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Anaerobic Speed and Power Reserve for Individualizing High Intensity Training in Trained Athletes: A Systematic Review

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Anaerobic Speed and Power Reserve for Individualizing High Intensity Training in Trained Athletes: A Systematic Review

Running heading：ASR and APR for Individualizing High Intensity Training

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Abstract

Background

Anaerobic speed reserve (ASR) and anaerobic power reserve (APR) locate prescribed exercise intensity between individual aerobic and maximal sprint performance boundaries. Whether they provide additional value over conventional individualized intensity anchors for high intensity training in trained athletes remains uncertain. This review evaluated acute response standardization, chronic adaptation magnitude and dispersion, prediction, training decisions, and longitudinal monitoring.

Methods

Six databases were searched from inception to 7 August 2026, with supplementary reference list screening. Eligible peer reviewed studies examined dimensionally compatible reserve constructs in trained athletes. Risk of bias was evaluated using design appropriate tools and certainty using GRADE. Chronic maximal oxygen uptake (VO_2max) adaptation was pooled as a postintervention mean difference (MD). Dispersion of individual percentage change was synthesized using the coefficient of variation ratio (CVR), with variability ratio (VR) sensitivity analysis.

Results

Twenty four reports representing 23 independent studies were included. Five randomized studies involving 104 participants in the primary comparisons contributed to the chronic VO_2max meta analysis. The pooled MD was 1.00 $mL \cdot kg^{-1} \cdot min^{-1}$ (95% CI −0.03 to 2.03), showing no clear mean advantage with low certainty. Reserve based prescription was associated with lower observed adaptation dispersion (CVR 0.311, 95% CI 0.229 to 0.423),

supported by VR analysis (0.38, 95% CI 0.29 to 0.51); certainty was moderate. Chronic sport specific performance showed no consistent mean advantage. Three direct acute comparisons yielded inconsistent response standardization findings. Evidence for incremental prediction, profile guided allocation, and longitudinal monitoring was limited. In nine active training arms with complete component data, the aerobic anchor improved more than the maximal anchor and the derived reserve narrowed.

Conclusions

Reserve based prescription was associated with lower observed between athlete dispersion in aerobic adaptation, while mean VO_2max improvements did not clearly differ from conventional individualized prescriptions. These findings suggest that ASR/APR may offer additional value in improving the consistency of aerobic training outcomes among trained athletes.

Registration

PROSPERO CRD420261471635.

Key Points

- Across five randomized studies, ASR/APR based training prescriptions were associated with approximately 69% lower observed relative dispersion in VO_2max adaptation, suggesting greater consistency in aerobic fitness gains across athletes.
- Reserve based prescription showed no clear advantage in average aerobic fitness or sport performance gains, highlighting response consistency as a potentially valuable dimension of individualized training.

- Whether ASR/APR profiles can improve the prediction of training responses, inform training selection, and guide prescription adjustments over time remains an open question.

Keywords

Anaerobic speed reserve; Anaerobic power reserve; High intensity interval training; Individualized exercise prescription; Interindividual variability; Training adaptation; Systematic review; Meta analysis

1 Background

High intensity interval training is widely used to develop aerobic fitness and the capacity to sustain high intensity exercise in trained athletes [1]. Exercise intensity can be individualized using different physiological and performance based anchors (e.g. ventilatory or lactate thresholds) depending on the targeted intensity domain [2]. At submaximal intensities, physiological thresholds (e.g. the second lactate threshold and respiratory compensation point), together with $\dot{V}O_2$ kinetics, can inform individualized training prescription [2-4]. Intensity can also be prescribed relative to two physiological boundaries using the delta concept (e.g., Δ50, corresponding to 50% of the range between the lower and upper landmarks within a given intensity domain) [4]. Different positions within the same domain can elicit different physiological responses, including differences in $\dot{V}O_2$ kinetics and the attainment of $\dot{V}O_2$max [5, 6]. These approaches illustrate that individualizing exercise intensity requires locating the prescribed workload within the athlete's physiological range, a consideration that becomes increasingly relevant as intensity approaches or exceeds the maximal aerobic boundary.

A recent systematic review showed that acute physiological and perceptual responses to short bout high intensity interval training vary with session design, aerobic fitness, and competitive level [7]. Between athletes' differences are also evident over longer training periods. In highly trained middle-distance runners exposed to the same progressive overload protocol, estimated muscle fibre typology was associated with training tolerance and subsequent adaptation. A greater estimated proportion of type I fibres was associated with a lower incidence of functional overreaching and greater performance supercompensation [8]. Training responsiveness may also depend on exercise dose, as individuals showing little improvement at one training dose can respond when training exposure is increased [9]. These observations become particularly relevant in high-intensity interval training, where work bouts often extend beyond maximal aerobic speed or power and therefore require the relative supramaximal demand to be standardized across athletes.

Supramaximal exercise involves concurrent aerobic and anaerobic energy provision, with their relative contributions varying substantially with exercise duration [10]. When exercise intensity extends above maximal aerobic speed or maximal aerobic power, external workload is often prescribed from a single performance reference (e.g. a fixed percentage of MAS or MAP or a percentage of sport specific race or field performance) [11, 12]. A prescription anchored to an aerobic reference (e.g. 110% or 120% MAS) scales the workload to aerobic capacity but does not indicate where that workload lies relative to maximal sprint speed or power [11, 13, 14]. Conversely, a prescription anchored to sport specific performance (e.g. a percentage of 1500 m race speed) reflect overall event performance but does not explicitly locate the workload relative to the athlete's aerobic boundary [12]. Consequently, the same

nominal percentage may represent different relative demands in athletes with different combinations of aerobic and maximal speed or power capacities [11].

This problem provides the rationale for using two individual performance boundaries rather than a single reference when prescribing supramaximal exercise. Athletes with similar MAS can differ substantially in MSS, such that the same percentage of MAS can occupy a different position within each athlete's available supramaximal range [11]. Anaerobic speed reserve (ASR) quantifies the difference between MAS and MSS and allows a prescribed speed to be located within this individual supramaximal range. The same principle extends to the power domain as anaerobic power reserve (APR), using MAP and maximal or peak sprint power [11]. As the evidence base has expanded, research on ASR and APR has progressed from athlete profiling and response prediction towards their practical application in individualized training prescription [11, 13-15]. This shift has raised the question of whether reserve-based prescription produces more consistent responses or greater training adaptations than established individualized approaches [11].

Direct evidence for lower between athlete variability with reserve-based prescription has already been reported. In running, ASR based prescription reduced intersubject variability in exercise tolerance and blood lactate compared with prescription at 110% MAS, while a subsequent field sport study showed a similar directional reduction in exercise tolerance variability with 20% ASR compared with 120% MAS, although the latter estimate was uncertain [13, 14]. Chronic studies have subsequently reported more homogeneous cardiorespiratory adaptations with ASR or APR based prescription than with single reference aerobic prescriptions, whereas effects on performance and hormonal outcomes have been less

consistent [16-20]. Reserve measures have also been used to characterize athlete profiles, examine associations with exercise responses, and define individualized training-load thresholds [21-25]. However, it remains unclear whether they improve prediction beyond established aerobic and maximal performance measures or whether reserve-based profiling leads to better training decisions [26, 27]. Studies also differ in how the reserve components are measured. Because reserve values depend directly on the tests used to determine the aerobic and maximal performance landmarks, variation in the assessment of MAS, MSS, MAP, or maximal sprint power can alter both the calculated reserve and the workload prescribed from it [15]. The validity and reliability of these component measures have been reviewed systematically [15], yet the additional training value of reserve-based prescription beyond conventional individualized aerobic anchors has not been systematically evaluated [11, 15].

The aim of this systematic review was to determine whether anaerobic speed and power reserve frameworks provide additional value beyond conventional individualized anchors for prescribing and interpreting high intensity training in trained athletes. We examined whether reserve-based prescription produces more consistent observed acute responses and whether it influences the magnitude or between athlete dispersion of chronic training adaptations. We also evaluated whether reserve information adds value beyond conventional anchors for response prediction, training decisions, and longitudinal monitoring. We hypothesized that reserve-based prescription would reduce the observed between athlete dispersion of acute responses and chronic adaptations compared with prescription based on a conventional aerobic anchor.

2 Methods

2.1 Reporting framework and protocol status

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 statement and PRISMA-S. Where quantitative pooling was inappropriate, synthesis and reporting additionally followed the Synthesis Without Meta-analysis guideline [28-30]. The review was prospectively registered in PROSPERO on 6 August 2026 before the formal database searches and study screening commenced (CRD420261471635). Eligibility criteria and planned synthesis procedures were established during review planning. Subsequent deviations were documented with their rationale and timing. The PROSPERO record was subsequently updated to reflect review completion.

2.2 Information sources and search process

A comprehensive literature search was conducted across PubMed, Embase via Embase.com, Web of Science Core Collection, Scopus, SPORTDiscus with Full Text via EBSCOhost, and the Cochrane Central Register of Controlled Trials. Searches covered each database from inception to the date of search and were completed on 6 and 7 August 2026. No restrictions were applied according to publication year, language, sex, age, or competitive level at the search stage.

Search strategies combined terms describing anaerobic speed reserve, anaerobic power reserve, speed reserve ratio, power reserve ratio, and related reserve constructs with terms

describing high intensity interval training, sprint interval training, repeated sprint training, exercise tolerance, acute physiological and perceptual responses, training adaptation, response variability, and prospective prediction. Controlled vocabulary was used where available and was combined with title, abstract, and keyword terms. Database specific syntax, field codes, truncation, and proximity operators were adapted for each platform.

Scopus records were retrieved through Publish or Perish using the Elsevier Scopus application programming interface because direct institutional access to the Scopus web interface was unavailable. SPORTDiscus was searched as a standalone database through EBSCOhost. CENTRAL was searched through the Cochrane Library Search Manager without additional filters. Full database-specific search strategies, search dates, platforms, and search syntax are provided in the Additional file 1.

Records from all sources were exported with available bibliographic information and abstracts, merged into a single database, and deduplicated using a reproducible scripted procedure. Duplicate identification was based sequentially on exact digital object identifier matches, PubMed identifier matches, standardized title matches, and bibliographic concordance. Potential near duplicates that could not be resolved automatically were retained for manual review. Distinct reports arising from the same underlying study were linked at the study level during full-text assessment rather than removed during bibliographic deduplication. Following initial screening, the reference list of Sandford et al., a key ASR/APR review and framework paper, was screened on 12 August 2026 as a supplementary completeness check [11]. This reference-list screening identified 14 unique records that were not present in the original database set. Because several early studies used terminology that was not consistently

represented in titles or abstracts, legacy terms including AnSR, anaerobic scope, peak anaerobic scope, mean anaerobic scope, and power reserve were used to verify candidate terminology and eligibility during the audit. These terminology checks did not generate additional unique records beyond the 14 identified from Sandford et al. [11]. Candidate reports were checked against the original database set before eligibility assessment; full details are provided in Additional file 1.

2.3 Eligibility criteria

Studies were eligible when they examined anaerobic speed reserve, anaerobic power reserve, speed reserve ratio, power reserve ratio, or an equivalent reserve construct in trained athletes or systematically trained individuals and used that construct to inform or evaluate high-intensity training prescription, acute exercise tolerance, response consistency, chronic training adaptation, or prospective training decisions.

Eligible participants were required to be described as athletes, participate in organised sport, or have undertaken structured sport-specific or physical training for at least three months. Mixed samples were eligible only when data for trained participants could be extracted separately. Participants could be of any sex, age, sport, or competitive level. However, generally active but untrained individuals, clinical populations, rehabilitation populations, and occupational or tactical populations without regular competitive sport training were excluded.

Eligible reserve constructs were required to be derived from physiologically and dimensionally compatible lower and upper component anchors within the same exercise mode. Standard frameworks included constructs based on MAS or MAP together with MSS or peak

power [11]. Extended frameworks using critical speed or critical power as the lower component anchor were also eligible but were analysed separately from standard reserve frameworks. Studies using incompatible exercise modes, dimensions, or units to derive a reserve measure were excluded.

