



Impact of Pre-exercise Hypohydration on Aerobic Exercise Performance, Peak Oxygen Consumption and Oxygen Consumption at Lactate Threshold: A Systematic Review with Meta-analysis

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Abstract

Background Progressive exercise-induced dehydration may impair aerobic exercise performance (AEP). However, no systematic approach has yet been used to determine how pre-exercise hypohydration, which imposes physiological challenges differing from those of a well-hydrated pre-exercise state, affects AEP and related components such as peak oxygen consumption ($\dot{V}O_{2\text{peak}}$) and $\dot{V}O_2$ at lactate threshold ($\dot{V}O_{2\text{LT}}$).

Objective To determine, using a systematic approach with meta-analysis, the magnitude of the effect of pre-exercise hypohydration on AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$.

Design This was a systematic review with meta-analysis of well-controlled studies.

Data Sources MEDLINE, SPORTDiscus and CINAHL databases and cross-referencing.

Inclusion Criteria for Selecting Studies (1) well-controlled human (≥ 18 years) studies; (2) pre-exercise hypohydration induced at least 1 h prior to exercise onset; (3) pre-exercise body mass loss in the hypohydrated, experimental condition was $\geq 1\%$ and $\geq 0.5\%$ than the well-hydrated, control condition; (4) following the dehydrating protocol body mass change in the control condition was within -1% to $+0.5\%$ of the well-hydrated body mass.

Results A total of 15 manuscripts were included, among which 14, 6 and 6 met the inclusion criteria for AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$, respectively, providing 21, 10 and 9 effect estimates, representing 186 subjects. Mean body mass decrease was $3.6 \pm 1.0\%$ (range 1.7–5.6%). Mean AEP test time among studies was 22.3 ± 13.5 min (range 4.5–54.4 min). Pre-exercise hypohydration impaired AEP by $2.4 \pm 0.8\%$ (95% CI 0.8–4.0%), relative to the control condition. Peak oxygen consumption and $\dot{V}O_{2\text{LT}}$, respectively, decreased by $2.4 \pm 0.8\%$ (95% CI 0.7–4.0%) and $4.4 \pm 1.4\%$ (95% CI 1.7–7.1%), relative to the control condition. Compared with starting an exercise hypohydrated, it is respectively likely, possible and likely that AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ benefit from a euhydrated state prior to exercise. Meta-regression analyses did not establish any significant relationship between differences in body mass loss and differences in the percent change in AEP or $\dot{V}O_{2\text{LT}}$. However, $\dot{V}O_{2\text{peak}}$ was found to decrease by $2.6 \pm 0.8\%$ (95% CI 0.7–4.5%) for each percent loss in body mass above a body mass loss threshold of 2.8%.

Conclusion Pre-exercise hypohydration likely impairs AEP and likely reduces $\dot{V}O_{2\text{LT}}$ (i.e., the aerobic contribution to exercise was lower) during running and cycling exercises ≤ 1 h across different environmental conditions (i.e., from 19 to 40 °C). Moreover, pre-exercise hypohydration possibly impedes $\dot{V}O_{2\text{peak}}$ during such exercises.

1 Introduction

Several physiological attributes contribute to the modulation of aerobic exercise performance (AEP), and peak oxygen consumption ($\dot{V}O_{2\text{peak}}$) as well as lactate threshold is known to play a predominant role [1]. Peak oxygen consumption and lactate threshold may be impacted by a myriad of endogenous physiological factors, one of which is hydration status. In this regard, numerous studies have demonstrated that hypohydration reduces plasma volume [2, 3] and stroke

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Key Points

Although information exists on the magnitude of the impact of exercise-induced dehydration upon endurance performance, it is still unknown whether and to what extent pre-exercise hypohydration impairs AEP and related components such as $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$.

Conditions such as illness, inadequate rehydration following exercise or lack of time to fully replace fluid losses between two closely-spaced events may lead athletes to begin training or competition hypohydrated.

From a statistical point of view, AEP, $\dot{V}O_{2\text{LT}}$ and $\dot{V}O_{2\text{peak}}$ are all significantly impacted by pre-exercise hypohydration across different environmental conditions (i.e., for ambient temperatures ranging from 19 to 40 °C).

From a practical point of view, end users of these findings should expect that pre-exercise hypohydration will (1) likely impair AEP by 2.4% and likely reduce $\dot{V}O_{2\text{LT}}$ by 4.4% and; (2) possibly impede $\dot{V}O_{2\text{peak}}$ by 2.4%.

The current findings highlight the importance of starting endurance exercises in a well-hydrated state.

volume [4–8], the combination of which may contribute to degrade $\dot{V}O_{2\text{peak}}$ [3, 9–11]. Moreover, hypohydration-associated increase in core temperature may contribute to enhance glycogenolysis [12–14] and lactate production [14, 15], thereby decreasing lactate threshold [16]. Given the above observations, it is legitimate to believe that a hypohydration-mediated decrease in $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ at lactate threshold ($\dot{V}O_{2\text{LT}}$) may contribute to the impairment of AEP [17], especially in warm/hot environments.

Exercise-induced dehydration has been shown to impair AEP under certain circumstances. For instance, both the American College of Sports Medicine [18] and National Athletic Trainers' Association [19] organisations indicate in their respective position statements on fluid replacement that AEP becomes compromised when exercise-induced dehydration reaches 2% of body mass. Holland et al. [20] report that cyclists should expect gains in performance of at least 2–3% when fluid is consumed during moderate intensity exercise > 1 h, compared with no fluid consumption. Goulet and Hoffman [21] observed that whether one decides to drink ad libitum or according to a plan is unlikely to be relevant during out-of-doors running or cycling exercises of 1–2 h. In fact, a difference in performance of less than 1% is to be expected between drinking strategies. This collection of findings suggests that it is important to consume fluid and limit the extent of dehydration during prolonged or moderate intensity aerobic exercise.

Singularly or in combination, favorable exercise durations, exercise intensities, ambient temperatures and fluid availabilities may prevent sufficient dehydration to develop during aerobic exercise such that AEP or related components such as $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ can be well preserved. But what if someone, even under the best exercise conditions possible, starts an aerobic exercise in a hypohydrated state? Could it impact AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$? Moreover, does pre-exercise hypohydration impact AEP to an extent similar to exercise-induced dehydration? It would be relevant to provide answers to these questions, because illness, extreme weather, inadequate rehydration following exercise or lack of time to fully replace fluid losses between two closely spaced events may lead athletes to begin a training session or a competition hypohydrated [22, 23].

Several studies have examined the impact of pre-exercise hypohydration on AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ [24–30]. However, nothing has been done to summarize and determine the magnitude of the effect of pre-exercise hypohydration on these variables, thereby indicating a need to use a systematic approach with meta-analysis to further our understanding of this research field. Consequently, this study aimed to use this methodological technique, known to yield the highest level of evidence about a treatment effect, and to overcome the difficulties in performing large-scale randomized controlled trials [31], to provide answers to the following questions: (1) What is the magnitude of the effect of pre-exercise hypohydration on AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$?; (2) Are there any associations between the magnitude of pre-exercise hypohydration and changes in AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$?; (3) Can confounding factors such as cardiorespiratory fitness, ambient temperature or the elapsed time between the end of the dehydration protocol and onset of testing impact the relationship between pre-exercise hypohydration and AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$?

2 Methods

2.1 Search Strategies

The strategy used for selecting research articles is described in Fig. 1. A thorough review of the literature, limited to the French- and English-language references, was made using MEDLINE, SPORTDiscus and CINAHL databases. To find the articles of interest the following keywords and MeSH headings were used alone or in combination, combining a “title field” and “abstract field” research: “hydration”, “dehydration”, “hypohydration”, “exercise performance”, “physical performance”, “endurance performance”, “aerobic performance”, “lactate threshold”, “oxygen consumption”, “work”, “time”, “power”, “cycling” and “running”. Case studies, published abstracts, manuscripts published

