A systematic review of arterial stiffness, wave reflection and air pollution

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Abstract. Arterial stiffening is associated with increased cardiovascular risk. Whether exposure to relatively high levels of air pollution is associated with arterial stiffening is unclear. We aimed to assess the association between exposure to major air pollutants and arterial stiffening. PubMed, SCOPUS and Web of Science databases (through 31 January 2017) were searched using a combination of terms related to exposure to gaseous [nitrogen dioxide (NO_2), nitrogen oxides (NO_x) and sulphur dioxide (SO_2)] or particulate matter pollutants ($PM_{2.5}$, PM_{10} and $PM_{10-2.5}$), arterial stiffness (pulse wave velocity) and reflected waves (augmentation index, augmentation pressure). Pertinent information were extracted from selected studies. In this systematic review were included 8 studies with available data on air pollution and arterial stiffness/reflected waves parameters (8 studies explored the effects of exposure to particulate matter pollutants, 3 studies the effects of exposure to gaseous pollutants); seven of them reported increased arterial stiffness/reflected waves after exposure to air pollution (6 of 8 studies after particulate matter pollutants; 2 of 3 studies after gaseous pollutants). Arterial stiffness and reflected waves were increased in the majority of the studies after both short- and long-term exposure to air pollutants. In conclusion, available evidence supports an association of main air pollutants with increased arterial stiffness and reflected waves. This finding may have implications for population-based strategies for the reduction of arterial stiffness, a vascular biomarker and an intermediate endpoint for cardiovascular disease.

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Introduction

Arterial stiffness is a vascular biomarker and an intermediate endpoint for cardiovascular disease (1). Similarly, pollution is an emerging cause of cardiovascular disease and mortality (2,3). We have recently reported in patients with inflammatory bowel disease, a model of a concomitant chronic inflammation and low burden of traditional cardiovascular risk factors (4), that arterial stiffness and reflected waves are increased (5-7), dependent upon inflammation and reduced by immunomodulatory drugs (8) but not by salicylates (9). Despite some methodology issues (10), these results were confirmed by an independent group (11). Several possible pathways exist that could link inflammation and arterial stiffening (12). Because pollution is associated with elevated levels of serum biomarkers of inflammation (13,14) and endothelial dysfunction (15-18), a condition associated with functional arterial stiffening (12), it is reasonable that inflammation could be a potential link between pollution and arterial stiffening and the consequent increase of the cardiovascular risk.

Pulse wave velocity (PWV) represents the speed at which the pressure wave generated by left-ventricular ejection is propagated within the arterial tree and is considered the gold standard for assessing regional arterial stiffness in clinical practice. Augmentation pressure and augmentation index are two measures of reflected pulse waves.

In this work, we aimed to perform a systematic review to investigate the role of air pollution on arterial stiffness and wave reflection.

Materials and methods

Review criteria. According to PRISMA guidelines, a systematic literature search of original studies in humans was performed using PubMed database (last accessed on January 31, 2017) without restrictions on the year of publication. The search terms were 'arterial stiffness' or 'pulse wave velocity' or 'augmentation index' or 'augmentation pressure' in combination with '$PM_{2.5}$' or 'PM_{10}' or 'nitrogen dioxide' or 'nitrogen oxides' or 'sulfur dioxide' or 'NO_2' or 'NO_x' or 'SO_2' or 'pollution'. The inclusion criteria included

i) peer-reviewed publications reporting original data; ii) a minimum of 10 adult subjects tested to maximize reliability; and iii) arterial stiffness measured with a well-accepted and validated technique [pulse wave velocity (PWV)]; reflected waves estimated by augmentation index or augmentation pressure. Two independent reviewers (L.Z. and P.L.) selected the studies for inclusion in this systematic review. First, the titles of the studies were screened for relevance. In case of doubt, conflict or discussion between the two independent reviewers, the article was retained. Second, publications with titles or abstracts appearing to meet these inclusion criteria were selected for detailed review. In cases of doubt on the inclusion of an article, a decision was achieved by consensus. The reference lists of the analysed studies were also searched. These studies were subjected to the same selection procedures. A narrative of the collected data has been reported. Because of the high heterogeneity of the studies, a meta-analysis was not performed. Whenever possible, data were presented as the mean ± standard deviation and percentages.

