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SYSTEMATIC REVIEW

# The Effects of Hyperoxia on Sea-Level Exercise Performance, Training, and Recovery: A Meta-Analysis

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**Abstract**

*Background* Acute exercise performance can be limited by arterial hypoxemia, such that hyperoxia may be an ergogenic aid by increasing tissue oxygen availability. Hyperoxia during a single bout of exercise performance has been examined using many test modalities, including time trials (TTs), time to exhaustion (TTE), graded exercise tests (GXTs), and dynamic muscle function tests. Hyperoxia has also been used as a long-term training stimulus or a recovery intervention between bouts of exercise. However, due to the methodological differences in fraction of inspired oxygen ($F_iO_2$), exercise type, training regime, or recovery protocols, a firm consensus on the effectiveness of hyperoxia as an ergogenic aid for exercise training or recovery remains unclear.

*Objectives* The aims of this study were to (1) determine the efficacy of hyperoxia as an ergogenic aid for exercise performance, training stimulus, and recovery before subsequent exercise; and (2) determine if a dose–response exists between $F_iO_2$ and exercise performance improvements.

*Data Source* The PubMed, Web of Science, and SPORTDiscus databases were searched for original published articles up to and including 8 September 2017, using appropriate first- and second-order search terms.

*Study Selection* English-language, peer-reviewed, full-text manuscripts using human participants were reviewed using the process identified in the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement.

*Data Extraction* Data for the following variables were obtained by at least two of the authors: $F_iO_2$, wash-in time for gas, exercise performance modality, heart rate, cardiac output, stroke volume, oxygen saturation, arterial and/or capillary lactate, hemoglobin concentration, hematocrit, arterial pH, arterial oxygen content, arterial partial pressure of oxygen and carbon dioxide, consumption of oxygen and carbon dioxide, minute ventilation, tidal volume, respiratory frequency, ratings of perceived exertion of breathing and exercise, and end-tidal oxygen and carbon dioxide partial pressures.

*Data Grouping* Data were grouped into type of intervention (acute exercise, recovery, and training), and performance data were grouped into type of exercise (TTs, TTE, GXTs, dynamic muscle function), recovery, and training in hyperoxia.

*Data Analysis* Hedges' $g$ effect sizes and 95% confidence intervals were calculated. Separate Pearson's correlations were performed comparing the effect size of performance versus $F_iO_2$, along with the effect size of arterial content of oxygen, arterial partial pressure of oxygen, and oxygen saturation.

*Results* Fifty-one manuscripts were reviewed. The most common $F_iO_2$ for acute exercise was 1.00, with GXTs the most investigated exercise modality. Hyperoxia had a large effect improving TTE ($g = 0.89$), and small-to-moderate effects increasing TTs ($g = 0.56$), GXTs ($g = 0.40$), and dynamic muscle function performance ($g = 0.28$). An $F_iO_2 \geq 0.30$ was sufficient to increase general exercise performance to a small effect or higher; a moderate positive correlation ($r = 0.47$–$0.63$) existed between performance improvement of TTs, TTE, and dynamic muscle function tests and $F_iO_2$, but not GXTs ($r = 0.06$). Exercise training and recovery supplemented with hyperoxia trended towards a large and small ergogenic effect, respectively,

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but the large variability and limited amount of research on these topics prevented a definitive conclusion.
*Conclusion* Acute exercise performance is increased with hyperoxia. An $F_iO_2 \geq 0.30$ appears to be beneficial for performance, with a higher $F_iO_2$ being correlated to greater performance improvement in TTs, TTE, and dynamic muscle function tests. Exercise training and recovery supplemented with hyperoxic gas appears to have a beneficial effect on subsequent exercise performance, but small sample size and wide disparity in experimental protocols preclude definitive conclusions.

**Key Points**

Hyperoxia acutely increases exercise performance in time trials (TTs), time to exhaustion (TTE), graded exercise tests (GXTs), and dynamic muscle function tests. Time-to-exhaustion tests showed the largest performance enhancement, followed equally by TTs and GXTs.

A fraction of inspired oxygen ($F_iO_2$) $\geq 0.30$ is sufficient to increase general exercise performance to a small effect or higher. A moderate positive correlation ($r = 0.47$–$0.63$) exists between performance improvement of TTs, TTE, and dynamic muscle function tests and $F_iO_2$, but not GXTs ($r = 0.06$).

The limited research into chronic training in hyperoxia suggests a benefit to subsequent normoxic performance by increasing the training stress, but more studies are needed to establish the most beneficial training modality and the appropriate mix of higher training stress and recovery.

Recovery with hyperoxic gas appears to be beneficial for subsequent exercise performance; however, due to the large variability in the time between subsequent bouts of exercise, more research needs to be conducted to determine the optimal length of time of hyperoxic gas delivery.

## 1 Introduction

### 1.1 General Introduction

Many endurance-type events rely on a high aerobic capacity, which is dependent on cardiac output and oxygen extraction. Aerobic exercise performance appears to be largely mediated through changes in arterial content of oxygen ($C_aO_2$) and subsequent oxygen delivery. Exercise capacity is also affected by, but not limited to, energy metabolism, neuromuscular function, motivation, and environmental conditions, among several other factors (the reader is referred to excellent reviews on this topic [1–3]). At rest, arterial oxygen saturation is $\sim$ 100%, but this can fall to $< 90\%$ during intense exercise [4–6]. Thus, increasing the availability of oxygen during intense endurance exercise may attenuate the reduction in arterial oxygen saturation and permit greater exercise capacity [7, 8]. Supporting this idea, either raising or lowering $C_aO_2$ has been shown to induce parallel changes to aerobic exercise performance, accompanied by the maintenance of $C_aO_2$ during intense exercise [7]. Richardson et al. [9] performed dynamic knee extensions until exhaustion under various fractions of inspired oxygen ($F_iO_2$) and found that hyperoxia facilitated greater work rate, maximal leg oxygen consumption ($\dot{V}O_{2max}$), and $C_aO_2$, with no changes to muscle blood flow or venous oxygen concentration, indicating that the increased work was due to increased oxygen delivery. Increasing the $F_iO_2$ above 0.21 is thus used by a wide variety of athletes, based on the premise of hyperoxia permitting greater exercise training stimulus and eventually greater adaptations.

While intuitive, actual demonstration of hyperoxia improving exercise performance through increasing oxygen delivery remains equivocal as hyperoxia may also paradoxically decrease muscle blood flow during rest and exercise in the forearm [10, 11] and leg [9, 12]. The mechanisms for why this occurs are still unclear but may be through downregulation to keep overall oxygen delivery stable despite an increased $C_aO_2$. This hypothesis is supported by observations that an increase in $C_aO_2$ with hyperoxia is coupled with reduced blood flow of a similar amount [10, 13]. It is important to note that the direct assessment of forearm muscle blood flow has been performed during isolated single-joint exercise involving an isometric hand grip to 20% of maximal force [10, 11], and a dynamic incremental knee extension exercise until exhaustion [9, 12]. However, it is notable that a reduction in blood flow does not necessarily mean reduced oxygen delivery to the muscle; rather, some have proposed that decreased flow could increase the time available for oxygen extraction while the cell is crossing the capillary bed, allowing for greater arteriovenous differences [14, 15]. The idea of $C_aO_2$ regulating local blood flow stems from hypoxia studies, where the notion of compensatory vasodilation suggests that, with decreased $C_aO_2$ from hypoxia, there is a matching homeostatic increase in blood flow to maintain oxygen delivery [13].

Sustained physical activity is reliant on adequate central nervous system activation, such that maintained cerebral

blood flow and oxygenation in the face of high metabolic demand may be another mechanism of hyperoxic benefit [7]. Cerebral oxygenation has been proposed to be a limiting factor for exercise performance [16] as increasing $F_iO_2$ to 0.30 maintained cerebral oxygenation and improved rowing time trial (TT) performance [17]. A reduction in cerebral oxygenation is coincident with voluntary exhaustion, and exercise is prolonged during hyperoxia when cerebral oxygenation is maintained, whereas hyperoxia does not affect muscle oxygenation [16, 17]. Indeed, one challenge in delineating the relative role of regional oxygenation is that breathing in hyperoxia will lead to increased systemic oxygen levels throughout all tissues (observed through arterial oxygen saturation and content, as well as through the tissue oxygenation index from near-infrared spectrometry); however, upon examining tissue oxygenation profiles during exercise, it has been suggested that hyperoxia has a larger effect attenuating the decrease in cerebral oxygenation, but has little to no effect on muscle oxygenation. As observed in muscle tissue, the brain may be able to increase oxygen extraction, as seen from increased deoxyhemoglobin [16] with no change to global cerebral blood flow [18], in order to maintain oxygen delivery [3, 19, 20]. Furthermore, Smith et al. [18, 21] demonstrated that hyperoxia and exercise elicit distinct regional differences in cerebral blood flow response during exercise. Hyperoxia augments the increase in posterior cerebral artery velocity with exercise, but does not affect middle cerebral artery velocity [21]. However, these changes to regional cerebral blood flow velocity did not affect incremental exercise performance.

### 1.2 Hyperoxia for Training and Recovery

Practical applications for hyperoxia include increasing training intensity for exercise performance, recovery from injury or previous exercise, increasing work efficiency, or potentially providing an ergogenic aid for a world-record attempt. It is generally well supported that acute hyperoxia administered during aerobic exercise increases performance (higher power output maintained during TTs, or prolonged exercise tolerance at a given workload), yet it is usually not practical in a field setting because it requires additional equipment in the form of gas tanks, tubing, and masks [8]. Alternately, breathing hyperoxic gas following exercise enhances mitochondrial consumption of metabolic byproducts [22], and it is common to see National Football League players breathing hyperoxic gas mixtures on the sideline after each play, especially at altitude (e.g. 1609 m above sea level in Denver, CO, USA). However, there have been conflicting findings regarding the efficacy of using hyperoxia to restore metabolic homeostasis [23–26] or acting as an ergogenic aid for exercise recovery [27–29]. A reason for the inconsistent findings may be due to individual factors affecting the responses to exercise, recovery, and hyperoxia. Nummela et al. [6] found that individuals who demonstrate large decreases in arterial saturation during exercise (exercise-induced hypoxemia) in normoxia had faster heart-rate recoveries and larger changes in blood pH during recovery with hyperoxic gas. Acutely, Grataloup et al. [30] demonstrated that participants who demonstrated exercise-induced hypoxemia attained larger performance benefits from hyperoxia than those who did not. Maeda and Yasukouchi [26, 31] found that people with higher anaerobic thresholds ($\sim$ 60% $\dot{V}O_{2max}$) respond more effectively to higher levels of $F_iO_2$ ($> 0.60$), likely from improved removal of harmful metabolites (hydrogen ions, inorganic phosphate) following exercise. When there is ineffective metabolic clearance, there is an accumulation of metabolites that have been shown/postulated to stimulate group III/IV muscle afferents, which downregulates exercise performance through decreased central motor drive [32].

Chronic training in hyperoxia may improve sea-level performance [33–36] by facilitating a greater power output during TTs and GXTs, or prolonging exercise tolerance at a given workload, thus imposing a larger training stress. However, due to the varied training methodologies used (steady state, interval, mixing hyperoxic and normoxic training), along with differences in $F_iO_2$ and duration of exposure and training, it is difficult to formulate a consensus of the efficacy of training in hyperoxia. An added consideration involving training in hyperoxia and eliciting greater physiological stress is that more time may be needed for adequate recovery and adaptation to prevent overtraining from occurring.

