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REVIEW

The role of ambient air pollution, exercise intensity and duration on the acute lung function and airway inflammation responses to exercise: a systematic review

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Abstract

Background More attention is required on the relations between air pollution and exercise characteristics.

Aims This systematic review aims to investigate the combined effects of exercise intensity and duration in ambient air pollution referenced against 2021 WHO Air Quality Guidelines, on lung function and fractional exhaled nitric oxide (FeNO).

Methods A search was conducted using PubMed, Sport Discus, Proquest, and Web of Science databases, up to August 2023, in accordance with the PRISMA guidelines.

Results From 1220 identified articles, 22 were included based on inclusion and exclusion criteria. Exercise intensity and duration were reported as described by authors of the original research. Pollutant concentrations were classified as above or below the 2021 24-h WHO Air Quality Guideline pollutant thresholds. Exercise intensities were inconsistent, ranging from "comfortable pace" walking to 90% maximum heart rate. Exercise duration ranged from 20 min to 8 h of intermittent exercise. Eighteen studies measured pollutants that the WHO provide 24 h thresholds for; 14 of those 18 studies had conditions that exceeded threshold for at least one pollutant, and 11 of the 14 reported significant associations between air pollution, exercise, lung function and/or FeNO.

Conclusions Adverse lung function and FeNO responses were associated with exercise in conditions with pollutant concentrations exceeding the 2021 24 h WHO thresholds. Longer duration exercise (> 2 h) was frequently associated with adverse acute responses, whilst the potential influence of exercise intensity was less clear. Evidence appears to suggest exercise ≥ 120 min in pollution concentrations exceeding WHO thresholds may result in reduced lung function.

Keywords Exercise · Air pollution · Lung function · Air quality · Airway inflammation

Introduction

Exercise is an effective non-pharmacological means for preventing and treating various non-communicable diseases including cardiovascular disease [1], type-2 diabetes [2], obesity [3] and more recently recommended to optimise the management of respiratory diseases including asthma [4]. Accessible forms of exercise such as running and walking are encouraged to increase physical activity participation for the majority of populations [5]. However, for exercise recommendations to be optimal for public health, it is important to consider environmental factors such as air pollution, that may threaten the health benefits associated with exercise [6]. It has been suggested that more attention needs to be focussed on the relations between air pollution and the characteristics of exercise such as duration and intensity [7, 8]. In the future, better understanding of these relationships would aid communication to the general public about air pollution concentrations and associated advice about exercise behaviour.

Ambient air pollution is the most potent environmental threat to global health [9]. From a public health perspective, numerous pollutants are associated with a many diseases

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[10], and as many as 16% of premature deaths associated with pollutant inhalation [8, 11]. Short-term exposures to moderate levels of traffic related air pollution (TRAP) including coarse (PM_{10}) and fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2), have been associated with an increase in inflammatory markers and reduced lung function, despite pollutant concentrations below former 2005 World Health Organisation (WHO) limits [12].

The respiratory system is the first organ system to be impacted by air pollution [13]. During exercise, pollutant inhalation is increased due to the ventilatory responses that occur to meet metabolic demand [14, 15]. These include a greater tidal volume compared to rest, and a reduction in nasal passage filtration, as air is typically inhaled through the mouth with increased air flow velocity causing deeper deposition of particles that subsequently penetrate the airway epithelium. Exposure to air pollution has frequently been associated with changes in lung function [16]. Fractional exhaled nitric oxide (FeNO) is a non-invasive biomarker of eosinophilic airway inflammation [17], and is commonly measured to investigate the airway response to air pollution exposure in real-world studies not involving chambers and controlled concentrations of pollutants [18]. Furthermore, repeated exposure to irritants including pollutants are associated with declines in lung function [19] and chronically increased FeNO [20], and both are associated with reduced exercise capacity and deteriorating control of asthma [21]. Unlike other biomarkers of airway inflammation that are measured via venous blood samples, FeNO is measured using a non-invasive test that can be used in different settings. This, along with FeNO being a valid tool to assess the airway inflammatory response to air pollution exposure [22], means measuring FeNO in studies investigating exercise in ambient air is an appropriate and practical choice to facilitate field testing. FeNO has therefore been widely used to assess the airway inflammation response to air pollution in adults [12, 23–26].

The revised WHO Air Quality Guidelines published in 2021 [27] provided lower 24 h and annual thresholds for key pollutants compared to the previous 2005 guidelines, and it has even been suggested there may not be a threshold of no effect [28, 29]. Studies have considered a potential paradox between the benefits of exercise and the health risks associated with increased pollutant inhalation. Whilst some studies suggest the benefits of exercise outweigh the adverse effects of increased pollutant inhalation [30, 31], previous reviews have strongly encouraged for levels of pollutant exposure, the population, and exercise intensity be investigated further as they may influence the interaction between exercise and air pollution [8, 32]. To progress from those reviews and their directions, it is imperative that we place greater emphasis on exercise prescription variables including intensity and duration and explore how they may modify the relationship between exercise and air pollution [32].

A recent review suggested that mild exercise in low levels of pollution may be detrimental to health [8]. However, there has been no systematic review of evidence that has evaluated the impact of air pollution exposure during exercise, on acute changes in lung function and airway inflammation against the revised 2021 WHO thresholds. Given research in this field is conducted in various parts of the world, using standardised guidelines will aid comparisons and enable clearer conclusions to be made. At present, there are differences in how pollutant concentrations are classified by national air quality information resources based on each nation's daily air quality index (AQI). For example, the current 24 h WHO thresholds for both PM_{10} and $PM_{2.5}$ would be classified as "Low" on the current UK Daily AQI [33], in the United States PM_{10} would be classified as "Good" and $PM_{2.5}$ would be classified as "Moderate" [34], and in the European Union, PM_{10} would be classified as "Moderate" and $PM_{2.5}$ would be classified as "Fair" [35], all with slightly different recommendations about exercise behaviour and associated risk or pollution exposure.

This study aimed to systematically review existing evidence to better understand the combined effect of exercise and ambient air pollution on both lung function and airway inflammation indicated by FeNO, with specific focus on two key factors: First, the principles of exercise prescription, given factors such as exercise intensity and duration will influence respiratory physiology. And second, the comparison of pollutant concentrations against WHO 2021 24 h thresholds, given there is currently no consistent method used to provide a real-world, global context for the pollutants reported.

Methods

To identify relevant studies that examined the effect of air pollution during exercise on lung function and/or airway inflammation, an electronic database search was performed using PubMed, Sport Discus, Proquest, and Web of Science. The PRISMA guidelines were followed [36], with the following key words and phrases included in the search criteria: ("air quality" OR "air pollution" OR pollution OR "particulate matter" OR PM OR $PM_{2.5}$ OR PM_{10} OR ozone OR "nitrogen dioxide" OR NO_2 OR diesel OR TRAP OR "traffic related pollution exposure" OR "nitrogen oxides" OR "nitrogen oxide" OR NO_x) AND (Exercise OR "physical activity"OR running OR cycling OR"active travel"OR walking) AND ("Lung function" OR "respiratory function" OR "pulmonary function" OR FEV_1 OR FVC OR "peak flow" OR "fractional exhaled nitric oxide" OR FeNO OR "airway

inflammation" OR "exhaled nitric oxide"). Subsequently, a secondary search was conducted by viewing the reference list of each article included to ensure all relevant articles had been obtained.

Inclusion criteria

The studies eligible for analysis adhered to the following inclusion criteria:

(i) participants were adults (≥ 18 years old);
(ii) participants were performing exercise at any intensity;
(iii) lung function and/or FeNO were measured;
(iv) intensity, duration, and type of exercise was described;
(v) air pollution concentrations were reported;
(vi) exercise was performed in ambient air pollution only.

Exclusion criteria

Studies were excluded if any of the following applied:

(i) the study was not written in the English language;
(ii) concentration of pollutant exposure was controlled in an environmental chamber or delivered via a mask;
(iii) the environment was artificially controlled (e.g. ice rinks);
(iv) participants declared any acute illnesses or chronic diseases apart from asthma;
(v) participants were smokers;
(vi) animal models were used.

Whilst chamber studies with regulated pollutant concentrations offer robust control, those concentrations are not representative of real-world scenarios as they are typically higher than those encountered in the real-world, not as variable, and/or based on a single pollutant [37]. Therefore, these methods were not included in this review. Studies including participants with asthma will not be excluded from the review as there is no sufficient reason, providing the physiological responses are reviewed with appropriate consideration.

Study selection

Figure 1 outlines the study selection process. Following this study selection process, 22 studies out of the original 1220 were included in this systematic review. Using a blinded approach, all identified articles were screened by two authors (SMl and GD). Any differences in included articles after the exclusion process were discussed and a final decision was made by comparing the article against the inclusion and exclusion criteria.

Results

Participant characteristics and study design

A total of 1524 participants with mean ages between 20 and 45 years old were included in the 22 studies; five of the studies included asthmatic participants [38–42] (Table 1). In all studies, participants were either described as 'healthy', or as 'regular' exercisers, apart from the two studies that involved asthmatic participants exclusively that described the population based on asthma severity [40, 42]. Seventeen of the studies utilised a cross-over design [24, 37, 40–54] with either two [24, 40, 42–46, 48–52, 54], three [41, 47] or five conditions [37]. One of these studies utilised a single environment to compare an exercise and rest condition [53]. Of the remaining five studies, one utilised a prospective cohort design [55], one used a prospective intervention design [56], and three used a before and after study design [38, 39, 57]. If the authors included a description of the study design, this has been used. However, in cases where this was not provided the authors of this review have used an appropriate description.

Table 1 also summarises the physiological outcomes in each of the included studies. Lung function was measured via spirometry in 20 of the studies [24, 37–43, 45, 47–57], of which FEV_1, FVC, FEV_1/FVC, PEFR and $FEF_{25–75\%}$ were used most frequently, with one study measuring PEFR only using a peak flow metre [46]. FeNO was measured in 12 of the studies [24, 37, 40–44, 47, 51, 52, 56, 57].