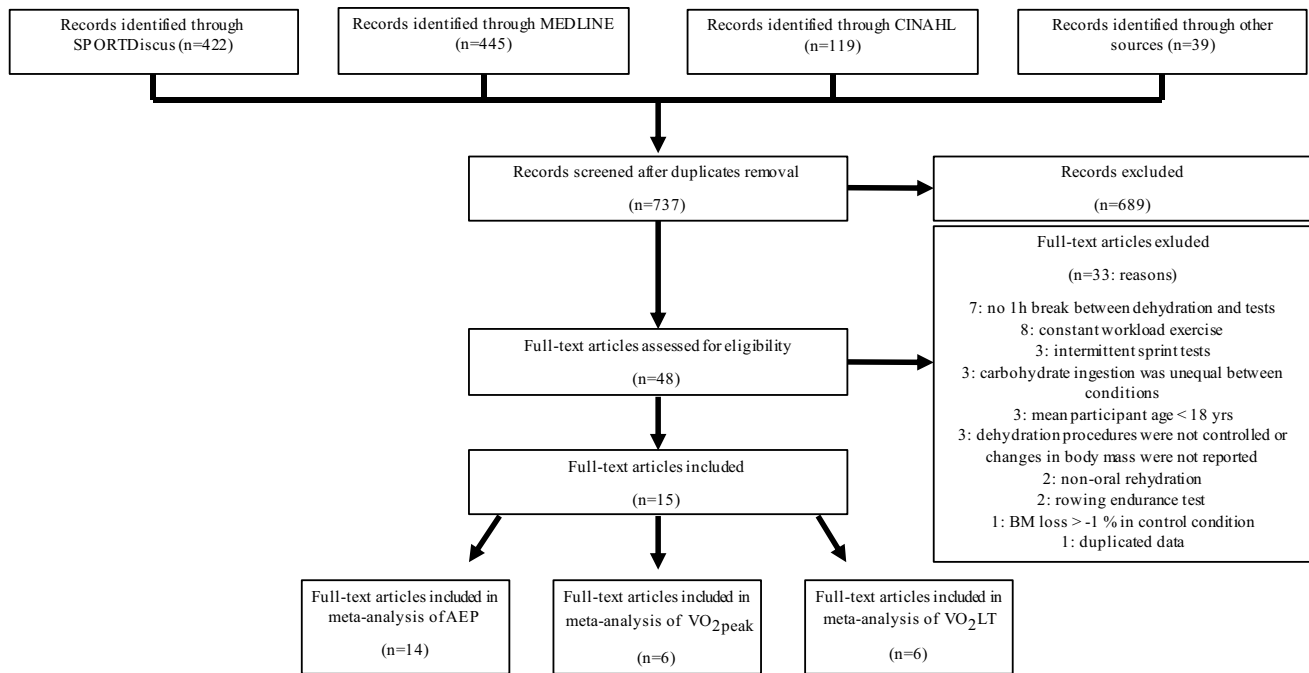


Fig. 1 Flowchart showing the selection process used for the inclusion and exclusion of research articles. *AEP* aerobic exercise performance, *BM* body mass, $\dot{V}O_{2peak}$ peak oxygen consumption, $\dot{V}O_{2LT}$ oxygen consumption at lactate threshold

in non-peer-reviewed journals and conference proceedings were not included. References included in retrieved articles as well as those from two narrative reviews [32, 33] were investigated. Abstracts of all potential articles were read; if they revealed an intervention for the determination of the impact of pre-exercise hypohydration on AEP, $\dot{V}O_{2peak}$ or $\dot{V}O_{2LT}$, then the methodological section was assessed for eligibility. When necessary, the corresponding authors of original studies were contacted to resolve statistical issues or determine whether they would agree to share raw experimental data or exact *p* values of a priori defined comparisons. The last day of the literature search was March 1 2019.

2.2 Inclusion Criteria

To be eligible for inclusion research manuscripts had to meet the following criteria: (1) well-controlled research design and dehydration protocol; (2) hypohydration was induced before, not during testing; (3) pre-exercise hypohydration was not induced by caloric restriction or diuretics; (4) pre-exercise hypohydration was induced at least 1 h prior to testing; (5) pre-exercise body mass loss in the hypohydrated experimental condition was $\geq 1\%$ and $\geq 0.5\%$ than the well-hydrated control condition; (6) following the dehydrating protocol body mass change in the control condition was within -1% to $+0.5\%$ of the well-hydrated body mass; (7)

if the control condition included a rehydration period, fluid replacement was given orally; (8) if food was provided, the amount was identical between conditions; (9) continuous (not intermittent) running or cycling aerobic exercises allowing performance measurement; (10) exercise performed $> 15\text{ }^{\circ}\text{C}$; (11) participants aged ≥ 18 years old and; (12) data required to compute changes in body mass and percent changes in AEP, $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$ were provided.

2.3 Assessment of Trial Quality

Assessment of trial quality was not performed so as not to bias findings. In fact, it has been demonstrated that the type of scale used to assess trial quality can dramatically influence the interpretation of meta-analytic studies [34].

2.4 Data Extraction

Upon articles selection and inclusion, data were extracted and coded in spreadsheets (Microsoft Office Excel) for (1) study characteristics; (2) participant characteristics; (3) exercise protocol characteristics; (4) dehydration protocol characteristics; (5) changes in body mass from before to after the dehydrating protocol; and (6) changes in AEP, $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$ between the experimental and control condition. Data were coded by TAD and EDBG and any disagreement was discussed, and a consensus reached.

2.5 Aerobic Exercise Performance and Measurement of Exercise Intensity

The intensities at which the AEP tests were performed are reported as mean percentages of the $\dot{V}O_{2\text{peak}}$ used to complete the testing protocol in both conditions. Caldwell et al. [11], Castellani et al. [26], Chevront et al. [35], England et al. [36], Kenefick et al. [25], Kozlowski et al. [37] and Stewart et al. [38] reported baseline participants' $\dot{V}O_{2\text{peak}}$ and the mean power outputs at which the exercise protocol was completed. Therefore, to estimate the percentage of $\dot{V}O_{2\text{peak}}$, the relationship between mean exercise power output and $\dot{V}O_2$ was used [39]. For Casa et al. [40], the mean exercise intensity was determined using the estimated maximal heart rate [41], the mean heart rate at which the exercise was performed, and by converting the percent maximal heart rate at which the exercise was completed to a mean percentage of the $\dot{V}O_{2\text{peak}}$ using the formula of Londeree et al. [42]. With respect to Webster et al. [43], Kenefick et al. [16] and Dengel et al. [44], exercise intensity was computed by averaging $\dot{V}O_2$ values observed at the commencement of exercise, at lactate threshold and at peak exercise. Finally, for Oliver et al. [24], exercise intensity could not be calculated due to a lack of data.

2.6 Aerobic Exercise Performance and Measurement of Exercise Duration

Exercise duration of the AEP tests represents the mean total exercise time (min) completed in the experimental and control conditions. Overall exercise duration includes the pre-load exercise period and represents the mean total exercise time (min) completed in the experimental and control conditions.

2.7 Changes in Body Mass from Before to After the Dehydrating Protocol

The acute change in body mass remains the most accessible method to estimate hypohydration level under laboratory and field conditions. Although it is recognized that when exercise is used as a means to produce hypohydration the difference between pre- and post-exercise body mass also includes a small amount of non-fluid mass loss (carbon exchange) [45], its impact on body mass is typically trivial over the duration and intensity of exercise protocols usually used to produce dehydration of 2–4% body mass [46]. Therefore, in the present meta-analysis, the extent of hypohydration incurred by a dehydration protocol was taken as the percent change in body mass from prior to after the dehydration protocol and computed with the following equation:

$$\frac{\text{Post-dehydration body mass} - \text{pre-dehydration body mass}}{\text{pre-dehydration body mass}} \times 100. \quad (1)$$

Percent changes in body mass were computed only for those studies for which these data had not been computed and reported by authors.

2.8 Confounding Factors

Confounding factors such as $\dot{V}O_{2\text{peak}}$, a surrogate index of training state [47], ambient temperature under which AEP was measured [25, 35] and the elapsed time between the end of the dehydration protocol and onset of testing [48] may impact the relationships between variables of interest. Therefore, these three parameters were extracted when accessible. A dehydration protocol was considered passive when it did not use exercise to induce hypohydration or when ≥ 8 h and an overnight sleep separated the exercise-induced dehydration procedures from the AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ tests. In fact, following this time point, any residual effects of exercise or heat stress were considered null [48].

2.9 Assessment of AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$

For the sake of this meta-analysis and to render findings easier to understand and interpret, all outcomes of interest were transformed and are reported as percentage changes.

2.9.1 Computation of the Percentage Changes in AEP

Aerobic exercise performance was computed as percent changes in power output between the experimental and control condition. When AEP was tested using a time-trial type exercise protocol and the mean power output during exercise was reported, the percent changes in AEP were calculated using the following equation [49]:

$$\frac{\text{Mean power output experimental group (W)} - \text{mean power output control group (W)}}{\text{mean power output control group (W)}} \times 100, \quad (2)$$

where W represents watts, and the experimental and control conditions represent the hypohydrated and euhydrated trials, respectively.