### 1.3 Aim of the Meta-Analysis

A recent narrative review by Sperlich et al. on the effects of hyperoxia during acute exercise highlighted the need for a meta-analysis by stating "the evidence concerning the ergogenic effects of hyperoxic training is inconclusive, perhaps due to the lack of standardization in the methodological approaches employed" [8, p. 434]. For example, exercise test protocols have included multiple sports (cycling, running, rowing) at a set workload to exhaustion; TTs to a set workload, duration, or distance; graded tests to voluntary exhaustion; and testing of isolated limb movements. Previous reviews have tended to group all exercise tests together, even though the primary determinants of fatigue or performance may differ depending on test modality [37, 38]. Even within one test modality (e.g. TTs), duration, and therefore intensity and physiological demands, can range widely, from very short efforts of $\sim$ 4 to 30 min or greater. Given such wide disparity, hyperoxia

may have different levels of effect due to the different metabolic, cardiovascular, and respiratory demands. This wide variability in methodology is ideally suited for an objective and quantified meta-analytic approach, especially when there is a sufficiently large database of studies available for analysis [39, 40]. We also aimed to take a broader approach to the utility of hyperoxia as an ergogenic aid, expanding analysis from use solely during acute exercise to use during recovery and also during chronic training in preparation for competitions in a normoxic environment. Therefore, the aim of this meta-analysis is to complete the most thorough review to date regarding the effects of hyperoxia on maximal exercise performance and recovery during repeated bouts of exercise, and to determine if chronic exercise training in hyperoxia provides an ergogenic benefit for exercise in a normoxic environment. For the purpose of our review, we define maximal exercise performance as a test where individuals perform an exercise to their maximal voluntary capacity, whether in a time trial (completing a set distance/work as rapidly as possible), maintain a constant or incremental workload to the point of voluntary exhaustion, or exert maximal force during an isolated joint movement. In addition, we aim to analyze hyperoxic effects on the likely pathway(s) that facilitate increases in performance. The secondary aim is to determine if there is an $F_iO_2$ threshold where increasing the oxygen content further does not elicit a greater performance improvement, therefore improving the efficacy and safety of hyperoxic training. Readers interested in the effect of hypoxia and hyperoxia on exercise performance in a clinical population are referred to an excellent review by Ulrich et al. [41].

## 2 Methods

### 2.1 Literature Search Parameters Used

An initial search of the PubMed, Web of Science, and SPORTDiscus databases was conducted on 4 May 2016. Across these databases, searches were broken into four categories: species AND oxygen condition AND exercise AND outcome variable(s). After removing duplicates, titles were screened by all authors. Additional manuscripts were added through reference list screening. The final literature search was conducted on 8 September 2017.

### 2.2 Inclusion/Exclusion Criteria

Initial filtering limited articles to full-text articles involving human participants published in English and in peer-reviewed journals; abstracts, conference proceedings, and theses were excluded. Manuscripts were limited to English due to this being the native language of the authors; it has been reported that language-inclusive meta-analyses do not differ when estimating the effectiveness of an intervention compared with language-exclusive versions [42]. Studies were excluded if they were not reported in English, were conducted at an altitude greater than 1600 m above sea level, used hyperbaria (simulated atmospheric pressure) to increase partial pressure of oxygen ($P_aO_2$), included non-healthy populations (e.g. those with chronic obstructive pulmonary disease), did not assess exercise performance, or did not include a normoxic control group.

We used the four-stage (identification, screening, eligibility, and inclusion) process identified in the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement [43] to reduce the number of initial search results. Data for the following variables were extracted from these articles: $F_iO_2$, wash-in time for gas, exercise performance modality, heart rate, cardiac output, stroke volume, oxygen saturation, arterial and/or capillary lactate, hemoglobin concentration, hematocrit, arterial pH, arterial oxygen content ($C_aO_2$), arterial $P_aO_2$, arterial partial pressure of carbon dioxide ($P_aCO_2$), oxygen consumption ($VO_2$), carbon dioxide consumption, minute ventilation ($V_E$), tidal volume, respiratory frequency, ratings of perceived exercise (RPE) of breathing and exercise, end-tidal oxygen and carbon dioxide. Of the extracted variables, only variables with more than five pieces of data were included.

### 2.3 Data Grouping

Data were divided into groups based on the type of exercise performed and how hyperoxia was utilized. The four types of exercise were: TTs, TTE, GXTs, and isolated muscle function of a large locomotory muscle (e.g. dynamic knee extension). Sprint exercise was originally planned as another category but was ultimately excluded from the analysis due to low data availability (two studies). Hyperoxia was administered three different ways: during an acute bout of exercise and categorized as above; during recovery before a subsequent bout of exercise; and during an exercise training intervention with subsequent performance tests in a normoxic environment. Recovery and training studies were each pooled across all exercise types due to the small number of studies. To analyze potential dose–response relationships, data were examined separately by exercise type (TTs, TTE, GXTs, and muscle performance) and were also pooled together to determine if the level of $F_iO_2$ affected the effect size of performance.

### 2.4 Data Analysis

The term ‘manuscript’ will refer to the individual published manuscript, whereas ‘studies’ will refer to the tests

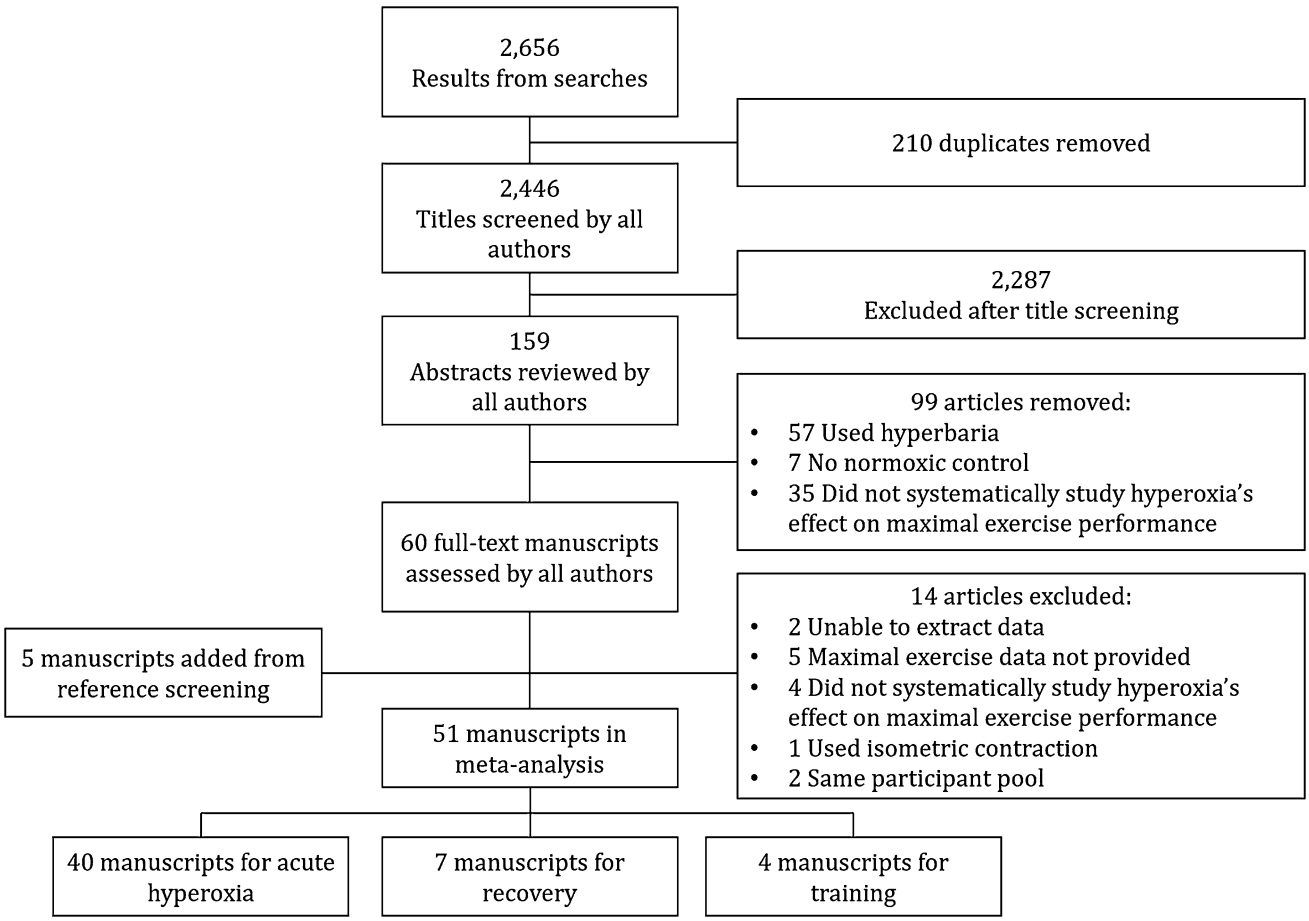


**Fig. 1** Overview of the selection process used for this meta-analysis

performed (e.g. one manuscript that performed a TT and a TTE will count for two studies). Ten manuscripts were randomly chosen and data [means, standard deviations (SD), and sample sizes] were extracted by all three reviewers to ensure consistency. Standard error of the mean was converted to SD. The remaining manuscripts were then analyzed by at least two reviewers. In cases of discrepancy, the third reviewer also analyzed the data and a group consensus was obtained.

Using Review Manager version 5.3 (The Nordic Cochrane Centre, The Cochrane Collaboration, Copenhagen, 2014), effect sizes (Hedges' $g$) and 95% confidence intervals (CIs) were calculated for each study and the weighted mean estimate of effect sizes and associated 95% CIs were calculated to account for the differences in sample sizes. Hedges' $g$ effect sizes were interpreted using the following classification: $g < 0.12$, negligible effect; $g = 0.2$–$0.49$, small effect; $g = 0.5$–$0.79$, moderate effect; and $g > 0.8$, large effect [44]. Correlations were performed in GraphPad Prism version 6.01 (GraphPad Software Inc., La Jolla, CA, USA). The risk of bias was assessed using a low- to high-risk scale, with low risk qualifying as a randomized sequence generated, a double-blind design, and all data presented. Data are reported in text as mean ± SD of the percentage change between normoxia and hyperoxia, effect size 95% CI lower bound, upper bound, significance, and study sample size for each type of exercise ($TT_n$, $TTE_n$, $GXT_n$, $muscle_n$).