Results

The PRISMA flow diagram is reported in Fig. 1, summarizing the number of studies included for analysis based on the search criteria of this systematic review. Using the search terms, a total of 76 studies were identified. In total, 52 studies were excluded based on a review of the title and the abstract only; 16 studies were excluded after the reading of the full text. The remaining 8 studies, published within the last 8 years, reported carotid-femoral PWV (2 studies), brachial-ankle PWV (2 studies), augmentation index and/or augmentation pressure measurements (6 studies) and were considered for the review (19-26). The main results of the studies included in this review are reported in Table I.

Exposure to air pollutants and arterial stiffness. The exposure to particulate matter pollutants ($PM_{2.5}$, PM_{10}) was evaluated in 8 studies (19-26). In 6 of them (19,22-26), the exposure was associated with an increased arterial stiffness and/or wave reflection (estimated by brachial-ankle PWV, augmentation index and augmentation pressure) whereas in the remaining two studies (20,21) the exposure was not associated with either carotid-femoral PWV or augmentation index. However, in one of these negative studies (20), the $PM_{2.5}$ exposure variability within the urban population was very low (mean $PM_{2.5}$ exposure: 21.4±1.1 $\mu g/m^3$); in the second negative study (21), despite the augmentation index was slightly increased (25.4±4.8% vs. 22.6±5.1%) in the group of patients with the higher $PM_{2.5}$ exposure, this difference was not significant, probably for the small sample size (n=23). The exposure to gaseous pollutants (NO_2, NO_x, SO_2) was evaluated in 3 studies (20,22,24) and was associated with increased arterial stiffness and wave reflection in two large studies (20,22) whereas no significant association with brachial-ankle PWV was observed for NO_2 in the third, smaller study (24).

Increased arterial stiffness and wave reflection were reported in 4 of the 5 studies that evaluated the short-term exposure to particulate matter pollutants (19,21,22,24,25) and in 2 of the 3 studies that evaluated the long-term exposure to particulate matter pollutants (20,23,26), whereas an increased wave reflection was reported in 1 of the 2 studies that evaluated the short-term (≤1 day) exposure to gaseous pollutants (22,24), whereas both increased arterial stiffness and wave reflection were reported in one article after long-term exposure to gaseous pollutants (20).

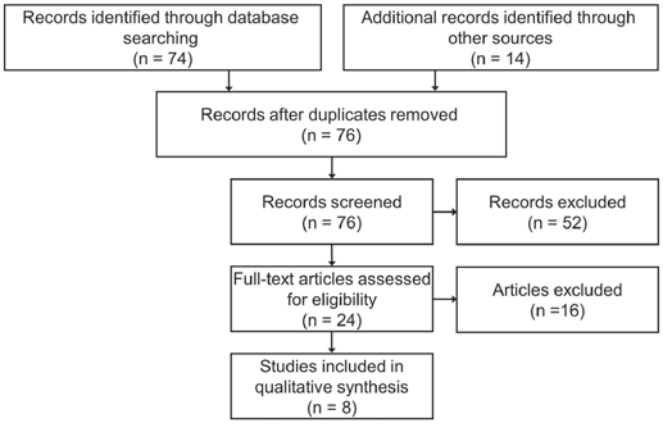

Figure 1. PRISMA flow diagram.

The association between serum biomarkers of vascular inflammation and air pollutants was evaluated in 3 studies (21,24). One study (24) evaluated the effect of NO_2 exposure on hsCRP and revealed a positive association between 1-day NO_2 exposure and hsCRP levels. No significant association was observed between $PM_{2.5}$ levels and hsCRP in either study (21,24). The association between serum biomarkers of inflammation and arterial stiffness was evaluated only in one study (26) in which hsCRP was positively correlated with brachial-ankle PWV.

Discussion

In the present systematic review of the effect of air pollution on arterial stiffness and wave reflection, we included 8 studies for discussion. A total of 6 of the 8 studies reported an increased arterial stiffness and/or wave reflection after particulate matter pollutants exposure. The exposure to gaseous pollutants was associated with increased arterial stiffness and wave reflection in 2 of the 3 studies with available data.

Increased arterial stiffness and wave reflection after exposure to air pollutants. Possible underlying mechanisms. During the last decades, several clinical settings, either physiological or pathological, has been associated with increased arterial stiffness and wave reflection. Most of them, such as the elderly and chronic diseases, are irreversible, other clinical conditions, such as chronic inflammation, are associated with increased arterial stiffness (4-12,27) and potentially reversible, as suggested in recent studies showing that the reduction of inflammation can be associated with a reduction of arterial stiffness (6,8,27). In this review, we reported that arterial stiffness is increased after exposure of air pollutants. Despite it remains to be demonstrated whether the reduction of chronic exposure to air pollution is associated with arterial destiffening, it was reported that wave reflection increased immediately after 1 h of exposure to diesel exhaust while there was no effect on arterial stiffness 40 min after exposure (28),

Table I. Studies that have evaluated the effect of gaseous or particulate matter pollutants on arterial stiffness.

