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Acute effects of short-term exposure to air pollution while being physically active, the potential for modification: A review of the literature

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Abstract

The science behind the combined effect of (and possible interaction between) physical activity and air pollution exposure on health endpoints is not well established, despite the fact that independent effects of physical activity and air pollution on health are well known. The objective of this review is to systematically assess the available literature pertaining to exposure to air pollution while being physically active, in order to assess statistical interaction. Articles published during 2000–2020 were identified by searching PubMed, Science Direct, and ProQuest Agricultural & Environmental Science Database for terms encompassing air pollution and exercise/physical activity. Articles were included if they examined the following four scenarios: at rest in clean air, physical activity in clean air, at rest in polluted air, and physical activity in polluted air. Risk of bias assessment was performed on all included articles. We identified 25 articles for inclusion and determined risk of bias was low to moderate. Nine articles identified evidence of statistical interaction between air pollution exposure and physical activity, while 16 identified no such interaction. However, pollutant levels, exercise intensity, and the population studied appeared to influence statistical interaction. Even in low levels of air pollution, low-intensity activities (i.e., walking), may intensify the negative impacts of air pollution, particularly among those with pre-existing conditions. However, among healthy adults, the review suggests that exercise is generally

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beneficial even in high air pollution environments. Particularly, the review indicates that moderate to high-intensity exercise may neutralize any short-term negative effects of air pollution.

Introduction

Short-term exposure to ambient air pollution is associated with considerable morbidity and mortality.^{1–4} The US Environmental Protection Agency (US EPA) has designated the criteria air pollutants (particulate matter, nitrogen dioxide, sulfur dioxide, carbon monoxide, and ozone) as having causal or likely causal associations with mortality, respiratory outcomes, and cardiovascular outcomes.^{5–9} In particular, short-term exposure to fine particulate matter (PM_{2.5}) and other traffic-related air pollutants (TRAP) may act as a triggering agent for adverse cardiovascular and respiratory health events.^{10–13}

Physical activity is known to be beneficial to general and cardiovascular health.^{14,15} Both the Department of Health and Human Services¹⁶ and the World Health Organization¹⁷ recommend all individuals engage in some form of physical activity, for health benefits. The benefits of physical activity include both short-term benefits such as improved brain health, including reduced short-term feelings of anxiety, and long-term benefits, such as reduced risk of all-cause mortality and cardiovascular disease, among several others.¹⁶

It is well established that physical activity increases the dose of environmental air pollutants that enter the lungs.¹⁸ During physical activity, increases in an individual's ventilation rate increases exposure to harmful air pollutants.^{19–21} As the ventilation rate increases, a larger proportion of air bypasses the nasal passageways, which typically provide protection against particles reaching the lower parts of the lung. Changes in nasal resistance, due to physical activity, can allow particle pollution to reach the lower respiratory tract.²² In healthy subjects, fractional deposition for the total respiratory tract of ultrafine particles increases during periods of exercise compared to rest.²³ Increased deposition of ultrafine particulate matter in the lungs is particularly concerning among persons with pre-existing conditions, such as cardiovascular disease,²⁴ yet physicians often recommend physical activity to high-risk patients, such as those in cardiac rehabilitation.²⁵ Intense physical activity may act as a short-term trigger for cardiovascular events,²⁶ particularly among those with pre-existing conditions.¹³ Since exposure to air pollutants is associated with cardiovascular outcomes and an increased mortality risk,⁵ the potential interactions between air pollution exposure and physical activity on acute health endpoints require evaluation, especially among susceptible subgroups.

In 2010, the Centers for Disease Control and Prevention (CDC) convened a workshop²⁷ to review the state of the science and existing public health guidance that jointly addressed physical activity and outdoor air pollution exposure. The workshop was motivated, in part, by The 2008 Physical Activity Guidelines for Americans, which included the risk-benefit statement, “the benefits of being active, even in polluted air, outweigh the risk of being inactive”.²⁸ Workshop participants, including international experts in the fields of physical activity and air pollution sciences, determined that the science behind the combined effect of (and possible interaction between) physical activity and air pollution exposure on health endpoints was not well established, despite the fact that independent effects of physical

activity and air pollution on health are well known. It was concluded that more interdisciplinary research was needed to expand the evidence base and provide a foundation for future public health guidance that would adequately inform the public, including susceptible subgroups, about the risks and benefits of air pollution exposure while being physically active. Current guidelines, published in 2018, refer readers to the US EPA's Air Quality Index for information about when air conditions are unhealthy for physical activity.¹⁶

In recent years, there have been several studies^{29–38} specifically examining potential interactive effects of air pollution and physical activity on various health endpoints. This review evaluates and summarizes the scientific literature published since the year 2000 pertaining to potential interactive effects between short-term air pollution (<24 hours of exposure) and physical activity exposure on acute health endpoints.