## 3 Results

### 3.1 Search Results Overview

Figure 1 shows the search process overview to obtain the 51 manuscripts included in the meta-analysis. Fifty-one studies from 40 manuscripts focused on the effect of hyperoxia and acute exercise performance. These studies were further grouped by exercise type: 9 TTs, 13 TTEs, 22 GXTs, and 7 muscle contraction-type exercises; the reason for the discrepancy between numbers is due to a manuscript performing multiple types of exercise. TTs ranged from as much work as possible performed in 4 min, to a 20 km TT (Table 1), which ranged in intensity

**Table 1** Summary of investigations that evaluated acute time-trial exercise

| Reference | Age, years | $\dot{V}O_{2max}$ (ml kg$^{-1}$ min$^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Wash-in time (min) | Performance[a,c] | $\dot{V}O_2$[a] | $V_E$[a] | HR[a] | Capillary/blood lactate[a] | $C_aO_2$/$P_aO_2$[a] | $S_pO_2$/$S_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Amann et al. [7] | 23 | 63 | 8 (0) | Cycling, 5 km | 1 | – | 13.6 | – | 20.7 | 0.8 | 15.1/– | 16.7/– | 9.6/– |
| Marwood and Bowtell [54] | 36 | 58 | 8 (NS) | Cycling, 4 min | 0.5 | 10 | 4.5 | 4.9 | – | – | –/−19.8 | –/– | –/– |
| Nielsen et al. [17] | 24 | – | 11 (0) | Rowing, 6 min | 0.3 | 5 | 6.2 | – | 1.1 | 1.6 | –/– | 7.7/111.5 | –/6.2 |
| Peltonen et al. [62] | 23 | – | 6 (0) | Rowing, 2.5 km | 0.622 | – | 6.5 | 11.0 | −4.0 | 6.2 | −3.6/– | –/– | –/– |
| Peltonen et al. [63] | 29 | – | 11 (0) | Cycling, 7 min | 0.325 | 3 | 3.0 | 10.7 | −5.1 | 0.5 | −14.8/– | –/– | 4.0/– |
| Petersen et al. [64] [b] | 28 | 52 | 17 (2) | Firefighter simulation | 0.4 | – | 3.7 | – | – | 0.6 | –/1.1 | –/– | –/– |
| Tucker et al. [51] | 24 | – | 11 (0) | Cycling, 20 km | 0.4 | – | 5.4 | – | – | 1.7 | –/10.0 | –/– | –/– |
| Volianitis et al. [65] | 32 | – | 6 (0) | Rowing, 2 km | 0.3 | 5 | – | 10.6 | 2.4 | −1.7 | –/−17.4 | 7.2/71.3 | –/6.9 |
| Weltman et al. [66] | 23 | 52 | 26 (0) | Cycling, 6 min | 1 | 0 | 5.9 | 27.2 | | – | –/– | –/– | –/– |

$\dot{V}O_{2max}$ maximal oxygen consumption, $F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, $V_E$ minute ventilation, *HR* heart rate, $C_aO_2$ arterial oxygen content, $P_aO_2$ partial pressure of oxygen, $S_pO_2$ estimated oxygen saturation, $S_aO_2$ arterial oxygen saturation, *NS* not stated

[a]Percentage change in variable of hyperoxia relative to control

[b]Not included in the meta-analysis

[c]Improved performance indicated by higher mean power output

from ~ 70–98% of peak power output. TTE tests ranged from the mean power output over the 20 km TT to 110% $\dot{V}O_{2max}$, corresponding to tests lasting between ~ 4 and 35 min. Seven manuscripts (eight studies) examined the effects of hyperoxia on exercise recovery, both short-term (< 10 min) and long-term (12 h). Furthermore, four manuscripts examined the effect of exercise training using hyperoxia and its effect on normoxic exercise performance. The characteristics of each study can be seen in Tables 1, 2, 3, 4, 5, 6. Hyperoxic gas mixtures were delivered through a face mask/mouth piece attached to either Douglas bags of desired gas concentrations or gas tanks via a mixing chamber. Risk of bias assessment showed that studies involving hyperoxia and exercise performance, recovery, and training are at a low-moderate level of bias due to most interventions using randomized, single-blind study designs blinding participants to the air mixture they are breathing (Fig. 2).

### 3.2 Effect of Hyperoxia on Exercise Performance While Breathing Gas Mixture

The effects of hyperoxia on acute exercise performance are presented in Fig. 3. When exercise (TTs, TTE, GXTs, and muscle function) was analyzed collectively, hyperoxia had a moderate beneficial effect on exercise performance (16.5 ± 24.1%, $g = 0.52$, 95% CI 0.40–0.64, $p < 0.001$, $TT_n = 7$, $TTE_n = 13$, $GXT_n = 19$, $muscle_n = 7$). Analyzed separately, TTE exercise benefited most from hyperoxia, with a large positive effect of 40.6 ± 34.1% ($g = 0.89$, 95% CI 0.64–1.14, $p < 0.001$, $TTE_n = 13$) and ranging from +9.6 to +137.9%. Of these studies, Amann et al. [7] was a notable outlier, with a significantly greater improvement (+137.9%) than all other TTE protocols. Nevertheless, even with Amann et al. [7] removed, hyperoxia still had a large positive effect on TTE performance (33.1 ± 20.2%, $g = 0.87$, 95% CI 0.61–1.12, $p < 0.001$, $TTE_n = 12$).

During GXTs, hyperoxia had a large effect, increasing $\dot{V}O_{2max}$ by 9.5 ± 5.8% ($g = 0.83$, 95% CI 0.41–1.26, $p < 0.001$, $GXT_n = 15$) and small positive effect on peak power output of 6.2 ± 4.2% ($g = 0.40$, 95% CI 0.22–0.58, $p < 0.001$, $GXT_n = 19$). Hyperoxia had a moderate positive effect during TTs, increasing performance by 6.1 ± 3.3% ($g = 0.56$, 95% CI 0.25–0.88, $p = 0.001$, $TT_n = 7$). Finally, hyperoxia administered during isolated muscle function had a small positive effect (8.0 ± 7.3%, $g = 0.28$, 95% CI −0.02 to 0.58, $p = 0.07$, $muscle_n = 7$). When exercise modalities were grouped together, hyperoxia had a negligible to small effect on decreasing ratings of perceived exertion during maximal exercise (−2.3 ± 2.6%, $g = -0.19$, 95% CI −0.40 to 0.02, $p = 0.07$, $TT_n = 2$, $TTE_n = 5$, $GXT_n = 4$, $muscle_n = 1$).

### 3.3 Effect of Hyperoxia on Cardiovascular Response During Acute Exercise

The effect of hyperoxia on cardiovascular responses can be seen in Fig. 4. Hyperoxia during exercise had an overall negligible effect on maximal heart rate (0.2 ± 2.2%, $g = 0.03$, 95% CI −0.10 to 0.16, $p = 0.67$, $TT_n = 6$, $TTE_n = 8$, $GXT_n = 17$, $muscle_n = 5$). Analyzed separately, hyperoxia had a negligible effect on maximal heart rate during TTE (−0.1 ± 1.5%, $g = 0.02$, 95% CI −0.27 to 0.32, $p = 0.89$, $TTE_n = 8$) and GXTs (0.7 ± 1.2%, $g = 0.08$, 95% CI −0.11 to 0.27, $p = 0.40$, $GXT_n = 17$). During TTs, hyperoxia facilitated a slightly higher heart rate with a negligible to small effect (1.4 ± 2.4%, $g = 0.19$, 95% CI −0.19 to 0.58, $p = 0.33$, $TT_n = 6$). In contrast, hyperoxia during isolated muscle contractions lowered heart rate with a small negative effect (−2.7 ± 3.0%, $g = -0.23$, 95% CI −0.56 to 0.09, $p = 0.16$, $muscle_n = 5$).

Hyperoxia had a small overall positive effect, increasing cardiac output during exercise by 1.8 ± 2.6% ($g = 0.33$, 95% CI 0.02–0.63, $p = 0.04$, $TT_n = 2$, $TTE_n = 1$, $GXT_n = 2$, $muscle_n = 4$). Hyperoxia had a large positive effect on other cardiovascular variables, increasing $C_aO_2$ by 9.1 ± 4.7% ($g = 1.19$, 95% CI 0.81–1.58, $p < 0.001$, $TT_n = 3$, $TTE_n = 2$, $muscle_n = 4$), $P_aO_2$ by 148.7 ± 139.9% ($g = 6.02$, 95% CI 3.37–8.67, $p < 0.001$, $TT_n = 2$, $TTE_n = 2$, $GXT_n = 1$, $muscle_n = 4$), and a moderate positive effect on $P_aCO_2$ by 10.0 ± 7.9% ($g = 0.51$, 95% CI 0.13–0.89, $p = 0.008$, $TT_n = 1$, $TTE_n = 2$, $GXT_n = 1$, $muscle_n = 2$). In addition, hyperoxia administered during exercise had a large effect on arteriovenous $O_2$ difference, increasing it by 4.6 ± 9.9% ($g = 0.88$, 95% CI −0.20 to 1.95, $p = 0.11$, $TT_n = 3$, $TTE_n = 1$, $muscle_n = 2$).

To increase power for the meta-analysis and allow us to analyze the data by exercise modality, arterial oxygen saturation from pulse oximetry and blood measurements were combined. Generally, hyperoxia had a large effect, maintaining higher arterial saturation of oxygen during exercise (4.2 ± 2.3%, $g = 2.77$, 95% CI 2.08–3.47, $p < 0.001$, $TT_n = 4$, $TTE_n = 6$, $GXT_n = 9$, $muscle_n = 4$). Individually, TTs (6.7 ± 2.3%, $g = 4.61$, 95% CI 1.57–7.66, $p = 0.003$, $TT_n = 4$), TTE (4.3 ± 1.7%, $g = 1.87$, 95% CI 0.91–2.82, $p < 0.001$, $TTE_n = 6$), GXTs (4.1 ± 2.2%, $g = 2.90$, 95% CI 1.64–4.14, $p < 0.001$, $GXT_n = 9$), and muscle function (1.6 ± 0.1%, $g = 3.24$, 95% CI 1.66–4.81, $p < 0.001$, $muscle_n = 4$) all

**Table 2** Summary of investigations that evaluated acute time-to-exhaustion exercise

| Reference | Age, years | $\dot{V}O_{2max}$ (ml kg$^{-1}$ min$^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Wash-in time (min) | Performance[a,b] | $\dot{V}O_2$[a] | $V_E$[a] | HR[a] | Capillary/ blood lactate[a] | $C_aO_2$/ $P_aO_2$[a] | $S_pO_2$/ $S_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Amann et al. [7] | 23 | 63 | 8 (0) | Cycling, mean PO 5 km TT | 1 | – | 137.9 | – | −15.8 | −1.7 | –/– | 15.0/– | 7.2/– |
| Bye et al. [50] | 31 | – | 7 (0) | Cycling, 80% $\dot{W}_{max}$ | 0.4 | 10 | 66.1 | – | −4.9 | 0.0 | –/– | –/– | –/5.3 |
| Ekblom et al. [67] | 27 | 58.0 | 9 (0) | Treadmill running | 0.5 | 45 | 68 | 12.6 | −11.1 | 1.1 | –/− 16.2 | 7.7/176.6 | –/4.3 |
| Linossier et al. [68] | 22 | 54 | 5 (0) | Cycling, 100% $\dot{W}_{max}$ | 0.6 | 15 | 44.6 | – | | – | –/− 8.5 | –/– | –/– |
| Manselin et al. [69] | 28 | 56 | 18 (4) | Cycling, 80% $\dot{W}_{max}$ | 0.31 | 5 | 9.6 | – | | – | −2.6/– | –/– | 2.8/– |
| Ohya et al. [70] | 20 | 59 | 8 (0) | Cycling, 90% $\dot{W}_{max}$ | 0.36 | 2 | 16.3 | – | | – | −30.7/– | –/– | 3.1/– |
| Plet et al. [71] | 24 | 51 | 11 (6) | Cycling, 80% $\dot{V}O_{2max}$ | 0.55 | – | 40.7 | 0.0 | −6.2 | −2.2 | –/− 36.1 | –/106.6 | –/– |
| Tucker et al. [51] | 24 | – | 11 (0) | Cycling, mean PO 20 km TT | 0.4 | – | 10.7 | – | | 1.7 | –/− 9.3 | –/– | –/– |
| Ulrich et al. [49] | 45 | 43 | 32 (12) | Cycling, 75% $\dot{W}_{max}$ | 0.5 | 2 | 51.6 | 10 | −5.5 | 1.2 | –/– | –/– | 3.1/– |
| Wilkerson et al. [72] | 26 | 47 | 7 (0) | Cycling, 105% $\dot{V}O_{2max}$ | 0.5 | 10 | 14.5 | – | | 0.6 | –/5.6 | –/– | –/– |
| Wilson and Welch [57] | 30 | – | 9 (0) | Treadmill running, 110% $\dot{V}O_{2max}$ | 0.4 | 1 | 17.9 | – | −1.5 | – | –/– | –/– | –/– |
| Wilson and Welch [57] | ~30 | – | 9 (0) | Treadmill running, 110% $\dot{V}O_{2max}$ | 0.6 | 1 | 22.2 | – | −5.3 | – | –/– | –/– | –/– |
| Wilson and Welch [57] | ~30 | – | 9 (0) | Treadmill running, 110% $\dot{V}O_{2max}$ | 0.8 | 1 | 29.8 | – | −6.1 | – | –/– | –/– | –/– |
| Wilson and Welch [57] | ~30 | – | 9 (0) | Treadmill running, 110% $\dot{V}O_{2max}$ | 1.0 | 1 | 38.1 | – | −6.0 | – | –/– | –/– | –/– |

