision strategies—such as

stratification by age, sex, or prognosis, or biomarker-defined subgrouping–serve as an intermediate step, enabling more targeted treatment than population-level guidelines, even if they fall short of true personalisation.

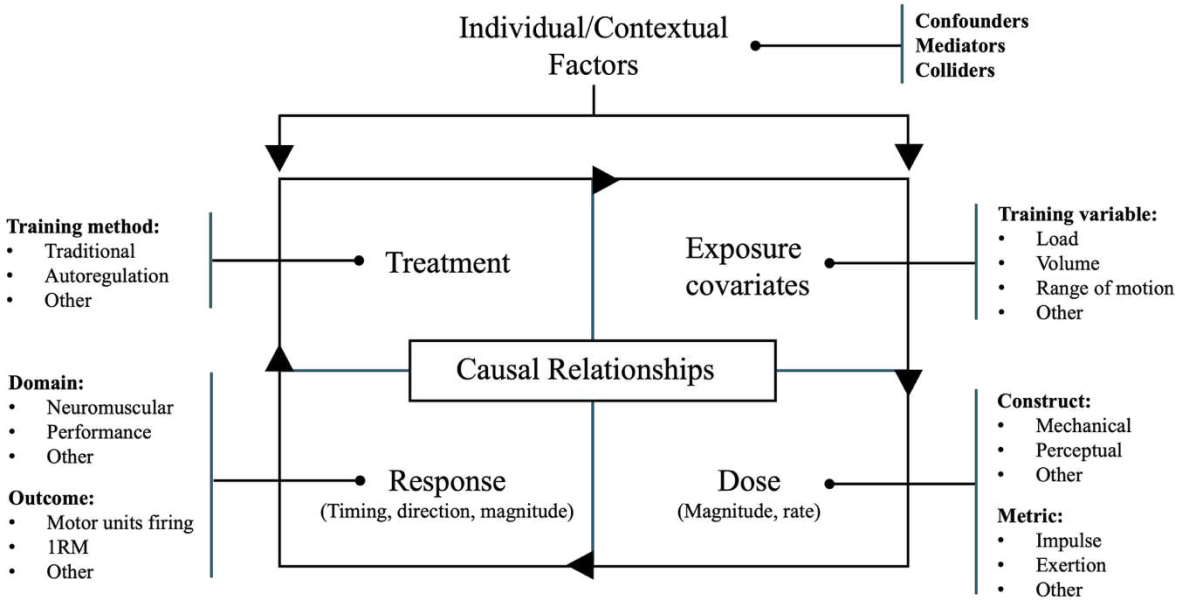
A parallel approach has been proposed in sport and exercise science [10–12]. While sport science currently lacks a widely validated, measurable set of biomarkers capable of predicting individual trainability or reliably forecasting an individual's response trajectory to a given training stimulus [13–15], the guiding principles remain comparable. Here, decision-making requires combining evidence-based insights on the causal effects of training interventions with a pragmatic approach that adjusts training variables according to the individual's unique needs, preferences, goals, and acute responses[16–18]. Just as patient responses in clinical settings are tracked using surrogate measures, the individual's acute responses to repeated training stimuli can be monitored using proxy measures of resistance training dose (see Section 3.1 – Resistance training exposure and dose). Conceptual models (e.g., the fitness–fatigue model) and computational approaches [19,20], developed to characterise the training process, can then be applied to prescribe personalised interventions and iteratively refine them over time [21]. By analogy with this medical framework, *personalised resistance training* can be defined as the continuous and iterative tailoring of resistance exercise prescription (i.e., prescribed and performed exposure, with the aim of influencing the internal dose) to the individual. This process should be based on an individual's baseline capacity, predicted and observed responsiveness to specific interventions, and intended outcomes (fitness, performance, or health).

3. FOUNDATIONS OF RESISTANCE TRAINING PRESCRIPTION: THE TREATMENT-EXPOSURE-DOSE-RESPONSE RELATIONSHIP FRAMEWORK AND OPERATIONAL DEFINITIONS

Sport and exercise practitioners across recreational, performance, and clinical domains commonly draw upon what is traditionally labelled the *dose–response framework* to operationalise causal relationships between resistance training and resulting physiological, biomechanical, psychological, anthropometric, and physical outcomes. However, as previously established, we distinguish between exposure and dose, and consider the response as the *magnitude* (acute), *accumulation* (chronic), and *direction* of change in the causal effects induced by training, reflecting both positive (fitness) and negative (fatigue) influences [20]. Thus, in the context of resistance training, a more precise formulation is the t*reatment-exposure-dose-response relationship framework* illustrated in Fig. 1.

This conceptual schematic highlights the overall process: programme-level treatment shapes the exposure performed, which in turn determines the proximal dose experienced (magnitude/rate) and the resulting response (domain → outcome [timing, direction, magnitude]). It also illustrates where key measurement choices enter the pathway (training variables → constructs → metrics) and how individual and contextual factors may shape both what is performed and how it translates into dose and response.

Fig. 1 Treatment-exposure-dose-response framework



To ensure conceptual clarity throughout this article, we establish the following operational definitions:

- Treatment: A resistance-training condition defined by the programme features and targets applied to an exercise/session/block (e.g., set structure, loading scheme, velocity-loss threshold, inter-set rest prescription, tempo constraints). Treatments may be contrasted as discrete methods (e.g., traditional vs intra-set rest-redistribution) or parameter levels (e.g., 30% vs 70% 1RM; 20% vs 40% velocity loss).
- Exposure: The external training performed under a treatment, characterised by the training variables delivered and accumulated over the relevant time scale (e.g., exercises, sets, repetitions, external load, range of motion, tempo/time under tension, inter-set rest, and session frequency). Exposure may be summarised (e.g., total repetitions, total lifted load, session duration) but is not assumed to reduce to a single product term.
- Dose: The proximal stimulus experienced by the individual arising from the interaction between the exposure and the organism. Dose is a latent construct and is therefore inferred from mechanistically grounded proxies (mechanical, metabolic, neuromuscular, perceptual), expressed in terms of magnitude and rate/time-course (e.g., force–time/impulse characteristics, time under tension, muscle activation proxies, metabolic disturbance proxies).
- Response: The change attributable to the dose in physiological, neuromuscular, perceptual, or performance state. Responses can be acute (immediate perturbation) and/or chronic (accumulated adaptation), and are characterised by their magnitude, direction, and timing, including both fitness-enhancing and fatigue-related components.
- Outcome(s): Measured variables used to operationalise and quantify the response in the target domain(s). Outcomes may be acute or chronic and may include, for example:

physiological (heart rate, blood pressure, recovery kinetics); biomechanical (ground reaction forces, kinematics/kinetics, joint moments); psychological/perceptual (RPE, pain, readiness/fatigue ratings); anthropometric (body mass, lean mass estimates); and physical performance (1RM, jump height, sprint time, power output). Notably, several measures (e.g., velocity, impulse, RPE) may act as dose proxies or outcomes depending on the analytic target and time scale.

- Individual/contextual factors (exposure covariates): Participant and setting characteristics that can influence what exposure is achieved and/or how exposure translates to dose and response (e.g., training status, fatigue/sleep, motivation, technique, equipment constraints, supervision).

As discussed in Section 2, the central challenge in resistance training is that a key element of the relevant causal processes—specifically the mechanical dose—cannot be monitored continuously at the muscle-fibre level. The latent mechanical dose captures the magnitude and rate of mechanical tension that produces force and/or length change, triggering the cascade of biological responses and adaptations [22–24]. In the absence of direct measurement, the resistance training literature has systematically conflated exposure with dose. The "dose" has typically been characterised using the mode of training, external load intensity, and training volume (sets and repetitions). However, this is conceptually problematic, as reducing the external exposure to summary metrics—such as calculating "volume load" as a single product term—falsely assumes that different combinations of sets, repetitions, and loads yield biologically equivalent stimuli. While these operational definitions are practical and widely used, they capture only the external work performed (the exposure*).* Thus, they offer an incomplete, and often misleading approximation of the latent internal dose that drives adaptation.

Therefore, in the following subsections, we seek to advance the discussion by revisiting and clarifying the concepts of resistance training exposure and dose, highlighting their frequent but problematic interchangeable use. Moreover, we propose a conceptually grounded set of resistance training dose metrics capable of more accurately capturing the causal pathways linking the training stimulus to the resulting response—that is, the acute and chronic changes, positive or negative, that constitute adaptation.

3.1. Resistance training exposure and dose

To contextualise the concepts of resistance training exposure and dose, we draw on definitions from epidemiology and those more recently proposed by Impellizzeri and colleagues within the sport and exercise science domain [25]. A standard definition of exposure in epidemiology is *"the amount of a factor to which a group or individual was exposed, sometimes contrasted with dose, the amount that enters or interacts with the organism [26]."* This definition highlights a key distinction: exposure refers to the external agent, factor, or distal cause of a response, whereas dose denotes the internal and more proximal cause of adaptations mediated by various physiological processes.