Four of the studies included one experimental exposure [38, 39, 44, 56], with one of those using an indoor filtered air condition as a control [44], whereas all other studies included two or more, which for the purpose of this review have been termed 'higher pollution' (HP) or 'lower pollution' (LP) with 'mid pollution' (MP) when necessary. This description was informed by the intention of the exposure rather than the measured pollution concentrations; for example a roadside condition would be labelled as HP, and a park as LP. Where studies have one condition, this has been referred to using the 'HP' description. In two studies, where five conditions or routes have been used, the higher or lower categorisations have not been used as their focus was to investigate the interaction of pollutant concentrations and health outcomes without the initial purpose to use high vs. low-pollution conditions [37, 55]. Nine of the included studies described statistical interactions between pollutants and health outcomes only [37–39, 42, 47, 52, 53, 56, 57], two of the studies provided

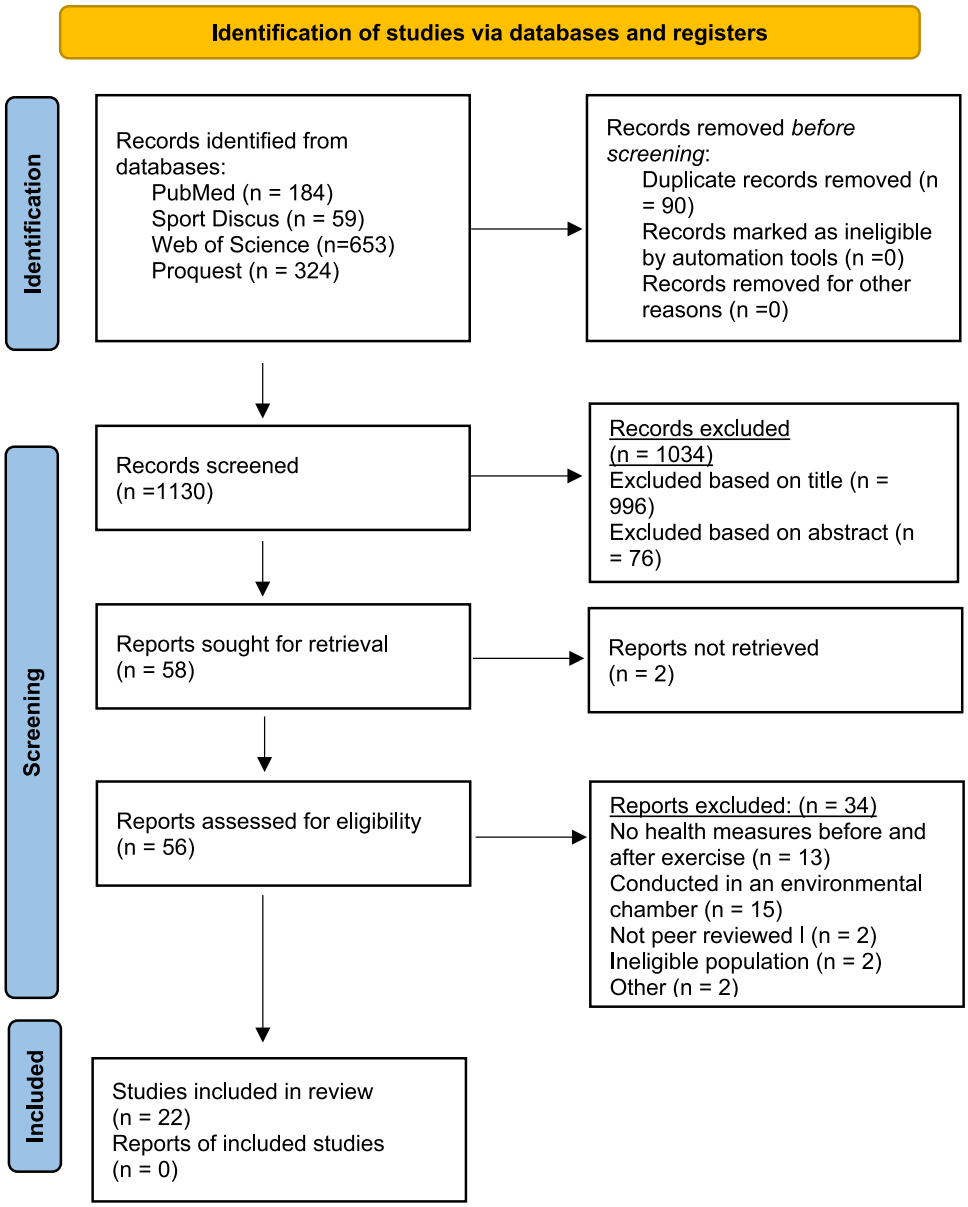

Fig. 1 Screening and selection process for identifying relevant studies

the pre- to post-exercise change in health outcomes only (either percentage or absolute values) [46, 51], and 11 studies included results from both types of analysis [24, 40, 41, 43–45, 48–50, 54, 55].

Characteristics of exercise performed

Cycling was the chosen mode of exercise in 14 of the studies [24, 37, 41, 44–46, 48–51, 53, 55–57], walking/hiking was used for seven [38–40, 42, 47, 52, 54], and running was used in a single study [43]. Exercise intensity was reported as: a relative percentage of maximum heart rate (HRmax) in seven of the studies, ranging between 50 and 90% of HRmax [24, 38, 43, 44, 48, 53, 56], as an absolute mean ± SD in beats per minute ($b.min^{-1}$) [46], or as $b.min^{-1}$ corresponding to VE (L/min^{-1}) [37, 50]. In other studies exercise intensity can be described by the average time it took to cover a given distance and is reported accordingly in km/h^{-1} [39, 41, 45, 47, 57]. Five studies provided a subjective description of exercise intensity such as 'walking at a steady pace' [40, 42, 52, 54, 55]. One described a resistance as percentage of body weight for a set duration [51], and another did not specify an intensity, but stated that pulse rate and cycling speed were included in analysis as covariates [49]. Duration of exercise ranged from 20 min up to 8 h, although some studies used intermittent rest periods, and therefore described total session time rather than continuous exercise time. All exercise characteristics are presented in Table 1.

Table 1 Study design, participant characteristics, exercise parameters and physiological outcomes for included studies

First author & year	Study design	Participants (M/F)	Mean age (years ± SD)	Number of experimental conditions	Control group? Y/N	Participant characteristics	Mode of Exercise	Exercise duration & distance	Exercise intensity	Time of exercise	Physiological outcome measurements
Cole et al. (2018) [50]	Crossover	$n = 38$ (28/10)	29 ± 5.6	1. Downtown (HP) 2. Residential (LP)	N	Healthy Adults	Cycling	HP = 63.9 ± 3.42 min (9.7 km) LP = 62.9 ± 3.57 min (12 km)	Mean V_E: HP = 44.8 ~ 14 L/min LP = 47.8 ~ 14.3 L/min	07.00–16.00	Lung function, endothelia function (IL6, IL8)
Cole-Hunter et al. (2013) [46]	Crossover	$n = 35$ (10/25)	39 ± 11	1. High proximity to traffic (HP) 2. Lower proximity to traffic (LP)	N	Healthy adults	Cycling	HP = 24 min 59 s LP = 24 min 37 s	HP = 136 ± 11 $b.min^{-1}$ LP = 132.5 ± 9 $b.min^{-1}$*	Not specified	Symptom reporting, peak flow metering, sputum collecting
Elliott and Loomis (2020) [55]	Prospective cohort	$n = 31$ (12/19)	34.7 (20.3–58.1)	Five routes available to participants	N	Healthy adults	Cycling	All = 51 min average (range: 25–100 min), 5.5–15.5 miles	"Comfortable pace"	Not specified	Lung function
Girardot et al. (2006) [39]	Before and after	$n = 354$ (154/200)	43.2 ± 12.6	Hiking trail	N	Regular day hikers, some asthmatic ($n = 62$)	Hiking (1,540 m summit)	5 ± 1.2 h (6.7 km)	2.8 km/h (approx.)	12.00–14.00	Lung function
Guo et al. (2023) [53]	Crossover	$n = 74$ (35/39)	19.9 ± 0.9	University campus outdoor recreational area	N	Healthy adults	Cycling	2 h (4 × 15 min exercise interspersed with 4 × 15 min rest)	70–75% HRmax	10.00–12.00	Lung function
Habre et al. (2018) [42]	Crossover	$n = 22$ (6/16)	27 ± 9.5	1. Park close to major roadway (HP) 2. Park further from immediate traffic (LP)	N	Adults with mild to moderate asthma	Walking	HP & LP = 2 h with rest every 15 min	"Mild walking"	12:00–14:00	Lung function, FeNO, IL-6 soluble tumour necrosis factor receptor II, von-Willebrand factor
Jacobs et al. (2010) [44]	Crossover	$n = 38$ (28/10)	43 ± 8.6	1. Real traffic road (HP) 2. Indoor (FA)	Y (FA)	Regular cyclists	Cycling	HP = 20.8 ± 1.6 min FA = 20.2 ± 1.9 min	HP = 74 ± 8.6% HRmax FA = 74.1 ± 8.8% HRmax	08.00–17.00	FeNO, differential leukocyte count, plasma IL-6, blood neutrophil counts
Jarjour et al. (2013) [45]	Crossover	$n = 15$ (11/4)	32.2 ± 6.67	1. High-traffic cycle route (HP) 2. Low traffic cycle route (LP)	N	Regular cyclists	Cycling	HP = 40.2 min (8–9.5 km) LP = 37.6 min (8–9.5 km)	HP = 13.6 km/h (approx.) LP = 13.9 km/h (approx.)	08.00–10.00	Lung function
Korrick et al. (1998) [38]	Before and after	$n = 530$ (375/155)	35 ± 10	Hiking trail	N	Recreational hikers, some asthmatic ($n = 40$)	Hiking (1,900 m summit)	8.0 ± 1.5 h	66 ± 14% HRmax	10.00–15.30 (approx.)	Lung function
Kubesch et al. (2015) [24]	Crossover	$n = 28$ (13/15)	34.4	1. High traffic (HP) 2. Low traffic (LP)	N	Healthy adults	Cycling	2 h (4 × 15 min exercise interspersed with 4 × 15 min rest)	50–70% HRmax	08:00–10:00	Lung function, FeNO, IL-1b, IL-6, IL-8, IL-10, TNFα
Lammers et al. (2020) [56]	Prospective intervention	$n = 21$(4/17)	23 (20–23)	Airport	N	Healthy adults	Cycling	5 h (20 min cycling per hour)	50–60% HRmax	10.00–15.00	Lung function, FeNO, electrocardiography, BP

Table 1 (continued)