Eligible acute studies compared reserve-based prescriptions, reserve intensities, doses, or conventional prescription approaches, quantified between-athlete response variation, or prospectively tested whether baseline reserve characteristics predicted subsequent exercise responses. Eligible chronic studies included repeated training exposure with preintervention and postintervention assessment. Prospective cohort studies were eligible when reserve measures were evaluated as predictors or moderators of subsequent acute responses, training adaptations, or differential responses to training programmes.

Comparators included MAS, MAP, velocity derived from intermittent fitness testing, critical speed, critical power, percentages of $\dot{V}O_2$max or maximal heart rate, incremental-test peak velocity or power, fixed absolute speed or power, or alternative reserve-based prescriptions. Noncomparative studies remained eligible when they evaluated dose-response relationships, between-athlete variation, or prospective predictive value.

Studies conducted under hypoxia, altitude, heat, cold, humidity, or other special environmental conditions were eligible when the effect of the reserve framework could be distinguished from the environmental exposure. Studies in which prescription method, environmental condition, exercise mode, training volume, or another major cointervention changed simultaneously and prevented attribution of the observed effect to the reserve framework did not contribute to the primary synthesis.

Cross-sectional descriptive studies, retrospective analyses without prospective training or response outcomes, case reports, case series, qualitative studies, test-validity or reliability studies without a training-decision component, and studies limited to sport, position, or performance profiling were excluded. Study design eligibility was based on the study methods and data structure rather than the authors' design label. Full peer-reviewed journal articles were eligible regardless of language. Conference abstracts, dissertations, preprints, protocols, reviews, editorials, commentaries, and book chapters were not included as primary evidence.

2.4 Study selection and data extraction

Two reviewers (S.X. and S.L.) independently screened titles and abstracts and assessed potentially eligible full texts against the prespecified eligibility criteria. Disagreements were resolved by discussion to consensus. A report that could not be retrieved was retained as not retrieved and no eligibility decision was assigned. For included reports, S.X. extracted data into structured study level and outcome level datasets, and S.L. checked study classifications, table entries, and quantitative values used in the synthesis against the source reports. Extracted items included participant and sport characteristics, reserve definition and component anchors, prescription or exposure, comparator, intervention duration, outcome means and variability, reported associations and model estimates, and information required for risk of bias assessment. S.L. checked review relevant quantitative values against source tables, figures, and available participant level data before analysis. Reports arising from the same or probably overlapping cohorts were linked and were not treated as independent evidence within the same synthesis. Multiple eligible reports from the same underlying study were retained

when they contributed distinct review relevant outcomes, but were linked at the study level and were not counted as independent studies or given duplicate quantitative weight.

Component anchor measurement was extracted separately for each reserve construct. For both lower and upper anchors, we recorded the test protocol and operational definition, whether the anchor was directly assessed or estimated, mode and dimensional compatibility, and reported reliability information. As an additional post hoc contextual methodological appraisal, component anchor methods were compared descriptively with available mode-specific external evidence for the validity and reliability of the corresponding anchors [15]. The external evidence was selected from the cited measurement review and other available mode-specific sources; no dedicated systematic search of measurement studies was undertaken [15]. Evidence alignment was judged separately for lower and upper anchors as supported, supported with caveat, limited support, or not assessable, considering exercise mode, protocol, operational definition, population, and measurement device; reliability evidence was evaluated separately for each anchor. These classifications were intended to make measurement uncertainty explicit and were not treated as a second systematic review or as formal quality scores. The appraisal did not affect study eligibility, risk of bias judgements, statistical weighting, meta-analytic estimates, or GRADE certainty. No numerical or composite anchor quality score was calculated (Additional file 1, Tables S10 and S11).

Eligibility was re adjudicated when data extraction identified internal reporting inconsistencies that prevented the review relevant intervention, analytic sample, timing, or outcome from being determined with sufficient reliability for synthesis. Heaney et al., which had initially met the screening criteria, was excluded at this post extraction adjudication stage

because the reported intervention endpoint or timing, preintervention and postintervention denominators, and reported percentage change and effect size summaries could not be reconciled reliably with the displayed data [31]. Because report level internal consistency had not been specified as a standalone screening exclusion criterion, this post extraction adjudication was documented as a review process deviation and its rationale and timing were retained in the review record.

2.5 Outcome prioritization and evidence structure

Additional value was defined as information or benefit added by reserve frameworks beyond conventional individualized anchors such as MAS, MAP, velocity from intermittent fitness testing, and critical speed or power. For direct prescription comparisons, additional value was operationalized as a between-prescription difference relative to an established individualized comparator. For predictive analyses, incremental predictive value required evidence that reserve information contributed beyond a conventional or component anchor included in the same model. Association or profile description alone was not treated as evidence of incremental prediction. Evidence was organized into acute direct prescription, chronic direct prescription and adaptation, predictor or profile, profile-guided decision or treatment moderation, and longitudinal individualized-threshold monitoring. Environmental-modifier reports were retained separately as exploratory contextual evidence. The primary chronic quantitative outcomes were postintervention relative $\dot{V}O_2max$ and observed relative dispersion in individual percentage change. Chronic high-intensity and sport-specific performance outcomes were examined as secondary outcomes, with quantitative pooling restricted to outcomes with sufficiently comparable definitions and exercise modes. Two post hoc

descriptive analyses additionally examined the outcome specificity of chronic observed response dispersion and longitudinal changes in the lower and upper component anchors and the derived reserve when complete preintervention and postintervention data were available. Acute direct evidence focused on exercise tolerance and within-study response dispersion. Predictor and profile evidence was organized according to association or profile description, normalization, adjusted prediction, treatment moderation, profile-guided decision, and longitudinal monitoring.

2.6 Risk of bias

Risk of bias was assessed at the level of the review-relevant result or evidence contribution. Randomized parallel trials were assessed with RoB 2 [32], randomized crossover trials with the RoB 2 crossover extension [32], the nonrandomized intervention study with ROBINS-I 2016 [33], and predictor, profile, and longitudinal exposure studies with QUIPS [34]. S.X. completed the initial domain level assessments and supporting rationales, and S.L. checked the judgements against the source reports. Disagreements were resolved by discussion to consensus. QUIPS domain judgements were retained without calculating a formal overall score. The label Higher concern was used only as an operational synthesis flag for QUIPS-assessed evidence and was not treated as an official QUIPS overall category. Companion reports arising from the same cohort were linked for overlapping evidence contributions so that the same participants were not treated as independent risk-of-bias units.

2.7 Statistical synthesis

For chronic mean adaptation, postintervention raw mean differences in $\dot{V}O_2max$ were synthesized because all contributing studies reported the outcome on the same scale in $mL \cdot kg^{-1} \cdot min^{-1}$. Quantitative analyses and figure generation were performed using reproducible Python scripts in Python version 3.13.5, with Pandas version 2.2.3, NumPy version 2.3.5, SciPy version 1.17.0, Matplotlib version 3.10.8, and statsmodels version 0.14.6. The primary random effects models used a custom inverse variance implementation with restricted maximum likelihood estimation of τ^2, with numerical optimization performed using SciPy [35]. Statsmodels was used for an independent numerical cross check of the Paule Mandel estimator. Postintervention values were selected for the primary model because variances of change scores could not be calculated without a within-group preintervention-to-postintervention correlation, which was not reported consistently. The primary model therefore avoided imputing this correlation. Positive mean differences favoured reserve-based prescription. For studies with multiple eligible conventional comparators sharing the same reserve prescription group, the primary comparison prioritized the conventional aerobic anchor that most directly represented the lower component anchor in that exercise mode, such as MAS, MAP, or power at $\dot{V}O_2max$, and only one comparator was entered into the primary model. Alternative eligible comparators were evaluated by substitution in sensitivity analyses so that shared reserve prescription groups were not double counted. Random-effects models used restricted maximum likelihood estimation of τ^2 and Wald 95% confidence intervals. Statistical heterogeneity was described using Q, I^2, and τ^2 [36]. Prespecified sensitivity analyses evaluated alternative eligible comparators, addition or

exclusion of studies with defined reporting or design caveats, leave-one-out analyses, change-score models across assumed within-group preintervention-to-postintervention correlations from 0.0 to 0.9, and alternative random-effects estimators or interval methods. Small-study-effect tests were not performed because fewer than 10 studies contributed to the pooled outcome [37].

$$MD_i = \overline{X}_{R,i,post} - \overline{X}_{C,i,post}$$

$$SD_\Delta = \sqrt{SD_{pre}{}^2 + SD_{post}{}^2 - 2rSD_{pre}SD_{post}}$$

Chronic response dispersion was synthesized primarily using the coefficient of variation ratio (CVR), defined as the ratio of the observed coefficient of variation of individual percentage change in the reserve-based condition to that in the comparator condition [38, 39]. The primary analysis used the bias-corrected log CVR with a first-order sampling-variance approximation [38, 39], a random-effects REML model, and Wald 95% confidence intervals before back-transformation to the ratio scale. Values below 1 indicated lower observed relative dispersion under reserve-based prescription. A complementary sensitivity analysis used the variability ratio (VR) of the standard deviation of individual percentage change [38, 39]. Standard deviations were recovered from the reported coefficient of variation and mean percentage change, and bias-corrected log VR values were synthesized with the same random-effects framework. Because VR does not contain the response mean in its denominator, this analysis tested whether the CVR finding persisted after removing the response mean from the variability metric. Mean percentage change was derived from group preintervention and postintervention means in two studies in which it was not directly reported, and a sensitivity analysis excluded those studies. CVR and VR were interpreted only as

observed response dispersion because measurement error and day-to-day biological variation could not be separated from the observed change distributions. Wald confidence intervals were retained for the primary model, while conventional and modified Hartung-Knapp intervals were examined as small-k sensitivity analyses [40]. The group mean percentage changes underlying the primary CV inputs and the recovered SDs used for the VR sensitivity are reported for transparency in Additional file 1, Table S4A. Full formulas and variance specifications are provided in Additional file 1, Section S3. Given the small chronic evidence base, exploratory stratified sensitivity analyses were limited to ASR studies in running and team sports and APR studies in rowing. Reserve construct and exercise modality were fully confounded across these strata. Pooled stratum estimates and Cochran Q between statistics were therefore treated only as descriptive robustness checks and not as formal moderator tests. The same study-level effect estimates and sampling variances as the primary models were used. For the CVR analysis, an additional comparator robustness check replaced the eligible MAS comparators with VIFT comparators in the two ASR studies for which both conventional anchors were available.

$$CVR_i = \frac{CV_{R,i}}{CV_{C,i}}$$

Chronic high intensity and sport specific performance outcomes were synthesized separately from $\dot{V}O_2max$. A single global performance effect was not calculated because outcomes represented different performance constructs, exercise modes, and units. Exploratory random-effects pooling was undertaken only when outcome definitions were sufficiently comparable across studies. Three rowing studies reported 2000 m time trial performance on the same scale and were combined using between-group differences in change, with lower time

representing better performance and positive effect estimates defined to favour reserve-based prescription. Because within-group preintervention-to-postintervention correlations were not reported, the main exploratory change-score model assumed $r = 0.5$ and sensitivity analyses examined r values from 0.0 to 0.9. Observed dispersion in 2000 m change was synthesized using the same bias-corrected log CVR framework as the primary chronic dispersion analysis. Other performance outcomes were retained in structured within-outcome synthesis because only one or two sufficiently comparable studies were available or operational definitions differed.

To examine outcome specificity, 68 matched chronic outcome-level CVR observations were organized into five descriptive domains. Multiple outcomes within studies were treated as dependent and were not pooled; study-by-domain median CVRs were used for evidence mapping, and hierarchical clustering of $\log_2$-transformed patterns was used only to reorder the heatmap. Full classification and clustering procedures are provided in Additional file 1, Section S3. Longitudinal reserve dynamics were examined in chronic study arms with complete preintervention and postintervention lower-anchor, upper-anchor, and reserve triads. Percentage change in each component and an illustrative posttraining reserve position for the original baseline absolute workload were calculated descriptively. These calculations did not reconstruct actual workload progression or establish that reserve-based training caused reserve contraction; full procedures are provided in Additional file 1, Section S3.