Methods

Following the PRISMA guidelines for systematic reviews,³⁹ we conducted a literature search in PubMed, Science Direct, and ProQuest Agricultural & Environmental Science databases for articles published during 2000–2020. Search terms included air pollution terms joined with an AND operator with physical activity terms (Table S1). We limited our search to include human subjects, peer-reviewed literature, and the English language. The search returned 1,348 articles after duplicates were removed. Articles were screened by S.D.B and K.M.R and selected for further review if the title indicated the study was related to air pollution and health, leaving 125 articles. Next, based on the review of the abstract, articles were retained if physical activity was incorporated into the study design, leaving 75 articles. For final inclusion in this review, the study design had to assess outcomes across all four of the following scenarios in order to address potential statistical interaction: 1) at rest in clean air, 2) physical activity in clean air, 3) at rest in polluted air, and 4) physical activity in polluted air. Without these four conditions, we would be unable to tease apart the independent effects of either physical activity or exposure to polluted air. A total of 25 articles were reviewed based on the literature search and expert recommendations. Twenty-two studies had crossover study designs and three had observational study designs.

We extracted the following data from each reviewed article: location, study population (e.g., healthy, persons with asthma, etc.), sample size, study location, type of physical activity, air pollutant measurements in filtered and unfiltered/polluted air, and the health endpoint measured under the four scenarios.

Articles were assessed for the presence of statistical interaction between air pollution and physical activity. We relied on the reported statistical significance in the few articles that directly measured the interaction but, the primary research aim of most articles was not to evaluate interaction between air pollution and physical activity. In these cases, we evaluated potential interaction by examining the health endpoints in each of the four scenarios. We defined interactions as either synergistic or antagonistic with regards to the effect of air pollution on health endpoints. Synergistic interaction implies the effect of two exposures combined is greater than the sum of their separate effects. We classified articles as having

‘synergistic interaction’ if the combined effect of physical activity in the presence of air pollution was higher than the effect of air pollution and physical activity alone. Alternatively, antagonistic interaction indicates the effect of two exposures combined is less than the sum of their separate effects. We classified articles as having ‘antagonistic interaction’, if the combined effect of physical activity in the presence of air pollution was lower than the effect of air pollution and physical activity alone. Consider the following example, if physical activity in clean air beneficially increases lung function by 10%, compared to baseline, and exposure to air pollution at rest detrimentally decreases lung function by 10% compared to baseline, three potential conclusions regarding interaction can be made based on the findings among those exercising in polluted air. First, if there is a 25% decrease in lung function compared to baseline, this would be considered synergistic interaction. If there is only a 5% decrease in lung function, this would be considered an antagonistic interaction. Lastly, if lung function was not impacted by physical activity while exposed to air pollution, or if there was no association with air pollution at all, then this would be considered a situation in which no interaction is present.

A risk of bias analysis evaluated methodology and study implementation of included articles. The 22 crossover study articles were assessed by following recommendations outlined in the Cochrane Handbook for Systematic Reviews of Interventions for Crossover Studies⁴⁰ and a modified Jadad scale.⁴¹ Risk of bias in the three observational study articles was evaluated based on the Newcastle-Ottawa Scale guidelines for assessing the quality of cohort studies.⁴² Each risk of bias category was assessed and scored as “low risk,” “unclear risk,” or “high risk” of bias by S.D.B. Studies were classified as “unclear risk of bias” if there was no discussion in the article about a specific criterion, or if the study used single, rather than double blinding.

Results

Most studies were conducted in North America and Europe. Six studies occurred in the United States,^{43–48} five in Canada,^{30–34} four in Spain,^{29,35,36,38} three each in Denmark,^{49–51} and Belgium,^{52,53,54} two in the United Kingdom^{55,56} and two^{37,57} were conducted in three different European cities (Antwerp, Belgium; Barcelona, Spain; London, United Kingdom). Three^{29,35,36} of the four studies occurring in Spain were part of the TAPAS (Transportation, Air Pollution, and Physical Activities) Project. Four of the five Canadian studies^{30–33} were from the same research group, and all three studies from Denmark were also part of the same research group.^{49–51} Both studies occurring in Antwerp, Barcelona, and London were part of the PASTA study (Physical Activity through Sustainable Transport Approaches).