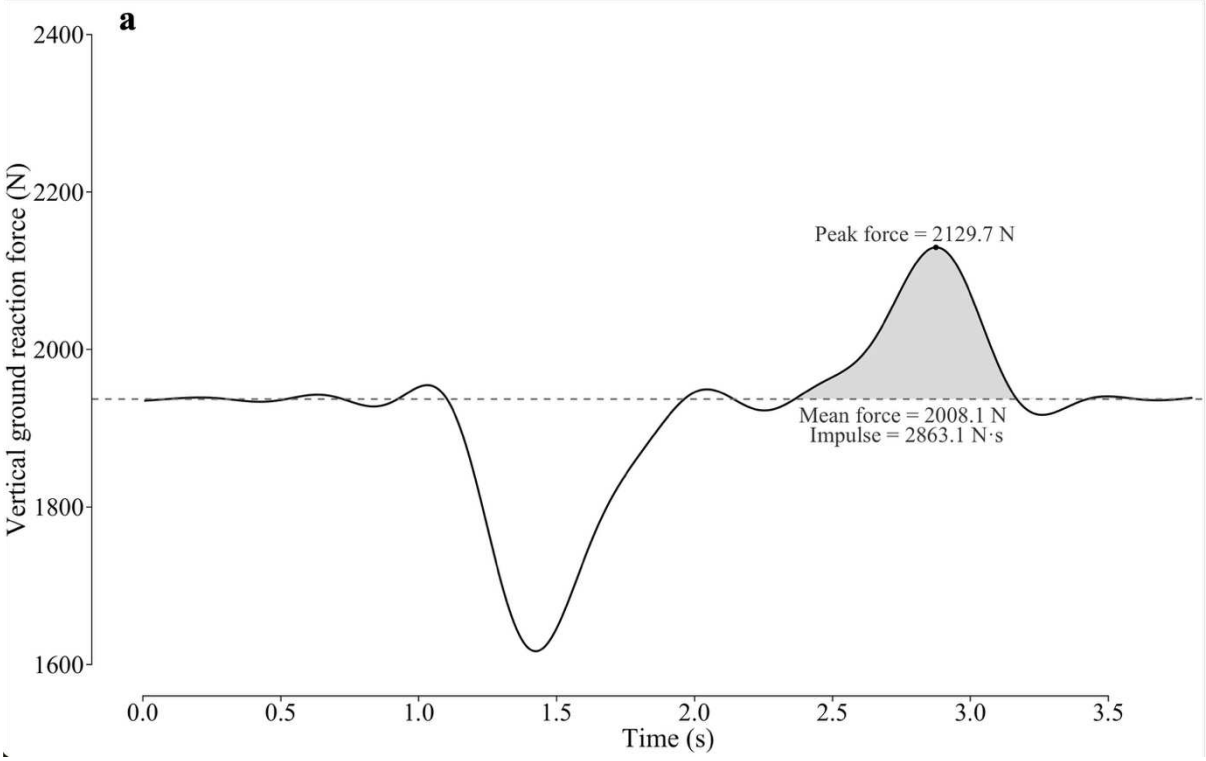
In resistance training, however, these two concepts are often used interchangeably. Typically, exposure is treated as if it were dose, with cumulative training stimuli operationalised solely through load intensity and volume. In this framing, what is labelled as dose is calculated as the product of load intensity (i.e., absolute load) and training volume (e.g., frequency, number of exercises, sets, and repetitions). This interpretation is adopted in meta-research to infer so-called dose–response relationships between resistance training and relevant performance or clinical outcomes [27–101]. In our view, while this interpretation aligns conceptually with *cumulative exposure [102]*—where the training variables function as

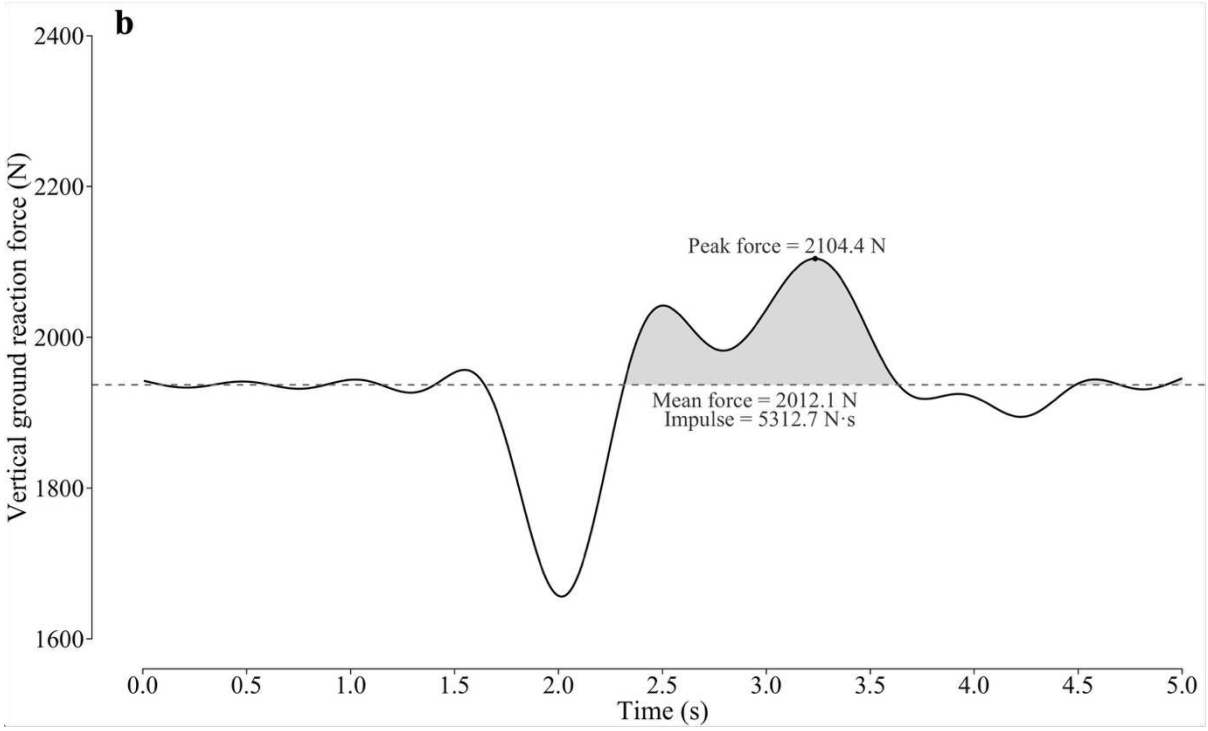
exposure covariates manipulated at the level of a single exercise, session, or training period—it does not translate directly to the concept of dose and carries important limitations [102,103].

The first limitation is the omission of a temporal component, which in epidemiology is routinely integrated as *duration* when characterising exposure or dose over time [104]. In the resistance training context, this temporal dimension manifests macroscopically as time under tension—the sum of eccentric (braking), isometric, and concentric (propulsive) phases across repetitions during which skeletal muscles are actively recruited to resist, hold, or overcome the external resistance across a full or partial length excursion [105]. Microscopically, at the level of a single repetition, this temporal dimension interacts with force to generate impulse (i.e., a *mechanical construct and dose metric*; Fig. 1). Given that the duration of mechanical tension contributes to the accumulated magnitude andtime-course of the mechanical stimulus experienced by the musculoskeletal system [24], and because biological responses to mechanical stress in humans are dynamic and non-linear [106], integrating *duration* is critical for capturing adaptive processes [23,96,107–110]. Therefore, neglecting this temporal dimension hinders both the accurate quantification of resistance training dose proxies and the valid investigation of the dose–response relationship. Moreover, omitting the temporal dimension implicitly assumes mechanical homogeneity across the prescribed training volume. This would imply identical force–time profiles across repetitions within a set and between sets performed at the same load intensity. Such an assumption is clearly flawed, as substantial evidence demonstrates large repetition-to-repetition variability in commonly used resistance training metrics, including force, velocity, power, and impulse [111,112]. For example, Fig. 2 illustrates the force–time profiles of the first (panel a) and last (panel b) repetitions in a barbell back squat set performed to failure at 75% 1RM. While only small differences in peak and mean force are evident, the impulse differs by approximately two-fold between repetitions. By analogy with pharmacology—where each unit of treatment

represents a dose and its effects are mediated by multiple interactions within the organism—the "dose" associated with each repetition cannot be considered constant. Instead, it depends on multiple training- and non-training-related factors.