Performance data deriving from fixed-intensity tests to exhaustion were transformed to percent changes in power output using the following equation [49]:

$$\frac{\text{Mean experimental group time to exhaustion (min)} - \text{mean control group time to exhaustion (min)}}{\text{mean control group time to exhaustion (min)}} \times 100 / (\% \dot{V}O_{2\text{peak}} \text{ at which the test was performed} / 6.4). \quad (3)$$

When studies used an incremental test to exhaustion to evaluate AEP, the percent changes in power output were calculated with the following equation [49]:

$$\begin{aligned} & \text{Mean experimental group time to exhaustion (min)} \\ & \quad - \text{mean control group time to exhaustion (min)/} \\ & \quad \text{mean control group time to exhaustion (min)} \\ & \quad \times 100 \times (1 - (\% \dot{V}O_{2\text{peak}} \text{ at which the test started}/100)). \end{aligned} \quad (4)$$

Performance data from Castellani et al. [26] and Kenefick et al. [25] were reported in kJ; therefore, they were transformed to Watts. Casa et al. [40] reported performance running time-trial times; these were converted to running speeds, considering that a 1% change in power output equals a 1% change in running speed [50]. Stewart et al. [38] tested performance using a cycling time trial. Performance times were converted to Watts on the basis that a 0.4% change in cycling time-trial time equals a 1% change in power output [51]. For the study of Caldwell et al. [11] the percent change in AEP was determined by (1) calculating baseline peak power output from baseline mean $\dot{V}O_{2\text{peak}}$ value and the formula by Hawley et al. [52] and; (2) computing a mean time to exhaustion by dividing mean peak power output by the 1-min stage load increase. In the study of Kozłowski et al. [37], the percent change in AEP was computed from the mean times to exhaustion obtained in the control and experimental conditions by dividing the total work performed on the bike (kgm/min) by the mean resistance at which the test to exhaustion was performed. The percent $\dot{V}O_{2\text{peak}}$ value at which the test was completed was determined by dividing the mean workload at which the bike test was performed by the participants' peak power output computed from the formula of Hawley et al. [52]. For Webster et al. [43], the percent $\dot{V}O_{2\text{peak}}$ value at which the test started was determined by dividing the participants' initial treadmill velocity by terminal treadmill velocity.

2.9.2 Computation of the Percentage Changes in $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$

When not provided in the manuscript, percent changes in $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ were computed using the following equation:

$$\begin{aligned} & \text{Mean experimental group } \dot{V}O_{2\text{peak}} \text{ or } \dot{V}O_{2\text{LT}} \text{ (L/min)} \\ & \quad - \text{mean control group } \dot{V}O_{2\text{peak}} \text{ or } \dot{V}O_{2\text{LT}} \text{ (L/min)/} \\ & \quad \text{mean control group } \dot{V}O_{2\text{peak}} \text{ or } \dot{V}O_{2\text{LT}} \text{ (L/min)} \times 100. \end{aligned} \quad (5)$$

2.9.3 Measurement of $\dot{V}O_{2\text{peak}}$

The explicit goal of all included studies was to determine the impact of hypohydration on maximal oxygen consumption ($\dot{V}O_{2\text{max}}$). However, as all could not confirm that criteria for attainment of $\dot{V}O_{2\text{max}}$ were met, and that several

$\dot{V}O_{2\text{max}}$ tests lasted more than 15 min [16, 43, 44], then it was decided to refer to the expression $\dot{V}O_{2\text{peak}}$. Peak oxygen consumption was either measured on an ergocycle or treadmill using fixed intensity [37, 53] or incremental tests to exhaustion with 1–4 min long stages [11, 16, 43, 44]. Studies that determined the effect of pre-exercise hypohydration on both $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ did so simultaneously using the same test.

2.9.4 Measurement of $\dot{V}O_{2\text{LT}}$

Oxygen consumption corresponding to the lactate threshold was designated as $\dot{V}O_{2\text{LT}}$. In all included studies, $\dot{V}O_{2\text{LT}}$ was measured using graded incremental exercise tests with stages lasting from 1 to 4 min, which elicits an exponential rise in lactate concentration. Various methodologies were used to determine lactate thresholds; up to now, no generally accepted fitting procedure has been established as a gold standard [54]. The lactate threshold represents different concepts used to determine the aerobic–anaerobic exercise transition and can be divided into three categories: (1) fixed lactate levels; (2) the first rise in lactate above baseline levels and; (3) a rapid and distinct change in the lactate curve [54]. In this meta-analysis, included studies verified the impact of pre-exercise hypohydration on concepts 2 and 3. Hence, the goal of this meta-analysis was not to identify the impact of pre-exercise hypohydration on a single lactate threshold concept (i.e., lactate or anaerobic threshold), but rather to report how the relationship between lactate and $\dot{V}O_2$ is impacted within the aerobic–anaerobic transition phase. England et al. [36] did not specifically determine values corresponding to a lactate threshold. Hence, $\dot{V}O_{2\text{LT}}$ was taken as the work rate at 120 W, which corresponded to the work rate at which lactate level increased 1 mmol/L above baseline level.

2.10 Statistical Analyses

2.10.1 Software Used for Analyses

Data were analyzed using the Microsoft Office Excel 2018 (version 1803, Redmond, WA, USA), Comprehensive Meta-analysis (version 2.2.064, Englewood, NJ, USA), IBM SPSS Statistics (version 21.0.0.0, Armonk, NY, USA) and Meta XL software [55] and SPSS macros provided by Lipsey and Watson [56].

2.10.2 Weighted Mean Effect Summaries

The weighted mean effect summaries were determined using the inverse variance heterogeneity (IVhet) model, which has been shown to be superior to the traditional random-effects model [55]. Specifically, simulation studies have shown

that the IVhet model retains correct coverage probabilities as well as a lower observed variance when compared with the random-effects model, regardless of heterogeneity. When possible, variances were directly calculated from the Δ standard errors or standard deviations of the net percent changes in AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ between the experimental and control condition. When no differences between the conditions were accessible, variances were calculated from the p values or confidence intervals (CI). When only $p \leq X$ was reported, p was considered equal to X , where X is any p value ≤ 0.05 . When only $p > 0.05$ was reported, individual variances for net percent changes in AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ were estimated as in Savoie et al. [48], using an imputed correlation coefficient of 0.84, which represents the weighted mean correlation coefficient computed from values of 9 individual studies.

Some research articles included more than one treatment effect for a given parameter of interest. To account for independency of research data, an important assumption of parametric statistical analyses, two separate statistical analyses were performed where, on one occasion, the weighted mean effect summary was determined with only one effect estimate and weighting factor per research article and, on the other, where each outcome was treated independently. If both approaches yielded similar figures, then the model treating each outcome independently was retained since it allows the retention of a maximum of information.

Results are reported as means \pm standard errors and were considered significant when the 95% CI did not include 0.

2.10.3 Practical Significance of the Mean Effect Summaries

The qualitative interpretation of the practical significance of the effect of pre-exercise hypohydration on AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ was made using the second-generation p value (SGPV) technique [57]. The SGPV requires specifying a CI around the null-hypothesis that would be considered equivalent to zero, from a practical point of view. If the statistically derived CI falls completely within the CI considered to be zero from a practical standpoint, then SGPV is 1; but if it falls completely outside of the zero range, the SGPV is 0. Otherwise, the SGPV is between 0 and 1, which expresses the amount of overlap between intervals. Therefore, the SGPV is an extension of the p value that, however, accounts for practical relevance. The amount of overlap between CIs was used to determine the chances for the effects to be considered practically relevant, which was estimated using the following descriptors [58]: $< 1\%$, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely or probably not; 25–75%, possibly or maybe; 75–95%, likely or probably; 95–99%, very likely; $> 99\%$, almost certainly. The smallest worthwhile percent differences in AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ were, respectively, taken as 1.5% [59], 2.5% [59] and 2.4%

[60]. They were computed from the product of the normal day-to-day variability coefficient of variations for these parameters $\times 0.5$, as recommended by Hopkins et al. [51].

2.10.4 Heterogeneity, Publication Bias and Sensitivity Analysis

Cochran's Q and I^2 statistic were both used to assess between-study heterogeneity and the degree of inconsistency among results of included studies [61]. Cochran's Q test was considered significant if $p \leq 0.1$ [62]. The following classification was used to interpret the I^2 statistic: low ($< 40\%$), moderate (40–59%), substantial ($> 60\%$) [63]. Whether there was evidence of literature bias was examined with a funnel plot visual inspection. The use of a cumulative forest plot classifying studies based on their precision was used to test if the overall observed weighted mean effect for AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ was an artifact of bias. A sensitivity analysis was performed by removing each study once from the model to determine whether this would change the magnitude of the outcome summary.

2.10.5 Meta-regression Analyses

Meta-regressions for AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ were performed using the IVhet model, with 95% robust (Huber–Eicher–White-sandwich) standard errors [55]. The IVhet model uses a method that allows for a multiplicative component of residual heterogeneity, unlike the random-effects model which uses an additive component of residual heterogeneity [55]. Multiple regression analyses were examined for the presence of multicollinearity between predictor variables (variance inflation factor). The alpha level for statistical significance was set at $p \leq 0.05$.