$F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, $V_E$ minute ventilation, *HR* heart rate, $C_aO_2$ arterial oxygen content, $P_aO_2$ partial pressure of oxygen, $S_pO_2$ estimated oxygen saturation, $S_aO_2$ arterial oxygen saturation, *TT* time trial, *PO* power output, $\dot{W}_{max}$ maximal workload, $\dot{V}O_{2max}$ maximal oxygen consumption

[a]Percentage change in variable of hyperoxia relative to control

[b]Improved performance indicated by longer exercise tolerance

**Table 3** Summary of investigations that evaluated acute graded exercise tests

| Reference | Age, years | $\dot{V}O_{2max}$ (ml kg$^{-1}$ min$^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Wash-in time (min) | Performance[a,b] | $\dot{V}O_2$[a] | $V_E$[a] | HR[a] | Capillary/blood lactate[a] | $C_aO_2$/$P_aO_2$[a] | $S_pO_2$/$S_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Byrnes et al. [48] | – | – | 6 (0) | Cycling, +33 W min$^{-1}$ | 0.7 | 30 | – | 13.0 | −5.5 | 0.0 | –/– 5.1 | –/– | –/– |
| Eves et al. [73] | 33 | 53 | 25 (0) | Treadmill walking at 93.9 m min$^{-1}$, +2% incline• 2 min$^{-11}$ | 0.4064 | 0 | 10.3 | 10.1 | −1.1 | 0.7 | 0.0/– | –/– | 6.8/– |
| Eves et al. [74] | 34 | 45 | 15 (0) | Treadmill walking at 3.5 or 4 mph, +2% incline • 2 min$^{-11}$ | 0.4 | 5 | 10.1 | 13.0 | 2.7 | −0.5 | –/– | –/– | 7.2/– |
| Grataloup et al. [30] (EIH) | 19 | 65 | 9 (0) | Cycling, +0.333 W • min$^{-1}$ • kg$^{-1}$ | 0.3 | 3 | 4.3 | 12.8 | −2.1 | −1.6 | −3.3/– | –/– | 6.5/– |
| Grataloup et al. [30] (non-EIH) | 22 | 63 | 7 (0) | Cycling, +0.333 W • min$^{-1}$ • kg$^{-1}$ | 0.3 | 3 | 2.5 | 4.2 | −3.6 | −0.5 | −20.3/– | –/– | 3.2/– |
| Hogan et al. [53] | 27 | 47 | 6 (0) | Cycling, +15 W • min$^{-1}$ | 0.5976 | 10 | 5.9 | 5.2 | −18.3 | −0.5 | –/– 12.8 | –/– | –/– |
| Hopman et al. [75] | 26 | 36 | 10 (0) | Arm cycling, +10 W • min$^{-1}$ | 0.5 | 15 | 2.2 | 5.6 | −3.6 | 0.9 | –/– | –/– | –/– |
| Hughson and Kowalchuk [76] | 30 | 53 | 6 (0) | Cycling, +15 W • min$^{-1}$ | 0.7 | 10–15 | 4.1 | 4.0 | – | – | –/– | –/– | –/– |
| Knight et al. [77] | 29 | 57 | 11 (0) | Cycling, +30 W • min$^{-1}$ | 1 | – | 8.7 | – | – | – | –/– 10.5 | –/– | –/– |
| Linossier et al. [68] | 22 | 54 | 5 (0) | Cycling, +37 W • 4 min$^{-1}$ | 0.6 | 15 | 4.2 | – | – | – | –/– 8.5 | –/– | –/– |
| Lovering et al. [78]. | 30 | 51 | 15 (5) | Cycling, +30 • 2 min$^{-1}$ | 1 | 0 | 9.9 | – | – | – | –/– | –/– | –/– |
| Manselin et al. [69] | 28 | 56 | 18 (4) | Cycling, males +25 • min$^{-1}$; females +20 • min$^{-1}$ | 0.31 | 5 | 2.3 | – | – | 0.5 | −2.5/– | –/– | 3.2/– |
| Oussaidene et al. [16] | 27 | 45 | 8 (0) | Cycling, +20 W•min$^{-1}$ | 0.3 | 5 | 5.6 | – | – | 1.6 | –/– | –/– | 1.3/– |
| Ozgurbuz et al. [79] | 21 | 51 | 46 (19) | Cycling, +50 W • 3 min$^{-1}$ | 0.744 | 15 | 2.5 | 1.2 | −1.2 | 0.5 | 5.1/– | –/– 1.5 | –/– |
| Peltonen et al. [80] | 24 | 63 | 6 (0) | Cycling, +100 W • 4 min$^{-1}$ until 400 W, then +50 W • 4 min$^{-1}$ | 0.32 | 5 | 5.5 | 13.6 | – | 2.8 | –/– | –/– | 2.1/– |
| Plet et al. [71] (females) | 23 | 44 | 6 (6) | Cycling, +30 W • 3 min$^{-1}$ | 0.55 | – | – | 11.4 | −6.2 | 2.7 | –/– 5.4 | –/– | –/– |
| Plet el al. [71] (males) | 25 | 57 | 5 (0) | Cycling, +30 W • 3 min$^{-1}$ | 0.55 | – | – | 3.7 | −9.8 | 1.1 | –/– 12.3 | –/– | –/– |

**Table 3** continued

| Reference | Age, years | $\dot{V}O_{2max}$ (ml kg$^{-1}$ min$^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Wash-in time (min) | Performance[a,b] | $\dot{V}O_2$[a] | $V_E$[a] | HR[a] | Capillary/blood lactate[a] | $C_aO_2$/$P_aO_2$[a] | $S_pO_2$/$S_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Prieur et al. [81] | 25 | 45 | 10 (0) | Cycling, +30 W • 2 min$^{-1}$ | 0.331 | 10 | 4.6 | 14.2 | −5.5 | 0.0 | –/– | –/– | 3.2/– |
| Smith et al. [21] | 22 | – | 10 (0) | Cycling, +30 W • 2 min$^{-1}$ until hyperventilation, then +30 W • min$^{-1}$ | 1 | 10 | 5.7 | – | – | 1.6 | –/– | –/– | –/– |
| Smith et al. [18] | 22 | – | 17 (6) | Recumbent cycling, +20 W • 3 min$^{-1}$ | 0.4 | – | 20 | – | – | – | –/– | –/– | –/– |
| Ulrich et al. [49] | 45 | 43 | 32 (12) | Cycling, +15–20 W • min$^{-1}$ | 0.5 | 2 | 5.1 | 23.3 | 1.9 | 0.6 | –/– | | 3.1/– |
| Walsh and Banister [82] | 29 | 54 | 7 (0) | Cycling, +20 W • min$^{-1}$ | 0.4 | 10 | 6 | 7.8 | −1.2 | 2.1 | –/– | –/– | –/– |

$\dot{V}O_{2max}$ maximal oxygen consumption, $F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, $V_E$ minute ventilation, $HR$ heart rate, $C_aO_2$ arterial oxygen content, $P_aO_2$ partial pressure of oxygen, $S_pO_2$ estimated oxygen saturation, $S_aO_2$ arterial oxygen saturation, $EIH$ exercise-induced hypoxemia

[a]Percentage change in variable of hyperoxia relative to control

[b]Improved performance indicated by greater power output achieved

demonstrated a large effect while breathing hyperoxic gas mixtures. Furthermore, hyperoxia had a negligible effect on hemoglobin concentration during maximal exercise (0.6 ± 1.6%, $g = 0.10$, 95% CI −0.31 to 0.51, $p = 0.64$, $TT_n = 1$, $TTE_n = 1$, $GXT_n = 2$, $muscle_n = 2$).

### 3.4 Effect of Hyperoxia on Respiratory Response during Acute Exercise

The effect of hyperoxia on respiratory responses can be seen in Fig. 5. Hyperoxia had a moderate effect on increasing oxygen consumption ($\dot{V}O_2$) during exercise (9.7 ± 7.3%, $g = 0.70$, 95% CI 0.44–0.96, $p < 0.001$, $TT_n = 5$, $TTE_n = 3$, $GXT_n = 15$, $muscle_n = 4$). Hyperoxia had a large effect on increasing $\dot{V}O_2$ during TTs (12.9 ± 8.4%, $g = 1.02$, 95% CI 0.63–1.42, $p < 0.001$, $TT_n = 5$), a moderate effect during GXTs (see Sect. 3.2), and a small negative effect during isolated muscle function (7.7 ± 12.5%, $g = 0.15$, 95% CI −0.72 to 0.42, $p = 0.60$, $muscle_n = 4$). TTE only had three studies reporting maximal oxygen consumption, therefore we did not analyze this individually. Hyperoxia had a large effect on increasing end-tidal $P_aO_2$ (284.8 ± 164.1%, $g = 10.6$, 95% CI 6.95–14.25, $p < 0.001$, $TT_n = 2$, $TTE_n = 2$, $GXT_n = 2$) and a small effect on increasing end-tidal $P_aCO_2$ by 4.8 ± 5.4% ($g = 0.33$, 95% CI 0.05–0.61, $p = 0.02$, $TT_n = 2$, $TTE_n = 3$, $GXT_n = 2$). Hyperoxia had a small effect on increasing maximal $CO_2$ consumption by 0.17 ± 0.23% ($g = 0.27$, 95% CI 0.02–0.55, $p = 0.06$, $TT_n = 2$, $TTE_n = 1$, $GXT_n = 4$).

Hyperoxia had a small effect on decreasing $V_E$ (−3.7 ± 6.9%, $g = -0.25$, 95% CI −0.45 to −0.05, $p = 0.02$, $TT_n = 5$, $TTE_n = 9$, $GXT_n = 13$) and ratings of perceived breathing exertion (−8.8 ± 4.1%, $g = -0.38$, 95% CI −0.67 to −0.09, $p = 0.01$, $TT_n = 1$, $TTE_n = 2$, $GXT_n = 2$) during exercise. During TTs, hyperoxia increased $V_E$ by 3.02 ± 10.4% ($g = -0.46$, 95% CI −0.51 to 1.43, $p = 0.35$, $TT_n = 5$); however, Amann et al. [7] had a significantly larger effect ($g = 3.33$, 95% CI 1.68–4.98) than the rest of the TT studies. With Amann et al. [7] removed, hyperoxia had a negligible effect on $V_E$ during TTs (−1.4 ± 3.7%, $g = -0.07$, 95% CI −0.55 to 0.41, $p = 0.78$, $TT_n = 4$). During TTE (−6.9 ± 4.1%, $g = -0.49$, 95% CI −0.78 to −0.21, $p = 0.001$, $TTE_n = 9$) and GXTs (−4.1 ± 5.4%, $g = -0.14$, 95% CI −0.35 to 0.07, $p = 0.19$, $GXT_n = 13$), hyperoxia had a small and negligible effect on decreasing $V_E$, respectively. Hyperoxia administered during exercise had a negligible effect on decreasing respiratory frequency by −3.7 ± 6.8% ($g = -0.08$, 95% CI −0.34 to 0.18, $p = 0.54$, $TT_n = 2$, $TTE_n = 3$, $GXT_n = 2$).

