Exposure to particulate matter: a brief review with a focus on cardiovascular effects, children, and research conducted in Turkey

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[Received in June 2021; Similarity Check in June 2021; Accepted in November 2021]

Exposure to environmental particulate matter (PM), outdoor air pollution in particular, has long been associated with adverse health effects. Today, PM has widely been accepted as a systemic toxicant showing adverse effects beyond the lungs. There are numerous studies, from those in vitro to epidemiological ones, suggesting various direct and indirect PM toxicity mechanisms associated with cardiovascular risks, including inflammatory responses, oxidative stress, changes in blood pressure, autonomic regulation of heart rate, suppression of endothelium-dependent vasodilation, thrombogenesis, myocardial infarction, and fibrinolysis. In addition to these and other health risks, considerations about air quality standards should include individual differences, lifestyle, and vulnerable populations such as children. Urban air pollution has been a major environmental issue for Turkey, and this review will also address current situation, research, and measures taken in our country.

KEY WORDS: air quality; cardiovascular toxicity; monitoring; regulations; vulnerable populations

It is difficult to attribute toxic and health effects of air pollution to one compound alone, since ambient air is a mixture of gases, particles, and liquids that interact with each other and may have synergistic effects. Environmental air pollution and its main component, particulate matter (PM), is an attractive subject of toxicological research ranging from in vitro to molecular epidemiology studies. Epidemiological research has revealed a relationship between PM in ambient air and serious chronic diseases. According to the World Health Organization (WHO), fine PM (of 2.5 microns or less in diameter) accounts for more premature deaths globally than other air pollutants (1). Furthermore, PM as a major constituent of outdoor air pollution has recently been classified as human carcinogen (Group 1) by the International Agency for Research on Cancer (IARC) (2). The detrimental health effects of airborne PM are not limited to respiratory diseases but encompass other systemic diseases, most notably the cardiovascular (CV) ones, including myocardial infarction, hypertension, atherosclerosis, thrombosis, cardiac arrhythmias, and stroke (3, 4). In addition, a large body of evidence demonstrates that PM is closely related to diseases in vulnerable populations such as the elderly and children (5).

The aim of this brief review was to sum up current knowledge on global and local exposure to PM in Turkey, its sources, mechanisms of action, and adverse effects on the CV system, especially in children. It will also address how regulatory and personal intervention can reduce health risks and what has been done in Turkey in this respect.

WHAT IS PARTICULATE MATTER?

Particulate matter is not a specific pollutant but a complex mixture of solid particles and liquid droplets (dust, dirt, soot, or smoke) of organic and inorganic substances suspended in the air (6). By size PM is divided into three main categories: coarse, fine, and ultrafine. Coarse particles (PM_{10}) have the aerodynamic diameter from 2.5 to 10 µm and are mostly composed of dust raised from disturbed soil and surfaces. Its major sources are industry and traffic, most notably mining, farming, unpaved roads, plant and animal fragments, and building construction (7). Fine PM is mostly anthropogenic, generated by fossil fuel combustion (e.g. power plants), residential heating, and automobile exhausts (diesel engine emissions, in particular) (7). Ultrafine particles come from natural and anthropogenic sources and precursor gases (10). As far as the particle size is concerned it overlaps with nanoparticles (NPs), which may also be associated with CV diseases (11). However, NPs are manufactured (for a variety of purposes) and not the consequence of emissions, which is why we shall focus on ambient (air-suspended) particles alone.
Physical and chemical properties of PM relevant for health outcomes depend on many parameters besides size, including PM components, season, meteorological conditions, industrial and combustion sources, and traffic intensity (12,13). Depending on emission source, the carbonaceous core of PM can have organic and inorganic compounds (e.g. transition metals, polycyclic aromatic- and nitro-hydrocarbons, acid aerosols) attached, whose composition determines its toxicity and associated health outcomes (14), while size determines its penetration capability in the organism (15).

**CARDIOVASCULAR OUTCOMES OF PARTICULATE MATTER: LESSONS LEARNED FROM EPIDEMIOLOGY STUDIES**

By some accounts, air pollution is responsible for 19% of all deaths caused by CV diseases and for 21% of all deaths caused by stroke (16). According to a recent Global Exposure Mortality Model based on cohort studies in Europe (including 28 European Union (EU) countries), annual excess mortality rate from ambient air pollution is estimated to 790,000, and 40–80% of this excess mortality is owed to CV events (17). A study in a Danish cohort of 49,564 people found increased risk of CV disease mortality from exposure to PM$_{2.5}$, black carbon, and nitrogen dioxide (NO$_2$) (18). One Chinese study, in turn, showed that PM$_{10}$ and sulphur dioxide (SO$_2$) were associated with higher CV disease mortality (19). In Iran higher relative risk of CV disease mortality was associated with exposure to PM (6.6%) and ozone (O$_3$) (2%) (20).