3 Results

3.1 Search Results

A total of 48 articles of interest were first identified as potentially eligible for the analyses; of them, 15 met all the inclusion criteria. Among the included studies 14 met the inclusion criteria for AEP [11, 16, 24–27, 35–38, 40, 43, 44, 53], 6 for $\dot{V}O_{2\text{peak}}$ [11, 16, 37, 43, 44, 53] and 6 for $\dot{V}O_{2\text{LT}}$ [11, 16, 27, 36, 43, 44]. Overall, 21 effect estimates were retrieved for AEP, 10 for $\dot{V}O_{2\text{peak}}$ and 9 for $\dot{V}O_{2\text{LT}}$.

3.2 Characteristics of Studies

Specific protocol characteristics associated with the assessment of AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$ are shown in Tables 1, 2 and

3, respectively. Reported below is an overview of the general characteristics of all included studies.

3.2.1 Characteristics of Publications

The included studies were published between 1964 and 2014 in 9 different peer-reviewed journals. Among the 15 studies, nine were performed in the USA [16, 25–27, 35, 36, 40, 43, 44], two in Australia [38, 47], one in Poland [37], one in Sweden [53], one in the UK [24] and one in Finland [11]. With one exception [16, 25], there was no overlap of authorship among the 15 publications.

3.2.2 Characteristics of Participants

A total of 186 individuals are represented in the 15 included studies (for a mean of 10 ± 3 individuals per study). Women represented 8.7% ($N = 16$) of the total sample. The mean

age, height, body mass and relative $\dot{V}O_{2peak}$ of the participants were 24 ± 2 years (range 20–31 years), 176 ± 4 cm, 75 ± 6 kg and 52 ± 6 mL/kg/min (absolute 3882 ± 306 mL/min), respectively. Nine studies reported the training state of their participants ($N = 116$): 71% were trained ($N = 82$) [11, 16, 36, 40, 43, 47], 24% were moderately trained ($N = 28$) [24, 35, 38] and 5% were untrained ($N = 6$) [47]. Saltin [53] reported marked differences between participants' training state ($N = 10$), while the other studies did not report any training state data ($N = 70$) [25–27, 44]. None of the included studies reported data about ethnicity.

3.2.3 Dehydration Protocol Characteristics

Pre-exercise hypohydration was induced passively in 15 individual studies [11, 16, 24, 26, 27, 35, 36, 40, 43, 44, 47, 53], whereas 8 used an active dehydration protocol [25, 37, 38, 53]. Of those 15 studies where hypohydration was

Table 1 Summary of protocol characteristics of included studies that evaluated aerobic exercise performance

References	Participants: N (M/W), age (years), peak oxygen consumption (mL/kg/min)	Dehydration protocol: elapsed time before testing: (0: < 3 h; 1: > 12 h)	% BM loss (CON/EXP)	Exercise protocol: exercise mode, duration (min), temperature ($^{\circ}\text{C}$), ecologically valid (0) or not (1)
Caldwell et al. [11]	32 (32/0), 21.6 (a) 16 (16/0), 21.4, 54.7 (b) 16 (16/0), 21.7, 57.6	(a) 0 (b) 0	(a) 0.0/–3.4 (b) 0.0/–4.7	Cycling (a) 11, –, 1 (b) 12.9, –, 1
Casa et al. [40]	17 (9/8), 27, –	1	–0.79/–2.27	Running, outdoor, 54.4, 27.2, 0
Castellani et al. [26]	7 (7/0), 25, 44.1	1	–0.6/–4.0	Cycling, 30, 27.5, 0
Cheuvront et al. [35]	8 (6/2), 24, 48.0	0	–0.4/–3.0	Cycling, 30, 20, 0
Dengel et al. [44]	9 (9/0), 26.4, 54.1	1	(a) –0.6/–3.3 (b) –0.6/–5.6	Cycling (a) 29.2, 22 1 (b) 28.8, 22 1
England et al. [36]	6 (6/0), 28.5, 54.0	0	0.0/–5.0	Cycling, 19.8, –, 1
Kenefick et al. [16]	14 (8/6), 20.9, 62.8	1	0.0/–3.9	Running, 30.45, 22, 1
Kenefick et al. [25]	24 (24/0), 23 (a) 8 (8/0), 23, 45.3 (b) 8 (8/0), 24, 46.3 (c) 8 (8/0), 22, 43.7	0	(a) 0.0/–4.2 (b) 0.0/–4.0 (c) 0.0/–4.1	Cycling (a) 15, 20, 0 (b) 15, 30, 0 (c) 15, 40, 0
Kozlowski (1966) [37]	10 (10/0), 23.5, 50.7	0	–0.5/–4.1	Cycling, 11.9, 20.3, 1
Merry et al. (2010) [47]	(a) 6 (6/0), 30.6, 64.0 (b) 6 (6/0), 25.2, 45.0	1	(a) –0.5/–1.8 (b) –0.2/–1.7	Cycling, 40, 24.3, 0
Oliver et al. [24]	13 (13/0), 21, 50.9	1	–0.6/–3.2	Running, 30, 19.7, 0
Saltin [53]	10 (10/0), 24.7, 54.7	(a) 0 (b) 0 (c) 0	(a) –0.3/–3.8 (b) –0.3/–3.6 (c) –0.3/–4.0	Cycling (a) 5.7, 19, 1 (b) 4.5, 19, 1 (c) 4.6, 19, 1
Stewart et al. [38]	7 (7/0), 23, 52.7	0	–0.2/–3.8	Cycling, 7.2, 21.5, 0
Webster et al. [43]	7 (7/0), 19.7, 57.0	1	0.0/–4.9	Running, 33.5, –, 1
Mean \pm SD	10 ± 3 (9/1), 24 ± 3 , 52.5 ± 5.8		CON: -0.3 ± 0.3 EXP: -3.7 ± 1.0	22.3 ± 13.7 , 23.4 ± 5.4

References are listed in alphabetical order

M male, *W* women, *BM* body mass, *CON* control group, *EXP* experimental group, – missing data

Table 2 Summary of protocol characteristics of included studies that evaluated $\dot{V}O_{2peak}$

References	Participants: <i>N</i> (M/W), age, peak oxygen consumption (mL/kg/min)	Dehydration protocol: elapsed time before testing: (0:< 3 h; 1:> 12 h)	% BM loss (CON/EXP)	Exercise protocol: exercise mode, duration (min), temperature (°C), ecologically valid (0) or not (1)
Caldwell et al. [11]	32 (32/0), 21.6 (a) 16 (16/0), 21.4, 54.7 (b) 16 (16/0), 21.7, 57.6	(a) 0 (b) 0	(a) 0.0/–3.4 (b) 0.0/–4.7	Cycling (a) 11, –, 1 (b) 12.9, –, 1
Dengel et al. [44]	9 (9/0), 26.4, 54.1	1	(a) –0.6/–3.3 (b) –0.6/–5.6	Cycling (a) 29.2, 22, 1 (b) 28.8, 22, 1
Kenefick et al. [16]	14 (8/6), 20.9, 62.8	1	0.0/–3.9	Running, 30.45, 22, 1
Kozlowski [37]	10 (10/0), 23.5, 50.7	0	–0.5/–4.1	Cycling, 11.9, 20.3, 1
Saltin [53]	10 (10/0), 24.7, 54.7	(a) 0 (b) 0 (c) 0	(a) –0.3/–3.8 (b) –0.3/–3.6 (c) –0.3/–4.0	Cycling (a) 5.7, 19, 1 (b) 4.5, 19, 1 (c) 4.6, 19, 1
Webster et al. [43]	7 (7/0), 19.7, 57.0	1	0.0/–4.9	Running, 33.5, –, 1
Mean ± SD	11 ± 3 (10/1), 23 ± 2, 55.5 ± 3.2		CON: –0.3 ± 0.3 EXP: –4.1 ± 0.7	17.3 ± 11.8, 20.5 ± 1.5

References are listed in alphabetical order

$\dot{V}O_{2peak}$ peak oxygen consumption, *M* male, *W* women, *BM* body mass, *CON* control group, *EXP* experimental group, – missing data

induced passively, 11 of them had subjects dehydrate with a combination of exercise and fluid restriction > 8 h prior to the exercise trials [16, 24, 26, 27, 40, 43, 44, 47] and one had subjects dehydrate with a passive heat exposition [35]. In the remaining 3 studies, sauna was used as a means to induce hypohydration [11, 36, 53]. When pre-exercise hypohydration was achieved using exercise with fluid restriction,

184 ± 35 min were required to achieve the targeted hypohydration level, and 90 to 180 min separated the end of the dehydration protocol from the onset of testing.