**Table 4** Summary of investigations that evaluated acute dynamic contractions of large locomotory muscle

| Reference | Age, years | $\dot{V}O_{2max}$ (ml $kg^{-1}$ $min^{-1}$) | Total no. of participnts (no. of females) | Exercise | $F_iO_2$ | Wash-in time (min) | Performance[a,b] | $\dot{V}O_2$[a] | $V_E$[a] | HR[a] | Capillary/ blood lactate[a] | $C_aO_2$/ $P_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Eiken and Tesch [83] | 27 | – | 8 (0) | KE, 60 MVCs @ 180° • $s^{-1}$ | 0.99 | 15 | 6.1 | – | – | –/– | –/– | –/– |
| Kleiner and Snyder [84] | 21 | – | 50 (30) | KE, MVC until exhaustion (70%) @ 60° • $s^{-1}$ | 1.0 | 5 | 5.8 | – | −2.3 | –/– | –/– | –/– |
| Mourtzakis et al. [85] (trained leg) | 23 | – | 8 (0) | KE, dynamic incremental | 0.6 | 10 | −2.0 | 3.7 | −4.7 | –/– | 3.9/178.4 | –/1.8 |
| Mourtzakis et al. [85] (non-trained leg) | 23 | – | 8 (0) | KE, dynamic incremental | 0.6 | 10 | 1.3 | −8.2 | −5.8 | –/– | 2.4/157.6 | –/1.5 |
| Richardson et al. [12] | 25 | 58 | 5 (0) | KE, dynamic, rate 60 • $min^{-1}$ + 5% increase | 1.0 | 0 | 12.0 | 18.5 | −2.9 | –/– | 9.1/394.6 | –/1.5 |
| Richardson et al. [9] | 26 | 65 | 7 (0) | KE, dynamic, rate 60 • $min^{-1}$ + 5% increase | 1.0 | 0 | 14 | 16.9 | 2.1 | –/– | 11.8/407.5 | –/1.6 |
| Vanhatolo et al. [86] | 30 | – | 7 (0) | KE, dynamic until exhaustion | 0.7 | 5 | 18.6 | – | – | –/– | –/– | –/– |

$\dot{V}O_{2max}$ maximal oxygen consumption, $F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, $V_E$ minute ventilation, *HR* heart rate, $C_aO_2$ arterial oxygen content, $P_aO_2$ partial pressure of oxygen, *KE* knee extensions, *MVC* maximal voluntary exhaustion

[a]Percentage change in variable of hyperoxia relative to control

[b]Improved performance indicated more work completed

**Table 5** Summary of investigations that evaluated hyperoxic training for normoxic exercise

| Reference | Age, years | $\dot{V}O_{2max}$ (ml $kg^{-1}$ $min^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Training frequency | Training intensity | Performance[a,b] | $\dot{V}O_2$[a] | HR[a] | Capillary/blood lactate[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Armstrong et al. [35] | 21 | 43 | 18 (6) | Cycling, incremental + 0.5–1 kp • $min^{-1}$<br>Cycling, TTE 80% $\dot{V}O_{2max}$ | 0.8249 | 40 min, 3 days/week, 5 weeks, | Cycling @ 60% $VO_{2peak}$ | 34.9 | 14.3 | – | –/– |
| Perry et al. [33] | 26 | 51 | 11 (3) | Cycling, incremental + 70 W•2 $min^{-1}$ until HR = 150, then + 35 W • 2 $min^{-1}$<br>Cycling, TTE<br>90% $\dot{V}O_{2max}$ | 0.585 | 60 min, 3 days/week, 6 weeks | 4 min @ 80% $VO_{2max}$ (2 min rest) × 10 | 106.9 | 200 | −22.2 | –/– |
| Perry et al. [34] | 23 | 48 | 9 (3) | Cycling, incremental + 70 W•2 $min^{-1}$ until HR = 150, then + 35 W • 2 $min^{-1}$<br>Cycling, TTE<br>90% $\dot{V}O_{2max}$ | 0.6 | 60 min, 3 days/week, 6 weeks | 4 min @ 90% $VO_{2max}$ (2 min rest) × 10 | −1.2 | −9.5 | – | –/– 68.8 |
| Ploutz-Snyder et al. [36] | 23 | 45 | 19 (0) | Cycling, incremental + 35 W • 2 $min^{-1}$ | 0.7 | 40 min, 5 days/week, 5 weeks | Cycling @ 70% $HR_{max}$ | – | 29.9 | – | –/– |

$F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, *HR* heart rate, *TTE* time to exhaustion, $\dot{V}O_{2max}$ maximal oxygen consumption, $\dot{V}O_{2peak}$ peak oxygen consumption, $HR_{max}$ maximal heart rate

[a]Percentage change in variable of hyperoxia relative to control

[b]Improved performance indicated by greater performance on post-test in normoxia

**Table 6** Summary of investigations that evaluated hyperoxia as a recovery intervention for subsequent performance

| Reference | Age, years | $\dot{V}O_{2max}$ (ml kg$^{-1}$ min$^{-1}$) | Total no. of participants (no. of females) | Exercise | $F_iO_2$ | Recovery intervention | Rest interval | Performance[a,b] | $\dot{V}O_2$[a] | HR[a] | Capillary/blood lactate[a] | $CaO_2$/$PaO_2$[a] | $S_pO_2$/$S_aO_2$[a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Kay et al. [27] | 21 | – | 12 (0) | Cycling, 30 s Wingate | 1.0 | 4 min | 4 min | 4.4 | –/– | –/– | –/– | –/– | –/– |
| Kay et al. [27] | 21 | – | 12 (0) | Cycling, 30 s Wingate | 0.6 | 4 min | 4 min | −3.3 | –/– | –/– | –/– | –/– | –/– |
| Peeling and Andersson [24] | 24 | – | 8 (3) | Swimming, 20 × 200 m | 0.995 | 2 × 10 min | 12 h | −0.5 | – | −2.7 | −7.5/– | –/– | –/– |
| Robbins et al. [29] | 28 | 60 | 13 (0) | Treadmill running, increasing +2% grade • 30 s$^{-1}$ | 1.0 | 4 min | 4 min | 3.9 | – 9.0 | −1.8 | –/– | –/– | –/– |
| Sperlich et al. [61] | 21 | – | 12 (0) | Swimming, 40 maximal 'butterfly strokes' | 1.0 | 6 min | 6 min | 4.2 | – | – | 0/– | –/– | –/3.1 |
| Sperlich et al. [28] | 25 | 65 | 10 (0) | Cycling, 30 s all-out @ 120 rpm | 1.0 | 6 min | 6 min | 3.6 | – | – | –/2.6 | –/20 | –/– |
| Winter et al. [87] | 26 | 65 | 12 (0) | Treadmill running, 8.5 mph, +2% grade • 60 s$^{-1}$ | 1.0 | 4 min | 3–4 h | 5 | – | – | –/3.6 | –/– | –/– |
| Zinner et al. [88] | 25 | 62 | 10 (0) | Double-poling, 3 min | 1.0 | 3 min | 3 min | 2.5 | – | – | –/– 8.5 | –/72.3 | –/5.5 |

$\dot{V}O_{2max}$ maximal oxygen consumption, $F_iO_2$ fraction of inspired oxygen, $\dot{V}O_2$ oxygen consumption, *HR* heart rate, $C_aO_2$ arterial oxygen content, $P_aO_2$ partial pressure of oxygen, $S_pO_2$ estimated oxygen saturation, $S_aO_2$ arterial oxygen saturation, *rpm* revolutions per minute

[a]Percentage change in variable of hyperoxia relative to control

[b]Improved performance indicated by greater performance in post-test

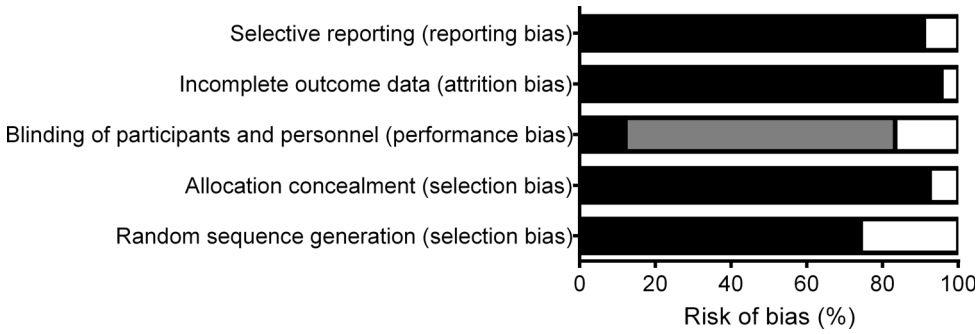


**Fig. 2** Risk of bias assessment. *Black* indicates low risk; *Grey* indicates moderate risk; *White* indicates high risk

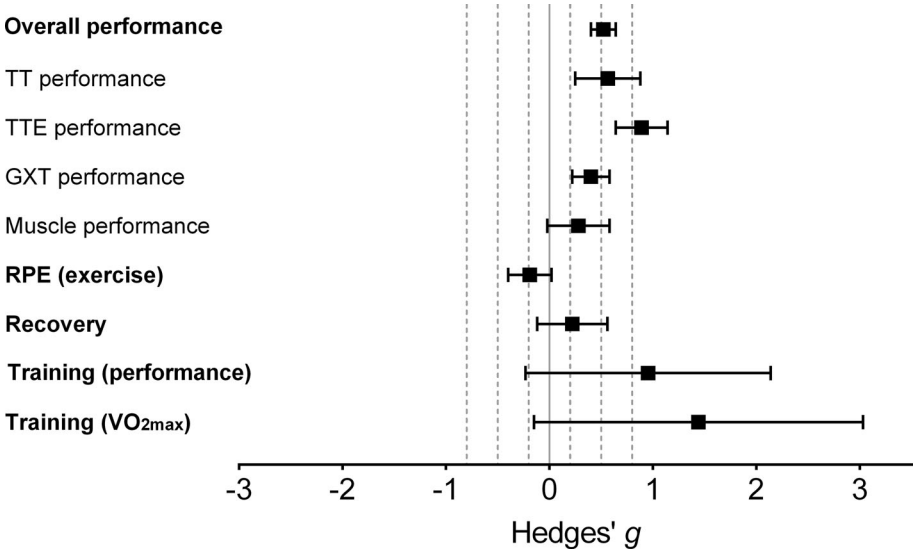


**Fig. 3** Forest plot summarizing the effect (± 95% confidence intervals) of hyperoxia on acute exercise performance, recovery (< 12 h), and training in hyperoxia for normoxic exercise performance and maximal aerobic capacity. *Bolded terms* include all exercise modalities. *Dashed lines* represent small (Hedges' $g = 0.20$–0.49), moderate (Hedges' $g = 0.50$–0.79), and large (Hedges' $g \geq 0.80$) effect sizes. *TT* Time trial, *TTE* time to exhaustion *GXT* graded exercise test, *Muscle* dynamic muscle tests, *RPE* ratings of perceived exertion, $\dot{V}O_{2max}$ maximal oxygen consumption

### 3.5 Effect of Hyperoxia on Metabolic Responses during Exercise

The effect of hyperoxia on the metabolic responses to exercise can be seen in Fig. 6. Capillary lactate and lactate calculated from a blood sample were grouped to increase power for the meta-analysis and allow us to separate exercise modalities. Overall, hyperoxia had a small effect on decreasing lactate build-up during maximal exercise ($-8.1 \pm 11.8\%$, $g = -0.26$, 95% CI $-0.46$ to $-0.07$, $p = 0.008$, $TT_n = 6$, $TTE_n = 7$, $GXT_n = 11$). During TTs, hyperoxia had a small effect on decreasing lactate concentration at completion by $-4.2 \pm 13.7\%$ ($g = -0.46$, 95% CI $-0.89$ to $-0.03$, $p = 0.04$, $TT_n = 6$) and TTE by $-14.0 \pm 14.9\%$ ($g = -0.49$, 95% CI $-0.99$ to 0.01, $p = 0.05$, $TTE_n = 7$), yet had a negligible effect during GXTs ($-6.9 \pm 6.9\%$, $g = -0.08$, 95% CI $-0.31$ to 0.15, $p = 0.51$, $GXT_n = 11$).