Overall, the studies examined a variety of outcomes (Table 1), with several evaluating more than one health endpoint. Twelve studies (48%) measured lung function changes,^{30,31,36–38,43–46,48,56,58} four measured heart rate variability (HRV),^{29,31,37,47} three systemic inflammation,^{33,36,50} three evaluated vascular effects,^{34,48,50} and two examined blood pressure.^{35,57} Lastly, one study each examined either measurements of cardiac stress,⁵³ blood-gas permeability,⁴⁹ endothelial function,³² brain-derived neurotrophic factor (BDNF),⁵² platelet activation,⁵⁴ or oxidative stress-induced DNA damage.⁵¹ The majority of the

studies evaluated (88%) had a crossover study design, with the study size ranging from 10 to 119. Three studies had observational study designs with $n=354$ ⁴⁵ and $n=122$.^{37,57}

Evidence of Interaction

Nine studies (36%) demonstrated evidence of an interaction between air pollution and exercise (Table 1, Table S2).^{29,30,35,37,38,44,52,54,56} Four studies showed evidence of synergistic interaction.^{30,44,54,56} One study, examining the relationship between lung function among adult males ($n=18$) cycling in filtered air and diesel exhaust (DE), observed that low and high intensity cycling were associated with beneficial increases in lung function in filtered air, while exposure to DE was associated with detrimental decreases in lung function. Yet, low intensity cycling in DE was associated with increases in certain lung function parameters that could be detrimental, particularly to those with cardiopulmonary disease.³⁰ Another study, evaluating healthy adults ($n=12$) and adults with asthma ($n=16$), found that cycling was associated with beneficial increases in certain lung function parameters, while exposure to ultrafine particles (UFP) had no evidence of an association with lung function. However, cycling in the presence of UFP was associated with decreased lung function, which was only significant in healthy adults.⁴⁴ A study evaluating the relationship between DE exposure and platelet activation among young healthy male subjects ($n=25$) found that neither exercise or exposure to DE independently had an impact on platelet activation. However, exercise in air polluted with DE was associated with increased platelet activation.⁵⁴ Lastly, a study of adults⁶⁰ evaluated lung function among those who were healthy ($n=40$), had chronic obstructive pulmonary disease (COPD) ($n=40$), or ischemic heart disease (IHD) ($n=39$). Walking along the clean air path showed beneficial increases in lung function compared to baseline (at rest in clean air), while walking along a path with polluted air resulted in worse lung function relative to baseline (at rest in polluted air).⁵⁶ This study met our inclusion criteria, but results were presented relative to baseline, preventing us from being able to directly assess the independent effects of walking and air pollution on lung function within this group.

Five studies showed evidence of antagonistic interaction (Table 1, Table S2).^{29,35,37,38,52} A study examining lung function among healthy adults ($n=30$) showed moderate intensity cycling was associated with increased lung function, while exposure to TRAP was associated with decreased lung function. However, moderate intensity cycling in the presence of TRAP was associated with beneficial increases in lung function among participants, compared to resting in polluted air.³⁸ A longitudinal study of healthy adults evaluated the short-term impacts of black carbon (BC) on HRV and lung function found that BC was significantly associated with decreased lung function, physical activity was associated with beneficial increases in lung function and significant increases in HRV, and physical activity in the presence of BC indicated significant increased lung function.³⁷ Two studies evaluated moderate intensity cycling among healthy adults ($n=28$) as part of the TAPAS Project.^{29,35} The first found moderate intensity cycling was associated with increased HRV, exposure to TRAP was associated with decreased HRV compared to baseline, and cycling in TRAP demonstrated increased HRV compared to baseline.²⁹ The second study from this group had similar conclusions, in that moderate intensity cycling was associated with decreased systolic blood pressure, exposure to TRAP was associated with

increased systolic and diastolic blood pressure compared to baseline, and physical activity in the presence of high TRAP was associated with decreased systolic blood pressure compared to rest in low TRAP conditions.³⁵ Lastly, a study examined the relationship between brain-derived neurotrophic factor (BDNF), a molecule that aids in regulating the proliferation and differentiation of cells in the central nervous system, and cycling in clean air and TRAP, among physically fit adults (n=38).⁵² Increases in BDNF are typically associated with physical activity⁵⁹ and BDNF may play an important role in memory formation and survival and growth of new neurons.⁶⁰ There was no association noted between exposure to TRAP while at rest, but cycling in clean air was associated with a significant increase in BDNF. However, there was no increase in BDNF observed among those cycling in polluted air, indicating that exposure to air pollution negated the positive benefit of physical activity.⁵²

Two studies indicated some presence of interaction, but were not statistically significant (Table 1, Table S3), and thus were classified as having no evidence of interaction.^{36,46} A study from the TAPAS Project, examining the same study population of healthy adults (n=31) as Cole-Hunter et al.²⁹ and Kubesch et al.,³⁵ reported no statistically significant differences in lung function and systemic inflammation whether participants were cycling at moderate intensity in low TRAP versus high TRAP, however, the point estimates show some evidence of antagonistic interaction.³⁶ A second study found decreased lung function following cycling in high concentrations of ultrafine particulate matter (UFP) along a high traffic route, but increased lung function when cycling in lower concentrations of UFP along a low traffic route, implying a potential synergistic interaction, but the difference between the two routes was not statistically significant.⁴⁶