Authors (refs.)	Year of publication	Subjects, n	Population	Measure of arterial stiffness	Inflammatory biomarkers	Measure of pollution	Main results
Fang *et al* (19)	2008	26	Welders	AIx		$PM_{2.5}$	6 h $PM_{2.5}$ exposure (median 390 $\mu g/m^3$, range 30-2620 $\mu g/m^3$) was associated with a slight 2.8% increase in AIx (95%CI -1.4 to 7.0%). Additional exposure the day before the monitoring was associated with a significant increase (5.1%; 95%CI 0.8-9.5%).
Lenters *et al* (20)	2010	745	Young adults, urban population	cf-PWV, AIx		$PM_{2.5}$	Long-term $PM_{2.5}$ exposure (21.4±1.1 $\mu g/m^3$) was not associated with either carotid-femoral PWV or AIx.
Shan *et al* (21)	2014	23	Rural population	cf-PWV, AIx	hsCRP	$PM_{2.5}$	Comparable carotid-femoral PWV, AIx and hsCRP in high (101±37 $\mu g/m^3$) vs. low (39±11 $\mu g/m^3$) 1-day $PM_{2.5}$ exposure group.
Mehta *et al* (22)	2014	370	Elderly men, urban population	AIx, AP		$PM_{2.5}$	Short-term changes in $PM_{2.5}$ (3.6 $\mu g/m^3$) were positively associated with AIx and AP.
Jiang *et al* (23)	2016	321	General population, urban population	AIx	IL-6	$PM_{2.5}$	Higher AIx and IL-6 in high $PM_{2.5}$ group (111 vs. 68 $\mu g/m^3$ exposure).
Wu *et al* (24)	2016	89	Healthy adults	ab-PWV	hsCRP	$PM_{2.5}$	A 10-$\mu g/m^3$ increase in $PM_{2.5}$ concentration at a 1-day lag was associated with 2.1% (95%CI 0.7-3.6%) increases in brachial-ankle PWV for 24 h of exposure. No significant association was observed between hsCRP and $PM_{2.5}$ levels.
Adamopoulos *et al* (25)	2010	1,222	Patients with hypertension	AP		PM_{10}	24 h mean PM_{10} concentrations were positively associated to the AP (Pearson r=0.06, p=0.04).
Weng *et al* (26)	2015	127	Patients undergoing hemodialysis	ab-PWV	hsCRP	PM_{10}	Previous 12-month average concentration of PM_{10} (57.9±5.7 $\mu g/m^3$) was positively correlated with brachial-ankle PWV (β=0.13, p=0.04). hsCRP was positively correlated with brachial-ankle PWV (β=0.23, p=0.01).
Lenters *et al* (20)	2010	745	Young adults	cf-PWV, AIx		NO_2, SO_2	Long-term exposure. 4.1% (95% confidence interval 0.1-8.0%) increase in carotid-femoral PWV and a 37.6% (2.2-72.9%) increase in AIx for a 25 $\mu g/m^3$ increase in NO_2, and a 5.3% (0.1-10.4%) increase in carotid-femoral PWV for a 5 $\mu g/m^3$ increase in SO_2.
Mehta *et al* (22)	2014	370	Elderly men, urban population	AIx, AP		NO_2, SO_2	Short-term changes in SO_2 (2.3±1.9 $\mu g/m^3$) were positively associated with AIx and AP. No associations were observed between NO_2 (0.02±0.01 ppm) and AIx.
Wu *et al* (24)	2016	89	Healthy adults	ab-PWV	hsCRP	NO_2	No significant association with brachial-ankle PWV was observed for NO_2. A 10 ppb increase in NO_2 was associated with 37% (95% CI 17-61%) increases in hsCRP for 1-day of exposure.

ab-PWV, ankle-brachial pulse wave velocity; AIx, augmentation index; AP, augmentation pressure; cf-PWV, carotid-femoral pulse wave velocity; hsCRP, high-sensibility C-reactive protein; IL-6, interleukin 6; NO_2, nitrogen dioxide; $PM_{2.5}$, fine particulate matter; SO_2, sulfur dioxide; Tr, time to wave reflection.

suggesting that acute exposure to pollutants could be associated with reversible (functional) arterial stiffening.