First author & year	Study design	Participants (M/F)	Mean age (years ± SD)	Number of experimental conditions	Control group? Y/N	Participant characteristics	Mode of Exercise	Exercise duration & distance	Exercise intensity	Time of exercise	Physiological outcome measurements
Matt et al. (2016) [48]	Crossover	$n = 30$ (15/15)	36 (19–57)	1. High traffic (HP) 2. Low traffic (LP)	N	Healthy adults	Cycling	HP & LP = 2 h (4 × 15 min exercise interspersed with 4× 15 min rest)	56% HRmax (range = 50%–62% HRmax)	08.00–10.00	Lung function
McCreanor et al. (2007) [40]	Crossover	mild asthma- $n = 31$ (17/14) moderate asthma-$n = 29$ (15/14)	Mild asthma = 31 Moderate asthma = 34	1. High traffic (HP) 2. Low traffic (LP)	N	Adults with mild and moderate asthma	Walking	HP & LP = 2 h walking, 6 km (15 min rest every 30 min)	"Steady pace"	10.30–12.30	Lung function, FeNO
Mirowsky et al. (2015) [47]	Crossover	$n = 23$ (11/12)		1. George Washington Bridge (HP) 2. Garden State Parkway (MP) 3.Rural Road in Sterling Forest (LP)	N	Healthy adults	Walking	All = 2 h (20 min walking, 5 min rest)	4.8 km/h	Not specified	Lung function, FeNO, HRV, BP, blood prick
Moshammer et al. (2019) [52]	Crossover	$n = 24$ (11/13)	24 (21–33)	1. Along a busy road (HP) 2. In a park (LP)	N	Healthy adults	Walking	HP & LP = 1 h	"Walk"	Not specified	Lung function, FeNO, HRV, BP
Park et al. (2017) [49]	Crossover	$n = 32$ (24/12)	45.1 ± 12.5	1. High traffic route (HP) 2. Low traffic route (LP)	N	Healthy adults	Cycling	HP & LP = 22.2 ± 3.1 km	Not specified but included as covariate in analysis	Not specified	Lung function, pulse rate
Rundell et al. (2008) [43]	Crossover	$n = 12$ (12/0)	20.5 ± 2.42	1. High traffic route (HP) 2.Low traffic route (LP)	N	Heathy adults	Running	HP & LP = 30 min	85–90% HRmax	"mid-morning"	Lung function, exhaled breath condensate, FeNO
Strak et al. (2010) [57]	Before and after (between-participant)	$n = 12$ (4/8)	30	1. High traffic (HP) 2. Low traffic (LP)	N	Regular cyclists	Cycling	HP & LP = 1 h (approx.) per exposure HP = 8 km LP = 7.7 km	HP = 8 km/h LP = 7.7 km/h	08.00–9.30	Lung function, FeNO
Strak et al. (2012) [37]	Crossover	$n = 31$ (10/21)	22 (19–26)	1. Underground train station 2. Continuous traffic location 3. Stop and go traffic location 4. Farm 5. Background urbans site	N	Healthy adults	Cycling	All = 5 h (20 min cycling per hour)	HR ($b.min^{-1}$) corresponding to minute ventilation rate of 20 L/min/body surface area (m^2)	Start 09:00–09:30 End 14:00–14:30	Lung function, FeNO, respiratory symptoms
Wagner and Clark (2018) [51]	Crossover	$n = 16$ (5/11)	31.5 ± 1.3	1. Higher $PM_{2.5}$ (HP) 2. Lower $PM_{2.5}$ (LP)	N	Healthy adults	Cycling	HP & LP = 20 min	20-min time trial against a resistance of approx. 3.5% of body weight	Not specified	Lung function, FeNO, c-reactive protein

Table 1 (continued)

First author & year	Study design	Participants (M/F)	Mean age (years ± SD)	Number of experimental conditions	Control group? Y/N	Participant characteristics	Mode of Exercise	Exercise duration & distance	Exercise intensity	Time of exercise	Physiological outcome measurements
Weichenthal et al. (2011) [41]	Crossover	$n = 42$ (28/14)	35 (19–58)	1. High traffic (HP) 2. Low traffic (LP) 3. Indoor	N	Healthy adults, 26 with self-reported allergies and 14 asthma	Cycling	All = 10 km (approx.) 1 h	20 km/h	11:30–12:30	Lung function, FeNO, HRV
Zhu et al. (2023) [54]	Crossover	$n = 56$ (25/31)	23.5 ± 2.4	1. High traffic (HP) 2. Low traffic (LP)	N	Healthy adults	Walking	4 h total (15 min walking, 30 min rest)	"Steady pace"	13:00–17:00	Lung function

BP blood pressure, *FA* filtered air, *FeNO* fractional exhaled nitric oxide, *HP* high pollution, *HRmax* maximum heart rate, *HRV* heart rate variability, *IL6* interleukin 6, *IL8* interleukin 8, *IL10* interleukin 10, *LP* low pollution, *MP* mid pollution, *TNFα* tumour necrosis factor alpha, V_E minute ventilation

Lung function and airway inflammation outcomes

Of the 20 studies that reported upon interactions between pollutants, lung function and airway inflammation (Table 2), six of them reported no interaction between any pollutant and lung function parameter and/or FeNO [39, 44, 45, 47, 50, 57]. The remaining 14 reported that an increase in particle number concentration (PNC) [37, 56], PM_{10} [24, 42, 48, 52, 53, 55], $PM_{2.5}$ [24, 38, 41, 42, 53], PM_1 [42, 43, 52, 53], ultrafine particulate matter (UFPM) [24, 40, 41, 49], NO_2 [37, 53, 54], O_3 [38, 53], and TRAP [54] were associated with a decline in at least one lung function measure, with time points ranging between immediately after exercise, through to 24 h after exercise (see Table 2). Regarding FeNO, PNC was associated with a significant increase at several time points up to 24 h post-exercise [37]. Conversely, one paper reported increases in NO_2 were associated with increased FEV_1 at both 2 h and 3 h post-exercise [41], and another reported an increase in UFPM to be associated with a decrease in FeNO [24].

For pre- to post-exercise comparisons of lung function and airway inflammation (Table 2), in most cases the outcome measurements were obtained from only one exercise trial [24, 38–40, 42–55], whereas others reported the average values produced from a range of 2 to 7 sessions [37, 56, 57]. For the HP conditions, nine out of the 13 studies that analysed the change in health outcomes post-exercise compared to pre-exercise, reported significant change [40, 43, 44, 46, 48–50, 54, 56]. Within those nine, three of them reported a significant decrease in FEV_1 [40, 43, 54], four reported a decrease in FVC [40, 49, 54, 56], two reported a decrease in $FEF_{25–75}$ or MMEF [43, 54] (refers to the same measurement), and two reported a decrease in FeNO [44, 52]. In contrast, two of these studies reported a significant increase in FEV_1 following exercise [46, 48], two reported an increase in $FEF_{25–75}$ [48, 50], and one reported an increase in FEV_1/FVC [48].

Out of 13 studies that used pre- to post-exercise comparison of health outcomes, 10 used a LP condition [40, 41, 43, 45, 46, 48–51, 54]. Of these, one study reported a decrease in FeNO [41], and another that included asthmatic participants exclusively reported a decrease in FEV_1 and FVC [40]. However, one study reported an increase in FEV_1 and FVC following exercise in a LP condition [49]. The remaining seven studies reported no significant differences in lung function or FeNO following exercise in a LP condition [43–46, 50, 51, 54].

The length of time between exercise cessation and post-exercise health measurements being obtained were reported yet varied between studies. Conversely, the frequency of post-exercise measurements across the studies varied with almost half of the studies including one post-exercise measurement [38, 39, 42–44, 49–51, 54, 56], ranging from

Table 2 Respiratory responses to exercise and air pollution

Author & year	Number of sessions per participant	Timing of outcome measurements	Δ in main outcomes following exercise in HP environment (if $P < 0.05$)	Δ in main outcomes following exercise in LP environment (if $P < 0.05$)	Evidence of interaction
Cole et al. (2018) [50]	1 in each exposure	Pre: 1 h before exercise Post: 15 min after exercise	$FEF_{25–75\%}$: + 110 mL/s (significance level not reported)	NSD	No
Cole-Hunter et al. (2013) [46]	1 in each exposure	Pre: Immediately before exercise Post: i. Immediately after exercise ii. 3 h after exercise	NSD	NSD	Not reported
Elliott and Loomis (2020) [55]	1 in one of the 5 bicycle routes	Pre: Before exercise Post: i. Immediately after exercise ii. 2 h after exercise	NSD	N/A (results combined for all 5 bicycle routes)	Peak exposure to PM_{10} was associated with decreases FEV_1^{**}, FVC^{**}, and $PEFR^{*}$
Girardot et al. (2006) [39]	1	Pre: 09:00–12:00 Post: 14:00- 19:00 (within 20 min of participant return to base)	Not reported	N/A	No
Guo et al. (2023) [53]	1 sedentary and 1 active session in the same environment	Pre: Before exercise Post: i. Immediately after exercise ii. 3 h after exercise iii. 5 h after exercise iv. 24 h after exercise	Not reported	Not reported	Increased PM_1 was associated with declines in FVC^{*}, FEV_1^{*}, FEF_{25}^{*}, FEF_{50}^{*}, and FEF_{75}^{*}. Increased BC was associated with declines in FVC^{*}, FEV_1^{*}, FEF_{25}^{*}, and $FEF_{25–75}^{*}$, $PM_{2.5}^{*}$ PM_{10}^{*} were associated with decreases in FEF_{25} and $FEF_{25–75}$. Increased NO_2 was associated with decreases in FVC^{*}, FEV_1^{*}, FEF_{25}^{*}, and FEF_{75}^{*}. And increased O_3 was associated with declines in FVC^{*}, FEV_1^{*}, and FEF_{75}^{*}
Habre et al. (2018) [42]	1 in each exposure	Pre: Before exercise Post: After exercise	Not reported	Not reported	Increased BC associated with a decline in FVC^{**}. And increasing $PM_{1,}$ $PM_{2.5,}$ PM_4 and PM_{10} exposures were associated with a decrease in predicted FEV_1^{**}
Jacobs et al. (2010) [44]	1 in each exposure	Pre: Before exercise Post: 30 min after exercise	FeNO: – 4.4%*	NSD	No

Table 2 (continued)

Author & year	Number of sessions per participant	Timing of outcome measurements	Δ in main outcomes following exercise in HP environment (if $P < 0.05$)	Δ in main outcomes following exercise in LP environment (if $P < 0.05$)	Evidence of interaction
Jarjour et al. (2013) [45]	1 in each exposure	Pre: Before exercise Post: i. Immediately after exercise ii. 4 h after exercise cessation	NSD	NSD	No
Korrick et al. (1998) [38]	1	Pre: 08:00–10:30 Post: 15:00–19:30 (90% of post hike tests were obtained withing 25 min of completion of the hike)	Not reported	N/A	Increased O_3 associated with declines in FVC* and FEV_1*. $PM_{2.5}$ associated with declines in FEV_1*, FVC** and PEFR*
Kubesch et al. (2015) [24]	1 in each exposure	Pre: Before exercise Post: i. 30 min after exercise ii. 2 h after exercise (lung function only) iii. 3 h after exercise (FeNO only) iv. 6 h after exercise	FEV_1: +34 ml** FVC: +29 ml** $FEF_{25–75\%}$: +91 ml*	FEV_1: +34 ml** FVC: +29 ml** $FEF_{25–75\%}$: +91 ml*	Increased PM_{10}* and $PM_{2.5}$* are associated with reduced FEV_1/FVC. Increased UFP associated with reduced FeNO*
Lammers et al. (2020) [56]	The mean of pre and post measurements of 2–5 occasions	Pre: Before exercise Post: After exercise	FVC: decrease* (values not reported)	N/A	Total PNC was associated with declines in FVC*
Matt et al. (2016) [48]	1 in each exposure	Pre: At 06:45 Post: i. Immediately after exercise ii. 7 h after exercise	FEV_1: +1.3%* $FEF_{25–75\%}$: +2.8%* FEV_1/FVC: +0.64%**	FEV_1: +1.3%* $FEF_{25–75\%}$: +2.8%* FEV_1/FVC: +0.64%**	Increased PM_{10} was associated with declines in FEV_1* and FVC*, immediately post-exercise
McCreanor et al. (2007) [40]	1 in each exposure	Pre: Before exercise Post: i. PEFR measured for 1 week after each exposure (by participants) ii. FeNO and spirometry measured regularly for 5 h after exercise iii. 22 h after exercise	FEV_1: – 6.1%* FVC: – 5.4%*	FEV_1: – 1.9%* FVC: – 1.6%*	Increased UFPM were associated with declines in FEV_1*
Mirowsky et al. (2015) [47]	1 in each exposure	Pre: Before exercise Post: i. After exercise ii. 24 h after exercise	Not reported	Not reported	No