No Evidence of Interaction

Sixteen articles (64%) indicated no evidence of interaction between physical activity and air pollution exposure and found no difference in effect of physical activity whether study participants were exposed to clean or polluted air (Table 1, Table S3).^{31,32,36,43,45–47,49–51,53,58} One study among obese (n=20) and non-obese women (n=20) found similar increases in lung function whether participants were lightly exercising on a treadmill in filtered air or in ozone.⁴³ Another study among male competitive runners (n=10) found neither high intensity running nor exposure to ozone influenced lung function relative to baseline.⁵⁵ A study examining the effects of dobutamine, a drug that mimics the effects of physical activity on the heart, among healthy non-smoking adults (n=10), found no difference in any of the cardiac stress test measurements whether exposed to ambient air or DE.⁵³ Another study of healthy non-smoking adults (n=24) found that, regardless of exposure to either air or UFP, there was no evidence of an association with HRV while cycling, compared to baseline.⁴⁷ One study evaluated vascular function and DE exposure, found no difference in vascular responses whether breathing in filtered air or DE.³⁴ Another study suggested healthy runners experienced more respiratory discomfort in high PM_{2.5} conditions, but there were no differences in lung function.⁴⁸ An observational study of adult hikers (n=354) found exposure to increasing concentrations of PM_{2.5} and ozone had no impact on lung function after hiking compared to pre-hike measurements. However, while this study contained all four scenarios for inclusion, the same group of people were not studied in each scenario.⁴⁵ The same longitudinal study³⁷ of healthy adults in three

European cities (Antwerp, Barcelona, and London) also evaluated short-term impacts of black carbon on blood pressure and found no difference in blood pressure whether active or at rest when exposed to varying levels of BC.⁵⁷ Three studies from Denmark evaluated exposure to DE among healthy adults (n=29).^{50,51} The first suggested exercise in the presence of air pollution does not increase DNA damage, repair activity, and oxidative stress⁵¹ and the second found that neither exercise nor exposure to polluted air had an effect on microvascular function or systemic inflammation.⁵⁰ The third study from this group found increases in blood gas permeability and lung function compared to baseline regardless of cycling in filtered versus non-filtered air.⁴⁹ Three Canadian studies evaluated high and low-intensity cycling among healthy adults exposed to DE^{31–33}, the same which were evaluated by Giles et al.³⁰ One found while there was an increase in throat and chest symptoms when exposed to DE, there was generally no difference in HRV, lung function, and plasma norepinephrine associations with DE exposure at rest compared to either level of physical activity.³¹ The other studies found that DE had no short-term impact on endothelial function³², nor an effect on adhesion molecules or markers of systemic inflammation,³³ whether at rest or during varying intensities of physical activity.

Differences Between Studies

Air pollutant concentrations varied widely from study to study. In many cases, what was considered “low” air pollution exposure in one study was equivalent to or greater than “high” air pollution exposure in another. We classified articles as either “high exposure” or “low exposure”, based on concentrations in the low pollution/filtered air scenarios. We defined a study as having overall “high exposure” if the measured concentrations in the low exposure/filtered air groups were greater than 12 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, 0.07 ppm for ozone (which correspond with the current US EPA’s National Ambient Air Quality Standards⁶¹), or 20,000 particles/ cm^3 for UFP (Table 1). Six studies were classified as “high exposure”^{29,35,36,38,53,54} and the mean concentrations for the low pollution/filtered air group ranged between 13.0–39.0 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, and approximately 32,000–45,000 particles/ cm^3 for UFP, whereas the mean concentrations for the high pollution/non-filtered air scenarios ranged between 80.76–309 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, and 164,000–167,000 particles/ cm^3 for UFP (Table S2, Table S3).^{29,35,36,38,53,54} Most “high exposure” studies occurred in Spain,^{29,35,36,38} while two occurred in Belgium.⁵³ Nineteen articles were classified as “low exposure”; in these studies the mean concentrations for the low pollution/filtered air scenarios ranged between 0–9.3 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, 0–0.001 ppm for ozone and approximately 0–17,000 particles/ cm^3 for UFP, yet the high pollution/non-filtered air scenario ranged between 24.6–302.8 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, 0.01–0.4 ppm for ozone, and 164,000–2,000,000 particles/ cm^3 for UFP (Table S2, Table S3).^{30–34,37,43–52,56–58}