Acute direct-prescription studies were not pooled across studies because the available variability metrics were not statistically commensurate and paired covariance was unavailable for some comparisons. Participant-level paired data from Julio et al. were used to calculate

paired mean contrasts and paired CV ratios with bias-corrected and accelerated bootstrap confidence intervals [13, 41]. For Collison et al. and Di Gennaro et al., available residual-dispersion or condition-level CV estimates were normalized to the study-specific conventional reference for descriptive within-study comparison only [14, 42]. For the environmental-modifier evidence, descriptive coefficients of variation for completed efforts were calculated from the reported means and standard deviations and expressed as percentages. These calculations were used only to characterize observed relative dispersion across oxygen conditions and were not treated as comparative evidence of a prescription effect or entered into quantitative synthesis.

$$d_z = \frac{\overline{D}}{SD_D}$$

Predictor, profile, decision, and longitudinal-threshold evidence was synthesized separately from direct prescription trials. Cross-study meta-analysis of correlations was not undertaken because studies evaluated different predictors, outcomes, session formats, and causal questions, and several studies contributed multiple statistically dependent effects from the same participants. Pearson correlations, partial correlations, profile-group contrasts, treatment-by-profile interactions, coefficients of determination, and longitudinal Spearman correlations were therefore not treated as a common effect-size family. Where source confidence intervals were unavailable for isolated Pearson correlations, 95% confidence intervals were calculated using Fisher r-to-z transformation. For Di Gennaro et al. [43], source partial-correlation coefficients were retained, while two-sided P values and 95% confidence intervals were recalculated uniformly using n = 19 and one controlled variable because the

published table contained internally inconsistent inferential values. Structured synthesis emphasized effect magnitude, consistency, inclusion of conventional anchors in the same model, and risk of bias rather than vote counting by statistical significance. For Fig. 7, evidence-signal categories were assigned using stage-specific operational rules. At the association or profile and normalization stages, a supportive signal indicated that reserve information characterized response heterogeneity or normalized relative demand and did not imply independent incremental prediction. Adjusted prediction required evidence after inclusion of conventional anchors; treatment moderation required profile-by-treatment evidence; profile-guided decision required prospective allocation; and longitudinal monitoring required prospective associations between individualized thresholds and subsequent change. Mixed or conditional indicated that direction or strength depended on intensity, outcome, profile, or model specification, whereas no clear or negative indicated absent, null, or contrary evidence at that stage. Statistical significance alone did not determine the category. Full coding rules and cell-level rationales are provided in Additional file 1, Section S3.8.

$$z = \frac{1}{2} ln(\frac{1 + r}{1 - r})$$

2.8 Certainty of evidence

Outcome-level certainty was assessed with GRADE [44] across risk of bias, inconsistency, indirectness, imprecision, and publication bias. Randomized intervention evidence began at high certainty and nonrandomized or observational predictor or exposure evidence at low certainty [44], with one or two level downgrades for serious or very serious concerns. Ratings were assigned separately for chronic mean $\dot{V}O_2max$ adaptation, chronic observed response dispersion, chronic high-intensity and sport-specific performance adaptation, acute direct

standardization, general predictor or profile evidence, incremental prediction beyond conventional anchors, profile-guided allocation, treatment moderation, and longitudinal individualized-threshold monitoring. Judgements were anchored to additional value beyond conventional individualized anchors, so association or profile evidence without a direct comparison could inform interpretation but did not increase certainty for incremental prediction. Environmental-modifier reports were retained as exploratory contextual evidence and were not assigned a separate GRADE rating because they did not directly compare reserve-based with conventional prescription. S.X. completed the initial ratings, and S.L. checked the domain judgements and final ratings; disagreements were resolved through discussion to consensus (Additional file 1, Table S12).

Table 1 PICOS eligibility framework

PICOS component	Inclusion criteria	Exclusion criteria
Population	Trained athletes or systematically trained individuals of any sex, age, sport, or competitive level. Participants must be identified as athletes, participate in organised sport, or have completed structured sport specific or physical training for at least three months. Mixed samples are eligible when trained participant data can be separated.	Untrained healthy individuals, generally active participants without structured training, clinical or rehabilitation populations, and mixed samples from which trained participants cannot be separated. Tactical or occupational populations are excluded unless they also undertake regular competitive sport training.
Intervention or exposure	ASR, APR, speed reserve ratio, power reserve ratio, or an equivalent reserve construct used for high intensity interval, sprint interval, repeated sprint, or related supramaximal training prescription, acute response evaluation, chronic adaptation assessment, or prospective training decision making. Standard and extended reserve frameworks are eligible.	Reserve measures used only for descriptive profiling, sport or position comparisons, test validation, competition performance prediction, or athlete classification without evaluation of training prescription, acute response, chronic adaptation, or prospective decision value. Reserve constructs derived from incompatible modes, dimensions, or units are excluded.
Comparator	Conventional prescription anchors including MAS, MAP, intermittent fitness test velocity, critical speed, critical power, percentages of $\dot{V}O_2max$ or maximal heart rate, peak incremental velocity or power, fixed absolute workload, alternative reserve definitions, reserve percentages, or training programmes. Eligible noncomparative studies must examine dose response,	Comparisons in which prescription method is inseparable from major differences in environment, exercise mode, training volume, or cointervention. Single exposure studies reporting only an average response without a comparator, dose response analysis, individual variation, or prospective prediction are excluded.

PICOS component	Inclusion criteria	Exclusion criteria
	individual variation, or prospective prediction.	
Outcomes	Acute exercise tolerance and completion, external workload, physiological and perceptual responses, between-athlete response consistency, chronic changes in aerobic capacity, maximal speed or power, high intensity performance, repeated sprint performance, sport related performance, between athlete variation in adaptation, and prospective predictive or moderating value of reserve measures.	Outcomes limited to descriptive athlete characteristics, competition prediction, sport classification, or test measurement properties without a direct relationship to training prescription, response, adaptation, or prospective decision making.
Study design	Randomised and nonrandomised parallel group trials, crossover trials, repeated measures acute experiments, controlled or uncontrolled longitudinal training interventions, and prospective cohort studies that evaluate prescription, response consistency, dose response, adaptation, or predictive value.	Cross sectional descriptive studies, retrospective analyses without prospective outcomes, case reports, case series, qualitative studies, methodological validation studies, reviews, protocols, editorials, conference abstracts, dissertations, preprints, and book chapters.

PICOS population, intervention or exposure, comparator, outcomes, and study design; APR anaerobic power reserve; ASR anaerobic speed reserve; MAP maximal aerobic power; MAS maximal aerobic speed; $\dot{V}O_2$max maximal oxygen uptake.

2.9 Use of artificial intelligence

ChatGPT (OpenAI) was used to assist with manuscript language editing. The authors retained responsibility for data extraction, analysis, interpretation, and the final manuscript.

3 Results

3.1 Study selection

Searches identified 454 records across the six information sources. After deduplication and screening, 26 reports underwent full text assessment. Three reports were excluded through the database pathway: two because the eligible trained population could not be separated from ineligible participants, and Heaney et al. [31] following post extraction eligibility adjudication because the intervention endpoint or timing, participant denominators, and reported summary calculations could not be reconciled reliably. Girard and Racinais [45] was verified following author contact and treated as a companion report to Soo et al. [46] from the same project and participant cohort. Supplementary reference list screening of Sandford et al. [11] identified 14 additional records. Three reports underwent full text assessment, of which Blondel et al. [47] and Barnett et al. [48] were excluded and Buchheit et al. [23] was included. Overall, 24 reports representing 23 independent studies were included (Fig. 1).

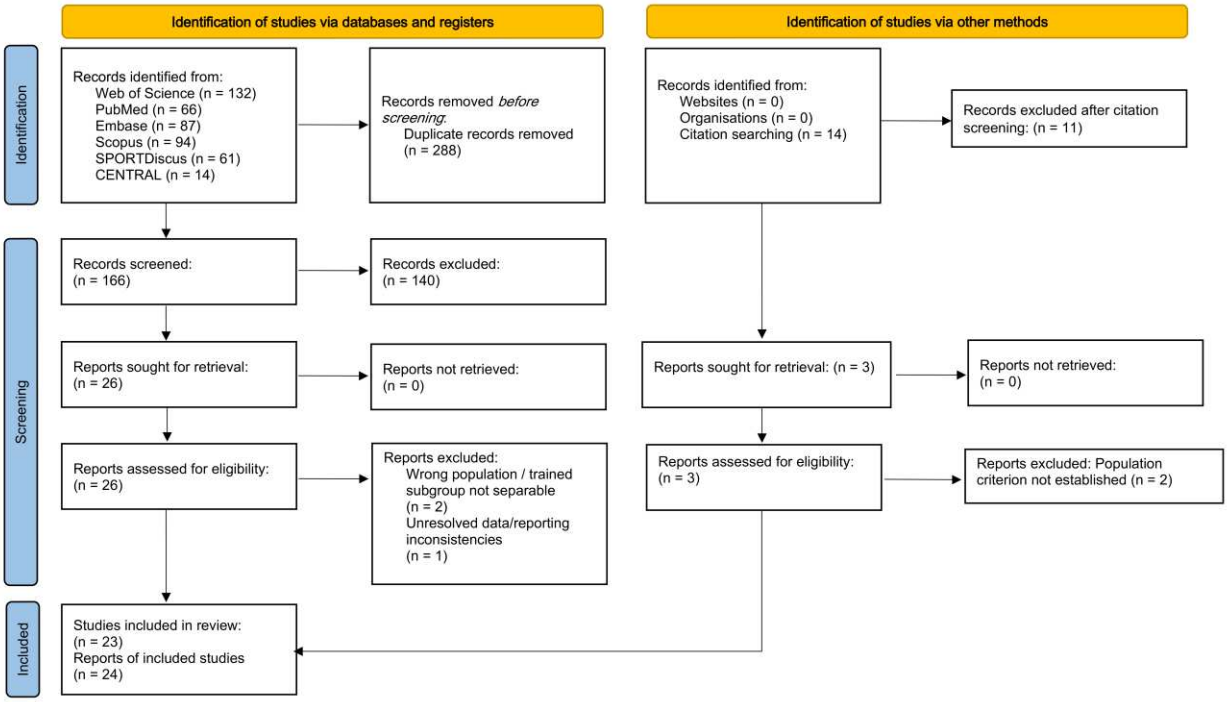


Fig. 1 PRISMA 2020 flow diagram of study selection.

Searches identified 454 records across six information sources. After deduplication, 166 records were screened, 26 reports were assessed through the database pathway, and 23 reports representing 22 independent studies were retained. Supplementary reference-list screening of Sandford et al. [11]. identified 14 additional records; 11 were excluded during citation screening, three reports were assessed, two were excluded, and one was included. Twenty-four reports representing 23 independent studies were included overall.

3.2 Study characteristics

Twenty-four reports representing 23 independent studies were included, comprising 12 acute and 11 chronic or longitudinal studies (Table 2). Soccer was the most frequently represented sport, followed by cycling, rowing, and running, while the remaining studies covered several individual and team sports. Sample sizes ranged from 7 to 117 participants. The evidence base included three acute direct prescription comparisons, seven chronic direct prescription and adaptation studies, seven acute predictor or profile studies, two profile guided decision or moderation studies, two longitudinal individualized load studies, and two environmental modifier studies. Most studies examined ASR or APR, with a smaller number evaluating relative reserve measures, profile classifications, or extended reserve constructs.

Component anchor measurement was examined as a post hoc contextual methodological appraisal (Additional file 1, Tables S10 and S11). Across the running based ASR studies, MAS was estimated using both field based and incremental running tests, while upper anchor methods also varied across studies. Many component measures were supported by directly applicable or closely related external measurement evidence, although important caveats remained where protocols, operational definitions, reference criteria, or measurement devices differed. The main uncertainties concerned several field derived MAS estimates and a small number of sport specific maximal speed or power definitions with limited directly matched validation evidence. Reliability evidence was also uneven and often derived from external validation studies rather than study specific repeatability data.