Fig. 2 Force-time profiles of first (a) and last (b) repetitions in a set performed to failure





Another fundamental limitation of equating cumulative external exposure with dose is that it fails to capture between-individual variability in task-specific capacity [113,114]. This is evident when considering the substantial differences in the maximum number of repetitions individuals can complete at a given relative load intensity. For instance, two individuals performing an exercise to task failure at 75% 1RM may be capable of completing different numbers of repetitions, with one terminating at 8 repetitions and another reaching 16 [114]. If both were prescribed 3 sets of 10 repetitions, the first individual would be unable to complete the prescribed set volume even at maximal effort, whereas the second would complete the same prescription with comparatively less effort. Thus, although the prescribed exposure (i.e., relative load, sets, and repetitions) is identical, the resulting dose—both mechanical (the cumulative magnitude and rate of mechanical tension across repetitions) and perceptual (the internal exertion)—that initiates the biological process of adaptation would remain markedly different for each individual. This example illustrates why distinguishing between exposure

and dose is essential, as the same prescribed exposure can produce substantially different effective doses across individuals.

The interchangeable use of exposure and dose is further challenged by evidence from alternative set structures, such as intra-set rest-redistribution training. When matched for external load intensity, exercise volume, and total rest duration, intra-set rest-redistribution and traditional set structures yield different mechanical, metabolic, perceptual, and performance outputs (i.e., *dose constructs and metrics*; Fig. 1) [115,116]. This demonstrates that identical external exposures can elicit markedly different doses depending on how the training stimulus is organised and delivered. Collectively, this evidence and other related research [117] highlight the mismatch between cumulative exposure and the true resistance training dose. It demonstrates that distinct underlying mechanisms may produce either similar adaptations through different pathways or divergent adaptations despite apparently identical exposure [22,118,119]. This underscores the need to refine the conceptualisation and measurement of dose in resistance training, just as precision medicine distinguishes between prescribed treatment exposure and the biologically active dose that drives the adaptive response.

3.2. Resistance training dose metrics

While the central role of mechanical stimuli in driving adaptation is well established [120,121], the direct in vivo measurement of mechanical dose at the cellular or muscle level remains infeasible in applied contexts [122]. Consequently, indirect approaches have been developed [123]. Kinetics combined with inverse dynamics can estimate net joint moments from ground reaction forces and motion capture, but these reflect only net torque and require assumptions about muscle-level distribution [124–127]. Musculoskeletal models incorporate anatomical and physiological parameters into optimisation techniques, yet their accuracy depends on individual-specific parameterisation and assumed cost functions, making them

highly sensitive to input uncertainty [125,128]. Electromyography-based methods can approximate muscle activation, but surface electromyography is prone to cross-talk and placement errors, while intramuscular electromyography is invasive and unsuitable for field use [129–131]. These limitations highlight the need for alternative strategies to quantify proxy measures of dose that are both mechanically valid and practically feasible.

In applied settings, resistance training practitioners rarely possess the means, nor the explicit need, to quantify individual muscle forces; instead, they aim to obtain reliable and actionable metrics that guide programming, provide immediate feedback, and track adaptation over time. Contemporary measurement systems—including force plates, strain gauges, position transducers, accelerometers, and gyroscopes—allow the measurement of force and velocity (or their rotational counterparts, such as torque and angular velocity), two fundamental physical quantities governed by Newton's laws of motion [132]. These measures, while not direct reflections of local tissue loading, represent systemic proxies for the mechanical dose delivered during training and align with practitioners' interests in evaluating task-specific force production and movement execution. This perspective mirrors the central aim of resistance training: to enhance the ability to produce higher velocities against a given load or to lift heavier loads at a given velocity, both of which relate to the generation of momentum in mechanical terms. According to Newton's second law of motion (eq.1), which can be rearranged to express momentum (eq.2) and further linked to the impulse–momentum relationship (eq.3), producing a given change in velocity requires maximising the force applied to the mass over the time available [133]:

$$\sum \boldsymbol{F} = m\boldsymbol{a}. \quad \text{(eq.1)}$$

$$\sum \boldsymbol{F} = m\frac{d\boldsymbol{v}}{dt} = \frac{d\boldsymbol{p}}{dt}, \text{where } \boldsymbol{p} = m\boldsymbol{v} \quad \text{(eq.2)}$$

$$\int_{t_0}^{t_1} \sum \boldsymbol{F}\, dt = \Delta \boldsymbol{p} \rightarrow \sum \boldsymbol{F}\, \Delta \boldsymbol{t} \text{ (impulse)} = \boldsymbol{m}\Delta \boldsymbol{v} \text{ (momentum)} \quad \text{(eq.3)}$$

where $\boldsymbol{F}$ is the force applied, $\boldsymbol{a}$ is the acceleration, $\boldsymbol{p}$ is the momentum, $\boldsymbol{t}$ is the duration of force application, $\boldsymbol{m}$ is the mass, and $\boldsymbol{v}$ is the resulting velocity. In line with this perspective, two metrics are particularly relevant:

- Total impulse is defined as the time integral of the net force applied throughout the duration of the task:

$$\text{RTDose}_{\text{magnitude}} = \int_{t_0}^{t_1} \boldsymbol{F}\,(t)dt \quad \text{(eq.4)}$$

- Time-restricted impulse is defined as the time integral of the net force applied within a predefined time interval of the task (e.g., early-phase [0-100ms] as in eq.5 below):

$$\text{RTDose}_{\text{rate}} = \int_{t_0}^{t_{100}} \boldsymbol{F}\,(t)dt \quad \text{(eq.5)}$$

Together, these metrics provide complementary insights: total impulse reflects the overall magnitude of the dose, while time-restricted impulse captures the temporal dynamics of its delivery. Both can be derived readily from existing measurement technologies, directly relate to performance outcomes, and can be normalised through scaling or allometric methods to facilitate within- and between-individual analyses, as well as investigations of chronic adaptations to training. The analogy with epidemiology is evident: just as cumulative and rate-based metrics distinguish the magnitude and temporal delivery of a therapeutic dose in determining outcomes, total and time-restricted impulse represent the resistance training counterparts that underpin movement execution and drive adaptation. Moreover, detailed analyses of the impulse curve—including its shape and partitioning across distinct movement phases—can enhance the specificity of these

metrics, offering a more contextualised understanding of the mechanical foundations of movement [134].

Leveraging the impulse–momentum relationship, these metrics can also be expressed in terms of momentum. Assuming a constant mass against which force is applied (as in free weights or weight-stack machines), the resulting mean velocity of the system reflects dose magnitude, while time-restricted velocity integrals (or early-phase kinematics) reflect the temporal delivery of the dose. When direct force measurements (e.g., force platforms) are unavailable, velocity-tracking technologies provide a feasible alternative. This constitutes the mechanical rationale for velocity-based resistance training, a widely adopted approach that traces its origins back to the pioneering work of González-Badillo [135], and has expanded with the availability of affordable, commercially accessible devices [136]. Importantly, load–velocity profiling shows strong longitudinal validity, as changes in velocity along the load-velocity profile correspond to changes in 1RM, the gold-standard measure of maximal strength [136,137].

Nevertheless, methodological considerations remain, particularly regarding the validity and reliability of velocity-tracking devices [137,138], and the fact that they usually measure only the uniaxial velocity of an external implement (e.g., barbell, weight stack). Whole-system velocity (body mass plus external load) is approximated only when the implement and lifter move together in the same direction [139]. Moreover, while mean velocity provides a practical proxy for dose magnitude when the system is in motion (i.e., absolute velocity greater than zero), it fundamentally fails to capture the overall dose during isometric phases under external loading, where substantial force and impulse are produced without displacement. This limitation can be addressed by accounting for the net impulse (e.g., beyond the impulse required to counteract body weight) required to sustain the external load over the duration of the isometric action. Finally, the equivalence between velocity-derived metrics and impulse

holds only under constant-resistance conditions. In training modalities where resistance varies dynamically with displacement, inertia, or pressure (e.g., flywheel devices, elastic bands, pneumatic machines), the direct mapping of velocity and acceleration onto impulse metrics is not straightforward, and alternative metrics or calculations are required.

In conclusion, when direct quantification of muscle-level dose is not feasible, the integration of total impulse and time-restricted impulse—or their velocity-based surrogates under constant-resistance conditions—offers a conceptually grounded and practically feasible approach for monitoring the training stimulus—provided their scope and limitations are made explicit. These metrics can capture essential dimensions of mechanical loading and align with the practical needs of practitioners, thereby bridging the gap between mechanistic understanding and applied monitoring.