The methods for measuring exposure to air pollution varied widely across studies. Most studies used on-site monitors, while some utilized personal monitors,^{37,46,52,57} and one relied on a central site monitor⁴⁸ (Table 1); there was no appreciable difference in type of monitor utilized and the presence or absence of interaction. While it is expected that personal monitors will capture spatial and temporal variations in air pollutants⁶² the use of on-site monitors likely captured personalized exposures, since many occurred in chambers,^{30–34,43,44,47,49–51,53–55} or on stationary bicycles.^{29,35,36,38}

The type and intensity of exercise also varied within each study. Cycling^{29–36,38,44,46,47,49–52,63} was the predominant (64%) type of exercise recorded in each of the studies; other studies assessed walking,⁵⁶ light exercise on a treadmill,⁴³ hiking,⁴⁵ running,^{48,58} dobutamine injection,⁵³ and daily activities measured using a personal accelerometer.^{37,57} Several (40%) studies described exercise intensity, with six indicating high intensity,^{30–33,48,58} four indicating moderate intensity,^{29,35,36,38} and six indicating or implying low intensity.^{30–33,43,56}

Risk of Bias Analysis

Two studies^{34,44} had high or unclear bias in at least one of the nine categories (Figure 1, Table S4). All studies were low risk of bias in categories indicating crossover design was appropriate, if more than the first period was used, utilizing the correct statistical techniques (paired analysis etc.) to assess crossover studies, and comparability of the results to other studies. Studies were only considered high risk of bias in the categories of randomization and blinding. Two studies did not indicate randomization,^{46,52} eight did not indicate double blinding,^{29,35,36,38,46,48,52,56} and four^{49–51,55} used single blinding. However, several studies that did not blind participants occurred outside the laboratory in locations obviously polluted or in clean air, making blinding impractical if not impossible. The three observational studies^{37,45,57} were low risk of bias in all assessed categories.

Discussion

This review summarizes the available literature on potential interactive effects of air pollution and physical activity on acute health endpoints. The potential interaction between air pollution and physical activity is complex and may depend on several factors. The included studies represent a wide range of air pollutant concentrations and types, varying degrees of physical exertion, and mixture of healthy adults and those with pre-existing conditions (Table 1, Table S2, Table S3).

To understand if physical activity modifies the relationship between air pollution and health endpoints, we defined statistical interactions as being synergistic or antagonistic. However, the presence of an interaction could imply an overall beneficial or detrimental effect. Here, we generally found synergistic interactions^{30,56} implied physical activity in the presence of air pollution was detrimental to one's health, while antagonistic interactions mostly indicated the benefits of physical activity outweighed the negative effects of air pollution.^{29,35,37,38} However, one antagonistic interaction implied the positive effects of physical activity on BDNF are eliminated in the presence of air pollution.⁵²

Since air pollutant concentrations varied so widely, we classified the studies as either “high exposure” or “low exposure”. In some studies what was considered “low” exposure to pollutants, were equivalent or higher than what was considered “high” exposure to pollutants in other studies. Thus, further complicating our ability to directly compare study results. Generally, antagonistic interactions were mostly observed in “high exposure” studies with moderate or high exercise intensity.^{29,35,38} Of the six studies^{35,36,38,53,54} with “high exposure”, three^{29,35,38} provided evidence of antagonistic interaction. However, one study among healthy adults suggested a synergistic interaction (Table 1, Table S2)⁵⁴. One “high

exposure” study was classified as having no evidence of interaction but had point estimates which suggested antagonistic interaction.³⁶ These findings could suggest that moderate intensity exercise may have benefits which may negate the negative impacts of high concentrations of air pollution.

Generally, synergistic interactions were observed in studies with “low exposure” pollution levels and low-intensity exercise.^{30,56} One study by Giles et al., measured low and high intensity cycling among healthy adults was classified as a “low exposure” study, since participants were exposed to $<12 \mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ in filtered air. However, in this study participants were exposed to a wide range of $\text{PM}_{2.5}$ concentrations. In the unfiltered DE condition, participants were exposed to high concentrations (over $300 \mu\text{g}/\text{m}^3$) of $\text{PM}_{2.5}$, which was similar to the upper range of $\text{PM}_{2.5}$ concentrations for the high pollution/non-filtered air group ($80.76\text{--}309 \mu\text{g}/\text{m}^3$) for “high exposure” studies.³⁰ Sinharay et al. found a synergistic interaction among elderly participants with either COPD or IHD.⁵⁶ These findings could suggest low intensity exercise, even in relatively low levels of air pollution can intensify negative impacts of air pollution, particularly among sensitive subgroups. It is also important to note that this review focused on short-term exposure to air pollution and acute health endpoints, however different lengths of exposure to air pollutants or physical activity could potentially interact in different ways. The authors plan to publish a follow-up to this review focusing on those regularly exercise compared to those who do not and could potentially include both short and long-term exposure to air pollutants.