Overall, with exercise, hyperoxia had a negligible effect on pH ($0.01 \pm 0.4\%$, $g = 0.07$, 95% CI $-0.21$ to 0.35, $p = 0.63$, $TT_n = 2$, $TTE_n = 2$, $GXT_n = 2$, $muscle_n = 1$). Hyperoxia had a moderate effect on decreasing respiratory exchange ratio ($-3.5 \pm 5.7\%$, $g = -0.51$, 95% CI $-1.05$ to 0.02, $p = 0.06$, $TTE_n = 1$, $GXT_n = 9$), a small effect on decreasing norepinephrine by $-7 \pm 19.7\%$ ($g = -0.29$, 95% CI $-0.74$ to 0.16, $p = 0.20$, $TT_n = 1$, $TTE_n = 1$, $GXT_n = 1$, $muscle_n = 2$), and a negligible effect on epinephrine by $-13.5 \pm 16.7\%$ ($g = -0.19$, 95% CI $-0.64$ to 0.25, $p = 0.40$, $TT_n = 1$, $TTE_n = 1$, $GXT_n = 1$, $muscle_n = 2$) during exercise.

### 3.6 Relationship between Fraction of Inspired Oxygen ($F_iO_2$) and Performance

To determine if there was a dose–response relationship between the level of $F_iO_2$ and acute exercise performance,

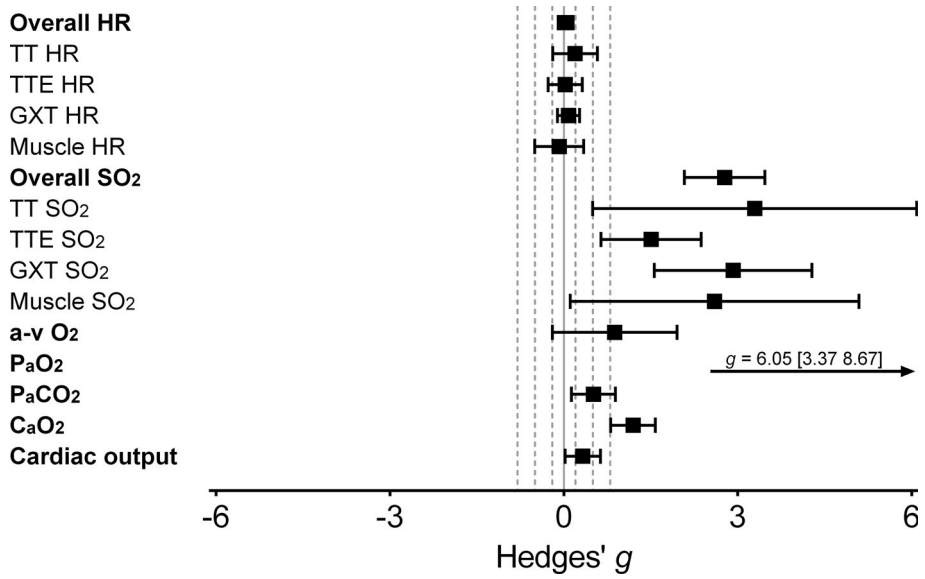


**Fig. 4** Forest plot summarizing the effect (± 95% confidence intervals) of hyperoxia on cardiovascular variables during acute exercise. *Bolded terms* include all exercise modalities. *Dashed lines* represent small (Hedges' $g = 0.20$–$0.49$), moderate (Hedges' $g = 0.50$–$0.79$), and large (Hedges' $g \geq 0.80$) effect sizes. The variable with a very large $g$ value is represented with an arrow to enhance legibility. *TT* Time trial, *TTE* time to exhaustion *GXT* graded exercise test, *Muscle* dynamic muscle tests, *HR* heart rate, $SO_2$ oxygen saturation, $a$–$v\ O_2$ arteriovenous oxygen difference, $P_aCO_2$ arterial partial pressure of carbon dioxide, $P_aO_2$ arterial partial pressure of oxygen, $C_aO_2$ arterial content of oxygen

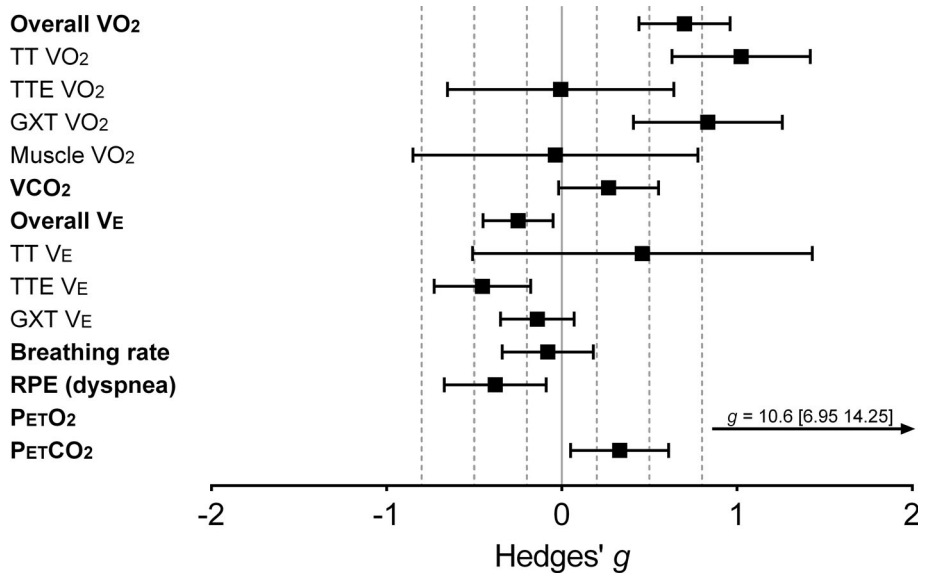


**Fig. 5** Forest plot summarizing the effect (± 95% confidence intervals) of hyperoxia on respiratory variables during acute exercise. *Bolded terms* include all exercise modalities. *Dashed lines* represent small (Hedges' $g = 0.20$–$0.49$), moderate (Hedges' $g = 0.50$–$0.79$), and large (Hedges' $g \geq 0.80$) effect sizes. The variable with a very large $g$ value is represented with an arrow to enhance legibility. *TT* Time trial, *TTE* time to exhaustion *GXT* graded exercise test, $VO_2$ oxygen consumption, $VCO_2$ carbon dioxide consumption, $V_E$ minute ventilation, $P_{ET}O_2$ end-tidal partial pressure of oxygen, $P_{ET}CO_2$ end-tidal partial pressure of carbon dioxide, *RPE* ratings of perceived exertion

we examined the effect size on exercise performance with the level of $F_iO_2$ (Fig. 7). We also explored whether different measures of oxygen availability or aerobic fitness of participants had a relationship with effect on performance. The $F_iO_2$ dose–response analysis was performed both with all acute exercise studies pooled together and then again separately for each of the four main exercise test modalities (TTs, TTE, GXTs, dynamic muscle function). Of the 51

studies, 20 had an $F_iO_2$ of 0.30–0.40, 16 studies had an $F_iO_2$ of 0.41–0.60, 5 studies had an $F_iO_2$ of 0.61–0.80, and 12 studies had an $F_iO_2$ of 1.00. When all acute exercise studies were pooled together, we found a very weak correlation between increasing levels of $F_iO_2$ and effect size of performance (Fig. 7a; $r = 0.22$, 95% CI −0.08 to 0.47, $p = 0.143$, number of pairs [$n_{pairs}$] = 47). Exercise performance was most correlated with $C_aO_2$ (Fig. 8a; $r = 0.62$, 95% CI −0.25 to 0.94, $p = 0.14$, $n_{pairs} = 7$), followed by $P_aO_2$ (Fig. 8b; $r = 0.55$, 95% CI −0.34 to 0.92, $p = 0.20$, $n_{pairs} = 7$), oxygen saturation (Fig. 8c; $r = 0.46$, 95% CI 0.03–0.74, $p = 0.04$, $n_{pairs} = 21$), and $\dot{V}O_{2max}$ of participants (Fig. 8d; $r = 0.32$, 95% CI −0.06 to 0.61, $p = 0.10$, $n_{pairs} = 29$).

When the relationship between $F_iO_2$ and performance effect size was examined separately by exercise modality, TTs (Fig. 7b; $r = 0.59$, 95% CI −0.30 to 0.93, $p = 0.16$, $n_{pairs} = 7$), TTE (Fig. 7c; $r = 0.63$, 95% CI 0.12–0.88, $p = 0.02$, $n_{pairs} = 13$), and dynamic muscle function (Fig. 7e; $r = 0.47$, 95% CI −0.44 to 0.90, $p = 0.29$, $n_{pairs} = 7$) had a positive linear relationship, whereas GXTs (Fig. 7d; $r = 0.06$, 95% CI −0.39 to 0.49, $p = 0.80$, $n_{pairs} = 20$) did not.

### 3.7 Effect of Hyperoxia during Recovery on Subsequent Exercise Performance

Eight studies examined the effects of breathing hyperoxic gas mixture on subsequent exercise performance, with all using an $F_iO_2$ of >0.995. The duration of hyperoxic exposure during recovery ranged from 4 to 10 min, with an interval before subsequent exercise from several minutes to 12 h. When the interval between exercise bouts lasted less than 12 h, there was a small ergogenic effect of hyperoxia in recovery (Fig. 3) of 2.9 ± 2.8% ($g = 0.22$, 95% CI −0.12 to 0.56, $p = 0.20$, $TT_n = 2$, $TTE_n = 1$, $GXT_n = 1$, $sprint_n = 3$). The only study that separated performance by 12 h did not demonstrate any ergogenic effect for a TT (−0.5%, $g = -0.12$, 95% CI −1.10 to 0.86) [24].

### 3.8 Effect of Training in Hyperoxia for Normoxic Exercise Performance

Four studies investigated whether exercise training in hyperoxia provided an ergogenic effect for competition in a normoxic environment (Fig. 3). There was a large overall effect of training in hyperoxia on exercise performance ($g = 0.95$, 95% CI −0.23 to 2.14, $p = 0.12$, $TTE_n = 3$), however due to the large variability in exercise training modalities, the range of effect sizes within each study was −0.4 to 2.10. Nonetheless, all four studies found a moderate effect for increasing normoxic $\dot{V}O_{2max}$ from training in hyperoxia ($g = 0.55$, 95% CI −0.05 to 1.04, $p = 0.19$, $TTE_n = 3$, $GXT_n = 1$).