Table 2 (continued)

Author & year	Number of sessions per participant	Timing of outcome measurements	Δ in main outcomes following exercise in HP environment (if $P < 0.05$)	Δ in main outcomes following exercise in LP environment (if $P < 0.05$)	Evidence of interaction
Moshammer et al. (2019) [52]	1 in each exposure	Pre: Before exercise Post: i. Immediately after exercise ii. 1 h after exercise iii. 24 h after exercise	Values not reported	Values not reported	Increased PM_{10} was associated with declines in PEF**, MMEF**, MEF 75**, MEF 50**, and MEF 25* immediately after exercise. FEV_1**, MMEF**, MEF 75*** and MEF 50** 1 h after exercise. And FEV_1*, MMEF**, MEF 50** and MEF 25*** 24 h after exercise Increased PM_1 was associated with declines in PEF*, MMEF**, MEF 75**, MEF 50**, and MEF 25** immediately after exercise. FEV_1**, MMEF**, MEF 75**, MEF 50**, and MEF 25* 1 h after exercise. And FEV_1**, MMEF***, MEF 50***, and MEF 25*** 24 h after exercise Increased PN was associated with declines in MMEF**, MEF 50**, and MEF 25** immediately after exercise. FEV_1*, MMEF**, MEF 50*** and MEF 25* 1 h after exercise. And FVC*, FEV_1*, and MEF 50** 24 h after exercise
Park et al. (2017) [49]	1 in each exposure	Pre: Before exercise Post: Immediately after exercise	FVC: -0.12 ± 0.33 L**	FVC: $+0.14 \pm 0.31$ L** FEV_1: $+0.11 \pm 0.18$ L**	Increased UFPM was associated with declines in FVC**
Rundell et al. (2008) [43]	1 in each exposure	Pre: Before exercise Post: 30–45 min after exercise	FEV_1: decrease** (values not reported) $FEF_{25-75\%}$: decrease** (values not reported)	NSD	Increased PM_1 associated with decreased FEV_1**, and FEF_{25-75}**
Strak et al. (2010) [57]	The mean of more than 1 session in each exposure (exact number not specified)	Pre: Before exercise Post: i. Pulmonary function measured immediately after exercise ii. FeNO measured 6 h after exercise cessation	Not reported	Not reported	No

Table 2 (continued)

Author & year	Number of sessions per participant	Timing of outcome measurements	Δ in main outcomes following exercise in HP environment (if $P < 0.05$)	Δ in main outcomes following exercise in LP environment (if $P < 0.05$)	Evidence of interaction
Strak et al. (2012) [37]	Average of 5.5 sessions across the exposures (range = 3–7 sessions per participants)	Pre: i. Before exercise ii. Before exercise at sampling location Post: i. Immediately after exercise ii. 2 h after exercise iii. "The next morning"	Not reported	Not reported	PNC was positively associated with FeNO at all post time points and decreases in FVC at immediately after exercise and the next morning NO_2 and NO_x were associated with decreased FVC at all post time points. And NO_x alone was associated with a decrease in FEV_1 2 h after exercise and the next morning
Wagner and Clark (2018) [51]	1 in each exposure	Pre: Before exercise Post: Immediately after exercise	NSD	NSD	Not reported
Weichenthal et al. (2011) [41]	1 in each exposure	Pre: i. Before exercise ii. Before exercise after 30 min of rest Post: i. Immediately after exercise ii. 1 h after exercise iii. 2 h after exercise iv. 3 h after exercise	NSD	FeNO, 1 h after exercise: – 1.9 ppb*	Increased $PM_{2.5}$ was associated with increased FeNO* 2 h after exercise. Increased UFP were associated with an increase in $FEF_{25–75}$* 1 h after exercise. Increased NO_2 was associated with increased FEV_1 at both 2 h* and 4 h* after exercise
Zhu et al. (2023) [54]	1 in each exposure	Pre: Before exercise Post: Immediately after exercise	FVC: – 8 m/L* FEV_1: – 7 m/L* MMEF: – 8 m/L*	NSD	Increased TRAP was associated with declines in FEV_1* and MMEF*. Increased NO_2 was associated with declines in FEV_1*, FEV_1/FVC* and MMEF*

AQI air quality index, $FEF_{25–75\%}$ forced expiratory flow at 25–75% of forced vital capacity, *FeNO* fractional exhaled nitric oxide, FEV_1 forced expiratory volume in the first second, *FVC* forced vital capacity, *HP* higher pollution, *LP* lower pollution, *MEF* mid expiratory flow, *MMEF* maximum mid expiratory flow, NO_2 nitrogen dioxide, NO_X nitrogen oxides, *NSD* no significant difference, O_3 ozone, *PEF* peak expiratory flow, *PEFR* peak expiratory flow rate, PM_1 ultrafine particulate matter, PM_{10} coarse particulate matter, $PM_{2.5}$ fine particulate matter, *PNC* particulate number counts, *TRAP* traffic related air pollution, *UFP* ultrafine particles, *UPFM* ultrafine particulate matter

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

immediately after to 45 min after exercise. The remainder of the studies measured health outcomes at additional time points at 1 [40, 41], 2 [24, 37, 41, 55], 3 [40, 41, 46, 53], 4 [24, 45], 5 [40, 53], 6 [24, 57], 7 [48], 20 [40], and 24 [47, 52, 53] h after exercise cessation.

Measured air pollution

Measured air pollutants and concentrations are presented in Table 3. Particulate matter was measured in all but one [46] of the studies and therefore is a consistent theme throughout this review. More specifically, UFPM was measured in eight of the studies [24, 40, 41, 44, 45, 48, 49, 54], five measured PM_1 [42, 43, 50, 52, 53], $PM_{2.5}$ was measured in 16 studies [24, 37–42, 44, 45, 47, 48, 50–54], and PM_{10} was measured in 13 studies [24, 37, 40, 42, 44, 46–48, 50, 52, 53, 55, 57]. Eight studies included the measurement of O_3 [37–39, 41, 42, 47, 53, 56], eight measured black carbon (BC) [24, 41, 45, 47, 48, 53, 54, 56], one reported measurements of NO [48], six measured NO_2 [37, 40, 41, 53, 54, 56], NO_X was reported in three studies [24, 37, 48], and some reported PNC [37, 46, 50, 52, 56, 57].

Air quality data were obtained using transportable devices [24, 37, 40–42, 44–47, 49, 50, 52–55] or fixed air quality sensors close to the site of exercise [38, 39, 43, 56]; in some studies however, the placement of the air quality sensor(s) was not clear [48, 57], and in one study pollution levels were taken from a state government website [51]. Table 4 presents studies as those that do and do not report associations, and whether they exceed the 2021 WHO 24 h threshold for pollutant concentrations.

Discussion

The evidence from the 22 included studies was synthesised to investigate the combined effect of ambient air pollution and exercise on parameters of lung function and fractional exhaled nitric oxide (FeNO), as a marker of airway inflammation. To our knowledge, this review is the first to (i) systematically appraise the influence of the principles of exercise prescription on the acute responses to exercise in the presence of air pollution, and (ii) reference pollution concentrations reported within those studies against the 2021 24 h WHO global thresholds.

Air pollution concentrations and physiological responses

As explained previously, we have categorised the exercise conditions using "higher pollution" and "lower pollution" where possible as intended by the authors of the original research, to keep the findings clear and accessible when reviewing the collection of studies. Using those classifications, declines in lung function and/or increases in airway inflammation were generally greater following exercise in higher compared to lower pollution conditions. The extent of these changes appeared to be greater in those that included asthmatic participants, although this is based on a limited number of studies. To provide consistent and objective comparisons, as per the aim of this review, when referencing the pollution concentrations against the 2021 24 h WHO Air Quality Guidelines (Table 4), we see a generally clear pattern when using the thresholds for both $PM_{2.5}$ and PM_{10}. These thresholds are often exceeded by the studies reporting associations between air pollution and lung function and/or airway inflammation [24, 37, 38, 40, 42, 44, 48, 52–54]. There is not such a clear pattern in those seven studies that do not report associations between air pollution and lung function and/or airway inflammation, as three [39, 47, 57] of the seven studies do show either $PM_{2.5}$ or PM_{10} above the 24 h threshold. However, it is worth noting the WHO threshold is only marginally exceeded in two [39, 57] of these three cases, by less than 1 $\mu g/m^3$.

The magnitude of change in pollutant concentration associated with a change in a lung function or FeNO are not consistent, which may be due to varied participant characteristics and underlying respiratory health at the point of study participation, and mixed relationships between individual pollutants and respiratory function [12]. Regression analyses do provide some context between studies and enable some comparison of existing evidence, but it is not uncommon to have a change in pollutant amount that is many multiples of the WHO threshold, associated with a change in a lung function or FeNO. For example, the interquartile range for $PM_{2.5}$ in one study [53], at 86.2 $\mu g/m^3$, is more than five times the 24-h threshold [27]. This 86.2 $\mu g/m^3$ was significantly associated with a 8.67% reduction in $FEF_{25\%}$. Whilst this reduction may be clinically meaningful, the interquartile range of pollutant concentration is likely never experienced by populations in many locations. For example, the peak $PM_{2.5}$ concentration seen in the city centre of London at 76.1 $\mu g/m^3$ [40] is lower than that interquartile range.

Whilst this is not a criticism of previous work using regression analysis, it will likely benefit the evidence if we refer to consistent thresholds, to enhance the ecological validity and practical meaning of what is being reported. National guidelines are important, and therefore should be reported, but the WHO Air Quality Guidelines are available as universal global thresholds in addition to those in each nation or region. In this review we have made comparisons against the 24 h thresholds as the included studies are cross-sectional in design, and therefore focus on the acute physiological responses to air pollution exposure. If future research includes more longitudinal and/or intervention methods, then there may be a debate as to when annual