4. MAPPING VARIABILITY IN RESISTANCE TRAINING: SOURCES OF VARIATION, TRIAL DESIGNS, ANALYTICAL APPROACHES AND INSIGHTS

4.1 Overview, motivation, and acute focus

With dose now defined and operationalised through conceptually defensible and practically measurable proxies, the focus shifts from what to quantify to what can be learned from quantifying it. Accordingly, in this section, we review study designs and statistical considerations for quantifying individual differences in delivered dose or dose-related proxies following a *single* resistance training session. The applied priorities are threefold: (i) optimisation, which leverages emerging theory, technology, and practice to improve dose delivery at the group level; (ii) individualisation, which seeks to understand stable between-individual differences in dose and substantive between-individual heterogeneity; and (iii) consistency, which evaluates the extent to which different

methods influence variability across sessions and across repeated units of work within a session. Addressing these priorities requires study designs and analytical approaches that can quantify not only average differences between training prescriptions, but also their stability, their dispersion across individuals, and the conditions under which they are most effective at producing the intended internal stimulus.

Our approach is informed by the methodological contributions of Senn [140], Atkinson [141,142], and Hecksteden [143], and more recently contextualised within sport, exercise, and nutrition science by Steele [144], Robinson [145] and Lolli [146], who have addressed the question of inter-individual variability in the long-term effects of exercise and nutritional interventions. We build on these perspectives by focusing on an acute, single-session context. Acute designs provide a resource-efficient way to characterise how a proposed alteration to training, relative to a reference method, changes the magnitude and stability of the delivered dose at both the group and individual level. This acute "screening" step can help prioritise which candidate treatment contrasts warrant longer-term evaluation. Importantly, we do not assume a linear relationship between acute dose magnitude and chronic phenotypic adaptation, as biological saturation and recovery kinetics also mediate long-term outcomes [106]. Rather, once the most promising contrasts (and potential effect modifiers) are identified in the acute setting, a smaller number of chronic trials can be used to evaluate whether these acute dose signatures persist with repeated exposure and translate into meaningful differences in adaptation.

Focusing on the acute setting also creates an opportunity to examine within-session variability, which has received comparatively little attention. Even when the prescribed external exposure is nominally repeated, the delivered dose can fluctuate across repeated units of work performed minutes apart under the same session context. Because resistance training is inherently multilevel, with repetitions nested within sets, sets within exercises, and exercises

within sessions, dose can be conceptualised and aggregated at multiple hierarchical levels (e.g., per repetition, per set, per exercise bout, or session total). At any chosen level of aggregation, repeating the same prescription yields a distribution of delivered doses rather than a single fixed value. Understanding this within-session distribution is essential; if the random intra-individual variability (noise) exceeds the systematic difference between treatments or individuals (signal), claims of individual-specific prescription effects cannot be statistically substantiated.

4.2 Mapping dose–response variability: variance sources and trial designs

With this motivation in place, a useful next step is to distinguish the main sources of variation that can contribute to observed differences in delivered dose within and between individuals, and to map each source to the minimal replication required for identification. In the acute-design setting, we treat delivered dose (or a dose proxy) as varying across multiple levels: between treatments, between individuals, across repeated sessions within individuals, and across repeated units (i.e., sets or repetitions) of work within a session. We organise this variability into five components that align naturally with mixed-effects decompositions:

- Source 1 (between-treatment effect): the mean shift in delivered dose attributable to the alternative training method.
- Source 2 (between-individual dispersion): stable between-individual differences in typical dose under a given method.
- Source 3 (individual-by-treatment interaction variance): heterogeneity in the treatment contrast (individual differences in how the alternative method changes delivered dose);
- Source 4 (day-to-day/session variability): within-individual fluctuation across repeated sessions under the same method.

- Source 5 (within-session variability): fluctuation across repeated units of work within a session.

A detailed treatment of the underlying variance-decomposition framework—including notation, mixed-effects specifications, identifiability conditions under common acute designs, and worked examples—is provided in the Supplementary Information File 1.

A distinctive feature of resistance training is that Source 5 can be conceptualised across multiple hierarchical levels because the training hierarchy is explicit: repetitions nested within sets, sets within exercises, and exercises within sessions. Accordingly, within-session variability may refer to repetition-to-repetition variation within a set, set-to-set variation within an exercise, or variation across exercise bouts within a session total. Despite this natural hierarchy, most studies implicitly treat a session's delivered dose as a single value (or average). This erroneously assumes mechanical homogeneity and therefore leaves the distribution of within-session dose—its spread, skew, and dependence on fatigue—largely unquantified. We argue that explicitly modelling within-session variability complements treatment-effect questions by revealing whether methods differ not only in mean delivered dose, but also in the stability and predictability with which a prescription translates into a realised stimulus.

4.2.1 Design targets in acute resistance training studies: mapping questions to variance sources

In line with previous literature, we highlight the need to consider study design and replication at multiple levels. A simple two-condition crossover with one session per condition can estimate the mean contrast (Source 1) and between-individual dispersion (Source 2) in a typical delivered dose, but cannot separate stable contrast heterogeneity (Source 3) from day-to-day variability (Source 4) without replication. Estimation of this variability component requires repeated sessions under the same method. Furthermore, within-session variability (e.g. set-level; Source 5) is only separable from residual noise when there is an additional nested

level beneath sets (e.g., repetitions within sets) that induces within-set correlation. This mapping between variance sources and replication level motivates increasingly informative acute designs and corresponding mixed-effects specifications, and clarifies what each component enables in practice—from identifying methods that increase dose on average, to understanding how individuals differ under a given method, determining whether a prescription stabilises dose across sessions or within sessions. From an applied standpoint, acute resistance training studies typically aim to answer four related design questions:

1. Which training method yields a greater average delivered dose across many individuals?
2. For a given individual, which training method is expected to yield a greater delivered dose?
3. Which training method yields a more consistent delivered dose across many individuals?
4. For a given individual, which training method is expected to yield a more consistent delivered dose?

These questions are best viewed as applied targets for the sources of variation represented by Sources 1–5. In brief, Question 1 primarily concerns the between-treatment effect (Source 1). Question 2 may initially be informed by the same population-average contrast, but individual-specific inference additionally requires designs that can quantify stable individual-by-treatment differences (Source 3). Questions 3 and 4 concern consistency in dose delivery and therefore depend on the extent to which designs can quantify between-individual dispersion in typical dose (Source 2), session-to-session variability (Source 4), and within-session variability (Source 5), alongside residual error.

Accordingly, we provide the detailed design–model correspondences, notation, and mixed-effects specifications in the Supplementary Information File 1, including worked examples

showing which components are identified under common acute designs and what assumptions are required when replication is limited. In the main text, we illustrate the framework using original and secondary resistance training data to show how these design choices translate into inference for the four core questions.

4.2.2 Single crossover trial

We begin with the simplest design, a single crossover trial in which each individual completes both treatments once, in a randomised order (AB/BA). This design supports straightforward estimation of the mean treatment contrast in delivered dose at the group level (Source 1), addressing the question: *"Which training method yields a greater average delivered dose when implemented across many individuals (Question 1)?"* What can be learned about other variance components, however, depends strongly on the level at which the dose is recorded. If the unit of interest is the set (e.g., whether delivered dose changes across sets within a session), then estimating within-session set-to-set variability separately from residual noise typically requires replication at the level below the set—most naturally, repetition-level measurements. In practice, this lower-level replication can make the variance decomposition identifiable and may reduce the risk of boundary estimates (e.g., variance components collapsing to zero) in mixed-effects models. The key limitation of a single crossover is the absence of replication across occasions: day-to-day/session variability (Source 4) cannot be estimated, and therefore cannot be separated from other sources of variation. This limitation most directly affects inference about contrast heterogeneity (individual differences in the treatment effect; Source 3), because with only one exposure to each treatment, the observed within-individual contrast is inevitably entangled with session-specific deviations. As a result, apparent between-individual variation in treatment effects should generally be treated as exploratory, and strong claims about stable individual differences typically require a replicate crossover design.

To illustrate the analytical boundaries of a single crossover, we reanalyse the data from Dello Iacono and colleagues [147]—comparing mean velocity (i.e., a proxy for delivered dose magnitude; See Section 3.2) in the barbell back squat performed under a traditional set structure (TRA: 3 × 8 repetitions) and an intra-set rest-redistribution structure (IRR: 6 × 4 repetitions) matched for total volume (24 repetitions), load intensity (75% 1RM), and overall rest duration (240 s). Because key training variables were deliberately controlled, this design naturally supports analyses that target the mean treatment contrast in delivered dose without adjustment for additional exposure covariates (e.g., sets or repetitions). At the same time, even in this simplified setting, the concept of "greater dose" can correspond to multiple, closely related estimands. For example, dose can be operationalised as an overall session-average (grand mean) magnitude, as the average dose within a defined segment of the session (e.g., early repetitions or the "fresh" portion of each set), or as differences in the dose profile across repetition position (i.e., how dose evolves over the set and/or session). Distinguishing these estimands clarifies what each analysis is estimating and makes explicit how different modelling choices map onto different substantive questions. In Table 1, we present the comparison of mean dose between treatments, operationalised as the rep-weighted average velocity across all 24 repetitions within a session. On average across individuals, the intra-set rest-redistribution structure produced a higher delivered dose than the traditional set structure.