47 **Table 2 Characteristics of included studies**

Study	Sport	Participants	Design	Reserve construct	Comparator or exposure	Primary review outcome family	Evidence role
Thron et al., 2025 [22]	Running	n=18; Male and female; 15.83 ± 0.86 y; Highly trained	Acute prospective predictor study with standardized HIIT exposure	ASR and relative ASR	MAS, MSS and relative intensity measures within the same cohort	Acute internal load and recovery	Predictor/profile
Buchheit et al., 2012 [23]	Mixed team sports	n=11; Sex NR; 30.5 ± 3.6 y; Recreational team sport players	Acute prospective predictor study with standardized fixed HIT exposure	ASR	vVO_2max, MSS and percentage of ASR used during fixed HIT	Acute HIT tolerance and metabolic response	Predictor/profile
Di Gennaro et al., 2025 [42]	Cycling	n=12; Male; 31 ± 6 y; Trained	Acute randomized counterbalanced crossover HIIT study	APR and GPR	120% MAP and GPR based prescription	Acute tolerance and response variability	Acute direct
Peng et al., 2025 [17]	Soccer	n=30; Male; 21.6 ± 4.8 y; Trained collegiate	Chronic randomized parallel intervention	ASR	130% MAS and load matched SIT	Chronic adaptation mean and variability	Chronic direct
Wang and Ye, 2024 [16]	Basketball	n=30; Male; 28.4 ± 5 y; National level	Chronic randomized parallel intervention	ASR	95% VIFT and 120% MAS	Chronic adaptation mean and variability	Chronic direct
Zhu et al., 2025 [26]	Soccer	n=46; Male; 16.5 ± 0.5 y; Competitive youth	Chronic randomized controlled parallel	ASR locomotor	HIIT, RST, and control	Treatment selection and performance adaptation	Decision/moderation

Study	Sport	Participants	Design	Reserve construct	Comparator or exposure	Primary review outcome family	Evidence role
			profile guided intervention	profile			
Luo et al., 2024 [18]	Rowing	n=24; NR; 24.8 ± 4.3 y; Well trained	Chronic randomized parallel intervention	APR	130% power at $\dot{V}O_2max$	Chronic adaptation mean and variability	Chronic direct
Dai and Xie, 2023 [19]	Soccer	n=30; Male; 19 ± 1.6 y; National level	Chronic randomized parallel intervention	ASR	95% VIFT and 120% MAS	Chronic adaptation mean and variability	Chronic direct
Yu Tongwu et al., 2025 [49]	Rowing	n=30; Male; 24.9 ± 3.1 y; Elite or national team	Chronic randomized controlled intervention	APR	130% MAP and volume matched maximal SIT	Chronic adaptation mean and variability	Chronic direct
Du and Tao, 2023 [50]	Sprint kayak	n=24; NR; Highly trained	Chronic randomized parallel intervention	ASR	100% MAS HIIT and endurance control	Chronic physiological and performance adaptation	Chronic direct
Aspin et al., 2024 [21]	Soccer	n=13; Female; 20.2 ± 4.6 y; Subelite	Acute randomized crossover response study with baseline ASR predictor	ASR	Multiple HIIT formats	Acute physiological and perceptual response	Predictor/profile
Julio et al., 2020 [13]	Running and rugby	n=21; NR; Trained athletes	Acute randomized crossover HIIE study	ASR	110% MAS	Acute tolerance and response variability	Acute direct

Study	Sport	Participants	Design	Reserve construct	Comparator or exposure	Primary review outcome family	Evidence role
Wang and Zhao, 2023 [20]	Rowing	n=20; Male; 22 ± 3 y; Highly trained	Chronic randomized parallel intervention	APR	130% MAP	Chronic adaptation mean and variability	Chronic direct
Soo et al., 2021; Girard and Racinais, 2023 companion report [45, 46]	Cycling	n=15; Male; 38.4 ± 7.1 y; Well trained	Acute randomized double blind crossover environmental study with companion report	APR	Sea level, moderate hypoxia and severe hypoxia	Environmental tolerance and neuromuscular response, including rate of torque development	Environmental
Collison et al., 2022 [14]	Australian Rules football	n=17; Male; 16.8 ± 0.4 y; Competitive youth	Acute randomized and counterbalanced repeated measures prescription study	ASR	120% MAS and 95% 30-15IFT	Acute tolerance variability	Acute direct
Di Gennaro et al., 2025 (APNM) [43]	Cycling	n=19; Male; 34 ± 9 y; Endurance trained	Acute prospective repeated measures predictor and construct study	APR and MPR	MAP, critical power, PPO, W′, and MAOD	Exercise tolerance and construct prediction	Predictor/profile
Salter et al., 2025 [51]	Multisport	n=117; Female; 13.6 ± 2.9 y, range 8 to 22; Competitive youth and	Acute pre post repeated sprint response by baseline	ASR-based locomotor profile	Slow, hybrid and fast profile groups	Acute neuromuscular fatigue by profile	Predictor/profile

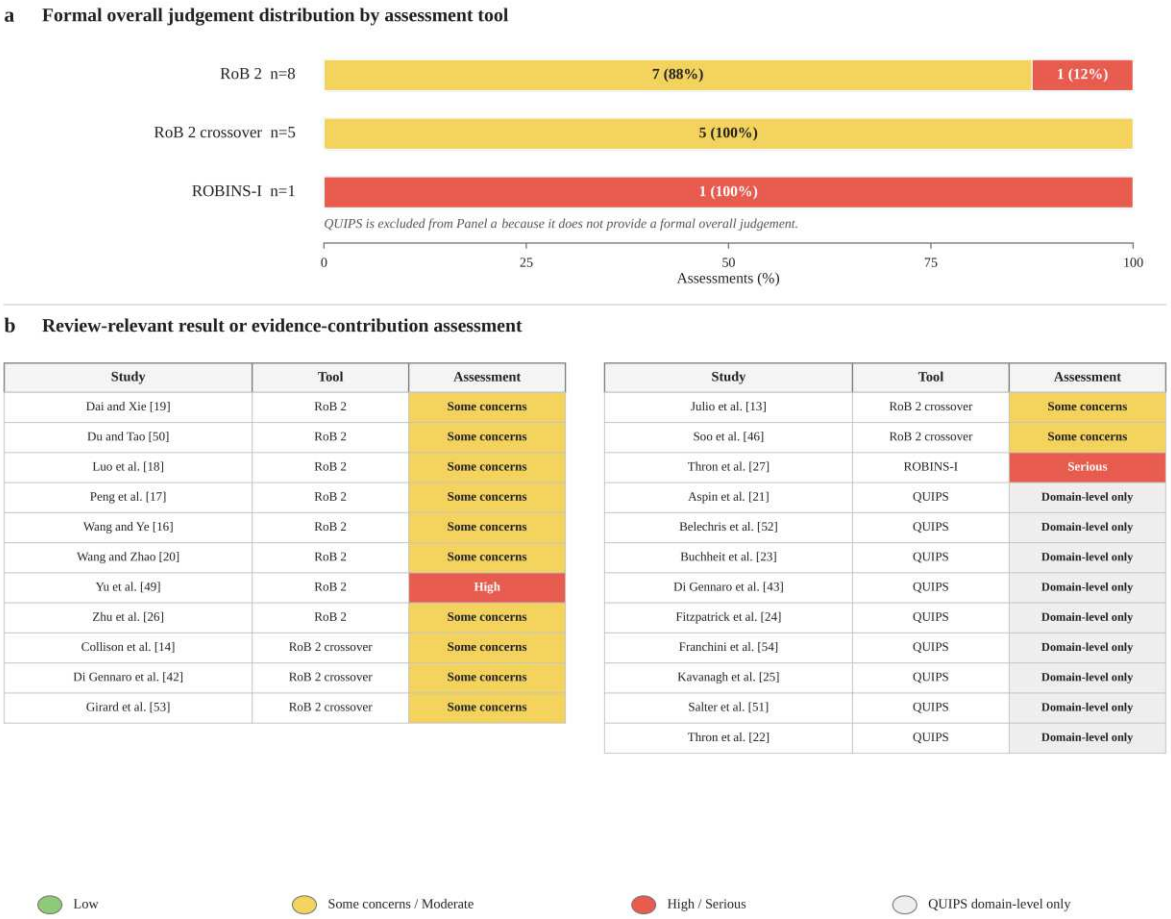
Study	Sport	Participants	Design	Reserve construct	Comparator or exposure	Primary review outcome family	Evidence role
		young adult	profile				
Thron et al., 2026 [27]	Running	n=27 completers (36 enrolled); Male and female; 27.7 ± 7.5 y; Trained	Chronic controlled 6 wk training trial with formal moderation analyses	ASR and SRR	HIIT at 90–95% MAS versus all-out SIT	Profile-by-training adaptation moderation	Decision/moderation
Belechris et al., 2025 [52]	Track sprint and middle distance	n=16; Male; Approximately 20.7 y; National level	Acute prospective predictor study	ASR	vVO2max and sprint mechanics variables	Repeated-sprint fatigue prediction	Predictor/profile
Girard et al., 2021 [53]	Cycling	n=11; Male; 38.1 ± 7.0 y; Well trained	Acute crossover graded hypoxia experiment	APR	Normoxia, moderate hypoxia at about 2200 m, severe hypoxia at about 4200 m	Environmental tolerance and neuromuscular response	Environmental
Fitzpatrick et al., 2018 [24]	Soccer	n=14; Male; 17.1 ± 0.5 y; Professional youth	Chronic prospective 6 wk observational training load cohort	ASR-based high speed threshold	Fixed HSR and MAS-based thresholds	Longitudinal training-load dose-response	Longitudinal
Franchini et al., 2022 [54]	Judo	n=28 overall; 12 high and 12 low ASR in	Acute prospective profile and	ASR	High and low ASR profiles	Sport-specific acute tolerance/performance	Predictor/profile

Study	Sport	Participants	Design	Reserve construct	Comparator or exposure	Primary review outcome family	Evidence role
		grouped analysis; NR; Approximately 24 ± 3 y; State and national	performance study				
Kavanagh et al., 2025 [25]	Soccer	n=7; Male; 20.1 ± 1.1 y; Professional youth	Chronic prospective longitudinal observational cohort	ASR threshold	MAS-based and fixed HSR thresholds	Longitudinal training-load dose-response	Longitudinal

APR anaerobic power reserve; ASR anaerobic speed reserve; GPR glycolytic power reserve; HIIT high-intensity interval training; HSR high-speed running; MAOD maximal accumulated oxygen deficit; MAP maximal aerobic power; MAS maximal aerobic speed; MPR maximal power reserve; MSS maximal sprint speed; PPO peak power output; RST repeated-sprint training; SIT sprint interval training; VIFT final velocity reached in the 30-15 Intermittent Fitness Test; W′ work capacity above critical power. NR not reported. Girard and Racinais (2023) is a companion report from the same project and participant cohort as Soo et al. (2021) and is represented within the same study level row [45, 46].

3.3 Risk of bias

Most randomized studies were judged as having some concerns for the review relevant results. One randomized parallel study was judged as high risk of bias because unresolved inconsistencies remained in the reserve prescription and review relevant quantitative reporting [49]. The single nonrandomized intervention study was judged as having serious risk of bias under ROBINS-I, primarily because treatment assignment was not clearly randomized and missing data were plausibly informative [27]. Among predictor, profile, and longitudinal exposure studies assessed with QUIPS, concerns were concentrated mainly in confounding and statistical analysis or reporting. All five studies contributing to the primary chronic $\dot{V}O_2max$ meta-analysis were judged as having some concerns, with none at high risk [16-20]. The companion report by Girard and Racinais was linked to Soo et al. for overlapping evidence contributions and was not counted as a separate risk of bias unit [45, 46]. Full domain level judgements are provided in Additional file 1, Supplementary Fig. S1.