Table 3 Measured air pollutants

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ± SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
Cole et al. (2018) [50]	Vancouver, Canada (residential route and downtown route) For PNC: P-Trak Ultrafine Particle Counter 8525 (TSI Inc., Shoreview, Minnesota, USA) For PM: GRIMM Dust Monitor, Model 1.108 (GRIMM Technologies, Inc. Douglasville, Georgia, USA)	**PM_{10}:** HP = 13 ± 7.3 µg/m^3; LP = 9.9 ± 5.6 µg/m^3 **$PM_{2.5}$**: HP = 7.3 ± 5.3 µg/m^3; LP = 5.8 ± 3.7 µg/m^3 **PM_1**: HP = 5 ± 4.2 µg/m^3; LP = 3.8 ± 2.9 µg/m^3 **PNC**: HP = 16,226 pt/cm^3; LP = 9,367 pt/cm^3	Not specified
Cole-Hunter et al. (2013) [46]	Brisbane, Australia For PNC: Aerasense NanoTracer (Philips, The Netherlands)	**PNC:** HP "Inbound" = 33,000 ± 15,700 pt/cm^3 and "Outbound" = 26,000 ± 13,500 pt/cm^3 **PNC:** LP = "Inbound" = 19,900 ± 12,000 pt/cm^3 and "Outbound" = 18,400 ± 8,400 pt/cm^3	HP = 13.3 ± 2.68 LP = 13.12 ± 2.7
Elliott and Loomis (2020) [55]	Reno, Nevada, USA For PM_{10}: Active personal particulate photometric monitor, MIE pDR-1500 (Thermo-Scientific, Massachusetts, USA)	**PM_{10}:** 5.66 µg/m^3 (*average of all routes)*	Not specified
Girardot et al. (2006) [39]	Great Smoky Mountains National Park, USA For $PM_{2.5}$: β-attenuation filter-based mass monitor (E-BAM; Met One Instruments, Grants Pass, Oregon, USA) For O_3: Ultraviolet-absorption-based model 202 (2B Technologies, Boulder, Colorado, USA)	**$PM_{2.5}$**: 15.0 ± 7.4 µg/m^3 **O_3:** 48.1 ± 12.0 ppb	20.4 ± 4.2
Guo et al. (2023) [53]	Beijing, China For PM: GRIMM Dust Monitor, Model 11-D Portable aerosol particle size spectrometer (GRIMM Technologies, Hamburg, Germany) For BC: MicroAeth Model AE51 Aethalometer (AethLabs, San Francisco, California, USA) For NO_2 and O_3: Electrochemical sensor-based monitor, Sniffer4D (Soarability Technologies Co., Ltd., Shenzhen, China)	**PM_{10}:** 122.8 ± 109 µg/m^3 **$PM_{2.5}$:** 59.4 ± 45.1 µg/m^3 **PM_1:** 38.8 ± 29.2 µg/m^3 **BC:** 1.94 ± 1.17 µg/m^3 **NO_2:** 59.5 ± 26.6 µg/m^3 **O_3:** 74 ± 30.3 µg/m^3	10.7 ± 8.8
Habre et al. (2018) [42]	Los Angles, USA HP = LAX Airport LP = Jesse Owens Park For PM: DRX 8534 (TSI Inc. Shoreview, Minnesota, USA) For BC: BC Aethalometer AE51 (Magee Scientific, Portland, Oregon, USA) For O_3: Model 205 (2B Technologies, Boulder, Colorado, USA)	**PM_{10}:** HP = 32.6 ± 28.7 µg/m^3; LP = 27.4 ± 12.3 µg/m^3 **$PM_{2.5}$:** HP = 13.7 ± 8.8 µg/m^3; LP = 10.1 ± 5.8 µg/m^3 **PM_1:** HP = 5.5 ± 4.2 µg/m^3; LP = 3.9 ± 2.7 µg/m^3 **BC:** HP = 631.9 ± 322.9 ng/m^3; LP = 410.4 ± 207.3 ng/m^3 **O_3:** HP = 46.7 ± 16.7 ppb; LP = 44.9 ± 12 ppb	HP = 27.7 ± 2.8 LP = 26.3 ± 2.5

Table 3 (continued)

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ± SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
Jacobs et al. (2010) [44]	Antwerp, Belgium For PM: GRIMM Dust Monitor, Model 1.108 (GRIMM Technologies, Hamburg, Germany) For UFPM: P-Trak Ultrafine Particle Counter 8525 (TSI Inc., Shoreview, Minnesota, USA)	**PM_{10}:** HP = 62.8 ± 23.6 μg/m^3; FA = 7.6 ± 3.3 μg/m^3 **$PM_{2.5}$:** HP = 24.2 ± 8.7 μg/m^3; FA = 2 ± 7.8 μg/m^3 **UFPM:** HP = 28,867 ± 8,479 pt/cm^3; FA = 496 ± 138 pt/cm^3	HP = 15.2 ± 1.6 FA = 21.6 ± 1
Jarjour et al. (2013) [45]	Berkeley, Northern California, USA For $PM_{2.5}$: DustTrak TSI Model 8520 (TSI Inc. Shoreview, Minnesota, USA) For UFPM: Condensation particle counter CPC Model 3007 (TSI Inc. Shoreview, Minnesota, USA) For BC: Microaethelometer, Model AE-51 (Magee Scientific, Berkeley, California, USA) For CO: Q-Trak TSI Model Veloci Calc Q-Trak7565 (TSI Inc. Shoreview, Minnesota, USA)	**$PM_{2.5}$:** HP = 4.53 ± 0.3955μg/m^3; LP = 4.88 ± 0.455μg/m^3 **UFPM:** HP = 19,945 ± 2,023 pt/cm^3; LP = 13,517 ± 826.28 pt/cm^3 **BC:** HP = 2.1 ± 0.15 μg/m^3; LP = 1.73 ± 0.9 μg/m^3 **CO:** HP = 0.93 ± 0.04 ppm; LP = 0.77 ± 0.03 ppm	Not specified
Korrick et al. (1998) [38]	Mt. Washington, New Hampshire, USA For $PM_{2.5}$: Teflon filter with a 10 l/min Harvard Impactor (Air Diagnostics and Engineering, Inc., Harrison, Maine, USA) For O_3 at base: Ultraviolet photometric O_3 analyzer, Model 49–100 (Thermo Environmental Corp., Franklin, Massachusetts, USA) For O_3 at summit in 1991: Chemiluminescent O_3 analyzer, Model 8410-E Ozone Analyzer (Monitor Labs, San Diego, California, USA) For O_3 at summit in 1992: Ultraviolet photometric O_3 analyzer, Model 1008-RS (Dasibi Environmental Corp., Glendale, California, USA)	**$PM_{2.5}$:** 15 ± 13 μg/m^3 **O_3:** 40 ± 12 ppb	17 ± 3 (range 8–25) measured at the base of the mountain 8 ± 3 (range – 2 to 16) at the summit
Kubesch et al. (2015) [24]	Barcelona, Spain For PM: Harvard Impactor (Air Diagnostics and Engineering, Inc., Harrison, Maine, USA) For UFPM: Optical particle counter CPC 3007 (TSI Inc. Shoreview, Minnesota, USA) For BC: portable aethalometer (Magee Scientific, Berkeley, California, USA) For NOx: NO_x analyser (2B Technologies, Boulder, Colorado, USA)	**PM_{10}:** HP = 129.68 μg/m^3 (117.16–142.20); LP = 67.79 μg/m^3 (55.48–80.10) **$PM_{2.5}$:** HP = 80.76 μg/m^3 (72.97–88.56); LP = 30.03 μg/m^3 (22.37–37.70) **UFPM:** HP = 164,464.3 pt/cm^3 (150,252.8–178,675.8); LP = 32,992.75 pt/cm^3 (19,020.1–46,965.39) **BC:** HP = 60.82 μg/m^3 (55.29–66.34); LP = 8.59 μg/m^3 (3.16–14.02) **NO_x:** HP = 722.18 ppb (665.31–779.04); LP = 71.62 ppb (15.71–127.53)	HP = 22.28 (20.83–23.72) LP = 20.78 (19.36–22.20)

Table 3 (continued)

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ± SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
Lammers et al. (2020) [56]	Schiphol, Amsterdam, Netherlands For PM: Tapered Element Oscillating Microbalance (TEOM) Series 1400a Ambient Particulate Monitor, Rupprecht & Patashnick, Teflo 2.0 µm 47 mm, R2PJ047 (PALL Life Sciences, Port Washington, New York, USA) For PM size distribution: Scanning mobility particle sizer (SMPS) TSI Model 3936, using a Model 3080 Electrostatic Classifier with a "Long-DMA" model 3081 and a Nano water-based TSI Model 3788 CPC. Particle size range between 6 (d_{50}) and 225 nm (TSI Inc. Shoreview, Minnesota, USA) For PNC: Condensation particle counter (CPC) water-based Model 3752, TSI with a d_{50} of 4 nm as lower size limit (TSI Inc. Shoreview, Minnesota, USA) For BC: Optically absorbing suspended particulates in a gas colloid stream using a aethalometer: microAeth® Model AE51, ETS (AethLabs, San Francisco, California, USA) For NO_2: Chemiluminescence Nitrogen Oxides Analyzer, model 200E (Advanced Pollution Instrumentation, San Diego, California, USA) For O_3: UV photometric analyzer, model 49 (Thermo Environmental Corp., Franklin, Massachusetts, USA)	*(Range values in parentheses* **PM:** 23.1 µg/m^3 (10.6–47.5) **PNC:** 53,500 pt/cm^3 (10,500–173,200) **BC:** 0.6 µg/m^3 (0.12–1.94) **NO_2:** 28.2 µg/m^3 (12.4–60.2) **O_3:** 35.7 (8.8–78.6)	23.3 (range = 15.7–28.6)
Matt et al. (2016) [48]	Barcelona, Spain For PM: DustTrack, DRX, Model 8534 (TSI Inc. Shoreview, Minnesota, USA) For UFPM: Condensation particle counter CPC, Model 3007 (TSI Inc. Shoreview, Minnesota, USA) For BC: Portable aetholometer, Model AE-51 (Magee Scientific, Berkeley, California, USA) For NOx and NO: Nitric oxide monitor, Model 410 Nitric Oxide Monitor in combination with a NO_2 converter, Model 401 NO_2 Converter (2B Technologies, Colorado, USA)	**PM_{10}:** HP = 123 µg/m^3 (99–146); LP = 65 µg/m^3 (37–92) **$PM_{2.5}$:** HP = 82 µg/m^3 (72–92); LP = 39 µg/m^3 (30–48) **UFPM:** HP = 164,708 pt/cm^3 (147,317–182,099); LP = 45,992 pt/cm^3 (32,608–59,376) **BC:** HP = 28.9 µg/m^3 (24–33); LP = 6.9 µg/m^3 (3–11) **NO_X:** HP = 685 ppb (555–815); LP = 102 ppb (61–143) **NO:** HP = 593 ppb (472–714); LP = 77 ppb (41–113)	HP = 11 (10–13) LP = 12 (10–14)

Table 3 (continued)