A wide variety of health endpoints were assessed across studies, which adds to the complexity of directly comparing studies. While lung function was most commonly assessed, even measurements taken to assess lung function varied greatly. Most articles measured common lung function parameters such as forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1),^{36–38,45,46,56,58} but some only evaluated minute ventilation (Ve) and tidal volume.^{30,43,44} Overall, more invasive and involved bodily measurements such as cardiac stress test measurements,⁵³ blood-gas permeability,⁴⁹ systemic inflammation,^{33,36,50} vascular effects,^{34,48} electrocardiogram parameters,⁴⁷ DNA damage,⁵¹ and endothelial function³² were assessed in articles with no evidence of interaction. Additionally, while physical activity in air pollution may invoke negative health outcomes, there also is generally a reduced benefit of physical activity in polluted versus clean air.^{38,64,65}

While most articles included in this review were among healthy adults, three were among more susceptible populations and two of these suggested synergistic interaction.^{43,44,56} The study by Sinharay et al. suggested possible synergistic interaction among elderly participants with COPD or IHD and low intensity exercise.⁵⁶ The study by Frampton et al., suggested negative health end points due to air pollution during physical activity among healthy individuals but was not statistically significant among adults with asthma.⁴⁴ The study by Bennett et al.⁴³ found no interaction between light exercise and certain lung function parameters among obese and non-obese women.

This review considers several important limitations. Several articles measured the effects of air pollution and exercise on a small number of people, therefore possibly limiting the power

to detect true statistical interaction. Additionally, most studies (88%) assessed healthy individuals, with only one study specifically examining the effects of physical activity and air pollution on those with pre-existing cardiovascular conditions,⁵⁶ who are among the most at-risk.^{10–13,66} In addition, many of the outcomes measured within the studies varied. Several evaluated lung function, yet there was noted inconsistency in lung function measurements, making comparisons between studies more complex. Measured concentrations of pollutants varied widely across studies and appeared to be closely related to region. Additionally, it is possible that rather than a single unified response to air pollution exposure, study populations express different responses based on where their exposure levels fall, for example a 1 unit increase at a generally low level of air pollution might produce a stronger effect than the same increase at a generally higher level of air pollution.⁶⁷ It is also possible that underlying mixtures of air pollutants, which are likely to be different at the various levels of pollution due to atmospheric conditions and chemical reactivity, could influence responses, and that observable interactions between air pollution and exercise may be dependent on these factors.

There are several factors that should be considered by those aiming to assess the risk and/or benefit of exercising in polluted environments in the future. First, inclusion of all four groups is necessary in order to discern between independent effects of exercise or exposure to polluted air. Second, evaluations of outcomes at several different exposure levels would help in gauging exposure response relationships. Third, many studies only evaluate young healthy adults, further research on susceptible populations is needed to fully determine if exercise in polluted air poses additional risks or benefits.

Conclusions

Pollutant levels, physical activity intensity, and the population studied (healthy adults vs. those with pre-existing conditions) may influence interaction effects between physical activity and air pollution exposure on different health endpoints. Even in relatively low levels of air pollution, low intensity exercise, such as walking, may intensify the negative impacts of air pollution, particularly among elderly persons with pre-existing conditions, such as COPD or IHD.⁵⁶ However, among healthy adults, our review suggests moderate to high intensity exercise may neutralize any short-term negative effects of air pollution exposure. Given the variability in study conditions and inconsistent findings among the studies included in our review, further research examining interaction between physical activity and air pollution on additional biomarkers of disease and health endpoints, especially among susceptible populations, can help guide future medical and public health recommendations related to being physically active in polluted environments.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1:**Summary of Risk of Bias for crossover studies**

The Cochrane criteria for risk of bias included 1) appropriateness of the crossover design, 2) carry-over effect, 3) period effect, 4) if analysis techniques considered the within-person design, and 5) the results of the study were comparable to other similar studies. The second risk of bias assessment followed a modified version of the Jadad scale, designed for randomized-control trials. For use in crossover studies, articles were assessed for 1) randomization, 2) if there was a description of how the study was randomized, 3) if either participants (single) or both participants and researchers (double) were blinded, 4) if the method of blinding was discussed, and 5) if there was a description of the withdrawals throughout the study period.

The three observational studies were critiqued based on 1) how representative the cohort was to the population of interest, 2) the ascertainment of exposure, 3) a demonstration that the outcome of interest was not present at the beginning of the study, 4) control for confounders, 5) how the outcome was determined, 6) if there was adequate follow-up time for the outcome to occur, and 7) if participants lost to follow-up were compared to those included in the study (results not shown).