Table 3 Summary of protocol characteristics of included studies that evaluated $\dot{V}O_{2LT}$

References	Participants: <i>N</i> (M/W), age, peak oxygen consumption (mL/kg/min)	Dehydration protocol: elapsed time before testing: (0:< 3 h; 1:> 12 h)	% BM loss (CON/EXP)	Exercise protocol: exercise mode, duration (min), temperature (°C), ecologically valid (0) or not (1)
Caldwell et al. [11]	32 (32/0), 21.6 (a) 16 (16/0), 21.4, 54.7 (b) 16 (16/0), 21.7, 57.6	(a) 0 (b) 0	(a) 0.0/–3.4 (b) 0.0/–4.7	Cycling (a) 11, –, 1 (b) 12.9, –, 1
Dengel et al. [44]	9 (9/0), 26.4, 54.1	1	(a) –0.6/–3.3 (b) –0.6/–5.6	Cycling (a) 29.2, 22, 1 (b) 28.8, 22, 1
England et al. [36]	6 (6/0), 28.5, 54.0	0	0.0/–5.0	Cycling, 19.8, –, 1
Kenefick et al. [16]	14 (8/6), 20.9, 62.8	1	0.0/–3.9	Running, 30.45, 22, 1
Papadopoulos et al. [27]	10 (10/0), 25, 47.4	1	(a) –0.1/–2.5 (b) –0.2/–2.6	Running (a) 11.5, 22.4, 1 (b) 8.7, 37.4, 1
Webster et al. [43]	7 (7/0), 19.7, 57.0	1	0.0/–4.9	Running, 33.5, –, 1
Mean ± SD	11 ± 4 (10/1), 24 ± 3, 54.3 ± 4.8		CON: –0.2 ± 0.3 EXP: –4.0 ± 1.1	20.6 ± 9.9, 25.2 ± 6.8

References are listed in alphabetical order

$\dot{V}O_{2LT}$ oxygen consumption at lactate threshold, *M* male, *W* women, *BM* body mass, *CON* control group, *EXP* experimental group, – missing data

3.2.4 Exercise Protocol Characteristics

The mean exercise test time among all included studies was 21.3 ± 13.5 min, with values of mean ambient temperature ($N=21$) and relative humidity ($N=11$) of, respectively, 24.1 ± 6.0 °C (range 19–40 °C) and $49.8 \pm 15.8\%$ (range 25–75%). More specifically, mean exercise test time for AEP was 22.3 ± 13.5 min (range 4.5–54.4 min), for $\dot{V}O_{2\text{LT}}$ was 20.6 ± 9.9 min (range 8.7–33.5 min) and for $\dot{V}O_{2\text{peak}}$ was 17.3 ± 11.8 min (range: 4.5 to 33.5 min). Aerobic exercise performance, $\dot{V}O_{2\text{LT}}$ and $\dot{V}O_{2\text{peak}}$ tests were performed under mean ambient temperature and relative humidity of, respectively, 23.4 ± 5.4 °C ($N=17$)/ $47 \pm 15\%$ ($N=9$) (range 19–40 °C; 25–73%), 25.2 ± 6.8 °C ($N=5$)/ $58.3 \pm 14.4\%$ ($N=3$) (range 22–37.4 °C; 50–75%) and 20.5 ± 1.5 °C ($N=7$)/ $44.5 \pm 7.8\%$ ($N=2$) (range 19–22 °C; 39–50%). Overall, $\dot{V}O_{2\text{LT}}$ corresponded to $64.5 \pm 7.1\%$ of $\dot{V}O_{2\text{peak}}$ (range 53.4–72.8% of $\dot{V}O_{2\text{peak}}$). On average, the AEP tests were performed at $72.4 \pm 15.9\%$ of $\dot{V}O_{2\text{peak}}$ ($N=22$) (range 45–100% of $\dot{V}O_{2\text{peak}}$). Only Chevront et al. [35], Kenefick et al. [25] and Merry et al. [47] reported having provided convective cooling, with a mean simulated air flow of 2.4 ± 1.9 m/s. Except for Casa et al. [40], who use an out-of-doors exercise protocol, all studies used laboratory-based tests. Of the 15 included studies, 9 (60%) reported that participants underwent a familiarisation trial [24–26, 35, 38, 40, 44, 47, 53], whereas 10 (67%) indicated standardization of food intake prior to the trials [11, 16, 24, 25, 27, 35, 40, 43, 44, 47].

3.2.5 Pre-exercise Hypohydration Levels Characteristics

Mean body mass loss of the hypohydrated experimental group was of the order of $-3.6 \pm 1.0\%$ (range -5.6 to -1.7%), whereas for the well-hydrated control group euhydration could not be maintained such that participants began the exercise trial with a body mass loss of $-0.3 \pm 0.3\%$ (range -0.8 to 0.0%). The mean difference in body mass loss between the hypohydrated and well-hydrated condition prior to starting the exercise tests was $-3.4 \pm 1.1\%$ (range -5.0 to -1.4%).

3.3 Performance

3.3.1 Weighted Mean Effect Summaries

3.3.1.1 Aerobic Exercise Performance, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ The impact of pre-exercise hypohydration on AEP is reported in Fig. 2. Results show that pre-exercise hypohydration decreases AEP by $2.4 \pm 0.8\%$ (95% CI 0.8–4.0%). Using only one single mean treatment effect summary per research manuscript did not change the outcome of the effect of pre-exercise hypohydration on AEP

($-2.9 \pm 0.9\%$, 95% CI -4.7 to -1.0%). Figure 3 reports the effect of pre-exercise hypohydration on $\dot{V}O_{2\text{peak}}$. Compared with the hypohydrated condition, being well-hydrated increased $\dot{V}O_{2\text{peak}}$ by a magnitude of $2.4 \pm 0.8\%$ (95% CI 0.7–4.0%) and using a single outcome per research manuscript did not substantially impact findings ($2.5 \pm 1.0\%$, 95% CI 0.5–4.5%). How hypohydration impacted $\dot{V}O_{2\text{LT}}$ is illustrated in Fig. 4. It can be observed that euhydration improved $\dot{V}O_{2\text{LT}}$ by $4.4 \pm 1.4\%$ (95% CI 1.7–7.1%). Using only one single mean treatment effect summary per research manuscript did not significantly change the outcome of the effect of pre-exercise hypohydration on $\dot{V}O_{2\text{LT}}$ ($5.1 \pm 1.5\%$, 95% CI 2.1–8.0%). From a practical standpoint, it can be concluded that compared with starting an exercise hypohydrated, it is respectively likely, possible and likely that AEP, $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ will benefit from a euhydrated state prior to exercise. A sensitivity analysis demonstrated that the deletion of each study one at a time from the different models did not substantially impact the practical effect of pre-exercise hypohydration on AEP, $\dot{V}O_{2\text{peak}}$ or $\dot{V}O_{2\text{LT}}$.

3.3.2 Heterogeneity

Statistically significant heterogeneity was observed for AEP ($Q=77.8$, $p<0.01$) with substantial degree of inconsistency among the included studies ($I^2=74\%$). Peak oxygen consumption and $\dot{V}O_{2\text{LT}}$ data were associated with no significant heterogeneity ($Q=11.9$, $p=0.22$ and $Q=10.1$, $p=0.26$, respectively) with I^2 values of 24 and 21%, respectively, which suggests low inconsistency among the included studies.

3.3.3 Publication Bias

Visual inspection of the funnel plots suggests a potential publication bias for $\dot{V}O_{2\text{peak}}$, $\dot{V}O_{2\text{LT}}$ and AEP, where studies with “no significant effect” of pre-exercise hypohydration on these parameters are missing to the right side of the mean summary effect. However, regarding AEP, limiting the analysis to the first 8 largest studies which are assigned a weight of 91% produces a summary effect estimate of $-2.1 \pm 0.4\%$ (95% CI -2.9 to -1.2%). The bias is, therefore, unlikely to be of real significance from a practical standpoint. With regard to $\dot{V}O_{2\text{peak}}$, a summary effect estimate of $-1.9 \pm 1.0\%$ (95% CI -3.8 to -0.1%) is obtained when limiting the analysis to the first 5 largest studies which are assigned a weight of 85%. Finally, keeping the computation of the summary effect estimate to the first 6 largest studies which encompasses 81% of the total weight reveals that $\dot{V}O_{2\text{LT}}$ would decrease by $4.4 \pm 1.8\%$ (95% CI 0.9–7.8%) if the exercise was to be started hypohydrated. The publication bias is, therefore, unlikely to be of real significance for $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ from a practical standpoint.