## 4 Discussion

The primary aim of this meta-analysis was to quantitatively assess the potential ergogenic effects of hyperoxia during acute exercise performance, recovery between exercise bouts, or with chronic training for subsequent competition in normoxic environments. The meta-analysis reviewed 40 manuscripts (51 studies) investigating hyperoxia and acute maximal exercise performance across a variety of modalities, including TTs, TTE, GXTs, or dynamic tests using large locomotory muscles. A further seven manuscripts (eight studies) investigated the use of hyperoxia as a

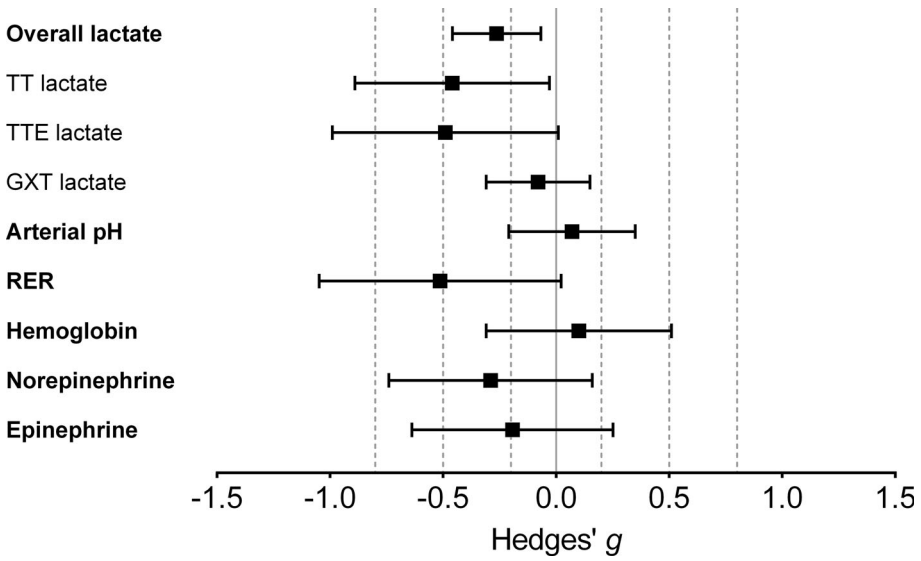


**Fig. 6** Forest plot summarizing the effect (± 95% confidence intervals) of hyperoxia on metabolic variables during acute exercise. *Bolded terms* include all exercise modalities. *Dashed lines* represent small (Hedges' $g = 0.20$–$0.49$), moderate (Hedges' $g = 0.50$–$0.79$), and large (Hedges' $g \geq 0.80$) effect sizes. *TT* time trial, *TTE* time to exhaustion *GXT* graded exercise test, *RER* respiratory exchange ratio

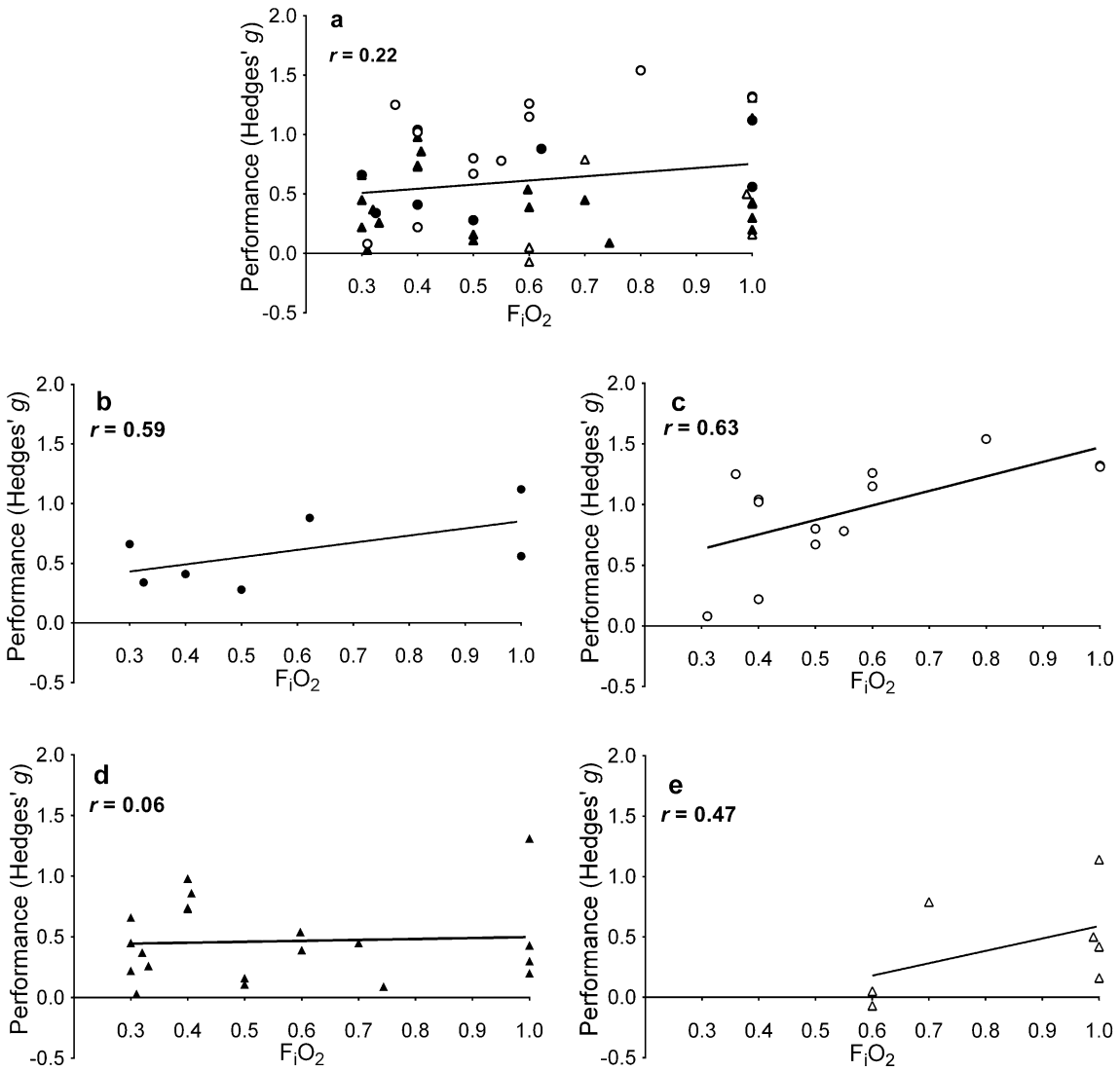


**Fig. 7** Pearson's correlations between $F_iO_2$ and effect size of performance when exercise modalities are grouped (**a**), and separated by exercise type: time trial (*filled circles*, **b**), time to exhaustion (*open circles*, **c**), graded exercise tests (*filled triangles*, **d**), or dynamic muscle function (*open triangles*, **e**). $F_iO_2$ fraction of inspired oxygen

recovery tool for subsequent exercise, with four manuscripts (four studies) testing the effect of training in hyperoxia for subsequent normoxic exercise. TTs and TTE ranged from ~ 4 and 5 min as the shortest, to ~ 28 and ~ 33 min as the longest, respectively. Due to the variety of TT distances in the examined studies, these tests were performed at a wide range of percentage of maximal physiologic capacity (~ 70–98% of peak power). However, regardless of the length of the TT, the nature of TTs is that they are performed at a maximal voluntary effort for each test. Dynamic muscle tests were either incremental leg extensions—similar to a GXT, just isolated for leg extensions—or were repeated leg extensions between 70 and 100% maximum voluntary force. We also quantified changes in physiological measures that are affected by altered $F_iO_2$. The secondary aim of this meta-analysis was to test for a dose–response relationship for the level of $F_iO_2$ and the corresponding effect on exercise performance. When used during a single bout of exercise, hyperoxia moderately improved performance by ~ 17%. An $F_iO_2$ of 0.3 was sufficient to elicit performance improvement corresponding to a small effect or higher, with small to moderate correlations between exercise performance and higher $F_iO_2$ with TTE ($r = 0.63$), TTs ($r = 0.59$), and dynamic muscle function ($r = 0.47$) tests. Hyperoxia offered a small effect as a recovery intervention, with eight studies observing a combined small effect when used in-between bouts of intermittent exercise. Chronic

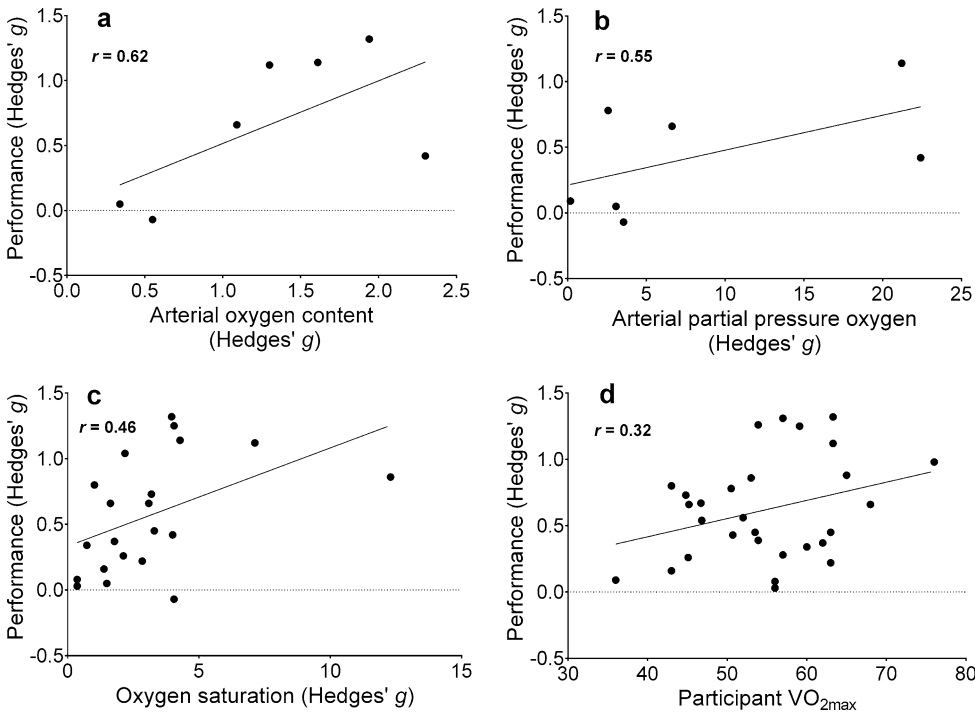


**Fig. 8** Pearson's correlations between effect size of performance and the effect sizes of **a** arterial oxygen content, **b** arterial partial pressure of oxygen, **c** oxygen saturation, and **d** participant $VO_{2max}$. $\dot{V}O_{2max}$ maximal oxygen consumption

training in hyperoxia was associated with a large effect on subsequent performance; however, with only three studies examining exercise performance and four for $\dot{V}O_{2max}$, this finding must be taken as very preliminary and with caution.

### 4.1 The Ergogenic Effect of Hyperoxia

Overall, hyperoxia consistently improved endurance exercise capacity and performance, although the degree of improvement varied widely across exercise types. TTE tests experienced the largest performance improvement ($\sim$ 40%), followed by dynamic muscle function ($\sim$ 8%), GXTs ($\sim$ 6%), and TTs ($\sim$ 6%). The TTE data included one outlier study with an improvement of 138% [7]; nevertheless, mean improvement without this study remained high at $\sim$ 32%. The wide difference in the magnitude of improvement between TTE and TT tests likely reflects the typically greater variability observed with TTE tests [37, 45, 46]. However, in terms of practical extrapolation to athletic performance, the sensitivity of both TTE and TT tests to changes in endurance, specifically to hypoxic and hyperoxic manipulations [46], and the ability of TTE data to predict TT performance [37] in trained individuals was confirmed.