Study	Tool	Assessment
Dai and Xie [19]	RoB 2	Some concerns
Du and Tao [50]	RoB 2	Some concerns
Luo et al. [18]	RoB 2	Some concerns
Peng et al. [17]	RoB 2	Some concerns
Wang and Ye [16]	RoB 2	Some concerns
Wang and Zhao [20]	RoB 2	Some concerns
Yu et al. [49]	RoB 2	High
Zhu et al. [26]	RoB 2	Some concerns
Collison et al. [14]	RoB 2 crossover	Some concerns
Di Gennaro et al. [42]	RoB 2 crossover	Some concerns
Girard et al. [53]	RoB 2 crossover	Some concerns

Study	Tool	Assessment
Julio et al. [13]	RoB 2 crossover	Some concerns
Soo et al. [46]	RoB 2 crossover	Some concerns
Thron et al. [27]	ROBINS-I	Serious
Aspin et al. [21]	QUIPS	Domain-level only
Belechris et al. [52]	QUIPS	Domain-level only
Buchheit et al. [23]	QUIPS	Domain-level only
Di Gennaro et al. [43]	QUIPS	Domain-level only
Fitzpatrick et al. [24]	QUIPS	Domain-level only
Franchini et al. [54]	QUIPS	Domain-level only
Kavanagh et al. [25]	QUIPS	Domain-level only
Salter et al. [51]	QUIPS	Domain-level only
Thron et al. [22]	QUIPS	Domain-level only

Fig. 2 Risk-of-bias assessment.

(a) Distribution of formal overall judgements for studies assessed with RoB 2, the RoB 2 crossover extension, or ROBINS-I 2016. Seven of eight randomized parallel studies assessed with RoB 2 were judged as having some concerns and one was judged as high risk. All five randomized crossover studies were judged as having some concerns. The single nonrandomized intervention study assessed with ROBINS-I 2016 was judged as serious risk. QUIPS-assessed predictor, profile, and longitudinal exposure studies are not included in the formal overall-judgement distribution because QUIPS does not provide a formal overall risk-of-bias category. (b) Review-relevant result or evidence-contribution assessment for the included evidence base. QUIPS-assessed studies are shown as domain-level only rather than assigned an overall category. The companion report by Girard and Racinais (2023) is linked to Soo et al. (2021) for overlapping evidence contributions and is not counted as a

separate risk-of-bias unit [45, 46]. Full domain-level assessments are provided in Additional file 1, Supplementary Fig. S1.

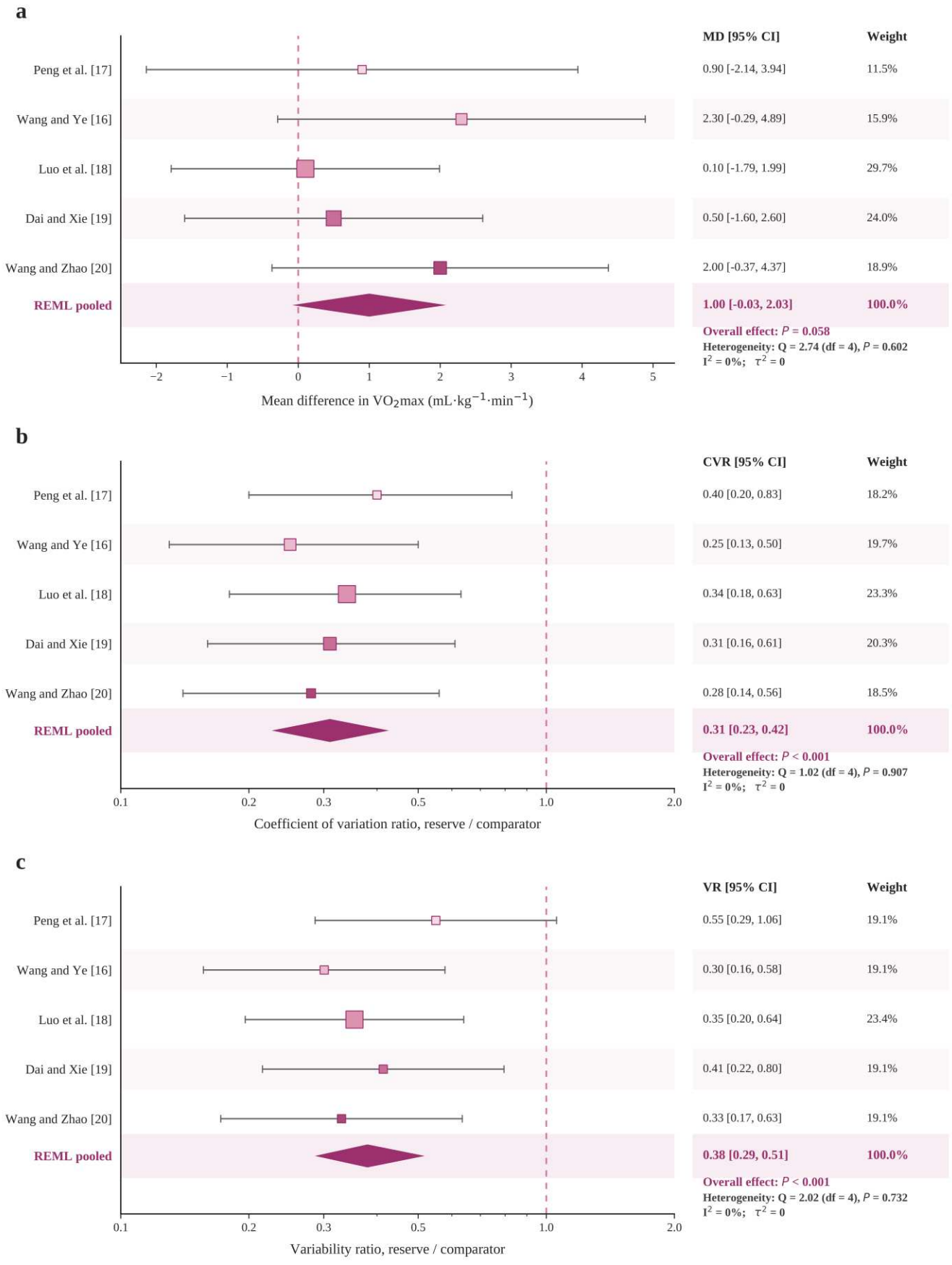
3.4 Chronic direct-prescription evidence

3.4.1 Mean $\dot{V}O_2$max adaptation

Five studies comprising 104 participants in the selected primary comparisons contributed to the chronic $\dot{V}O_2$max meta-analysis [16-20]. The pooled postintervention MD was 1.00 $mL \cdot kg^{-1} \cdot min^{-1}$ (95% CI −0.03 to 2.03; $P = .058$), with no observed statistical heterogeneity ($I^2 = 0\%$), providing no clear evidence of greater mean $\dot{V}O_2$max adaptation with reserve-based prescription (Fig. 3a). Sensitivity analyses did not materially alter this interpretation. Exclusion of Luo et al. [18] yielded a larger estimate with a confidence interval excluding zero, whereas alternative interval methods retained confidence intervals spanning zero. Change-score analyses were also consistent with the overall interpretation, with the confidence interval excluding zero only when a preintervention-to-postintervention correlation of 0.9 was assumed (Additional file 1, Supplementary Fig. S2).

3.4.2 Observed response dispersion

The same five studies contributed to the chronic observed dispersion synthesis [16-20]. Reserve-based prescription was associated with lower observed relative dispersion in $\dot{V}O_2$max adaptation (CVR = 0.311, 95% CI 0.229 to 0.423; $P < .001$; $I^2 = 0\%$; Fig. 3b). The complementary VR analysis showed the same direction (VR = 0.38, 95% CI 0.29 to 0.51; $P < .001$; $I^2 = 0\%$; Fig. 3c). Sensitivity analyses were consistent with the primary finding, including alternative comparators, source data rounding, leave one out analyses, and exclusion of studies in which mean percentage change was derived from group means (Additional file 1, Supplementary Fig. S3 and Table S4). Exploratory stratification did not materially alter the primary findings. Mean $\dot{V}O_2$max adaptation remained similar between the ASR running and team sport stratum and the APR rowing stratum, while lower observed dispersion remained evident in both strata (Additional file 1, Table S3).


a
MD [95% CI]
Weight
Peng et al. [17]
0.90 [-2.14, 3.94]
11.5%
Wang and Ye [16]
2.30 [-0.29, 4.89]
15.9%
Luo et al. [18]
0.10 [-1.79, 1.99]
29.7%
Dai and Xie [19]
0.50 [-1.60, 2.60]
24.0%
Wang and Zhao [20]
2.00 [-0.37, 4.37]
18.9%
REML pooled
1.00 [-0.03, 2.03]
100.0%
Overall effect: P = 0.058
Heterogeneity: Q = 2.74 (df = 4), P = 0.602
I² = 0%; τ² = 0
−2
−1
0
1
2
3
4
5
Mean difference in VO2max (mL·kg−1·min−1)
b
CVR [95% CI]
Weight
Peng et al. [17]
0.40 [0.20, 0.83]
18.2%
Wang and Ye [16]
0.25 [0.13, 0.50]
19.7%
Luo et al. [18]
0.34 [0.18, 0.63]
23.3%
Dai and Xie [19]
0.31 [0.16, 0.61]
20.3%
Wang and Zhao [20]
0.28 [0.14, 0.56]
18.5%
REML pooled
0.31 [0.23, 0.42]
100.0%
Overall effect: P < 0.001
Heterogeneity: Q = 1.02 (df = 4), P = 0.907
I² = 0%; τ² = 0
0.1
0.2
0.3
0.5
1.0
2.0
Coefficient of variation ratio, reserve / comparator
c
VR [95% CI]
Weight
Peng et al. [17]
0.55 [0.29, 1.06]
19.1%
Wang and Ye [16]
0.30 [0.16, 0.58]
19.1%
Luo et al. [18]
0.35 [0.20, 0.64]
23.4%
Dai and Xie [19]
0.41 [0.22, 0.80]
19.1%
Wang and Zhao [20]
0.33 [0.17, 0.63]
19.1%
REML pooled
0.38 [0.29, 0.51]
100.0%
Overall effect: P < 0.001
Heterogeneity: Q = 2.02 (df = 4), P = 0.732
I² = 0%; τ² = 0
0.1
0.2
0.3
0.5
1.0
2.0
Variability ratio, reserve / comparator

100

Fig. 3 Chronic effects of reserve-based prescription on mean aerobic adaptation and observed response dispersion [16-20].

(a) Random-effects meta-analysis of postintervention maximal oxygen uptake comparing reserve-based prescription with conventional individualized aerobic anchors. Positive mean differences favour reserve-based prescription. (b) Random-effects meta-analysis of the coefficient of variation ratio for observed individual percentage change in $\dot{V}O_2$max. CVR values below 1.0 indicate lower observed relative dispersion under reserve-based prescription. (c) Sensitivity analysis using the variability ratio of the standard deviation of individual percentage change in $\dot{V}O_2$max. VR values below 1.0 indicate lower absolute observed dispersion under reserve-based prescription and test whether the CVR finding persists after removing the mean term contained in the coefficient of variation. Squares represent study-specific estimates and are scaled by inverse-variance weight; horizontal lines indicate 95% confidence intervals and diamonds indicate pooled estimates. For the VR analysis, excluding the two studies in which mean percentage change was derived from group means yielded VR = 0.42 (95% CI 0.29 to 0.62). Dispersion estimates describe observed response dispersion and do not isolate true biological response variance from measurement error or day-to-day variation.

3.4.3 High intensity and sport specific performance adaptation

Three rowing studies provided directly comparable 2000 m time trial outcomes [18, 20, 49]. Exploratory pooling showed no clear advantage of APR-based prescription over conventional aerobic power-based prescription for mean performance change (MD = 0.08 s, 95% CI −2.72 to 2.89; P = .953). Observed dispersion in 2000 m performance change was also uncertain (CVR = 0.87, 95% CI 0.44 to 1.70; P = .680). Sensitivity analyses did not materially alter the interpretation, although exclusion of the high risk study [49] shifted the pooled CVR to approximately 1.02 (Additional file 1, Supplementary Fig. S5).