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Table 1. Model-based estimates addressing applied questions on average delivered dose and dose consistency comparing intra-set rest-redistribution (IRR) with traditional (TRA) resistance training in a single crossover trial

Question	Model	Estimate	Value (95% CI)
Which training method yields a greater average delivered dose when implemented across many individuals (Question 1)?			
Rep-weighted	Session_Mean ~ Treatment + (1|ID)	Session mean contrast (IRR-TRA)	0.05 (0.02 to 0.07) $m \cdot s^{-1}$
Which training method yields a more consistent delivered dose across many individuals (Question 3)?			
Set-to-set variability across all sets	log(set_sd) ~ Treatment + (1|ID)	Session SD (IRR/TRA)	1.9 (1.4 to 2.4) ratio
Set-to-set variability across sets 1-3	log(set_sd) ~ Treatment + (1|ID)	Session SD (IRR/TRA)	1.7 (1.3 to 2.2) ratio
Repetition-level velocity across all sets	Velocity ~ Treatment + (1|ID) + (1|ID:Treatment) + (0 + IRR + TRA || ID:Treatment:Set)	Set-to-set variability (IRR-TRA)	0.02 (0.01 to 0.03) $m \cdot s^{-1}$
Repetition-level velocity across sets 1-3	Velocity ~ Treatment + (1|ID) + (1|ID:Treatment) + (0 + IRR + TRA || ID:Treatment:Set)	Set-to-set variability (IRR-TRA)	-0.00 (-0.03 to 0.01) $m \cdot s^{-1}$
Repetition-level velocity across all sets	Velocity ~ Treatment + f(sets) + (1|ID) + (1|ID:Treatment) +	Set-to-set variability (IRR-TRA)	-0.02 (-0.04 to -0.00) $m \cdot s^{-1}$

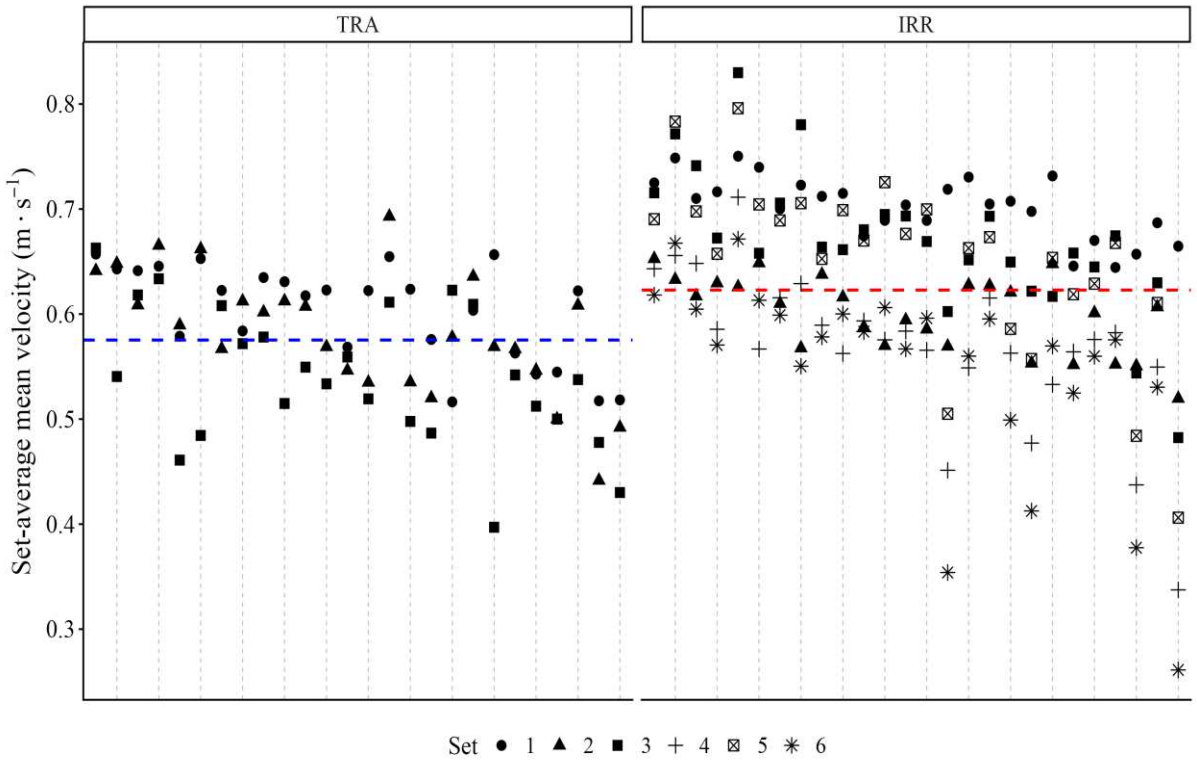
	(0 + IRR + TRA || ID:Treatment:Set)

For models fitted to log(set_sd), the estimand is naturally expressed on the ratio scale; accordingly, estimates and 95% confidence intervals were obtained by exponentiating the treatment coefficient, such that values greater than 1 indicate greater set-to-set variability under IRR than TRA. For repetition-level models, set-to-set variability (IRR − TRA) represents the absolute difference between the treatment-specific estimated set-level standard deviations, expressed in $m \cdot s^{-1}$. In the model syntax, ID, ID:Treatment, and ID:Treatment:Set denote the grouping structures used for the random effects. The term (0 + IRR + TRA || ID:Treatment:Set) specifies separate treatment-specific set-level variance components, allowing estimation of different set-to-set variability for IRR and TRA. CI: confidence interval.

Within-session dose consistency

Moving from mean effects to variability, the question becomes more nuanced. In addition to increasing the average delivered dose, an applied consideration is whether a training method alters the consistency of dose delivery across sets (Question 3). In this context, a natural question is whether set-to-set variation differs under IRR compared with TRA. A simple and transparent approach is to compute, for each individual and treatment, the standard deviation of set-average velocity across sets within the session (Fig. 3).

Fig. 3 Set-by-set mean velocity in traditional and intra-set rest-redistribution structures during single crossover



Because standard deviations are strictly positive and often skewed, and typically scale with the mean (heteroscedasticity), the logarithm of this quantity can be modelled, with a random intercept for individuals to account for the correlated nature of the data within individuals. This transformation is important as it ensures we are evaluating true relative consistency rather than

simply capturing scaling artefacts driven by the higher mean velocity in one of the set structures, as in this example. Different estimands arise depending on how sets are treated. One option is to restrict the analysis to sets common to both protocols (e.g., sets 1–3), providing a like-for-like comparison of set positions. Alternatively, all sets can be included, acknowledging that IRR contains more sets when the total volume is fixed, and that this structural difference is itself part of the training intervention. As shown in Table 1, both approaches indicate greater set-to-set variation under IRR relative to TRA.

Formal variance decomposition

To examine within-session variability more formally, mixed-effects models can be used to decompose variance into components attributable to individuals, sessions, sets, and residual variability. When only set-average values are available (one observation per individual × treatment × set), a random effect for set within session is not identifiable, because there is no replication within each set. A heteroscedastic model can instead be fitted, allowing residual variability to differ by treatment (see Supplementary Information File 1). However, this approach estimates differences in residual variability of set means rather than isolating a distinct set-to-set variance component, and therefore provides a broader measure of dispersion rather than a direct estimate of between-set variability.

In the present dataset, repetition-level velocity values are available, providing replication within sets. This additional structure allows estimation of a set-level variance component within a multilevel model. When a model is fitted without accounting for systematic differences across set number or repetition position, the estimated set-to-set variance is larger under IRR (Table 1: 0.02; 0.01 to 0.03 $m \cdot s^{-1}$). However, once fixed effects for set number are included, much of this apparent excess variability is explained by predictable structural features of the protocol, and the difference in residual set-to-set variance diminishes or reverses (Table

1: -0.02; -0.04 to -0.00 $m \cdot s^{-1}$). These findings illustrate an important principle, that reliability is not a single, intrinsic property of a treatment. Rather, it depends on how variability is defined and what aspects of the structure are treated as systematic versus random. Consequently, selection of an estimand should be guided not only by statistical considerations, but by the practical decision context—specifically, whether the concern is total within-session fluctuation in delivered dose, or unpredictable variability beyond expected fatigue patterns. Ultimately, variance components must be interpreted within a broader conceptual model linking the distribution of acute dose to long-term adaptation.