Table 1:

Summary of Articles

Article and citation	Pollutant Type (component)	Pollutant Monitor Type	Pollutant Level ^d	Study Population	Study Location	Type of Physical Activity	Health Endpoint	Air Pollution Associated with Health Endpoint	Evidence of Interaction
<i>Articles suggesting interaction between air pollution and exercise</i>									
Sinharay et al. 2017⁵⁶	TRAP (PM ₁₀ , PM _{2.5} , BC, NO ₂ , UFP)	On-site monitor	Low	Older adults aged 60 and over, n=119 (Healthy (n=40, 53% Female), COPD (n=40, 53% Female), and IHD (n=39, 10% Female))	United Kingdom	Walking	Lung Function	Yes	Synergistic
Giles et al. 2014³⁰	Diesel Exhaust (PM _{2.5} , NO ₂ , NO, CO)	On-site monitor (chamber-facemask)	Low	Recreationally active adult males (mean age 24.5 years), n=18	Canada	Low and high intensity cycling	Lung function	Yes	Synergistic (low intensity) None (high intensity)
Frampton et al. 2004⁴⁴	UFP	On-site monitor (chamber)	Low	Healthy subjects (mean age 26.9 years, n=12, 50% Female) and those with asthma (mean age 23 years, n=16, 50% Female)	United States	Cycling	Lung function	Yes	Synergistic
Wauters et al. 2014⁵⁴	Diesel Exhaust (PM ₁₀ , PM _{2.5} , PM ₁ , NO, NO ₂ , NO _x)	On-site monitor (chamber)	High	Healthy male subjects (mean age 23.0 years, n=25, 11 also completed exercise testing)	Belgium	Unspecified exercise	Platelet Activation	No	Synergistic
Matt et al. 2016³⁸	TRAP (PM ₁₀ , PM _{2.5} , BC, UFP, NO _x , NO)	On-site monitor	High	Healthy, non-smoking adults (aged 19–57), n=30 (50% Female) EXPOSOMICS Project	Spain	Cycling, moderate intensity	Lung Function	Yes	Antagonistic
Kubesch et al. 2015–1³⁵	TRAP (PM ₁₀ , PM _{2.5} , BC, UFP, NO _x)	On-site monitor	High	Healthy, non-smoking adults (aged 21–53), n=28 (54% Female) (TAPAS)	Spain	Cycling, moderate intensity	Blood Pressure	Yes	Antagonistic
Cole-Hunter et al. 2016²⁹	TRAP (PM _{2.5} , BC, UFP)	On-site monitor	High	Healthy, non-smoking adults (aged 21–52), n=28 (46% Female) (TAPAS)	Spain	Cycling, moderate intensity	Heart Rate Variability	Yes	Antagonistic
Bos et al. 2011⁵²	TRAP (PM ₁₀ , PM _{2.5} , UFP)	Personal monitor (attached to bicycle)	Low	Non-asthmatic, physically fit adults (mean age 43 years), n=38 (26% Female) (SHAPES injury surveillance System)	Belgium	Cycling	Brain-derived neurotrophic factor	No	Antagonistic

Article and citation	Pollutant Type (component)	Pollutant Monitor Type	Pollutant Level ^a	Study Population	Study Location	Type of Physical Activity	Health Endpoint	Air Pollution Associated with Health Endpoint	Evidence of Interaction
Laeremans et al. 2018 ³⁷	Black Carbon ^b	Personal monitors	Low	Healthy, non-smoking adults (mean age 35 years), n=122 (55% Female)	Belgium, United Kingdom, Spain	Personal monitors measured in MET hours	Heart Rate Variability, Lung function	Yes	Antagonistic
Park et al. 2017 ⁴⁶	UFP	Personal monitor (attached to bicycle)	Low	Frequent bicyclists (aged 23–68), n=32 (25% Female)	United States	Cycling	Lung function	Yes	None ^b
Kubesch et al. 2015–2 ³⁶	TRAP (PM ₁₀ , PM _{2.5} , BC, UFP, NO _x)	On-site monitor	High	Healthy, non-smoking adults (aged 21–53 years), n=28 (54% Female) (TAPAS)	Spain	Cycling, moderate intensity	Lung function & Systemic Inflammation	No	None ^c
<i>Articles suggesting no interaction between air pollution and exercise</i>									
Bennett et al. 2016 ⁴³	Ozone	On-site monitor (chamber)	Low	Obese and non-obese, smoking, 18–35 year-old females, n=40 (20 obese, 20 non obese)	United States	Light exercise on treadmill	Lung function	No	None
Girardot et al. 2006 ⁵	Ozone and PM _{2.5}	On-site monitor	Low	Nonsmoking Adult Hikers that met ATS standards for acceptable spirometry (aged 18–82), n=354 (56% Female)	United States	Hiking	Lung function	No	None
Gomes et al. 2011 ⁵⁸	Ozone	On-site monitor (chamber)	Low	Male competitive runners (mean age 24 years), n=10	United Kingdom	High intensity running	Lung function	No	None
Wauters et al. 2015 ⁵³	Diesel Exhaust (PM ₁₀ , PM _{2.5} , PM ₁ , NO, NO ₂ , NO _x , CO)	On-site monitor (chamber)	High	Healthy non-smoking male subjects with normal spirometry and ECG (mean age 22.2 years), n=10	Belgium	8 µg/kg/min dobutamine*	Cardiac stress test measurements	No	None
Bräuner et al. 2007 ⁵¹	TRAP ^d (NC _{Total} , PM ₁₀ , PM _{2.5} , BC, UFP, NO _x , NO, CO, O ₃)	On-site monitor (chamber)	Low	Caucasian nonsmokers (mean age 25 years), n=29 (31% Female)	Denmark	Cycling	Oxidative stress–induced DNA damage	Yes	None
Bräuner et al. 2008 ⁵⁰	TRAP ^d (NC _{Total} , PM ₁₀ , PM _{10-2.5} , PM _{2.5})	On-site monitor (chamber)	Low	Caucasian nonsmokers (mean age 25 years), n=29 (31% Female)	Denmark	Cycling	Microvascular function & inflammation	No	None
Bräuner et al. 2009 ⁴⁹	TRAP ^d (NC _{Total} , PM ₁₀ , PM _{2.5} , BC, CO)	On-site monitor (chamber)	Low	Caucasian nonsmokers (mean age 25 years), n=29 (31% Female)	Denmark	Cycling	Blood-gas permeability	No	None