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ± SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
McCreanor et al. (2007) [40]	London, UK HP = Oxford Street LP = Hyde Park For $PM_{2.5}$: Quartz-fibre filters to determine mass concentration gravimetrically, then analysed for elemental carbon according to National Institute for Occupational Safety and Health guidelines (method 5040; Sunset Laboratory) For UFPM: Real-time condensation particle counter, Model 3007 (TSI Inc. Shoreview, Minnesota, USA) For NO_2: C_{18} Sep-Pak cartridges coated with potassium hydroxide and triethanolamine and subsequently analysed using ion chromatography	*Median values, with range in parentheses* **PM_{10}:** HP = 125 µg/m^3 (62–161); LP = 72 µg/m^3 (60–100) **$PM_{2.5}$:** HP = 28.3 µg/m^3 (13.9–76.1); LP = 11.9 µg/m^3 (3–55.9) **UFPM:** HP = 63.7 thousands/cm^3 (39.5–92.4); LP = 18.37 thousands/cm^3 (10.3–37.1) **NO_2:** HP = 142 µg/m^3 (10.7–289); LP = 21.7 µg/m^3 (2.4–146)	HP = 10.8 (4–17.1) LP = 9.1 (2.5–17.2)
Mirowsky et al. (2015) [47]	North New Jersey & Southern New York, USA HP = George Washington Bridge MP = Garden State Parkway LP = Sterling Forest For PM: Aerotec-2 cyclone, with Teflon filter and quartz filters (Pall Corporation, Ann Arbor, Missouri, USA) For BC: Real-time micro-Aethalometer (microAeth model AE51, Magee Scientific, Berkeley, California, USA) For O_3: Advanced Sense Analyzer (GrayWolf Sensing Solutions, Shelton, Connecticut, USA)	*Range values in parentheses* **PM_{10}:** HP = 38 µg/m^3 (21–50); MP = 26 µg/m^3 (17–48); LP = 16 µg/m^3 (6–29) **$PM_{2.5}$:** HP = 31 µg/m^3 (11–45); MP = 21 µg/m^3 (9–50); LP = 13 µg/m^3 (7–24) **BC:** HP = 7.2 µg/m^3 (4.1–10.9); MP = 2.8 µg/m^3 (1.3–4.1); LP = 1.5 µg/m^3 (0.2–3.6) **O_3:** HP = 0.04 ppm (0.02–0.06); MP = 0.04 ppm (0.01–0.06); LP = 0.04 ppm (0.02–0.06)	HP = 23.8 (21.1–32.8) MP = 23.8 (18.9–30.6) LP = 21.7 (10.0–27.2)
Moshammer et al. (2019) [52]	Vienna, Austria HP = Hernalser Gürtel–busy road route LP = Augarten–park route For PM: Fixed monitor operated by the City of Vienna (Station near the General Hospital: AKH)	**PM_{10}:** 28 ± 26.5 µg/m^3 **$PM_{2.5}$:** 38.7 ± 43.5 µg/m^3 **PM_1:** 31 ± 38.9 µg/m^3 **PN:** 21,347.8 ± 18,826.5/cm^3	Not specified
Park et al. (2017) [49]	Sacramento/Davis region of California, USA For UFPM: Condensation particle counter, Model 3007 (TSI Inc. Shoreview, Minnesota, USA)	**UFPM:** HP = 49,369 ± 11,812 pt/cm^3; LP = 17,474 ± 6,600 pt/cm^3	Not specified
Rundell et al. (2008) [43]	Scranton, Pennsylvania, USA LP = inner campus free of traffic HP = soccer field 20-50 m away from highway PM_1: CPC, P-Trak Ultrafine Particle Counter, Model 8525 (TSI Inc. Shoreview, Minnesota, USA)	**PM_1:** HP = 252,290 ± 77,529 pt/cm^{-3}; LP = 7,382 ± 1,727 pt/cm^{-3}	Not specified

Table 3 (continued)

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ± SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
Strak et al. (2010) [57]	Utrecht, Netherlands For PNC: Condensation particle counter, TSI Model 3007 (TSI Inc. Shoreview, Minnesota, USA) For PM_{10}: Particles collected on Teflon filters (37 mm diameter, 2 mm pore size, PVC support ring (Pall Life Sciences, Port Washington, New York, USA). To determine particle mass, filters were pre- and post-weighed in a climatised room using a Mettler MT5 microbalance (Mettler-Toledo, Greifensee, Switzerland)	**PM_{10}:** HP = 44.01 ± 29.4 µg/m^3; LP = 45.6 ± 28.4 µg/m^3 **PNC:** HP = 44,090 ± 10,036 pt/cm^3**;** LP = 27,813 ± 5,919 pt/cm^3	HP = 13.3 ± 2.68 LP = 13.12 ± 2.7
Strak et al. (2012) [37]	Utrecht, Netherlands For PM: Particles collected on Teflon filters (37 mm diameter, 2 mm pore size, PVC support ring (Pall Life Sciences, Port Washington, New York, USA). To determine particle mass, filters were pre- and post-weighed in a climatised room using a Mettler MT5 microbalance (Mettler-Toledo, Greifensee, Switzerland). The concentration of coarse fraction was calculated as the difference between PM_{10} and $PM_{2.5}$ For PNC: Real-time condensation particle counter, CPC model 3022 A (TSI Inc. Shoreview, Minnesota, USA) For O3: real-time monitors (U.V. Photometric O3 Analyzer model 49 (Thermo Environmental Instruments, Franklin, Massachusetts, USA) For NO_2 and NOx: Chemiluminescence NO/NO_2/NOx Analyzer model 200E (Teledyne API, San Diego, California, USA)	*Range values in parentheses; average of all 5 sites* **PM_{10}:** 76 µg/m^3 (18–450) **$PM_{2.5}$:** 39 µg/m^3 (8–167) **PNC:** 23 10^3/cm^3 (7–74.7) **NO_2:** 20 ppb (9–34) **O_3:** 7 ppb (0.3–32) **NO_x:** 36 ppb (14–96)	Not specified
Wagner and Clark (2018) [51]	Cache Valley, USA For $PM_{2.5}$: Utah Department of Environmental Quality, Division of Air Quality website	**$PM_{2.5}$:** Intended HP = 13–35 µg/m^3; intended LP = 0–12 µg/m^3; measured range for all = 2.1–17.7 µg/m^3; difference = 9.3 ± 3.0 µg/m^3; mean ± SD not reported for HP and LP conditions	4.5 ± 2.7 4.5 ± 2.4 *Not specified which is HP and which is LP*

Table 3 (continued)

First author & year	Location and pollution measuring device	Air pollutant & mean exposure Mean with ±SD, or (95% confidence intervals) *Unless otherwise stated*	Temperature (°C)
Weichenthal et al. (2011) [41]	Ottowa, Canada For $PM_{2.5}$: TSI Dust Trak (TSI Inc. Shoreview, Minnesota, USA) For UFPM: TSI Model 3007 (TSI Inc. Shoreview, Minnesota, USA) For BC: MicroAeth Model AE51 (Magee Scientific, Berkeley, California, USA) For NO_2 and O_3: Fixed site monitoring station in downtown Ottawa (approximately 2 km from the main study site) For SO_2: Not stated For CO: Langan Enhanced CO Measurer Model T15n (Langan Products, San Francisco, California, USA) For VOCs: 1-L SUMMA canisters placed in panniers located on technicians' bicycles travelling directly in front of participants. VOC analysis was conducted by GC–MS according to the United States Environmental Protection Agency method TO-15	*Range values in parentheses* **$PM_{2.5}$:** HP = 12.2 μg/m^3 (3–34); LP = 8.14 μg/m^3 (2.2–26); Indoor = 2 μg/m^3 (1.5–4.1) **UFPM:** HP = 19,747 pt/cm^3 (6834–27,800); LP = 10,882 pt/cm^3 (3590–34,000); Indoor = 1162 pt/cm^3 (413–3210) **BC:** HP = 2520 ηg/m^3 (890–5670); LP = 1079 ηg/m^3 (173–3197); Indoor = 138 ηg/m^3 (1–720) **O_3:** HP = 34 ppb (13–52); LP = 32 ppb (3–51); Indoor = 27 ppb (9–52) **NO_2:** HP = 4.8 ppb (1–11); LP = 4.6 ppb (1–10); Indoor = 4.8 ppb (1–11) **SO_2:** HP = 1 ppb (0–2); LP = 1.2 ppb (0–4); Indoor = 1.1 ppb (0–2) **CO:** HP = 1.4 ppm (0.6–2.6); LP = 0.915 ppm (0.5–1.5); Indoor = 1.08 ppm (0.78–1.6) **VOCs:** HP = 63.8 μg/m^3 (18–246); LP = 33.4 μg/m^3 (15–132); Indoor = 98.7 μg/m^3 (22–204)	16–36 (range)
Zhu et al. (2023) [54]	Shanghai, China For $PM_{2.5}$: MicroPEM (RTI International, North Carolina, USA) For BC: MicroAeth AE51 (AethLabs, San Francisco, California, USA) For NO_2: Dynamic baseline electrochemical sensors, Sapiens PEK Lite A1 (Sapiens Environmental Technology Co. Ltd., Kowloon, Hong Kong) For UFPM: NanoTracer XP (Oxility, Breda, The Netherlands) For CO: Pair differential filter technology, Sapiens PEK Lite A1 (Sapiens Environmental Technology Co. Ltd., Kowloon, Hong Kong)	**$PM_{2.5}$:** HP = 27 ± 19 μg/m^3; LP = 19 ± 9 μg/m^3 **BC:** HP = 4 ± 1μg/m^3; LP = 2 ± 0 μg/m^3 **NO_2:** HP = 44 ± 9 ppb; LP = 14 ± 3 ppb **UFPM:** HP = 33,467 ± 6,678 pt/cm^3; LP = 14,996 ± 4,549 pt/cm^3 **CO:** HP = 948 ± 196 ppb; LP = 333 ± 156 ppb	HP = 22 ± 4 LP = 22 ± 3

BC black carbon, *CO* carbon monoxide, *FA* filtered air, *HP* high pollution, *LP* low pollution, *MP* mid pollution, *NO_2* nitrogen dioxide, *NO_x* nitrogen oxides, *O_3* ozone, *PM_1* ultrafine particulate matter, *$PM_{2.5}$* fine particulate matter, *PM_{10}* coarse particulate matter, *PNC* particulate number counts, *SO_2* sulphur dioxide, *UFPM* ultra-fine particulate matter

Table 4 Air pollution concentrations referenced against WHO thresholds and main conclusions for lung function and/or airway inflammation

First author & year	Mean Concentration Exceeds World Health Organisation 2021 Air Quality Guideline Level? (Yes/No) *BC, NO, NOx, PM1, PNC, UFPM not included in WHO thresholds, CO not included for annual threshold*		Main conclusions
	24-h	Annual	
Studies reporting associations between increasing air pollution and decreased lung function and/or increased airway inflammation			
Elliott and Loomis (2020) [55]	PM_{10} = N	PM_{10} = N	No change in pulmonary function following exercise along either route, however peak PM_{10} exposures were associated with short-term decrements in lung function measures
Guo et al. (2023) [53]	PM_{10} = Y $PM_{2.5}$ = Y NO_2 = Y O_3 = N	PM_{10} = Y $PM_{2.5}$ = Y NO_2 = Y O_3 = Y	Physical activity alleviates the detrimental effect of pollutants on lung function, even in areas of high pollution. Measurements indicative of small airway function increased following exercise, but they did not do so after sedentary time in the same environment. Whilst some lung function measures improved after exercise, increases in PM, BC and O_3 were associated with decreases in several lung function parameters
Habre et al. (2018) [42]	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y O_3 HP = N O_3 LP = N	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y O_3 HP = N O_3 LP = N	Decreases in FEV_1 associated with measured PM, BC and modelled 'traffic exposure'. There were no consistent associations observed with FeNO
Jacobs et al. (2010) [44]	PM_{10} HP = Y PM_{10} FA = N $PM_{2.5}$ HP = Y $PM_{2.5}$ FA = N	PM_{10} HP = Y PM_{10} FA = N $PM_{2.5}$ HP = Y $PM_{2.5}$ FA = N	Decreased FeNO immediately following cycling along HP route but not LP condition. No interaction between Δ FeNO and pollutant exposure. Healthy individuals should not be discouraged from cycling to work even in heavy traffic
Korrick et al. (1998) [38]	$PM_{2.5}$ = Y O_3 = N	$PM_{2.5}$ = Y O_3 = N	Declines in several measures of lung function were associated with low levels of $PM_{2.5}$ and O_3 after prolonged exercise. Participants with asthma had a greater percentage decline in FEV_1 associated with increases in O_3, compared to non-asthmatic counterparts
Kubesch et al. (2015) [24]	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y	Intermittent exercise is more beneficial compared to rest, even in high pollution for healthy participants, as lung function measures increased following exercise. Increasing PM_{10} and $PM_{2.5}$ are associated with attenuated improvements in lung function, however
Lammers et al. (2020) [56]	NO_2 = N O_3 = Y	NO_2 = N O_3 = Y	Increased PNC was associated with decreases in FVC, but no other lung function parameters or FeNO in single pollutant models
Matt et al. (2016) [48]	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y	Physical activity was associated with significant increases in FEV_1, FEV_1/FVC, and $FEF_{25–75\%}$. High TRAP exposure compared to low TRAP exposure attenuated those immediate respiratory benefits after exercise