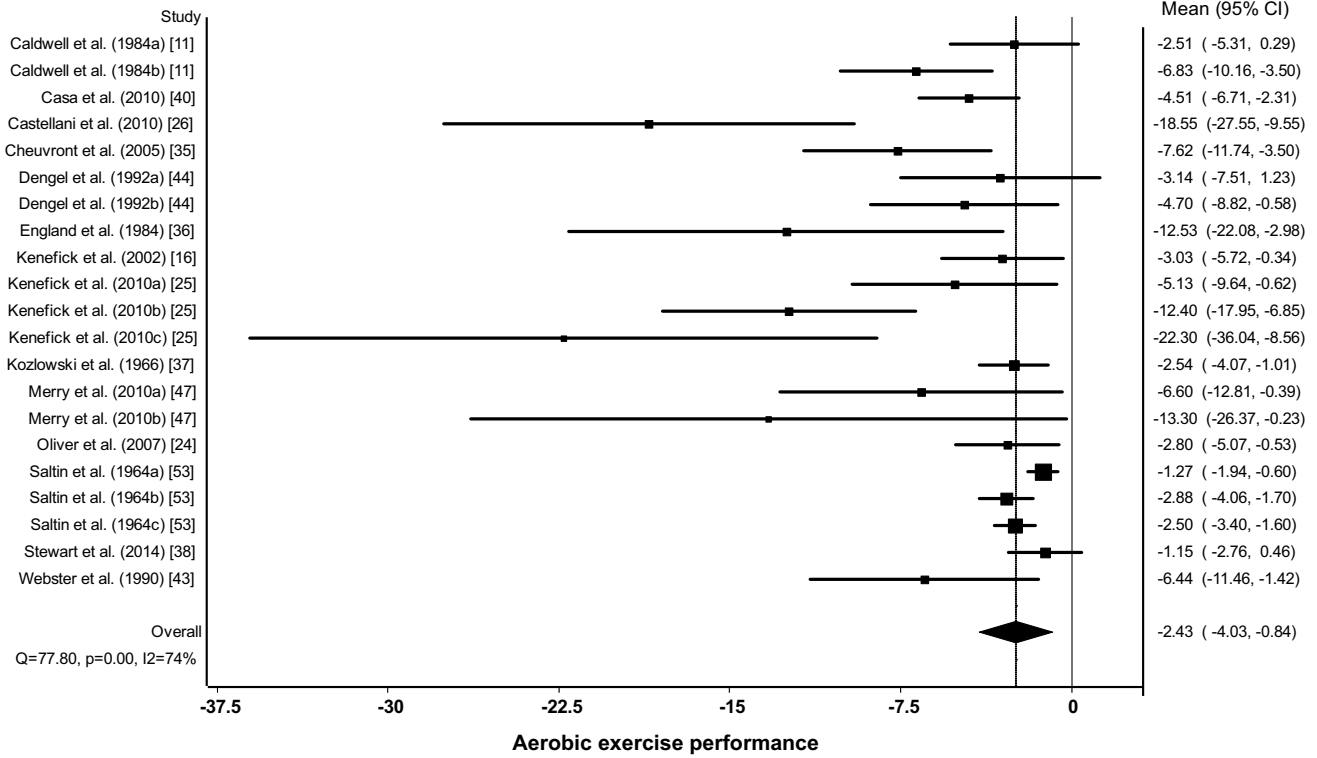


Fig. 2 Forest plot reporting the impact of pre-exercise hypohydration on aerobic exercise performance. *CI* confidence interval. Filled diamond symbol represents the weighted mean percentage change in

aerobic exercise performance between conditions. Size of squares is proportional to the weight of each study

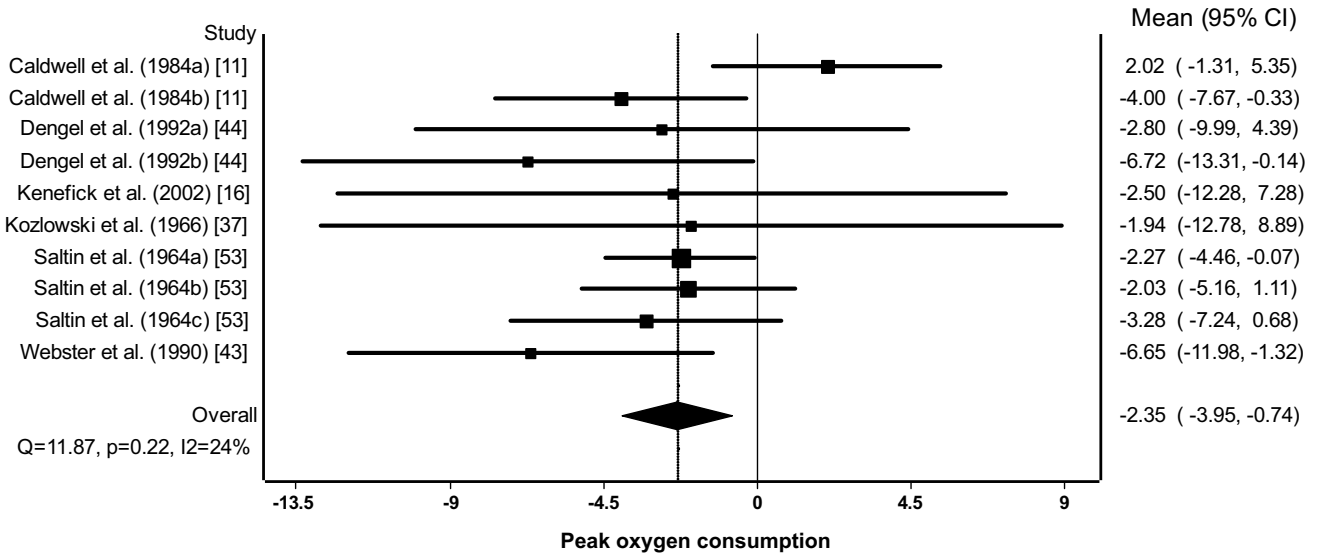


Fig. 3 Forest plot reporting the impact of pre-exercise hypohydration on peak oxygen consumption. *CI* confidence interval. Filled diamond symbol represents the weighted mean percentage change in peak oxy-

gen consumption between conditions. Size of squares is proportional to the weight of each study

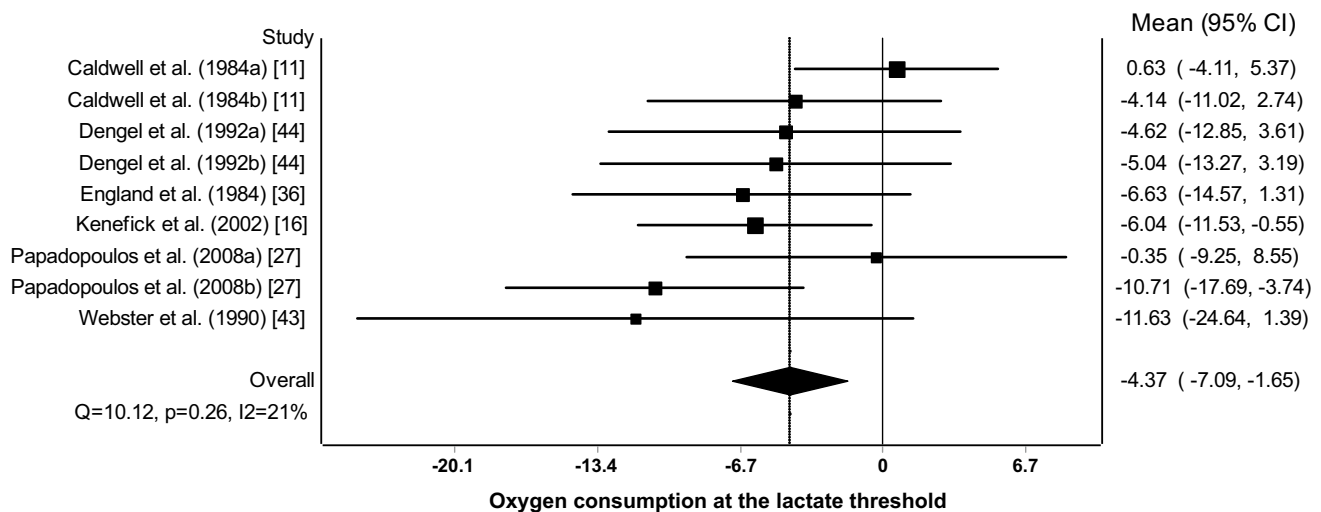


Fig. 4 Forest plot reporting the impact of pre-exercise hypohydration on oxygen consumption at lactate threshold. *CI* confidence interval. Filled diamond symbol represents the weighted mean percentage

change in oxygen consumption at lactate threshold between conditions. Size of squares is proportional to the weight of each study

3.3.4 Meta-regression Analyses

3.3.4.1 Aerobic Exercise Performance As demonstrated in Fig. 5, there was no significant association observed between the differences in AEP and the differences in body mass loss between conditions. However, as demonstrated in Fig. 6, which exposes the potential impact of confounders on AEP, there was a significant relationship between the latter variable (body mass loss) and temperature and the elapsed time between the end of the dehydrating protocol and the onset of the AEP test. There was no significant relationship between humidity levels and the changes in AEP between conditions ($p=0.56$). Controlling for the effect of temperature ($p=0.63$) or the elapsed time between the end of the dehydrating protocol and the onset of the AEP ($p=0.46$) test did not change the relationship between the differences in AEP and the differences in body mass loss between conditions.