Other chronic studies examined maximal sprint speed or power, Wingate performance, 60 s power, critical power, sprint and change of direction performance, jumping, and sport specific time trials [16-20, 49, 50]. No consistent between group advantage in the magnitude of performance adaptation was observed for reserve-based prescription. Response dispersion was also outcome dependent, with lower

dispersion under reserve-based prescription for some Wingate and power outcomes, but lower dispersion under conventional prescription for several outcomes in one rowing study [18]. In sprint kayakers, 500 m and 1000 m performance improved within the ASR group, although the corresponding between group differences were not significant and the ASR and MAS training protocols differed in work duration and recovery structure [50]. Overall, the chronic performance evidence did not show a consistent additional benefit of reserve-based prescription for mean high intensity or sport specific performance adaptation, and any effect on response standardization appeared to be outcome specific.

3.4.4 Outcome specificity of observed response dispersion

Across the chronic direct comparison evidence, 68 matched outcome level CVR observations were available for descriptive outcome domain mapping [16-20, 49, 50]. Lower observed dispersion under reserve based prescription was most consistent for maximal aerobic or cardiorespiratory and threshold or submaximal aerobic outcomes, whereas performance outcomes were more heterogeneous and hormonal or other outcomes showed no consistent advantage (Fig. 4; Additional file 1, Tables S6 and S7).



Fig. 4 Outcome-domain evidence map for observed response dispersion in chronic direct-comparison studies [16-20, 49, 50].

Rows represent studies and columns represent outcome domains. Each populated cell reports the median coefficient of variation ratio across outcomes within that study by domain combination and the number of contributing outcomes. Values below 1 indicate lower observed relative dispersion under reserve-based prescription, whereas values above 1 indicate lower observed relative dispersion under the comparator prescription. Very pale cells indicate combinations for which no eligible outcome was available. The observed-data colour scale is centred at CVR = 1.00. Study and domain order were determined by hierarchical clustering of $\log_2$-transformed CVR patterns for visualization only.

3.4.5 Longitudinal reserve dynamics

Four chronic studies provided complete preintervention and postintervention lower anchor, upper anchor, and reserve data for ten study arms [18-20, 50]. Across all nine active training arms, the lower aerobic anchor improved more than the upper maximal anchor, with corresponding median changes of +6.57% and +0.65%, respectively. The derived ASR or APR consequently decreased in all nine active arms, with a median change of −4.85% (Fig. 5). This pattern occurred under both reserve based and conventional individualized training. In the four reserve based arms with an explicit baseline reserve target, retaining the original absolute workload would have shifted the postintervention workload to a lower relative position within the updated reserve in all four arms [18-20, 50] (Additional file 1, Tables S8 and S9).

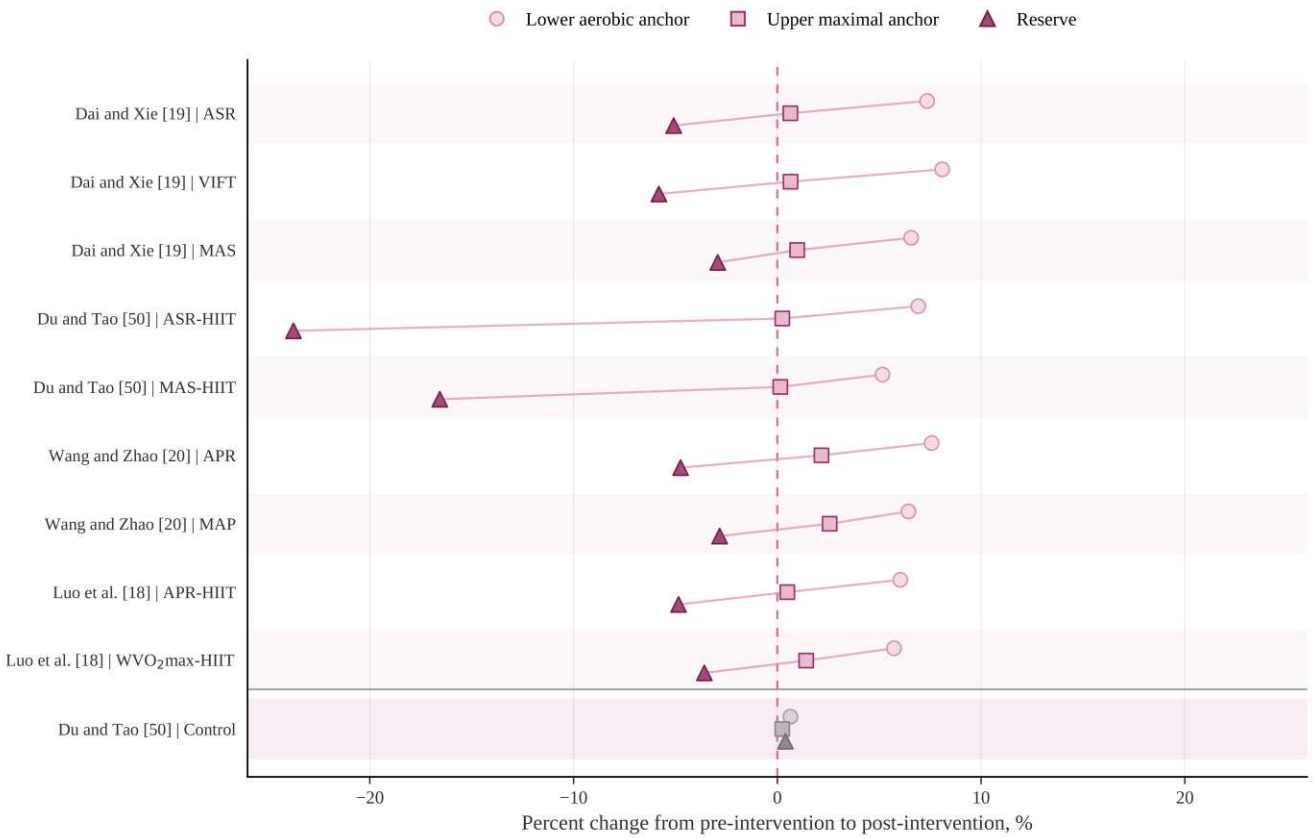


Fig. 5 Longitudinal changes in component anchors and reserve across chronic training arms [18-20, 50].

Each row represents one study arm. Circles denote the lower aerobic anchor, squares denote the upper maximal anchor, and triangles denote the derived reserve. Values are percentage change from preintervention to postintervention. The control arm is shown separately below the divider. Across the available active training arms, the lower aerobic anchor increased more than the upper maximal anchor and the observed ASR or APR consequently decreased. This pattern occurred under both reserve-based and conventional individualized prescriptions.

3.5 Acute direct-prescription evidence

Three studies directly compared acute reserve based and conventional prescription approaches [13, 14, 42] (Fig. 6). Julio et al. [13] reported longer time to exhaustion at 25% and 50% ASR than at 110% MAS, together with lower paired response dispersion under both ASR conditions (CV ratios = 0.49 and 0.47, respectively). Collison et al. [14] reported directionally lower residual dispersion with 20% ASR than

with 120% MAS, although the contrast was uncertain (SMD = −0.47, 95% CI −1.03 to 0.09; $P = .09$). In Di Gennaro et al. [42], mean time to exhaustion did not differ among APR, GPR, and MAP prescriptions, while observed CVs were higher under APR and GPR than under MAP. Overall, acute direct comparisons produced inconsistent evidence that reserve based prescription improved response standardization.

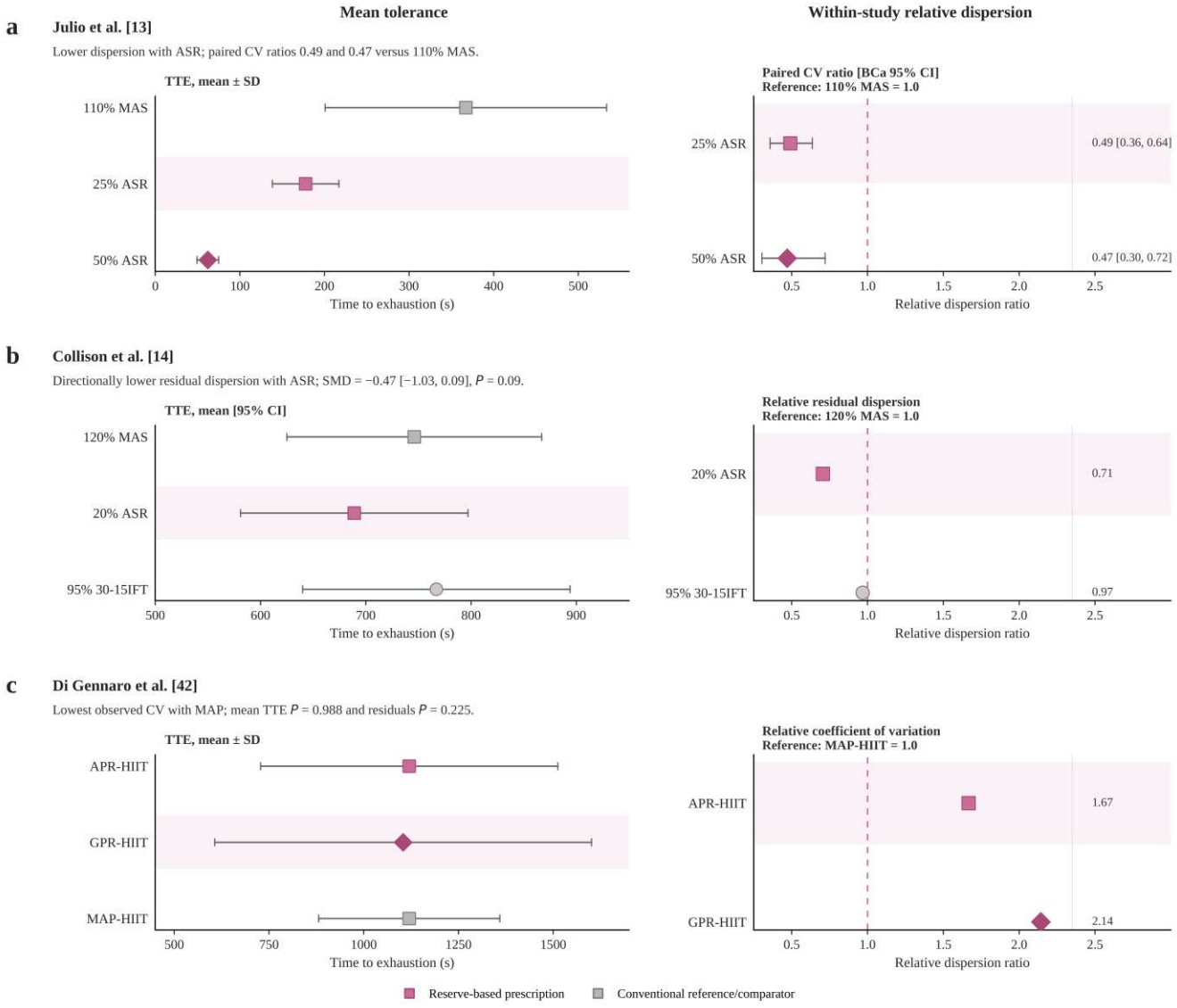


Fig. 6 Structured quantitative synthesis of acute direct-prescription evidence for mean tolerance and response standardization [13, 14, 42].

Each study occupies one row. The left column summarizes mean time to exhaustion. The right column expresses the available within-study dispersion metric relative to the study-specific reference condition. Dispersion metrics differ across studies and are shown only for within-study directional comparison; they do not represent a common effect measure and were not pooled. Horizontal positions should not be compared quantitatively across studies because the dispersion metrics are not commensurate.

3.6 Predictor, profile, decision, and longitudinal evidence

Eleven studies contributed predictor, profile, decision, moderation, or longitudinal monitoring evidence. Acute predictor and profile studies generally showed that ASR or APR was associated with exercise tolerance, metabolic responses, neuromuscular responses, or relative supramaximal demand, although the strength and direction of these associations varied across exercise formats and athlete profiles [21-23, 51, 54].