Clearly, combined exposure to toxic compounds within PM and other common air pollutants such as SO$_2$, NO$_2$, and O$_3$ is expected to exacerbate CV problems (21), yet, cardiovascular toxicity from exposure to combinations of air pollutants is poorly understood, and health effects are usually evaluated for each component alone (22). Given that combined exposure is difficult to evaluate, the last few decades of research have seen a shift of focus toward the relationship between PM size and systemic effects beyond respiratory, such as cardiovascular and neurological (3, 22).

**PM sources and size as determinants of cardiovascular toxicity**

Short and long-term exposure to PM$_{10}$ and PM$_{2.5}$ is a major contributor to CV toxicity. In fact, PM$_{2.5}$ is the 12th leading risk factor for morbidity and mortality worldwide, mostly due to CV diseases (23). In terms of PM sources, of particular interest are the so called “supersites” – large cities with dense population, traffic, and pollution such as those in China (24, 25). In the US, the National Morbidity, Mortality and Air Pollution Study (NMMAPS) conducted in 90 largest cities found that a 10 mg/m$^3$ increase in PM$_{10}$ concentration caused a 0.5% increase in total mortality and 1% increase in hospital admissions due to CV diseases (26). Similar was reported for Europe (27), and strong associations were found between short-term exposure to PM$_{2.5}$ and PM$_{10}$ and daily all-cause, CV, and respiratory mortality in more than 600 cities across the globe (28). The European Study of Cohorts for Air Pollution Effects (ESCAPE) (29) showed that long-term exposure to additional 5 µg/m$^3$ of PM$_{2.5}$ levels leads to a 13% increase in non-fatal acute coronary events. Another meta-analysis (30) singled out PM$_{2.5}$ as more harmful for the CV system than other air pollutants, including PM$_{10}$. Similarly, two German studies (31, 32) established an association between PM$_{2.5}$ and coronary atherosclerosis in densely populated and highly industrialised areas with dense traffic. High PM$_{2.5}$ concentrations were also associated with blood pressure changes in women due to the traffic pollution in Montreal, Canada (33). In contrast, no significant association was found between PM$_{2.5}$ exposure and heavy traffic on New York, Los Angeles, and Chicago roadways (34).

**Toxicity mechanisms of particulate matter in cardiovascular diseases**

It is a general agreement that PM affects the CV system through direct and indirect toxicity mechanisms (4). Indirect effects can be triggered by particles of every size, yet only fine and ultrafine PM can affect the heart and blood vessels directly as they enter the alveoli and pass into the systemic circulation (35, 36). Another determinant of toxicity mechanisms is exposure duration. Epidemiologic data suggest that short-term PM exposure triggers autonomic response, while long-term exposure triggers inflammatory response (21).

Although the mechanisms of direct PM toxicity to the CV system have not been clearly identified, growing evidence is pointing at reactive oxygen species (ROS) produced by reactions between organic hydrocarbons and transition metals attached to PM (37, 38). PM can also directly affect the CV system by disturbing the calcium ion (Ca$^{2+}$) channel controlling myocardial function, as its imbalance may lead to arrhythmias (39) and can cause vasoconstriction by inhibiting endothelial nitric oxide synthase (eNOS) through the uncoupling action of ROS (7). This mechanism has been supported by findings of impaired nitric oxide-driven vasodilation in humans (40), mice (41) and rats (42) due to exposure to PM from diesel exhaust.

As far as indirect PM mechanisms are concerned, lung oxidative stress and inflammation play a critical role (3, 4). PM can trigger inflammation cascade following inhalation (3, 43) by upregulating pro-inflammatory cytokines and chemokines, including interleukin (IL-6, IL-8, IL-1β, granulocyte macrophage colony-stimulating factor (GM-CSF), and tumour necrosis factor-alpha (TNF-α). As these mediators are released into the circulation, the resulting
systemic inflammatory response can lead to atherosclerosis, which is the cause of many CV diseases (4, 22, 25). One study (44) found significant association between PM$_{2.5}$ from wood combustion and traffic and inflammation markers C-reactive protein, IL-12, and myeloperoxidase, suggesting that PM$_{2.5}$ promotes systemic inflammation and risk of development of CV diseases. In addition, ROS-induced pulmonary inflammation was shown to be linked with atherosclerosis, vascular dysfunction, cardiac arrhythmias, and myocardial infarction (3).