Between-individual dispersion

Between-individual dispersion in delivered dose is another source of variation that may be of practical interest in delivered dose. In applied settings, such as team environments, it may be advantageous to implement training methods that reduce dispersion across individuals, thereby promoting a more uniform stimulus within the group. The example dataset provides another clear illustration of how conclusions depend on the estimand. When between-individual dispersion is estimated across the full protocol, including all IRR sets (1–6), the variability across individuals is greater under IRR than under TRA (0.062 and 0.042 $m \cdot s^{-1}$, respectively). However, when the analysis is restricted to the sets common to both treatments (sets 1–3), this pattern reverses, and between-individual dispersion is smaller under IRR (0.042 and 0.043 $m \cdot s^{-1}$, respectively). Additionally, when all sets are included but fixed effects for set position are incorporated to account for average set structure, the reverse pattern becomes clearer, and between-individual dispersion is now even greater under TRA compared to IRR (0.019 and 0.044 $m \cdot s^{-1}$, respectively). Taken together, these findings indicate that modelling set structure alters estimates of between-individual dispersion, but does not remove between-individual differences in how individuals respond to the additional sets in the IRR protocol. In other words, IRR may produce a more uniform dose across individuals in the

680 comparable early portion of the session, yet simultaneously amplify divergence in later sets
681 that are unique to the protocol. As with within-session variability, between-individual
682 dispersion is therefore not an inherent property of a treatment; it depends on how exposure
683 structure is defined and which portion of the protocol is considered.

684 *Treatment-contrast heterogeneity and limits for personalisation*

685 The sources of variation described above provide substantive information for comparing
686 treatments and informing training prescription at the group and session level. However, for
687 personalisation, a central quantity of interest is treatment-contrast heterogeneity, describing the
688 extent to which the within-participant difference between treatments varies across individuals.
689 If this heterogeneity is substantial, a further applied question is which individuals exhibit the
690 largest estimated contrasts, since these individuals would, in principle, be those for whom
691 protocol selection matters most. In the present dataset, treatment-contrast heterogeneity can be
692 quantified, and several estimands can be used to compute individual-specific treatment
693 differences and rank individuals accordingly. These estimates can also be interpreted relative
694 to the mean treatment difference and other variance components to assess whether between-
695 individual differences are likely to be practically meaningful. However, as previously
696 established, with a single crossover, any individual-specific contrast necessarily conflates true
697 individual differences with day-to-day variability and measurement error. In resistance training
698 settings, day-to-day fluctuations in performance are often non-trivial; together with
699 measurement error, these fluctuations inflate apparent contrast heterogeneity and substantially
700 reduce confidence in ranking individuals or concluding that any one individual has a reliably
701 larger (or smaller) treatment difference than another. It is for this reason that a replicate
702 crossover design is required to more rigorously evaluate treatment-contrast heterogeneity and
703 support personalisation.
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4.2.3 Replicate crossover trial

In a replicate crossover trial, each participant completes both treatments on at least two occasions across two or more randomised cycles. Conceptually, this design allows researchers to estimate not only the mean treatment contrast for each individual (Question 2), but also whether mean treatment contrasts are stable across repeated exposures (Question 4). In practice, replication enables more defensible estimation and separation of three key sources of variability: (a) the individual-by-treatment interaction standard deviation or variance component (Source 3), reflecting contrast heterogeneity in individual responses; (b) day-to-day or session variability (Source 4), reflecting within-individual fluctuations across repeated sessions under the same treatment; and (c) within-session variability (Source 5), reflecting fluctuations across repeated units of work within a session (e.g., multiple sets or repetitions).

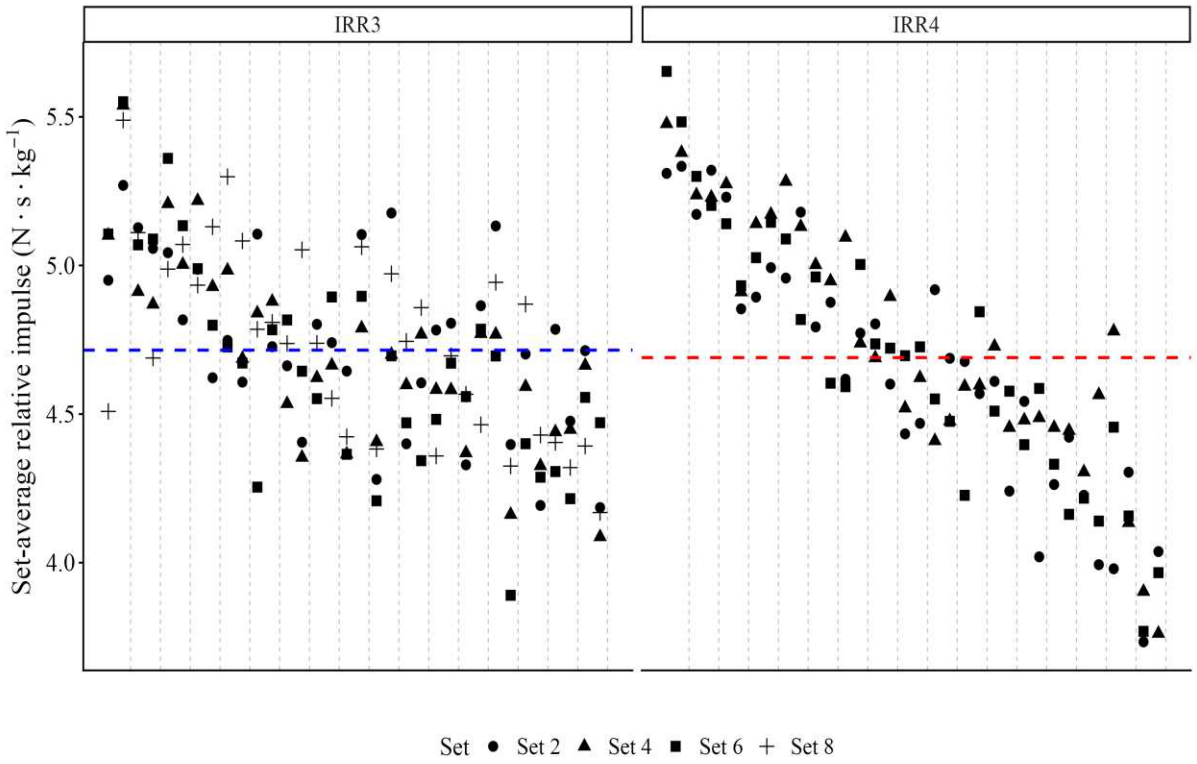
Contrast heterogeneity and the role of replication

In this example, we focus on contrast heterogeneity and illustrate why replication is important for obtaining stable estimates of the parameter most relevant for personalisation. We analyse unpublished real-world data of relative impulse collected during Smith machine back squats performed by seventeen male professional soccer players. The study used a replicate crossover design comparing two intra-set rest-redistribution structures: 6 × 4 (repetitions) with 48 s rest between sets (IRR4) and 8 × 3 (repetitions) with ~34 s rest between sets (IRR3). The protocols were matched for total volume, load intensity (80% 1RM), and total rest duration (240 s).

The set-average raw data are presented in Fig. 4. At the group level, there appears to be minimal difference in mean relative impulse between the two protocols. However, a small mean difference does not imply a lack of meaningful variability across individuals. That is,

some athletes produce greater relative impulse under IRR3, whereas others produce greater relative impulse under IRR4. When averaged across the group, these opposing mechanical dose outputs can cancel out, yielding a small overall mean difference. From an applied perspective, the important question is therefore whether such individual differences are substantive and, crucially, whether they are stable across repeated exposures.

Fig. 4 Set-by-set mean impulse comparing two intra-set rest-redistribution structures during a replicate crossover



Note: Grey vertical lines separate participants for the replicated crossover in each condition. To assist with clarity, only sets 2/4/6/8 are illustrated.