Several mechanisms involved in the stiffening of large arteries after exposure to air pollution could be involved. In this regard, acute and chronic inflammation could play a role in this process. Despite only one of the studies included in this review explored the association between air pollution and a biomarker of vascular inflammation (24), the negative effects of air pollution on inflammatory state are well known and widely documented in literature.

In another model of chronic inflammation it was reported that inflammation may be associated with functional or structural arterial stiffening (12). Functional arterial stiffening could be the consequence of endothelial dysfunction, reduced production/increased inactivation of nitric oxide and reduced vasodilation whereas structural arterial stiffening can be associated with the production of uncoiled stiff collagen, degradation of elastin, smooth muscle cell migration and intima proliferation, vascular calcification and stiffening of the extracellular matrix. The increased arterial stiffness after short-term exposure to air pollutants is in agreement with the finding that even an acute, mild and transitory inflammatory stimulus is associated with arterial stiffening (29) and suggests a functional arterial stiffening. In support of this hypothesis, there is evidence that particulate matter pollutants exposure is associated with acute arterial vasoconstriction (16) and endothelial dysfunction (14,16-18). Moreover, the increase in augmentation index and augmentation pressure, two indices of increased backward reflected waves, after exposure to air pollution (19,22,23,25) may be the consequence of the vasoconstriction at the level of the microcirculation (25). Whether the long-term exposure to air pollutants is associated with functional or structural arterial stiffening is not known because none of the studies included in this review explored the effect of the reduction of long-term exposure to air pollution.

In presence of increased arterial stiffness, the destiffening of large arteries is an objective with important implications on the cardiovascular risk, as suggested by the increased survival after the reduction of arterial stiffness (30). To obtain an arterial destiffening, three levels of treatment can be used. The first is the prescription of a drug that reduces the arterial stiffness, such as an antihypertensive with an independent effect on arterial stiffness. Unfortunately, using this option the causal factors of the arterial stiffening remain active. The second level of treatment consists in the reduction, most probably, or elimination, hardly, of a known causal factor of arterial stiffening, i.e. the chronic inflammation (6,8,27). This approach, acting in a step before the arterial wall stiffening, can be potentially highly effective in single patients. Regarding this point, interventional multicentre studies are ongoing. The model studied in this review has a great clinical importance because, reducing the air pollution, we can reduce the arterial stiffening already occurred and also prevent the development of chronic inflammation, a causal factor of arterial stiffening and several cardiovascular complications, acting at a population-based level of treatment. Consequently, the evidence that air pollution is associated with increased arterial stiffness has also a potential great epidemiological relevance and could represent a warning for the lawmakers and the occupational physicians.

Methodological issues. The present study has several strengths. First, to the best of our knowledge, no systematic review studies have determined whether arterial stiffness is increased after air pollution. Second, we used widely accepted measures of arterial stiffness and wave reflection. PWV represents the speed at which the pressure wave is generated by left-ventricular contraction is transmitted within the arterial tree. The carotid-femoral PWV represents the gold standard for arterial stiffness assessments in daily practice whereas ankle-brachial PWV represents the stiffness of muscular and elastic arteries considered as a whole. Augmentation index and augmentation pressure are measures of wave reflection. This study may have also a number of potential limitations. First, all of the studies included in this review did not explore the effect of the reduction of exposure to air pollutants on arterial stiffness and reflection waves. Future prospective studies need to be performed to confirm the hypothesis of a destiffening effect of a population-based reduction of the air pollution. Second, due to the high heterogeneity of the time of exposure and the concentration of air pollutants, outcomes and population studied included in this systematic review, a meta-analysis was not performed. Third, we con not exclude that the presence of comorbidities could have influenced the results of the studies included in this review. Adamopoulos *et al* (25) included patients with hypertension, Weng *et al* included patients undergoing haemodialysis (26), whereas other authors (19-23) included patients from the general population, and did not report any exclusion criteria or included patients with several cardiovascular risk factors.

In conclusion, available evidence supports an association of gaseous and particulate matter pollutants with an increased arterial stiffness and wave reflection. These findings may have important clinical implications for population-based strategies aimed at the reduction of arterial stiffness, an intermediate end-point of cardiovascular diseases.

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