Inflammation can also occur through changes in microRNA (miRNA) expression caused by PM. Several studies have reported that PM can modulate miRNAs involved in inflammation, endothelial dysfunction, and atherosclerosis (45–47).

Another indirect pathway of PM$_{2.5}$ action involves autonomic imbalance and activation of the sympathetic nervous system (35, 48), which affects heart rate variability (HRV) necessary for proper CV function, as cardiac output may vary in response to signals from the autonomic nervous system. In one study (35), ceramic factory workers exposed to PM showed lower HRV and parasympathetic activity and an increased sympathetic activity.

Role of individual differences in particulate matter-related cardiovascular risks

Not all individuals are equally exposed to or affected by PM in everyday life, even if they share the same environment (49). Individual exposure may vary with lifestyle (such as indoor and outdoor activities), exposure duration, socio-economic circumstances, genetic setup, age, and sex. In fact, according to the 2017 *Lancet* report by Landrigan et al. (50), about 92 % of pollution-related deaths occur in low- and middle-income countries and minorities, or as the authors succinctly put, “pollution disproportionately kills the poor and the vulnerable”. In addition, individuals with pre-existing lung and heart diseases, obesity, diabetes, high levels of LDL cholesterol, high blood pressure, and those physically inactive, smoking tobacco, or having a poor diet are more vulnerable to PM exposure (4, 8, 37, 51). For these and other vulnerable groups, there is no safe level of exposure or threshold below which no adverse health effects occur (52).

Furthermore, there is a general agreement that long-term (chronic) exposure to PM has more deleterious effects and higher CV mortality than short-term (acute) exposure (3). The latter can destabilise or rupture atherosclerotic plaques and cause cardiac arrhythmias, hypertension, and myocardial infarction (4, 22, 53). A meta-analysis of reports from China revealed that short-term exposure to both PM$_{10}$ and PM$_{2.5}$ was associated with higher mortality, while long-term exposure to PM$_{10}$ increased the risk of hospital admissions over CV problems (54). Long-term PM$_{2.5}$ exposure is also associated with significantly higher mortality due to hypertension (55) and carotid artery thickness (56).

For long, individual exposure to PM had not been measured but relied on environmental indoor or outdoor air monitoring. This has recently changed with the introduction of personal air samplers, which allows us to better evaluate the relations between personal exposure and CV risk biomarkers (49, 57, 58) such as blood pressure, systemic inflammation, endothelial function, oxidative stress, antioxidant activity, cardio metabolism, and neuroendocrine stress response established with a variety of methods (59). Vulnerable populations

The elderly and children are high-risk populations in terms of exposure to environmental chemicals, even at low doses, since organ systems in children are still under development and the elderly experience the loss of organ function.

A landmark study which involved 22 cities in Europe, 90 in the USA, and 12 in Canada under the Air Pollution and Health: A European Approach (APHEA) project has confirmed an association between acute PM$_{10}$ exposure and higher CV mortality among subjects over 75 years of age in all three regions, with the greatest effect in Canada (60–62). Similar vulnerability to PM and association with increased all-cause mortality, including CV, and increased rate of CV diseases in men was observed in the population older than 65 years (63). There is further evidence of association between exposure to PM$_{2.5}$ and emergency hospitalisations for CV diseases in a US population over 65 years of age (63). In Denmark, the association with CV effects was particularly strong in overweight middle-aged and elderly subjects (36). In elderly Chinese men, PM$_{2.5}$ was associated with increased risk of CV mortality (64), and in aged Hongkongers PM$_{2.5}$ was associated with increased risk of CV diseases and ischaemic heart diseases (65).

As a vulnerable group, children are of particular concern, as PM exposure can start in utero and adversely affect health for the rest of their lives (16, 66, 67). Every day, about 93 % of children under the age of 15 from all over the world (1.8 billion children) are exposed to polluted air, including PM$_{2.5}$ levels above the WHO guidelines (68). Cumulative lifetime exposure to air pollution in children is dramatically increasing, and recent data indicate that PM exposure, even in utero, leads to increased risk of CV diseases and associated burden for future generations (69).