Single crossover: conflation of variance sources

To illustrate the conflation of variance sources in a single crossover, we first analyse the data as if only one exposure to each treatment were available. Using mean relative impulse for each set (i.e., multiple observations per session) allows estimation of contrast heterogeneity

745 even without replication of the crossover, and these results are presented in Table 2. It is
746 important to note that within the treatment–exposure–dose pathway, many practitioners would
747 consider the different set–repetition structures—and the individual-specific performance
748 patterns that may arise from them—as the intervention itself. Consequently, statistically
749 controlling for set position would inappropriately regress away the treatment contrast we want
750 to estimate. In this analysis, the model without fixed effects for set position indicates substantial
751 treatment contrast heterogeneity, with the estimated standard deviation of the individual-by-
752 treatment interaction (0.45; 0.28 to 0.61 $N \cdot s \cdot kg^{-1}$) exceeding both the between-individual
753 dispersion (0.20; 0.11 to 0.27 $N \cdot s \cdot kg^{-1}$) and the residual standard deviation (0.19; 0.17 to 0.20
754 $N \cdot s \cdot kg^{-1}$).
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Table 2. Model-based estimates addressing applied question on contrast heterogeneity and scope for personalisation comparing IRR3 versus IRR4 in a replicate crossover trial

Question	Model	Estimate	Value (95% CI)
For a given individual, which training method is expected to yield a greater delivered dose (Question 3)?			
Contrast heterogeneity (Single crossover)	Set_mean ~ Treatment + (1 + Treatment |ID)	Between-individual dispersion	0.20 (0.11 to 0.27) $N \cdot s \cdot kg^{-1}$
		Contrast variability	0.45 (0.28 to 0.61) $N \cdot s \cdot kg^{-1}$
		Residual variability	0.19 (0.17 to 0.20) $N \cdot s \cdot kg^{-1}$
Contrast heterogeneity + day-to-day variability (Replicate crossover)	Set_mean ~ Treatment + (1 + Treatment |ID) + (1|ID:Treatment:Session)	Between-individual dispersion	0.18 (0.09 to 0.27) $N \cdot s \cdot kg^{-1}$
		Contrast variability	0.38 (0.22 to 0.53) $N \cdot s \cdot kg^{-1}$
		Day-to-day variability	0.14 (0.09 to 0.18) $N \cdot s \cdot kg^{-1}$
		Residual variability	0.19 (0.17 to 0.20) $N \cdot s \cdot kg^{-1}$

For variance-component models, estimates and 95% confidence intervals are presented on the standard deviation scale, such that larger values indicate greater dispersion in the corresponding source of variability. In the single crossover model, the random intercept for ID estimates between-individual dispersion, the random slope for Treatment estimates contrast variability, and the residual term represents within-individual variability that cannot be decomposed further in the absence of replication. In the replicate-crossover model, the additional term (1 | ID:Treatment:Session) estimates day-to-day/session-to-session variability, allowing this source to be separated from stable between-individual differences in the treatment contrast. Accordingly, contrast variability in the replicate crossover model reflects stable between-individual differences in the treatment effect after accounting for day-to-day fluctuations, whereas in the single crossover model these sources remain partially conflated. Table 2 therefore summarises contrast heterogeneity rather than individual treatment rankings. Individual-specific treatment contrasts can, however, be derived from the same mixed-effects model as BLUPs, which more directly address the applied question for a given individual and are displayed in Fig. 5. CI: confidence interval.

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Replicate crossover: separating variance components

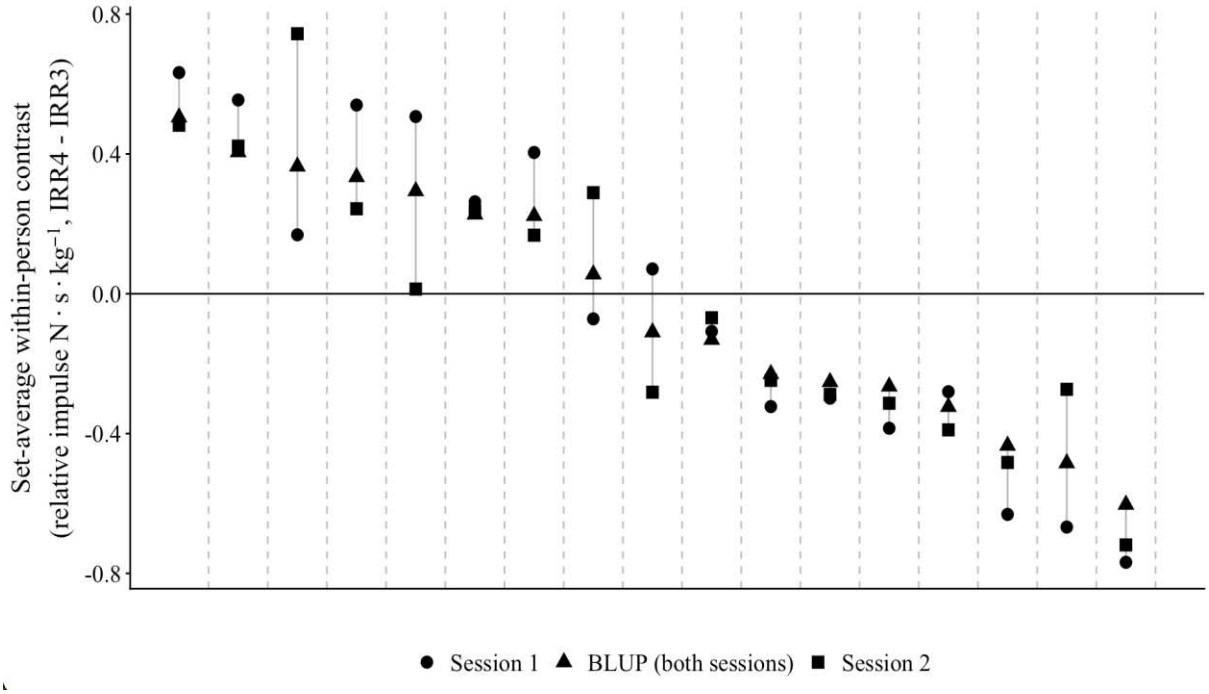
However, with only a single exposure per treatment, the estimated contrast heterogeneity also contains day-to-day fluctuations and measurement error. These stochastic components do not reflect stable individual differences in treatment response but instead inflate the apparent spread of individual-specific contrasts. With replication, the model separates these variance components. Specifically, the inclusion of a session-specific term captures day-to-day fluctuations within each individual and treatment, allowing this variability to be distinguished from stable individual-by-treatment interaction effects. In the present data, daily fluctuations are substantial (0.14; 0.09 to 0.18 $N \cdot s \cdot kg^{-1}$) and reduce the estimated contrast heterogeneity (0.38; 0.22 to 0.53 $N \cdot s \cdot kg^{-1}$) compared with the single-crossover analysis (0.45; 0.28 to 0.61 $N \cdot s \cdot kg^{-1}$). Nevertheless, the individual-by-treatment interaction standard deviation remains the largest estimated variability component, indicating meaningful and persistent individual differences in the delivered dose under the two protocols.

Estimating individual effects: BLUPs and shrinkage

Fig. 5 illustrates these results by showing treatment contrasts calculated separately for the first session, the second session, and the combined estimate across both sessions. For several individuals, the calculated contrasts differ substantially between sessions, highlighting the influence of day-to-day variation. To obtain more stable individual estimates, we use best linear unbiased predictors (BLUPs) derived from the mixed-effects model. These estimates represent model-based conditional predictions of each individual's treatment contrast after accounting for session-specific fluctuations and other sources of variability. Importantly, BLUPs are not simply the average of the two observed session contrasts; rather, they combine information from both sessions while partially pooling across individuals according to the estimated variance components of the model. A defining feature of BLUPs is statistical shrinkage; extreme individual estimates that are driven largely by day-to-day

variation are pulled (shrunk) back toward the group mean. This shrinkage reduces the risk of practitioners from falsely categorising an individual as a "high responder" simply because they had one exceptionally "noisy" testing or training session. Consequently, BLUP-based estimates are more appropriate for ranking individuals and informing personalisation decisions than raw contrasts derived from single observations, although their uncertainty should still be considered when making individual-level decisions.

Fig. 5 Individual treatment contrasts across sessions and model-based estimates (BLUPs) in the replicate crossover design



Implications for personalisation

From a research perspective, comparing the magnitude of contrast heterogeneity with the mean treatment difference and other variance components provides insight into whether personalisation is likely to be meaningful in practice. When contrast heterogeneity is large relative to other sources of variability, practitioners may benefit from collecting individual data and comparing treatments directly to inform the selection and refinement of training protocols

over time. In summary, replicate crossover designs provide an essential methodological tool for distinguishing stable individual differences in treatment response from transient fluctuations due to day-to-day variability or measurement error, yielding more credible estimates for personalisation.

4.3 Design issues in randomised crossover trials

The design of single and replicate randomised crossover trials is not exempt from challenges. Different research questions, target populations and subpopulations, treatments, and outcomes of interest demand tailored design choices. The purpose of this section is to highlight key methodological issues that warrant careful consideration when designing such trials in resistance training research.

4.3.1 Population and familiarisation

Participants must be familiarised with resistance training and at (or close to) a steady state before trial initiation. A steady state refers to a condition in which performance and dose responses are sufficiently stable across repeated exposures, thereby minimising confounding from early-phase learning or rapid adaptations. It is well established that force production capacity can increase within a few weeks of training, primarily through neural mechanisms, even in the absence of substantial morphological changes [148–152]. Consequently, to study treatment-specific effects and individual contrasts between resistance training treatments, participants should first undergo a familiarisation phase—typically a few weeks of training under both the reference and experimental modalities—to ensure stable baseline performance across all testing conditions. Without this, observed differences may reflect learning effects or novelty rather than stable treatment responses.