Table 4 (continued)

First author & year	Mean Concentration Exceeds World Health Organisation 2021 Air Quality Guideline Level? (Yes/No) *BC, NO, NOx, PM1, PNC, UFPM not included in WHO thresholds, CO not included for annual threshold*		Main conclusions
	24-h	Annual	
McCreanor et al. (2007) [40]	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = N NO_2 HP = Y NO_2 LP = N	PM_{10} HP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y NO_2 HP = Y NO_2 LP = Y	Reductions in FEV_1 and FVC were greater after walking in HP compared to LP. The magnitude of decrease in FEV_1 remained different between sites until 22 h post exercise. No differences between changes in $FEF_{25–75\%}$ or FeNO between conditions
Moshammer et al. (2019) [52]	PM_{10} = Y $PM_{2.5}$ = N	PM_{10} = Y $PM_{2.5}$ = Y	PM_{10} exposure was associated with a 5.47 ml/s decrease in PEF for every 1 $\mu g/m^3$ immediately following walking. PM_{10} and PM_1 concentrations were negatively associated most consistently with measurements indicative of small airway function. For some measurements, the significant effect remained persistent for 24 h post-exercise. PM concentrations had a stronger association with changes in lung function compared to the setting (road vs. park). FeNO was reduced immediately following and 1 h after exercise along both routes but increased 24 h after walking beside the road
Park et al. (2017) [49]	n/a	n/a	There were small increases in FVC and FEV_1 following cycling along the LP route, and small increases in FVC and FEV_1 following the HP route
Rundell et al. (2008) [43]	n/a	n/a	Exercise in high, but not low PM was associated with a dose-dependent decrease in FEV_1 and $FEF_{25–75\%.}$ FeNO did not change following exercise compared to baseline in either environment
Strak et al. (2012) [37]	PM_{10} = Y $PM_{2.5}$ = Y NO_2 = Y O_3 = Y	PM_{10} = Y $PM_{2.5}$ = Y NO_2 = Y O_3 = Y	An interquartile increase in PNC (33,000 particles/cm^3) was associated with an 11%, 12% and 7% increase in FeNO compared to baseline, immediately, at 2 h and 24 h after exercise respectively. Several pollutants including PNC, NO_2, NO_X, absorbance associated with changes in FeNO, FVC and FEV_1 at all post-exercise time points indicating airway inflammation and declines in lung function

Table 4 (continued)

First author & year	Mean Concentration Exceeds World Health Organisation 2021 Air Quality Guideline Level? (Yes/No) *BC, NO, NOx, PM1, PNC, UFPM not included in WHO thresholds, CO not included for annual threshold*		Main conclusions
	24-h	Annual	
Weichenthal et al. (2011) [41]	$PM_{2.5}$ HP = N $PM_{2.5}$ LP = N $PM_{2.5}$ Indoor = N O_3 HP = N O_3 LP = N O_3 Indoor = N NO_2 HP = N NO_2 LP = N NO_2 Indoor = N SO_2 HP = N SO_2 LP = N SO_2 Indoor = N CO HP = N CO LP = N CO Indoor = N	$PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y $PM_{2.5}$ Indoor = N O_3 HP = Y O_3 LP = Y O_3 Indoor = N NO_2 HP = N NO_2 LP = N NO_2 Indoor = N	Reduced FeNO in the LP condition, but not in HP. No strong relationships between TRAP and acute changes in lung function. Increased $PM_{2.5}$, associated with increases in FeNO, UFP and NO_2 were associated with decreased lung function
Zhu et al. (2023) [54]	$PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y NO_2 HP = Y NO_2 LP = N CO HP = N CO LP = N	$PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y NO_2 HP = Y NO_2 LP = N	TRAP was associated with decreased lung function. HP compared to LP environment was associated with some lower lung function measures following exercise
Studies reporting no associations			
Cole et al. (2018) [50]	PM_{10} HP = N PM_{10} LP = N $PM_{2.5}$ HP = N $PM_{2.5}$ LP = N	PM_{10} HP = N PM_{10} LP = N $PM_{2.5}$ HP = Y $PM_{2.5}$ LP = Y	No changes in lung function after cycling along a HP or LP route, apart from $FEF_{25–75\%}$ which increased after cycling along the HP route. Cycling either route promotes the benefits of physical activity
Cole-Hunter et al. (2013) [46]	n/a	n/a	PEFR did not change from baseline immediately following or 3 h post-exercise and were not different following exercise in a HP or LP environment
Girardot et al. (2006) [39]	$PM_{2.5}$ = Y O_3 = N	$PM_{2.5}$ = Y O_3 = N	No acute changes in lung function associated with $PM_{2.5}$ or O_3
Jarjour et al. (2013) [45]	$PM_{2.5}$ HP = N $PM_{2.5}$ LP = N CO HP = N CO LP = N	$PM_{2.5}$ HP = N $PM_{2.5}$ LP = N	No changes in lung function at any time point compared to baseline following cycling along the HP or LP route
Mirowsky et al. (2015) [47]	PM_{10} HP = N PM_{10} MP = N PM_{10} LP = N $PM_{2.5}$ HP = Y $PM_{2.5}$ MP = Y $PM_{2.5}$ LP = N O_3 HP = N O_3 MP = N O_3 LP = N	PM_{10} HP = Y PM_{10} MP = Y PM_{10} LP = Y $PM_{2.5}$ HP = Y $PM_{2.5}$ MP = Y $PM_{2.5}$ LP = Y O_3 HP = N O_3 MP = N O_3 LP = N	Change in lung function measurements did not differ between environments. Although insignificant, FeNO increased in the highest pollution environment following exercise, but decreased in the other two environments
Strak et al. (2010) [57]	PM_{10} HP = N PM_{10} LP = Y	PM_{10} HP = Y PM_{10} LP = Y	There were no significant results reported. Weak associations were discussed for UFP with increases in FenO and decreases in lung function 6 h after exercise

Table 4 (continued)

First author & year	Mean Concentration Exceeds World Health Organisation 2021 Air Quality Guideline Level? (Yes/No) *BC, NO, NOx, PM1, PNC, UFPM not included in WHO thresholds, CO not included for annual threshold*		Main conclusions
	24-h	Annual	
Wagner and Clark (2018) [51]	Unable to report (see Table 3)	Unable to report (see Table 3)	FVC, FEV_1 and FeNO were not impacted when exercising vigorously in ambient $PM_{2.5}$ concentrations that corresponded to an AQI of "yellow"

BC black carbon, *CO* carbon monoxide, *FA* filtered air, *HP* high pollution, *LP* low pollution, *MP* mid pollution, NO_2 nitrogen dioxide, NO_x nitrogen oxides, O_3 ozone, PM_1 ultrafine particulate matter, $PM_{2.5}$ fine particulate matter, PM_{10} coarse particulate matter, *PNC* particulate number counts, SO_2 sulphur dioxide, *UFPM* ultra-fine particulate matter

thresholds become more relevant, as studies could use multiple weeks or months of repeated data collection sessions.

Exercise intensity and duration

Generally, there is a lack of consistency amongst the studies for how exercise is described. This makes direct comparisons difficult, which is important given simple exercise characteristics such as intensity and duration will at least partly determine the acute physiological responses to exercise, irrespective of air pollution exposure. Of the seven studies that report no association between air pollution exposure during exercise and changes to lung function and/or FeNO [39, 45–47, 50, 51, 57], exercise was prescribed and/or reported in five different ways. There is more consistency amongst the 15 studies that do report associations [24, 37, 38, 40–44, 48, 49, 52–56], as there are four methods of prescription and/or reporting between them, however one of those is verbal description such as "steady" or "comfortable pace", which is one of the least reliable forms of exercise intensity prescription and monitoring.

There are two studies that use high-intensity exercise, one using 85–90% HRmax [43] and another using a 20 min time trial effort [51]. The former reports reductions in FEV_1 and $FEF_{25–75\%}$ after exercise in their HP condition, whereas the latter reports no difference between acute responses following exercise in their HP and LP conditions. Different pollutants were measured in these two studies, and it is therefore difficult to compare the conditions in which participants exercised in. For those that report intensity clearly as %HRmax, reducing intensity to ≈ 74% HRmax [44] has shown reduced FeNO after exercise in their HP condition, indicating smooth muscle relaxation and reduced airway inflammation [58], taken to be a positive acute response to exercise. Reducing further to ≈ 66% HRmax [38], we see no differences pre- to post-exercise, but do see associations between increased pollutant concentrations and decreased lung function, which would be taken as an adverse acute response. In studies that are firmly placed in the moderate exercise intensity range between 50 and 70% HRmax, there are inconsistent findings with both increases [24, 48] and decreases [56] in lung function following exercise in areas of higher air pollution. An unclear pattern exists across other studies using 50% to 70% HRmax, with increased lung function post-exercise [24, 48], although specific pollutant exposure was associated with reduced lung function in the very same studies, suggesting that whilst exercise is still beneficial in the presence of air pollution, the positive effects can be attenuated as pollution concentrations increase. The average pollutant concentrations for both studies [24, 48] exceed the 24 h WHO thresholds for both $PM_{2.5}$ and PM_{10} by approximately 5-times and 3-times respectively. For those using the lightest intensities (walking) with non-asthmatic healthy participants only, increased air pollution was associated with decreased lung function [52, 54], or no differences or associations found [47].

Regarding duration of exercise, 11 studies involve 2 h or more of exercise (some with interspersed rest), and nine of them show negative interaction between increased air pollution concentration, decreased lung function and/or increased airway inflammation [24, 37, 38, 40, 42, 48, 53, 54, 56] although three of those [38, 40, 42] do include asthmatic participants. And it should also be noted that two [24, 48] show positive, increased lung function following exercise with pre- to post-exercise changes. All nine studies report at least one pollutant (either $PM_{2.5}$, PM_{10} or O_3) above the 24 h WHO threshold. Only two studies, one using 2 h [47] and one using 5 h [39] do not show associations. For those using 1 h or less of exercise (or within 2 to 3 min of 1 h [50]), five report differences and/or interactions [41, 43, 44, 52, 55], and five report no difference and/or interaction [45, 46, 50, 51, 57] between air pollution and lung function and/or airway inflammation. Of interest however, two of the studies showing associations with an hour or less of exercise report what would

be interpreted as positive acute responses, as they show either reduced FeNO [44] or increased lung function [41].