Another contributing factor to greater performance improvements in TTE than GXTs or TTs may be because TTE tests are typically performed at a percentage of normoxic $\dot{V}O_{2max}$. As hyperoxia increases $\dot{V}O_{2max}$ [8, 47, 48], TTE tests with hyperoxia are therefore performed at a relatively lower percentage of maximal aerobic capacity, decreasing the stress on cardiorespiratory, metabolic, and hormonal response to exercise. Ulrich et al. [49] performed a TTE at a relative percentage of normoxic $\dot{V}O_{2max}$. At the same time that exercise cessation occurs in normoxia, they observed a decreased heart rate, $V_E$, breathing rate, and respiratory exchange ratio, and increased oxygenation (assessed via pulse oximetry and near-infrared spectrometry), oxygen uptake, end-tidal atrial pressure of oxygen and carbon dioxide in hyperoxia [49, 50]. Upon fatiguing in hyperoxia, heart rate, breathing rate, and $V_E$ were not different compared with normoxia, yet all other differences remained. Byrnes et al. [48] performed submaximal exercise in normoxia and two trials in hyperoxia ($F_iO_2 = 0.70$), one at the same absolute workload as normoxia and another at the same relative intensity as normoxia. They found attenuated cardiorespiratory, lactate, and central catecholamine levels at the same absolute workload, however no differences were observed during exercise at the same relative intensity [48]. To control for this potential confounder, future TTE tests should perform a $\dot{V}O_{2max}$ test at both normoxia and hyperoxia, and perform the TTE at similar relative $\dot{V}O_{2max}$ levels.

Multiple mechanisms likely contribute to increased acute exercise performance in hyperoxia, with most of these mechanisms revolving around increased oxygen availability at the muscles. Alterations in $C_aO_2$ paralleled exercise performance [7, 51, 52], and Amann et al. [7] demonstrated that 5 km TT performance mirrored $C_aO_2$ throughout a range of $F_iO_2$ from hypoxia to hyperoxia. Exhaustion occurs at similar concentrations of metabolic

byproducts (e.g. lactate, hydrogen ions, inorganic phosphate) in both normoxia or hyperoxia, but these critical levels were reached later in exercise or with a greater power output, supporting previous work that hyperoxia attenuates the accumulation of metabolic byproducts [53, 54]. This attenuation may be from greater oxygen availability facilitating aerobic metabolism. Linnarsson et al. [55] demonstrated that hyperoxia facilitates aerobic metabolism by decreasing the oxygen deficit at the onset of exercise, resulting in decreased lactate, inorganic phosphate, hydrogen ion, and pyruvate production [14]. As hyperoxia increases the amount of oxygen available, and thereby energy available, it does not appear to stress the oxidative pathways more than normoxia.

Although the small number of available studies precluded its examination in the present meta-analysis, hyperoxia may affect central motor output to the muscle. Higher $C_aO_2$ was associated with increased electromyographic activity and neural drive during a TT, whereas the opposite occurred with reduced $C_aO_2$ [7, 51, 52]. Amann et al. [7] also observed the same level of peripheral fatigue at the end of both TT and TTE tests throughout their range of $F_iO_2$, despite differences in mean power output or exercise time. Tucker et al. [51] found that an $F_iO_2$ of 0.4 improved 20 km TT performance by enhancing oxygen availability, and thereby energy availability, which increased muscle activation during the second half of the TT, as indicated by increased electromyography activity in the vastus lateralis.

Because of increased muscular activity and higher power outputs, exercise performed in hyperoxia facilitates higher training intensities, thus increasing the training stimulus and potentially promoting greater physiological adaptations. We found a beneficial effect on normoxic exercise performance from training in hyperoxia; however, due to the large variability and low power, we were unable to definitively say that training in hyperoxia acts as an ergogenic aid [33–36]. Maladaptation may exist with excessive exercise intensity while training in hyperoxia. As our current analysis has established an increased acute exercise capacity in hyperoxia, perhaps by increasing intensity there is a greater risk of overtraining and seeing no ergogenic effect when that higher training stimulus is paired with inadequate recovery. Perry et al. performed two training studies where participants performed 4 min intervals at either 80% [33] or 90% of normoxic $\dot{V}O_{2max}$ [34]. The 80% $\dot{V}O_{2max}$ protocol greatly increased performance ($\sim$ 105%, $g = 2.10$) [33], but no change ($\sim -1.2\%$, $g = -0.04$) occurred from the more intense protocol [34]. Another potential reason for the lack of a performance benefit while training at 90% $\dot{V}O_{2max}$ is that the higher-intensity exercise may have stressed the anaerobic energy system more than the aerobic system, resulting in less of an aerobic improvement with normoxic exercise. Performing exercise training in hyperoxia increases the training workload but does not lead to different metabolic adaptations [34, 36], suggesting potential neuromuscular adaptions with hyperoxic training; however, this hypothesis has yet to be tested. The timing of gas delivery also appears to be important for training. Murray et al. [56] had participants breathe hyperoxic gas during rest periods of interval workouts, and found that the hyperoxic group did not increase training stress (as assessed by distance ran) compared with the normoxic group. Thus, albeit with limited evidence, it appears that hyperoxia needs to be delivered during exercise and at a training intensity of $\leq$ 80% $\dot{V}O_{2max}$ to facilitate the greatest performance increase. Although preliminary research seems promising in that training in a hyperoxic environment increases performance in a normoxic environment, more research needs to be conducted to determine the optimal training intensity, frequency, and duration and rest interval to maximize physiological adaptations and exercise performance.

If inadequate oxygen delivery limits intense endurance exercise through arterial hypoxemia [4], Wilson and Welch [57] proposed that hyperoxia may increase performance in a dose-dependent fashion, reporting that running TTE progressively increased with $F_iO_2$ between 0.4 and 1.0. Our pooled analysis of all acute exercise studies reported only a weak correlation effect between increased $F_iO_2$ and performance effect size; however, this weak effect may have been an artefact of mixing multiple testing protocols. When separated by exercise type, TT, TTE, and dynamic muscle function tests all showed significant positive correlations between $F_iO_2$ and exercise performance, while GXTs showed none. Therefore, the decision of what $F_iO_2$ to use may come down to the testing modality or application. For athletic applications, where the training goal may be to maximize the duration or intensity of training stimulus, our analysis suggests that higher levels of $F_iO_2$, if properly supervised, may be effective.

One additional question to consider with the athletic application of hyperoxia is the fitness level of potential users. While some studies reported that individuals with higher levels of fitness respond stronger and more sensitively to hyperoxia and greater $F_iO_2$ [6, 26, 30, 31], our overall meta-analysis found no significant relationships. One caveat with the existing literature is that the relatively homogenous population of male participants with high aerobic fitness may have precluded a full examination of the relationship between fitness or sex and response to hyperoxia.

The role of hyperoxia in altering perceptual responses to exercise remains difficult to delineate from this meta-

analysis. TT protocols feature the capacity to self-regulate power output, and the higher average power in hyperoxic conditions likely elicited similar whole-body ratings of perceived exertion due to a relative intensity matching that during normoxia [58–60]. In contrast, while RPE may be lower with hyperoxia during TTE or GXT protocols, almost all surveyed studies only recorded RPE at the point of voluntary exhaustion, where perceptual exertion would a priori be expected to be similar. However, a small decrease in perceived breathing exertion was found, which also reduces perceived discomfort and prolongs exercise tolerance [2]. Future studies can better investigate the perceptual response to hyperoxia by measuring these variables throughout TTE and GXT protocols, especially at identical timepoints such as the iso-time point during hyperoxia relative to when exhaustion was reached in normoxia.

### 4.2 Recovery for Subsequent Exercise

Hyperoxia is commonly delivered as a recovery aid in-between bouts of exercise, despite minimal scientific investigations into its efficacy. As one of the main benefits of hyperoxic exercise is facilitating aerobic metabolism by increasing the amount of oxygen in the blood, hyperoxia should enhance recovery by facilitating the reversal of metabolic acidosis. Only eight studies fit within our inclusion criteria of hyperoxia during recovery between exercise bouts with the presence of a normoxic control group; these studies ranged widely in the period between exercise bouts, from 4 min through to 12 h, making direct comparisons and a consensus difficult. Almost all manuscripts evaluated the effect of hyperoxic recovery on very short, high-intensity activities, using protocols that generated large amounts of harmful metabolic byproducts [24, 27, 28, 61]. Overall, a small but non-significant positive effect ($g = 0.22$) was found, suggesting that hyperoxia may mildly benefit recovery but with a high amount of uncertainty as to its true efficacy. Most studies also focused primarily on performance measures, with very few exploring any mechanistic insights. Thus, hyperoxic recovery appears to be an area where much research is required to definitively determine its efficacy and then to understand potential mechanisms of effect. Future studies may also consider longer duration and aerobically dominant tests as hyperoxic recovery may also be useful in-between competition heats (e.g. track and field, track cycling, speed skating).

## 5 Conclusions and Practical Considerations

Hyperoxia consistently and substantially increases acute exercise performance, with TTE protocols showing the largest improvement. Other exercise modalities, such as TTs, GXTs, and dynamic tests of large locomotory muscles, all demonstrate increased exercise performance with acute hyperoxic exposure. Athletes can therefore confidently expect to be able to train at higher absolute workloads or extend the duration of hard efforts when using hyperoxia. Theoretically, this will enable greater neuromuscular stimulus to generate these higher workloads, but, due to the greater oxygen availability and a potentially similar relative exercise intensity as during normoxia, whether hyperoxia translates to greater metabolic stimulus remains unclear. An $F_iO_2 \geq 0.30$ will elicit an increase in performance but a dose-dependent relationship may depend on the type of exercise. Whereas GXTs showed no correlation with $F_iO_2$, TTs, TTE, and dynamic muscle function performance demonstrated a positive relationship with $F_iO_2$. As most actual training efforts (e.g. interval sessions) and competitions resemble a TT or TTE style of effort, it thus appears that, where feasible and safe, higher $F_iO_2$ may enable a greater training stimulus. Training in hyperoxia allows athletes to exercise at higher intensities, thus imposing a larger physiological training stress. Importantly, there appears to be a ceiling on improvement at high training intensities as there is a chance of athletes becoming overtrained more easily if proper and careful recovery is not implemented. However, more work needs to be done to optimize exercise training intensity and duration in order to achieve maximal performance gains while minimizing the risk of maladaptation or overtraining. Based on the current available research, it appears that hyperoxic training at intensities above 80% $\dot{V}O_{2max}$ may be counterproductive. Hyperoxia as a recovery modality for subsequent exercise performance remains unclear. There may be a small benefit of exercise recovery in hyperoxia, but more research must be conducted in order to prove the efficacy of using hyperoxia as a recovery modality.

Hyperoxia applied acutely is quite clearly an ergogenic aid; however, the precise mechanisms of how hyperoxia acts as an ergogenic aid remains yet to be elucidated. There is emerging evidence of a central/neural effect from hyperoxia; however, with only three manuscripts reporting these data in maximal exercise, it is difficult to make a firm conclusion. Importantly, care must be taken when performing set workload tests as the relative intensity of the workload is not the same between normoxia and hyperoxia. Perhaps the areas that will benefit most from further research in hyperoxia are training programs and recovery. To maximize physiological adaptations from training in hyperoxia, the optimal training exercise intensity, duration, and recovery interval still needs to be elucidated. Likewise, more research needs to be conducted in the area of recovery modalities, especially combining hyperoxia with other recovery modalities, such as contrast water therapy, or active recovery.

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**Compliance with Ethical Standards**

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