3.3.4.2 $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$ Figure 7 shows the relationship between the differences in body mass loss between conditions and the differences in $\dot{V}O_{2peak}$ (a) and $\dot{V}O_{2LT}$ (b). It can be observed that the differences in body mass loss between conditions correlated with the differences in $\dot{V}O_{2peak}$, but not $\dot{V}O_{2LT}$. Result of the meta-regression analysis suggests that pre-exercise hypohydration decreases $\dot{V}O_{2peak}$ by 2.6% for each percent loss in body mass above a body mass loss threshold of 2.8%.

4 Discussion

The main objective of this analysis was to use the meta-analytic approach to determine the magnitude of the effect of pre-exercise hypohydration on AEP, $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$. From a statistical point of view, our results show that pre-exercise hypohydration impairs AEP, $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$ by, respectively, 2.4, 2.4 and 4.4%. From a practical point of view, when considering their respective mean day-to-day variability, pre-exercise hypohydration was found to likely impede AEP and $\dot{V}O_{2LT}$ and to possibly impair $\dot{V}O_{2peak}$. The

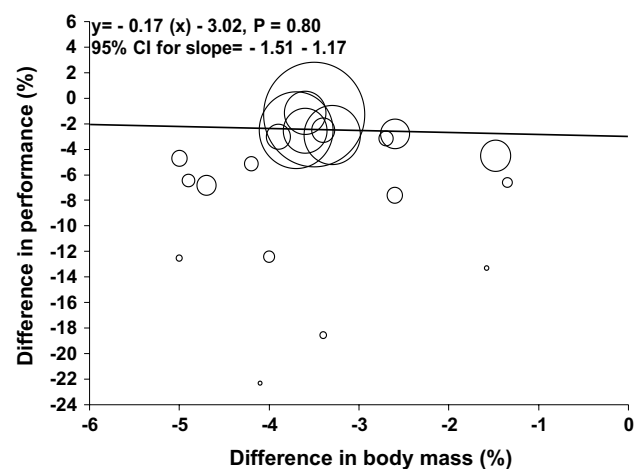


Fig. 5 Correlation between the percent differences in aerobic exercise performance and percent differences in body mass loss between conditions. Diameters of circles are proportional to the weight of the studies. *CI* confidence interval

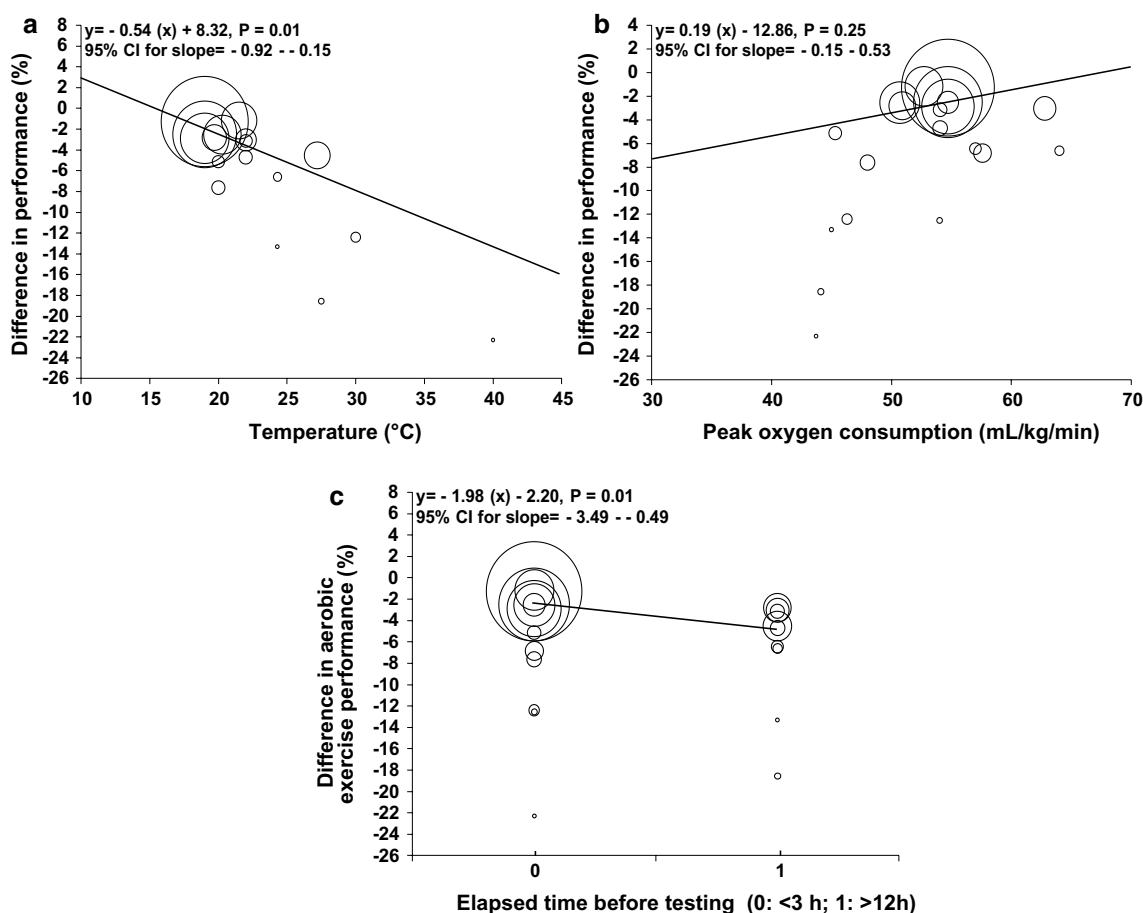


Fig. 6 Correlations of the percent differences in change in aerobic exercise performance between conditions with temperature (**a**), peak oxygen consumption (**b**) and elapsed time between the end of

the dehydrating protocol and the onset of the aerobic exercise performance test (**c**). Diameters of circles are proportional to the weight of the studies. *CI* confidence interval

present results, therefore, indicate that it is of paramount importance to begin aerobic exercise in a well-hydrated state and may have implications for the valid assessment of $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\text{LT}}$ and for the optimization of aerobic performance during exercises of ≤ 1 h in duration.

Substantial heterogeneity in the percent changes in AEP was observed among the included studies. This could potentially be explained by several factors, the first of which needing to be considered was the pre-exercise hypohydration level. However, we observed no statistically significant association between the percent changes in AEP and the percent losses in body mass. Although it cannot be ruled out, it is unlikely that the inability to detect a relationship between these variables was related to a lack of statistical power, as ample of comparisons was included in the model. In the face of the wide spectrum of hypohydration levels incurred by the different studies included in the analysis, the absence of relation could be explained by the high variability in individual responses for a given level of pre-exercise

hypohydration. For instance, variations in AEP ranging from -1.2 to -22.3% have been observed for a pre-exercise hypohydration level of $4 \pm 0.2\%$ of body mass. It is well documented that the impact of hypohydration is amplified in a warm environment [32]. Indeed, the physiological stress induced by the heat-related higher skin blood flow and hypohydration-related reduction in plasma volume could interact to impair AEP [3, 25]. Because studies have investigated the impact of pre-exercise hypohydration on AEP under different ambient temperatures, this factor was therefore considered as a potential confounding variable. A significant relationship was indeed found between the percent changes in AEP and the changes in ambient temperature among studies. However, no relationship was found between the changes in AEP and body mass loss, even after controlling for the effect of ambient temperature. We also observed that the longer the elapse time between the end of the dehydration protocol and the onset of the AEP test, the greater was the decrease in performance. The higher discomfort induced