Only two studies directly evaluated incremental prediction beyond conventional or component anchors [43, 52]. In endurance trained cyclists, associations between reserve measures and exercise tolerance became stronger at higher supramaximal intensities, but adjusted APR relationships were weakened after peak power output was considered, whereas MPR retained associations at selected higher intensities [43]. In repeated sprint exercise, vVO_2max was retained as an independent predictor of fatigue resistance, whereas ASR was not retained in the same model [52]. Overall, the available evidence did not show a consistent incremental predictive advantage of reserve measures beyond their component anchors.

Evidence for training decisions and longitudinal monitoring was also limited. Profile guided allocation did not show a clear advantage in one randomized study [26], while one nonrandomized study reported several profile by training interactions [27]. Longitudinal monitoring findings were inconsistent, with one cohort showing stronger associations for MAS based exposure [24] and another reporting exploratory associations for 30% ASR thresholds in seven athletes [25]. These evidence

patterns are summarized in Fig. 7, with supporting quantitative results in Additional file 1, Supplementary Fig. S4.



Fig. 7 Evidence landscape for predictor, profile, decision, and longitudinal applications [21-27, 43, 51, 52, 54].

The matrix maps the subset of included studies contributing to association or profile description, normalization, adjusted prediction, treatment moderation, profile-guided decision, or longitudinal monitoring evidence. Marker shape denotes design family and marker colour denotes a stage-specific evidence signal. A supportive signal at the association or profile or normalization stages indicates characterization or normalization of relative demand and does not imply independent predictive value. Mixed or conditional indicates that direction or strength depended on intensity, outcome, profile, or model specification. No clear or negative indicates absent, null, or contrary evidence at that stage. Statistical significance alone did not determine classification. Blank cells indicate that no eligible study directly addressed that stage. Full coding rules and cell-level rationales are provided in Additional file 1, Section S3.8. Direct chronic prescription evidence is summarized in Figs. 3 to 5 and acute direct-prescription evidence in Fig. 6.

3.7 Environmental-modifier evidence

Three reports from two independent cycling cohorts examined intermittent cycling prescribed at 30% APR under graded hypoxia [45, 46, 53]. Soo et al. [46] and the companion report by Girard and Racinais [45] arose from the same project and participant cohort. In this cohort, completed efforts during the initial exhaustive bout decreased from 39 ± 30 in normoxia to 22 ± 13 in moderate hypoxia and 13 ± 6 in severe hypoxia [46]. The companion report showed reductions in rapid torque development after exhaustive exercise, with no clear differences in these decrements across oxygen conditions [45]. In a separate cohort, completed efforts similarly decreased from 23 ± 9 in normoxia to 16 ± 6 in moderate hypoxia and 9 ± 3 in severe hypoxia [53]. Despite the reduction in mean exercise tolerance, the descriptive coefficient of variation for completed efforts did not increase with greater hypoxic severity in either cohort. Neither cohort included a direct MAP based prescription comparator.

3.8 Certainty of evidence

Certainty was rated moderate for chronic observed response dispersion and low for chronic mean $\dot{V}O_2max$ adaptation, chronic high intensity and sport specific performance adaptation, and profile guided allocation. Certainty was very low for acute direct standardization, general predictor or profile evidence, incremental prediction beyond conventional anchors, treatment moderation, and longitudinal individualized threshold monitoring (Table 3). Environmental modifier reports were exploratory contextual evidence and were not assigned a separate GRADE rating.

236 **Table 3 Summary of findings and certainty of evidence**

Review question	Evidence base	Main result	Certainty	Main certainty considerations
Chronic mean $\dot{V}O_2max$ adaptation	5 randomized parallel studies [16-20]; N = 104 in primary comparisons	MD = 1.00 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% CI -0.03 to 2.03	Low	Risk of bias was generally some concerns and the pooled interval remained compatible with no clear mean difference.
Chronic observed response dispersion	5 randomized parallel studies [16-20]; N = 104 in primary comparisons	CVR = 0.311; 95% CI 0.229 to 0.423. VR sensitivity = 0.38; 95% CI 0.29 to 0.51.	Moderate	Consistent CVR and VR signals support lower observed dispersion, but contributing studies had some risk-of-bias concerns, the effect was not uniform across outcome domains, and neither metric isolates true biological response variance from measurement error. The Moderate rating applies specifically to the observed dispersion contrast and does not imply moderate certainty that reserve-based prescription reduces true biological response variability or improves athlete-level training decisions.
Chronic high intensity and sport specific performance adaptation	Several chronic direct studies [16-20, 49, 50]; 3 rowing studies in exploratory 2000 m time trial synthesis [18, 20, 49]	No consistent mean advantage; pooled 2000 m change MD = 0.08 s, 95% CI -2.72 to 2.89; performance dispersion was outcome dependent	Low	Small and heterogeneous evidence base, one high risk study in the pooled 2000 m analysis, imprecision, and inconsistent performance-dispersion direction.
Acute direct standardization	3 direct acute crossover or repeated-measures studies [13, 14, 42]	Lower dispersion in Julio et al. [13]; directional reduction in Collison et al. [14]; higher observed CV under reserve conditions in Di Gennaro et al. [42]	Very low	Sparse studies, some risk-of-bias concerns, inconsistent direction, and noncommensurate variability metrics.
General predictor or profile	Acute predictor and profile studies	Reserve and profile measures frequently described	Very low	Higher risk-of-bias concern, heterogeneous predictors and

Review question	Evidence base	Main result	Certainty	Main certainty considerations
evidence	[21-23, 43, 51, 52, 54]	response heterogeneity, with strong context dependence		outcomes, small samples, and limited direct testing against conventional anchors.
Incremental prediction beyond conventional anchors	Direct adjusted prediction evidence from Di Gennaro et al. [43] and Belechris et al. [52]	Some MPR associations persisted after adjustment in Di Gennaro et al. [43], whereas ASR showed no independent value in the Belechris et al. model [52]	Very low	Small samples, higher risk-of-bias concern, intensity dependence, and inconsistent incremental findings.
Profile-guided allocation	1 randomized parallel trial [26]	No clear advantage of ASR-profile-guided allocation [26]	Low	Single study with some risk-of-bias concerns and limited precision for decision-level effects.
Treatment moderation	1 nonrandomized training study [27]	Several profile-by-training interactions; adjusted R^2 up to about 0.52 [27]	Very low	Serious risk of bias, small sample, and numerous tested interactions.
Longitudinal individualized thresholds	2 prospective observational cohorts [24, 25]	MAS based thresholds showed stronger associations in Fitzpatrick et al. [24], whereas exploratory ASR associations were reported in Kavanagh et al. [25] with n = 7	Very low	Higher risk-of-bias concern, very small samples, and inconsistent results.

APR anaerobic power reserve; ASR anaerobic speed reserve; CI confidence interval; CV coefficient of variation; CVR coefficient of variation ratio; MAP maximal aerobic power; MAS maximal aerobic speed; MD mean difference; MPR maximal power reserve; PPO peak power output; $\dot{V}O_2max$ maximal oxygen uptake.

4 Discussion

The present review found no clear evidence that reserve-based prescription improves mean $\dot{V}O_2max$ or sport-specific performance adaptation more than conventional individualized aerobic anchors. Lower observed dispersion in chronic aerobic adaptation was the most consistent finding, with less consistent patterns across performance and hormonal outcomes. Acute direct comparisons produced mixed findings, while evidence for incremental prediction, profile-guided training decisions, treatment moderation, and longitudinal monitoring remained limited. These findings suggest a possible role for ASR and APR in aligning supramaximal training demands across athletes with different aerobic and maximal performance profiles, although this has not yet been shown to improve training decisions or reduce true biological response variability.

4.1 Mean adaptation, response dispersion, and individualization

Similar mean adaptations across prescription methods indicate that both reserve based and conventional individualized approaches can provide substantial training stimuli when incorporated into structured high intensity training. This may be practically relevant in team settings, where coaches often apply a common session structure across athletes with different aerobic and maximal performance profiles. The concordant CVR and VR findings further indicate that this dispersion pattern was not dependent solely on the response mean contained in the coefficient of variation [16-18, 20].

This pattern is consistent with the physiological rationale for using the reserve at supramaximal intensities. Athletes with similar MAS or MAP can differ substantially in maximal sprint speed or power, so the same percentage of an aerobic anchor can place them at different positions within their available supramaximal range. ASR and APR account for this difference by locating the prescribed workload relative to both aerobic and maximal performance landmarks [11]. Acute studies have shown that this approach can reduce variation in exercise tolerance under some supramaximal conditions, although the effect depends on

the exercise format [13, 14]. The inconsistent acute findings may reflect differences in how well the reserve components match the physiological demands of the exercise task. In running, ASR is derived from MAS and MSS, and both anchors are closely related to the speed range used during short supramaximal intervals. In cycling, however, APR and GPR are constructed from MAP and sprint or Wingate based upper anchors, whereas tolerance during repeated longer intervals is also influenced by the power duration relationship, recovery between bouts, and the capacity to sustain work above the aerobic boundary. A fixed fractional position within APR or GPR may therefore represent different physiological demands between athletes even when the mathematical prescription is identical. Measurement characteristics may further contribute to this variation because error or protocol dependence in either component is propagated into the derived reserve. Acute standardization should therefore be interpreted as task specific rather than as an inherent property of reserve-based prescription. Future studies should match the reserve definition and component measurements more closely to the duration and energetic demands of the prescribed intervals. Over repeated training, a more comparable relative supramaximal exposure provides a plausible basis for the narrower distribution of aerobic adaptation observed in the present review.

The absence of an equally clear advantage for maximal speed, power, and sport-specific performance may partly reflect the training stimulus delivered in the available studies. Most chronic reserve based interventions used repeated supramaximal intervals designed to accumulate substantial work. Such sessions provide substantial supramaximal metabolic work but relatively limited exposure close to maximal sprint speed or maximal sprint power. This interpretation is consistent with the longitudinal analysis in the present review: in every active training arm with complete component data, the aerobic anchor improved more than the maximal anchor, and the derived reserve decreased as a mathematical consequence of the component difference. The performance findings point in the same direction. Load matched sprint interval training produced clearer improvements in linear sprint speed and several biomotor outcomes than ASR based HIIT in soccer players, while APR based rowing produced lower observed dispersion in maximal cardiorespiratory adaptations without a consistent advantage for critical power or 2000 m performance[17,

18]. The current chronic literature has therefore examined ASR and APR primarily as methods for scaling supramaximal interval training. Evidence remains much less developed for using the reserve specifically to improve the maximal performance landmark itself.

These findings generate a specific hypothesis that the optimal position within ASR or APR depends on the intended adaptation, but the available studies do not establish target reserve fractions for practice. Lower or moderate reserve fractions combined with sufficient accumulated work should be tested prospectively when the target is cardiorespiratory adaptation and lower response dispersion. Higher reserve positions, shorter work bouts, longer recovery, and explicit maintenance of high speed or power should be tested when the target is MSS or maximal sprint power. Sport-specific performance also requires the exercise mode and interval structure to reflect event demands. Future trials should therefore manipulate reserve fraction, work duration, recovery, and exercise mode factorially or in directly comparable prescriptions to determine which combinations best match specific physiological and performance targets.

4.2 Predictive and decision value of reserve profiling

The current evidence supports a clearer role for reserve profiling in describing relative supramaximal demand than in guiding prospective training decisions. Most studies have examined associations with exercise responses or the normalization of relative demand, while fewer have tested whether reserve information improves prediction beyond its component anchors or changes the training selected for an athlete. The central question is whether reserve information explains variation already contained in its aerobic and maximal components or adds prospective information beyond those measures.

Several studies have used ASR or APR to examine why the same external workload may impose different relative demands across athletes. During a standardized intermittent running session, the proportion of ASR represented by the prescribed workload was related to exercise tolerance and early metabolic responses [23]. In judo athletes, expressing exercise intensity relative to ASR reduced the apparent differences between athletes that were evident when intensity was expressed relative to MAS, although

MAS and MSS remained at least as strongly related to performance as ASR itself [54]. Acute studies have also shown that baseline reserve characteristics are associated with subsequent biochemical and neuromuscular responses. These relationships were evident after a standardized 110% MAS session [22], but varied across interval formats in female soccer players [21]. Profile-dependent changes in sprint and maximal speed performance have also been reported after repeated sprint exercise [51]. Overall, these studies support the use of reserve information to interpret relative supramaximal demand, while also showing that the observed response depends on the exercise task and does not necessarily indicate independent predictive value.