We could therefore draw the conclusion that exercise intensity may not consistently influence the relationship between acute physiological responses following exercise and air pollution concentrations, but it is perhaps best to refrain from definitive statements given the disparity in methods used to prescribe and/or report the intensity of exercise performed. Regarding duration of exercise, of the 11 studies using 2 or more hours of total session time (including intermittent rest), all nine studies that showed differences and/or interactions report what would be interpreted as adverse acute responses, as either increased airway inflammation measured by FeNO, or decreased lung function. Therefore, total duration does appear to influence the acute physiological responses following exercise in areas of higher air pollution. It appears that long duration exercise (or intermittent exercise) in areas of higher pollution is more likely to result in increased airway inflammation and/or decreased lung function compared to shorter duration exercise, irrespective of exercise intensity. Whilst decreases in FEV_1, FVC and PEF have been reported following prolonged periods of exercise [59], it is important to recognise that this is controlled for within this review, as the same duration of exercise was used in both lower and higher pollution concentrations with the same participants in several of the included studies.

These observations are supported by toxicological mechanisms that suggest the total number of particles deposited in the respiratory tract can increase during exercise, as the exhalation of particles is not as efficient during exercise compared to a rested state [14, 60]. Deposition fraction increases from approximately 0.6 at rest to approximately 0.8 during exercise, and 0.9 for the smallest ultrafine particles [14]. In addition, the rapid absorption of particles within the alveolar region is modulated by ventilation volume [61, 62] which will of course be at least partly dictated by duration of exercise; simply, a greater volume of particles are likely to be absorbed into cellular targets in the lung system when the duration of exercise is greater. This stimulates an inflammatory response which may lead to a decline in lung function [61–63].

Considering an increase in tidal volume during exercise, and thus, the likely volume of inhaled pollutants [64], it is logical to assume that a dose-dependent inflammatory response contributed to the acute, generally negative interactions and pre- to- post changes within the current review. Nevertheless, duration of exercise cannot be held solely accountable for the variation in response to exercising in air pollution, as a complex interplay of factors including participant characteristics, intermittent rest periods, exercise intensity and the amount of pollution exposure dictate the combined impact of air pollution and exercise on lung and airway inflammation. This is epitomised in the current review, considering the greatest decline in lung function was observed in a study with asthmatic participants, despite only walking at a "steady pace" [40].

To enhance our ability to compare evidence and maximise practical application of findings, there needs to be a more consistent approach in how the characteristics of exercise are observed or prescribed and reported in the literature. We know the basic principles of exercise prescription including intensity and duration are vital in determining the acute response to exercise [65, 66], irrespective of air pollution exposure. Given the relevance of minute ventilation for the exposure of any air pollutant, this method may be preferred where possible. But, given HR will ordinarily be used as the practical means of prescribing and monitoring exercise intensity in the field, even when minute ventilation is being used as the basis of prescription, it is likely that relative HR (e.g. HRmax) provides the most practicable solution to ensuring consistency across separate pieces of original research. HR, whilst not perfect, is also the most used variable to describe exercise intensity in exercise physiology literature [65, 67], again irrespective of air pollution.

Lung function and fractional exhaled nitric oxide

Decreases in lung function following exercise in air pollution may be explained by impairment of the regular defensive mechanisms of the respiratory tract by stimulating a host of pro-inflammatory mediators including, cytokines, chemokines, and adhesion molecules [68]. The heightened activation of alveolar macrophages following the airway epithelial cells being exposed to pollutants causes the secretion of histamines, leukotrienes and prostaglandins from mast cells, eosinophils, and neutrophils [69]. This inflammatory process causes bronchoconstriction, which is associated with a decrease in lung function, often FEV_1, even in the absence of asthma [70]. However, it should be noted that two studies [24, 48] report increased lung function after exercise in both HP and LP conditions and support the notion that intermittent exercise is beneficial even in higher pollution for healthy participants. In these cases, it is thought that the bronchodilatory effects of exercise are caused by β2-receptor activation by endogenous catecholamines [71]. One of the studies did however state that higher levels of TRAP attenuated the respiratory benefits of exercise [48].

For FeNO, the pre- to post-exercise changes and interactions were inconsistent; this may be partly influenced by the variance in participant and exercise stimulus characteristics, and the concentration of pollutant exposure across the studies within the review. Whilst it is expected that exercise in a polluted environment may cause FeNO to increase [18], of the 12 studies measuring FeNO there were only two

significant interactions reporting that type of relationship [37, 41], one of which included asthmatic participants [41]. Nitric oxide (NO) when elicited from inducible nitric oxide synthase, is suggested to be a key proinflammatory mediator [18]. PM inhalation facilitates the oxidation of L-arginine and resultantly, the production of NO is expected to increase [72]. This process involved in the production of NO under oxidative stress stimulates potent oxidising mediators that may influence the significance of the subsequent inflammatory response [20]. As a reliable marker of eosinophilic inflammation, FeNO measurements are generally greater amongst asthmatics compared to non-asthmatics [73], as seen within the current review.

One other study reported decreasing FeNO as ultrafine particles increased [24]. And pre- to post-decrease in FeNO was reported in two of the studies following a bout of exercise in their HP condition [44], and LP condition [41]. A reduction in FeNO in a HP condition may be explained by particle suppression of NO synthase and the production of peroxynitrate, which can attenuate the vasodilatory effects of NO [43]. This response may explain why Jacobs and colleagues [44] observed decreased FeNO after exercise in their HP condition, which involved pollutant concentrations of approximately 140% for PM_{10}, and 160% for $PM_{2.5}$ of the current WHO 24 h thresholds. Diminished NO availability can cause a sympathetic-induced bronchoconstriction [74], therefore it would have been interesting if Jacobs and colleagues [44] had measured lung function in addition to FeNO and had used additional time points to observe the responses in the hours that followed exercise. In the LP condition, given the indoor environment, and very low pollutant concentrations (e.g. $PM_{2.5} = 2\ \mu g/m^3$) [41], it is more likely the decreased FeNO was due to constitutive nitric oxide synthase aiding smooth muscle relaxation and bronchodilation after exercise had finished. Therefore the fraction of exhaled NO can be temporarily reduced [20, 75].

Lung function outcomes for asthmatic participants

Air pollution has been shown to elicit exacerbations in pre-existing asthma [76–78], and in the development of asthma [78, 79]. Exposure to environments with a high PM for even a transient period leads to a greater fractional deposition of ultrafine particles in asthmatic compared to non-asthmatic individuals [80]. Arguably, it is important to consider individuals with and without asthma separately, due to the altered pathophysiological mechanisms that characterise asthma [81]. These may lead to an underestimation of the extent to which air pollution can impair lung function and exercise capacity for individuals with asthma [82]. The method of reporting the diagnosis and severity of asthma was not consistent, and participant self-reporting was most common [38, 39, 41]. These three studies adjusted for self-reported asthma in the regression models involving all asthmatic and non-asthmatic participants together. Two studies categorised the severity of the condition using mild or moderate asthma and used asthmatic individuals exclusively [40, 42]; therefore, the analysis and findings are clearly based on asthma alone. One of the studies involving participants with asthma exclusively reported the largest reductions in lung function in this review and were more pronounced in those with moderate asthma compared to those with mild asthma [40]. Another controlling for asthma in regression analysis observed a 4-fold greater reduction in FEV_1 in asthmatic participants compared to non-asthmatics after a bout of recreational hiking [38], suggesting the effects of exercising in higher levels of pollution will indeed be exacerbated in asthmatics due to their hypersensitive predisposition [81].

Limitations and future directions

In theory, this topic would be ideal for meta-analysis, however the inconsistent method of measuring and/or reporting exercise intensity, pollution concentration and lung function means this is not possible at this stage. A second limitation is that the methods used to date do not consistently use a control when the experimental conditions are outdoors in ambient air; therefore this requirement was not part of our inclusion criteria.

To provide consistent and comparable methods, future studies should aim to measure exercise intensity using relative HR, either as %HRmax, or corresponding to a desired minute ventilation. In the latter case, HR should still be reported alongside minute ventilation. It is also recommended that pollution concentrations are reported using the WHO guidelines, as it was difficult to provide context for all studies in this review. Use of WHO thresholds will increase the ecological validity of future studies and provide greater context for the findings seen after exercise in different locations.

Third we suggest where health outcomes are measured before and after exercise, as in all the studies in this review, the percentage change from pre- to post-exercise should be reported in addition to any interactions resulting from regression analysis. Whilst reporting interactions between air pollutants and health outcomes are useful, it can be difficult to put this into relative terms when there are such different interquartile ranges across studies. Importantly, much like two studies in this review [24, 48] were able to, reporting the pre- to post-exercise change in health outcomes enables us to see if the benefits of exercise (i.e. positive changes in lung function and/or airway inflammation after exercise) may be attenuated by higher pollutant concentrations. Lastly, it is recommended that more research is conducted using asthmatic participants considering it appears that air pollution inhalation causes greater decreases in lung function in these individuals.

Conclusions

Overall, the beneficial effects of exercise on lung function and airway inflammation appear to be attenuated as pollution concentrations increase. Adverse lung function and FeNO responses were often associated with exercise in conditions with pollutant concentrations exceeding the 2021 24 h WHO Air Quality Guideline thresholds, or those that showed positive lung function responses after exercise report that those beneficial effects may be reduced by increased pollutant exposure. Declines in lung function were most frequently associated with particulate matter of varying aerodynamic diameter. Longer duration exercise (≥ 2 h) was more frequently associated with adverse acute health outcomes despite a lower total session intensity due to interspersed rest periods. A range of exercise intensities were used, although the method of measuring was inconsistent, therefore its potential influence was not as clear. In summary, on the available evidence it would appear that exercise sessions ≥ 120 min in duration in air pollution concentrations that exceed WHO thresholds are more likely to result in reduced lung function. Therefore, recommendations to modify exercise behaviour when high concentrations of air pollution are present should be based on reducing the duration of planned exercise. For example, if an individual were planning to undertake exercise in higher pollution conditions, but the duration of exercise is intended to be short (e.g. <60 min), it may be that the benefits of exercise are likely to outweigh the potential risks of pollution exposure, at least in terms of acute responses. However, should the exercise session last for ≥ 120 min, then it may be that exercising in a lower pollution environment, and/or for a shorter duration in that same environment may reduce the potential negative impact of acute pollution exposure on lung function. However, it should be noted that the evidence available to date often involves pollution concentrations that are multiple times the 2021 WHO thresholds. Therefore, further research is required to better understand the dose–response relationship between pollution concentration and acute physiological response to exercise. This knowledge would help us understand when recommendations about modifying exercise behaviour are necessary as a protective measure.

Author contributions All authors contributed to the study conception and design. Database searching, and initial analysis were performed by Scarlett Moloney and Gavin Devereux. The first draft of the manuscript was written by Scarlett Moloney and Gavin Devereux and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Conflicts of interest The authors declare no competing interests.

Human and animal rights Not applicable (systematic review).

Informed consent Not applicable (systematic review).

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