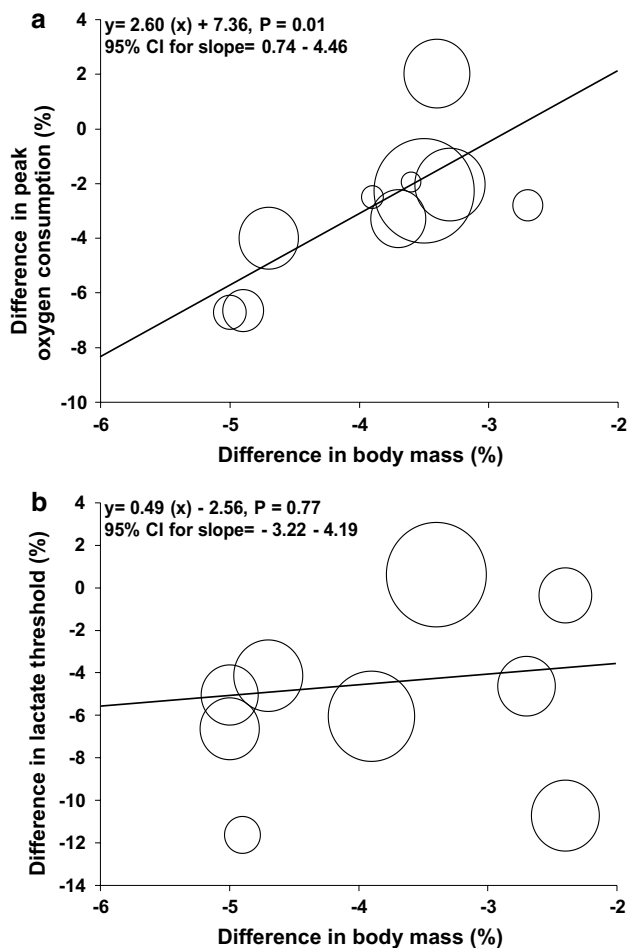


Fig. 7 Correlation between the percent differences in peak oxygen consumption (**a**) and percent differences oxygen consumption at lactate threshold (**b**) with the percent differences in body mass loss between conditions. Diameters of circles are proportional to the weight of the studies. *CI* confidence interval

by a longer time spent in a hypohydrated state, as well as a more pronounced nocebo effect (negative placebo) [64], could potentially explain this relationship. But again, even after adjusting for this parameter, the level of pre-exercise hypohydration was not found to relate to AEP.

Pre-exercise hypohydration of, on average, 3.6% of body mass reduced AEP by a magnitude of 2.4%. In comparison, Goulet [65] showed in a meta-analysis that exercise-induced dehydration of 2.2% incurred through sweating during cycling time-trial exercise of 1–2 h is associated with a non-significant increase in endurance performance of 0.06%, compared with the maintenance of euhydration. On the other hand, in comparison to maintaining a well-hydrated state, the same author [49] observed a performance impairment of 1.9% when athletes are deprived from fluid ingestion and dehydrate by 2.1% body mass during non-ecologically valid running or cycling exercise protocols of > 1 h. Interestingly,

Holland et al. [20] showed through a meta-analytic study that fluid consumption impairs high-intensity cycling exercise of 1 h by 2.5%. This latter observation reiterates the importance of starting an exercise well-hydrated (i.e., while not being thirsty, with a clear to yellow pale urine color or with a stable day-to-day body mass), as the consumption of excess fluid during short duration exercise to compensate for the pre-exercise losses would unlikely lead to a performance recovery. Controlled research is needed to determine whether the impact of pre-exercise hypohydration is similar to that of exercise induced dehydration for an identical level of body mass loss.

In addition to demonstrating that pre-exercise hypohydration reduces $\dot{V}O_{2\text{peak}}$, our results also suggest that there is a relationship between the magnitude of pre-exercise hypohydration and the extent of the decrease in $\dot{V}O_{2\text{peak}}$. More exactly, results show that for each decrease in 1% in body mass, $\dot{V}O_{2\text{peak}}$ declines by 2.6% above a threshold body mass loss of 2.8%. This observation supports the claim made by Trangmar et al. [66] in a recent narrative review where they suggested that a hypohydration level of 3% body mass impairs $\dot{V}O_{2\text{peak}}$. The mechanisms underlying the hypohydration-induced impairment in $\dot{V}O_{2\text{peak}}$ have already been discussed elsewhere [33, 66]. Briefly, it is proposed that the reduction in plasma volume caused by hypohydration acts to decrease venous return to the heart, stroke volume and thus cardiac output [67], leading to a compromised muscle oxygen delivery to the active musculature [66]. When the arteriovenous oxygen difference has been maximally widened, any further reductions in cardiac output will result in impaired $\dot{V}O_{2\text{peak}}$ as predicted by the Fick equation. In this regard, one could reasonably expect that cardiorespiratory fitness, as mirrored by one's $\dot{V}O_{2\text{peak}}$, could moderate the impact of pre-exercise hypohydration on AEP. In fact, high $\dot{V}O_{2\text{peak}}$ values have generally been associated with greater plasma volume. However, our results suggest that cardiorespiratory fitness does not influence the impact of pre-exercise hypohydration on AEP. This observation is in accordance with that of Sawka et al. [68] who showed no relationship between cardiovascular fitness and plasma volume. Given that both endurance training [69] and heat acclimatization [70] enhance plasma volume, we speculate that they could potentially be better moderators of AEP than $\dot{V}O_{2\text{peak}}$, although the present study did not assess this possibility.

Pre-exercise hypohydration was also shown to lower $\dot{V}O_2$ at the lactate threshold. However, no statistically significant relationship was observed between the changes in hydration status and the changes in $\dot{V}O_{2\text{LT}}$, implying that $\dot{V}O_{2\text{LT}}$ did not change systematically with the change in body water levels. It was not necessarily surprising to observe a decline in $\dot{V}O_{2\text{LT}}$ with pre-exercise hypohydration, as the latter was also observed to decrease $\dot{V}O_{2\text{peak}}$ and, hence, maximal oxygen flow to the muscles [71–73]. Furthermore, factors

that decrease oxygen delivery to the tissues will also act to decrease $\dot{V}O_{2LT}$ [74]. On that point, we observe a statistically significant relationship between the percent declines in $\dot{V}O_{2LT}$ and $\dot{V}O_{2peak}$ (results not shown). Some mechanisms can be proposed as to why pre-exercise hypohydration impacted $\dot{V}O_{2LT}$. Hemoconcentration [16] or the increase in catecholamines [75] induced by hypohydration could explain the shift observed in $\dot{V}O_{2LT}$. In that regard, increases in glycogenolysis and lactate production have been observed as a result of progressive dehydration induced during prolonged submaximal exercise in the heat [14, 76]. Core temperature increases in a graded manner with hypohydration [67]. Fernández-Elías et al. [12] have demonstrated that hypohydration-driven hyperthermia increases glycogen use during intense exercise, which has been demonstrated to increase lactate concentration for a given exercise intensity [15].

The results of the current meta-analysis need to be interpreted with the following considerations and limitations in mind. The article search was limited to English publications. Although we observed publication biases for $\dot{V}O_{2peak}$ and $\dot{V}O_{2LT}$, they were unlikely to be of real significance from a practical standpoint. The practical relevance of findings was calculated based on a smallest worthwhile enhancement of performance of 0.5 x the typical day-to-day coefficient of variation of the variable of interest, i.e., AEP, $\dot{V}O_{2LT}$ and $\dot{V}O_{2peak}$. Having used a different factor may have yielded different conclusions. However, a coefficient of 0.5 was used considering that the current results will not only be of interest for the elite athletes, but also for the recreational athletes. Taken together, the included studies examined levels of pre-exercise hypohydration ranging from 1.7 to 5.6% of body mass, with most targeting losses $\geq 3\%$ body mass. It is unlikely that a healthy, trained and hydration-preoccupied athlete would start an exercise with a hypohydration level $\geq 3\%$ body mass, especially given that the effects of thirst are generally felt before such a loss of body mass has been reached. Research is needed to examine the impact of more realistic or probabilistic level pre-exercise hypohydration (1–1.5% body mass) on aerobic performance-related components. Data regarding thirst sensation, perceived exertion, heart rate, core temperature and changes in plasma osmolality and plasma volume would have provided insight into the possible mechanisms linking pre-exercise hypohydration to the decline in AEP, $\dot{V}O_{2LT}$ and $\dot{V}O_{2peak}$. However, their impact could not be considered as they were reported by too few studies. Finally, the results of the current meta-analysis apply to cyclists or runners aged between 20 and 31 years old and for exercise duration < 1 h across a broad spectrum of environmental conditions ranging from 19 to 40 °C.

5 Conclusion

The results of the present meta-analysis showed that starting an exercise hypohydrated by an average of 3% statistically reduces $\dot{V}O_{2LT}$ and impairs AEP and $\dot{V}O_{2peak}$. However, from a practical point of view, which speaks to and fundamentally helps those working with field athletes, the current results indicate that pre-exercise hypohydration is likely to impair AEP and likely to reduce $\dot{V}O_{2LT}$ by, respectively, 2.4 and by 4.4% during running and cycling exercises lasting ~ 20 min across different environmental conditions (from 19 to 40 °C). Furthermore, a decrease in $\dot{V}O_{2peak}$ of 2.4% is to possibly be expected if one begins an exercise while hypohydrated.

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Data Availability Statement The data will be made available from the corresponding author upon reasonable request. Data used to conduct this meta-analysis can be found within each individual study included in this meta-analysis.

Compliance with Ethical Standards

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Conflict of interest Thomas A. Deshayes, David Jeker and Eric D. B. Goulet declare that they have no potential conflicts of interest that are directly relevant to the content of this article

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