Article and citation	Pollutant Type (component)	Pollutant Monitor Type	Pollutant Level ^a	Study Population	Study Location	Type of Physical Activity	Health Endpoint	Air Pollution Associated with Health Endpoint	Evidence of Interaction
	UFP, NO _x , NO, CO, O ₃								
Zareba et al. 2009⁴⁷	UFP	On-site monitor (chamber-facemask)	Low	Healthy non-smoking subjects with normal spirometry and ECG (aged 18–40 years), n=24 (50% Female)	United States	Cycling	Heart Rate Variability and electrocardiogram parameters	No	None
Giles et al. 2018-1³¹	Diesel Exhaust (PM _{2.5} , PNC, NO ₂ , NO, CO)	On-site monitor (chamber-facemask)	Low	Recreationally active adult males (mean age 24.5 years), n=18	Canada	Low and high intensity cycling	Lung function & heart rate variability	Yes	None
Giles et al. 2018-2³²	Diesel Exhaust (PM _{2.5} , PNC, NO ₂ , NO, CO)	On-site monitor (chamber-facemask)	Low	Recreationally active adult males (mean age 24.5 years), n=18	Canada	Low and high intensity cycling	Endothelial function	No	None
Giles et al. 2019	Diesel Exhaust (PM _{2.5} , PNC, NO, CO)	On-site monitor (chamber-facemask)	Low	Recreationally active adult males (mean age 24.5 years), n=18	Canada	Low and high intensity cycling	Systemic inflammation	No	None
Avila-Palencia et al. 2019	Black Carbon ^b	Personal monitors	Low	Healthy, non-smoking adults (aged 18–61), n=122 (55% Female)	Belgium, United Kingdom, Spain	Personal monitors measured in MET hours	Blood Pressure	No	None
Koch et al. 2020	Diesel Exhaust (PM _{2.5} , NO _x , NO, CO, CO ₂ , TVOC)	On-site monitor (chamber)	Low	Healthy adults (aged 22–33), n=18 (50% Female)	Canada	Cycling	Vascular effects	No	None
Wagner et al. 2020	PM _{2.5}	Central site monitor	Low	Healthy adults with regular performance of at least 150 mins/wk of aerobic physical activity (average age 20.9 years), n=10 (20% Female)	USA	Running	Lung function	No	None

^a“High” exposure studies were defined as such if the measured concentrations in the low exposure (or filtered air) groups were greater than 12 µg/m³ for PM_{2.5}, 0.07 ppm for ozone (which correspond with the current US EPA’s National Ambient Air Quality Standards⁶¹), or 20,000 particles/cm³ for UFP

^b Point estimates suggest synergistic interaction, but not statistically significant

^c Point estimates suggest antagonistic interaction, but not statistically significant

^d Air samples collected from busy street

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COPD: Chronic obstructive pulmonary disease, IHD: Ischemic heart disease

TRAP: Traffic-related air pollutants, UFP: Ultra fine particles, UFPM: Ultrafine particulate matter

TAPAS: Transportation, Air Pollution, and Physical Activities Project