ASR and APR are derived from aerobic and maximal performance measures, so their associations with exercise responses may partly reflect information already contained in those components. Reserve variables are deterministic functions of their component anchors. For standard ASR, the reserve equals MSS minus MAS, with analogous dependence for APR. Including both component anchors and their difference in the same unconstrained regression would create exact linear dependence. When a reserve is added to a model containing only one component anchor, any improvement in prediction may partly represent information carried by the omitted component rather than a distinct biological construct. Incremental prediction should therefore be judged relative to a clearly specified baseline model and interpreted as added predictive information, not as evidence that the reserve is an independent physiological mediator. In endurance trained cyclists, relationships between reserve measures and exercise tolerance became stronger as intensity approached and exceeded MAP. However, the independent contribution of APR was reduced after maximal power was considered, while maximal power itself remained strongly related to tolerance at the higher intensities [43]. A comparable finding was reported during repeated sprint exercise, where vVO_2max was retained as an independent predictor of fatigue resistance whereas ASR was not [52]. Reserve information may therefore become more relevant as exercise moves further into the supramaximal domain, but it is not yet clear whether it improves prediction beyond MAS, MAP, MSS, or maximal power.

ASR based locomotor profiling describes the balance between aerobic and maximal speed capacities, but current evidence does not yet show that athletes should be assigned different training solely on the basis of these profiles. Profile guided training did not produce a clear advantage in adaptation in youth soccer players [26]. More recent work has reported interactions between baseline locomotor characteristics and adaptation to HIIT and SIT [27]. These findings raise a more specific question: whether different locomotor profiles respond differently to HIIT, SIT, or other high intensity training formats. Demonstrating such profile by training interactions would provide a stronger basis for using reserve profiling to select training.

In one cohort, MAS based exposure explained subsequent changes in aerobic fitness better than exposure above 30% ASR [24], whereas a later study reported relatively strong associations between fitness change and the time and distance accumulated above 30% ASR [25]. The available evidence therefore does not identify a consistent advantage of ASR derived thresholds over conventional MAS based monitoring. Future work should compare whether ASR or APR adds predictive information beyond established aerobic and maximal performance measures and whether this value changes across exercise intensities. For training selection, studies should test prospectively whether athlete profile modifies the response to different training formats. If these interactions are reproducible, reserve profiling could begin to inform which athletes are more likely to benefit from HIIT, SIT, or other forms of high intensity training.

4.3 Component measurement and longitudinal recalibration

ASR and APR depend directly on how their component measures are determined. A recent systematic review showed that methods used to assess MAS and MSS are not interchangeable and that both test protocol and operational definition can affect the resulting reserve value [15]. In the present review, recent included studies more often used MSS procedures aligned with externally supported protocols, whereas several field-based MAS estimates had more limited or indirect support. This pattern describes measurement alignment within the included evidence base and does not establish a temporal improvement in testing practice.

ASR and APR are calculated from two independently measured components, so error or protocol-related variation in either component is carried into the reserve. For the same athlete, ASR may differ depending on whether MAS is obtained from a VO_2-based treadmill criterion, a time trial, or a field test, even when MSS is measured in the same way. The same applies to APR, as MAP and maximal sprint power are also sensitive to test protocol and operational definition. Field tests remain practical in applied settings, but component definitions and testing procedures should be reported clearly and kept consistent across repeated assessments.

Across chronic studies with complete component data, the aerobic component improved more than the maximal performance component. Because ASR and APR are derived from the difference between these components, the reserve narrowed as a mathematical consequence of their unequal changes. The same component pattern occurred after both reserve based and conventional individualized training, indicating that reserve contraction primarily reflected differential adaptation of the two component anchors. As training progresses, a workload set from the baseline reserve may therefore occupy a different relative position within the athlete's updated performance range.

The available evidence does not indicate how often the component measures should be reassessed during training. Single-anchor prescriptions also require updating as fitness changes, but reserve-based recalibration requires reassessment of both component anchors. Its practical value therefore depends on whether this additional testing burden produces measurable improvements in observed response dispersion or training decisions. Future longitudinal studies should repeat both component tests during longer training periods and interpret observed changes against the expected measurement error of each test. Recalculated ASR or APR values could then be used to determine whether the intended relative training intensity is maintained as fitness changes. This would also help establish whether longitudinal changes in the reserve exceed normal test-to-test variation.

4.4 Reserve based prescription under hypoxia

The lower observed dispersion in chronic aerobic adaptation with ASR/APR prescription may be particularly relevant in hypoxic training, where responses to the same external stimulus can vary considerably. Such variability may affect the exercise dose achieved and subsequent training adaptations. More consistent responses could help coaches deliver the intended training stimulus across athletes while retaining a common session structure.

A major practical challenge in hypoxic training is the large inter-individual variability in response to a given external hypoxic stimulus. Even at the same inspired oxygen fraction, athletes can show substantial differences in peripheral oxygen saturation and repeated sprint performance, and such differences may influence the amount of mechanical work completed during training and subsequent adaptation [55, 56]. Monitoring SpO_2 may help individualize the hypoxic dose, while exercise intensity prescription represents another source of variability. Conventional supramaximal prescriptions based on a single aerobic anchor (e.g., 120% MAS) do not account for the athlete's upper performance boundary. This issue may become more relevant under hypoxia because the two components of ASR or APR can be modified to different degrees. Maximal aerobic capacity and endurance performance decrease as oxygen availability is reduced [57], whereas single sprint performance and maximal force or power during isolated efforts are generally preserved under acute hypoxia [58, 59]. A larger reduction in MAS or MAP than in MSS or maximal sprint power would therefore widen the available reserve and change the relative supramaximal demand represented by a fixed aerobic anchor. Barometric conditions may further modify this relationship. At terrestrial altitude, reduced air density lowers aerodynamic drag and may increase sprint velocity, particularly in locomotor tasks where air resistance contributes substantially to performance. This effect may produce a larger change in ASR under hypobaric than normobaric hypoxia, whereas it has little relevance to the prescribed external power of stationary cycling [58, 60].

Findings from the two independent cycling cohorts included in this review provide preliminary support for this rationale [45, 46, 53]. During intermittent cycling prescribed at 30% APR, exercise tolerance

decreased substantially as hypoxia became more severe, whereas the descriptive CV of completed efforts did not increase in either cohort. These findings describe observed dispersion during APR-prescribed exercise under hypoxia, but do not establish that APR improves response standardization relative to conventional prescription.

Future studies should reassess both reserve components under the hypoxic condition in which training is prescribed, particularly when transferring prescriptions from normoxia to hypoxia or between normobaric and hypobaric environments. Direct comparisons between conventional aerobic anchor and ASR/APR prescription should determine whether environment-specific recalibration improves exercise tolerance and between-athlete response consistency, with exercise mode considered where aerodynamic effects may alter MSS. Longer interventions are needed to establish whether any improvement in acute response standardization translates into more homogeneous chronic adaptations.

4.5 Strengths and limitations

The review searched six databases, supplemented database retrieval with reference-list screening of a key ASR/APR review and legacy-terminology checks, and linked companion reports to avoid duplicate weighting of overlapping cohorts. Direct prescription, predictor, decision, longitudinal, and exploratory environmental evidence were analysed according to the questions each design could address. Chronic effects were examined for both mean adaptation and observed between-athlete dispersion, with sensitivity analyses for comparator selection, model specification, rounding, and alternative estimators. Component-anchor methods were also examined in a separate post hoc contextual methodological appraisal to make measurement uncertainty explicit.

The evidence base was small and heterogeneous in exercise mode, reserve definition, and comparator. ASR and APR strata were confounded with exercise modality, and acute dispersion outcomes could not be pooled because variability was reported using different metrics. Predictor, profile, and longitudinal studies were mostly small and observational, limiting conclusions about incremental prediction and training

decisions. Performance analyses were exploratory, and most outcome families contained too few comparable studies for robust pooling. Women and girls were underrepresented across the evidence base, with many studies including only male participants or not reporting sex.

CVR and VR quantify observed dispersion and cannot separate true differences in training adaptation from measurement error or normal biological variation. Repeated baseline testing and participant-level measurement error were rarely available. Because dispersion was derived from individual percentage change, these estimates also inherit the statistical dependence of percentage change on baseline values. The primary mean-effect model used postintervention values to avoid imputing unreported preintervention-to-postintervention correlations. Given the small group sizes, chance baseline imbalance could still influence postintervention contrasts; change-score sensitivities produced smaller pooled estimates and did not alter the conclusion of no clear mean advantage. Supplementary reference-list screening and legacy-terminology checks were added after initial screening. The outcome-domain map, reserve-dynamics analysis, component-anchor appraisal, and prescription-drift calculations were post hoc and descriptive. The anchor appraisal relied on selected external measurement evidence rather than a dedicated systematic search and should therefore be interpreted as contextual. Formal assessment of publication bias was not possible for the small quantitative evidence bodies. Environmental evidence was limited to two hypoxic cycling cohorts without a direct conventional prescription comparator.

5 Conclusion

Current evidence does not establish greater mean aerobic or sport-specific performance adaptation with reserve-based prescription than with conventional individualized aerobic anchors. Lower observed dispersion in chronic aerobic adaptation was the most consistent supportive finding, but it does not establish lower true biological response variability. Evidence for independent prediction, profile-guided training allocation, and longitudinal monitoring remains limited. Future studies should repeatedly assess both reserve

components, compare reserve-based prescription directly with established individualized approaches, and test whether reserve information improves training selection or response standardization beyond its component anchors. Hypoxic conditions that affect the two components differently provide a useful experimental setting for these comparisons.

List of abbreviations

APR, anaerobic power reserve

ASR, anaerobic speed reserve

CI, confidence interval

CV, coefficient of variation

CVR, coefficient of variation ratio

GRADE, Grading of Recommendations Assessment, Development and Evaluation

GPR, glycolytic power reserve

HIIT, high intensity interval training

MAP, maximal aerobic power

MAS, maximal aerobic speed

MD, mean difference

MPR, maximal power reserve

MSS, maximal sprint speed

PICOS, population, intervention, comparator, outcomes, and study design

PPO, peak power output

QUIPS, Quality in Prognosis Studies

REML, restricted maximum likelihood

RoB 2, revised Cochrane risk of bias tool for randomized trials

ROBINS-I, Risk Of Bias In Non-randomized Studies of Interventions

SIT, sprint interval training

VIFT, final velocity reached in the 30-15 Intermittent Fitness Test

VO_2max, maximal oxygen uptake

VR, variability ratio

Declarations

Ethics approval and consent to participate

Not applicable. This systematic review synthesized data from previously published studies and did not involve new participant recruitment or collection of identifiable personal data.

Consent for publication

Not applicable.

Availability of data and materials

The data supporting the findings of this review are provided in the article and its supplementary information. Additional extracted datasets and Python analysis scripts are available from the corresponding author upon reasonable request.

Competing interests

Suyan Xu, Shiwu Li, Jianhao Lu, Yan Chen, Guoyan Zhang, Zhenxiang Guo, Dapeng Bao, Øyvind Sandbakk and Grégoire P. Millet declare that they have no competing interests.

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Authors' contributions

SX conceived the review, developed the methodology, extracted data, performed the statistical analyses, and drafted and revised the manuscript. SL screened studies, extracted data, and contributed to drafting the Discussion section. JL contributed to drafting the manuscript. YC provided supervision and advice. GZ contributed to the conceptualization of the review and development of the Discussion section. ZG contributed to the conceptualization of the review and writing of the manuscript. DB supervised the work, provided advice, and revised the manuscript. ØS and GPM revised the manuscript and provided scientific advice. All authors read and approved the final manuscript.

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