4.3.2 Carry-over and sequence effects

A second consideration is carry-over, defined as the extent to which the effects of one treatment session persist and influence outcomes in subsequent sessions. Addressing acute physiological carry-over requires establishing an adequate wash-out period, which in acute resistance training contexts generally ranges between 48 and 96 hours, depending on the training modality and the outcomes of interest [153–155]. In practice, resistance training recovery kinetics may be monitored using the load–velocity profile, examining the velocity associated with submaximal loads during the warm-up or the velocity of the fastest repetition at the fixed external load selected during the experimental session [134,156,157]. Practically relevant thresholds may be used to operationalise sufficient recovery and adjust session timing or scheduling if recovery is incomplete. However, it must be noted that while wash-out periods mitigate transient fatigue, they cannot erase asymmetric factors such as strategic learning (e.g., altered pacing strategies) acquired during the first session. Researchers should therefore counterbalance treatment orders and sequence and period effects when evaluating whether first-order carryover or learning may be influencing the treatment contrast.

4.3.3 Time-varying confounding

Resistance training is rarely performed in isolation; it is often combined with other training modalities within the same day (e.g., concurrent endurance and strength sessions) or across consecutive days. While a primary advantage of crossover designs is that they control for stable, time-invariant confounders (e.g., genetics, training history) by having participants act as their own controls, they remain vulnerable to time-varying confounding. This occurs when external factors fluctuate between sessions—such as prior training load, accumulated fatigue, or poor sleep—acting as common causes that influence both the delivered treatment exposure and the outcome of interest (e.g., dose measure). For example, in a trial comparing traditional

and autoregulation training methods on mean relative impulse in highly trained distance runners, failing to account for fluctuations in cumulative training load or pre-session fatigue between testing or training days may bias conclusions. Fatigue can alter the number of repetitions performed before terminating a set, modify responses under autoregulated protocols (e.g., repetitions in reserve), and impair mechanical outputs [111,158–160]. Thus, without accounting for time-varying confounders that simultaneously influence exposure and outcome, estimates of treatment effects and inter-individual variability may be biased. Careful consideration of the causal pathway, as well as explicit assessment of measured and unmeasured confounders, is required. While a comprehensive discussion of causal inference is beyond the scope of this article, we encourage readers to consider causal inference frameworks and graphical causal models (e.g., directed acyclic graphs) to inform appropriate analytical strategies [161].

4.3.4 Sample size

Sample size planning is critical. Replicate crossover designs, while better suited for disentangling individual response variability from within-individual variability, require substantial sample sizes and/or repeated observations to achieve the desired precision in estimating variance components. It is important to note that studies adequately powered to detect a mean treatment effect (Source 1) are almost universally underpowered to detect individual contrast heterogeneity (Source 3); hence, estimating variance components requires a substantially larger sample size than estimating group means. This is because precision for variance components depends not only on the number of participants but also on the number of replicate cycles per participant. Although no dedicated guidelines currently exist for acute resistance training research, principles of power analysis and trial design have been extensively discussed by Senn [162], Zucker [163] and Swinton (currently only available as a non-peer

reviewed preprint [164]). This literature provides detailed guidance on sample size estimation, thresholds for practically relevant responses, and the balance between trial feasibility and statistical power. Their principles can be directly applied to resistance training contexts and should form the basis for planning robust studies in this area.

5. SUMMARY AND FUTURE DIRECTIONS IN PERSONALISED RESISTANCE TRAINING

This paper provides researchers and practitioners with a conceptual framework to guide the design and evaluation of treatment-exposure-dose-response relationships in resistance training. Our aims are threefold: 1) to stimulate critical reflection, 2) to encourage methodological rigour, and 3) to inform best practice.

Adopting an epidemiological perspective, we challenge the prevailing definition of resistance training dose, highlight the frequent misinterpretation of cumulative external exposure as dose, and propose a conceptually grounded and operationally valid set of proxy measures for quantifying resistance training dose. Focusing on core research questions related to optimisation and individualisation, we present methodological designs and analytical strategies capable of decomposing the multiple sources of variance inherent in resistance training interventions. Specifically, we illustrate how acute single and replicate crossover trials can quantify the mean treatment contrast and multiple variance components, including between-individual variance, treatment-contrast heterogeneity, day-to-day/session-to-session variance, within-session dose variability (e.g., set-to-set and repetition-to-repetition), and residual variance. Such variance decomposition is essential for distinguishing stable between-individual differences in treatment contrasts from random within-individual fluctuations. We argue that greater precision, repeatability, and predictability in delivered dose or dose proxies are important prerequisites for credible individualised prescription. Accordingly, reducing unwanted variability—both within sessions (exercise-, set-, and repetition-level) and

across repeated sessions (day-to-day fluctuations)—is fundamental. Greater dose precision enhances our capacity to refine training methods through deliberate manipulation of exposure covariates and improves the accuracy with which we contrast reference methods against alternative comparators.

To advance personalised resistance training, we propose two primary directions for future research:

1) Autoregulation as a path to precision. We propose autoregulation as a primary direction for future research to improve the precision and individualisation of training prescription. Autoregulation operates at two complementary levels [19]: 1) Between-session autoregulation, whereby training prescriptions are adjusted based on prior knowledge of an individual's predicted susceptibility to training or their longitudinal response profile, and 2) Within-session autoregulation, whereby individuals modify load, volume, rest, or other exposure covariates in real time according to objective proxies for dose (e.g., impulse), performance indicators (e.g., repetitions performed against velocity loss thresholds), or subjective proxies for dose (e.g., perceived exertion or repetitions in reserve). By dynamically aligning exposure covariates and delivered dose with an individual's current physiological and performance state, autoregulation has the potential to reduce unnecessary variability (both between- and within individuals), improve dose precision, and enhance the repeatability and predictability of resistance training outcomes.

Future research should therefore aim to:

- Quantify how autoregulatory strategies influence variance components of the delivered dose.
- Determine whether such strategies reduce individual-by-treatment interaction variability or instead amplify stable and practically relevant between-individual differences.

- Establish whether improvements in dose precision translate into superior long-term adaptations.

2) Methodological and analytical frontiers. Advancing personalised resistance training will require the integration of mechanistically informed training paradigms, rigorous experimental designs, and robust statistical modelling. Finally, the skillsets and knowledge of researchers, practitioners, and data analysts must be harmonised to align relevant research questions, appropriate study designs, estimand selection, and analytical strategies. It is also important to note that, whilst mixed-effects models provide a flexible framework for addressing a wide range of practical questions, the accurate estimation of variance components is particularly sensitive to the quantity and structure of the available data. In resistance training research, this challenge is often exacerbated by relatively small sample sizes and limited replication, which can lead to imprecise or unstable estimates of key parameters. Future research is therefore required to identify typical study designs and resource requirements necessary to obtain sufficiently precise estimates of these variance components. In addition, Bayesian statistical approaches warrant further investigation in this context, as they allow prior information—derived from previous studies, mechanistic understanding, or expert knowledge—to be incorporated into the analysis. Even when such information is used primarily to regularise estimates, Bayesian methods may improve the stability and interpretability of variance component estimates in small-sample settings that are common in resistance training research.

6. CONCLUSIONS

The pursuit of personalised resistance training has historically been constrained by a fundamental methodological limitation, namely, the conflation of external exposure (i.e., the prescribed training parameters) with internal dose (the latent biological stimulus experienced by the individual). As long as the field continues to treat nominal prescriptions—such as sets,

repetitions, and load—as the biological dose, claims of individualisation remain conceptually and statistically difficult to substantiate.

Inspired by frameworks from medicine and epidemiology, this article presents a conceptual framework for moving beyond this limitation. By redefining dose as a latent internal stimulus that can be inferred using mechanistically relevant proxies, such as impulse, the pathways linking training exposure to adaptation can be investigated more precisely. Importantly, quantifying this dose or its proxies represents only one part of the challenge; validating personalised interventions demands analytical models capable of separating stable individual differences from random day-to-day noise and measurement error. As demonstrated, single crossover trials are insufficient for this task when the aim is to make strong claims about stable individual treatment contrasts. Consequently, the advancement of precision resistance training relies on great use of replicate crossover designs, variance decomposition, and autoregulatory paradigms that dynamically align the prescribed exposure with the fluctuating biological state of individuals. Ultimately, personalisation necessitates a methodological shift: the assumption that a uniform prescription delivers a uniform dose must be abandoned in favour of rigorously quantifying the variance that shapes the precision, repeatability and interpretation of responses to and adaptations from resistance training.

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