These health concerns are heightened by the fact that children are not little adults, as their exposure pathways and toxicokinetics differ from those of the adults (70). Children have higher absorption due to intense anabolic processes, higher number of alveoli, and more permeable respiratory tract. In other words, PM will enter the lungs in children more easily than in adults (71). Moreover, children are more susceptible to the health effects of PM, because their underdeveloped systems and organs are less effective...
at dealing with pollutants (72). Any disruption during these critical years of development can result in severe and lifelong damage, and poor dietary and healthcare conditions can only worsen this damage (73).

Children spend most of their time outdoors, and outdoor air pollution determines the level of PM exposure. There are other factors, including location where they live or go to school, breathing zone, and oxygen consumption, that lead to different responses between children individually and between children and adults. Around two billion children live in areas that exceed the WHO annual limit of 10 µg/m³ for PM₂.₅ (74). Schools may be located in highly polluted places, near highways, power plants, or in industrial sites (71). As for the breathing zone, it is 120–180 cm above ground with adults and much closer to the ground with children (75), which implies higher exposure to heavier chemicals (such as mercury) and large particles. Another reason for the greater vulnerability of children is their higher breathing rate and higher oxygen consumption for their body size (71).

Only a few studies have investigated PM-related CV outcomes in children, mostly by measuring blood pressure, as high blood pressure early in life may predict serious CV outcomes later (58, 76, 77). Recent studies of long- and short-term PM exposure in China have shown an association with hypertension in children and adolescents (78–80). In Belgium, schoolchildren had higher systolic blood pressure on days with higher PM₁₀ concentrations (81). In the Netherlands, children aged 12 years had increased systolic and diastolic blood pressure associated with long-term exposure to PM₉₀ or PM₂.₅ in combination with NO₂ (82).

Besides blood pressure, one prospective pilot study (83) with 70 children aged 6–18 investigated their carotid artery intima-media thickness (CIMT; a measure of carotid plaque build-up and atherosclerosis) progression and established its association with traffic-related exposure to NO₂ but not PM and O₃.

Air quality in Turkey

Table 1 shows current daily and annual mean limit values for PM₂.₅ and PM₁₀ established by various organisations and governments (6, 84–86). Turkey has adopted the EU air quality regulations as its goal in its concept of “Air Quality Assessment and Management Regulation” (86).

Table 1: Current air quality limit values for PM₂.₅ and PM₁₀

| Country / Organisation (reference) | PM₂.₅ Limit values (mean) | PM₁₀ Limit values (mean) |
|-----------------------------------|--------------------------|--------------------------|
| WHO (84)                          | Daily: 25 µg/m³          | Annual: 10 µg/m³         |
|                                   | Daily: 50 µg/m³          | Annual: 20 µg/m³         |
| US EPA (6)                        | Daily: 35 µg/m³          | Annual: 12 µg/m³         |
|                                   | Daily: 150 µg/m³         | Annual: No data          |
| EU (85)                           | -                        | 25 µg/m³                 |
|                                   | Daily: 50 µg/m³          | Annual: 40 µg/m³         |
| Turkey (86)                       | -                        | -                        |

Urban air pollution is a significant environmental problem for Turkey. Since 1990, it has seen a decrease in PM and SO₂ concentrations in urban areas, as a result of the switch from coal (with high sulphur content) to natural gas in residential heating and power generation (87). Even so, according to the 2010 WHO report, Turkey ranked the second most polluted among 34 countries with PM₁₀ levels above the WHO air quality standard (52). In 2012, the European Environment Agency (EEA) estimated that 97.2% of the Turkish urban population was exposed to PM₁₀ levels above the daily limit of 50 µg/m³ (88).

According to the 2014 WHO Ambient Air Pollution Database, Turkish annual mean levels of PM₁₀ were 39 µg/m³ and 58 µg/m³, respectively, and exceeded the EU and WHO standards (84, 85, 89).

With increased concern about ambient air PM, so has increased the monitoring and research of PM₁₀ and PM₁₀ levels in Turkey over the last two decades. In 2003, Istanbul as its largest city and Kocaeli as one of the country’s most industrialised area in north-western Turkey exceeded the EU annual limits (90, 91). Research of PM composition in other air-polluted cities of Turkey, such as Ankara (92), Izmir (93), Sinop (94), Bursa (95), and Kayseri (96) revealed high amounts of toxic trace metals, organic chemicals, and elemental/organic carbon due to a variety of PM sources, including industrial emissions, vehicle exhausts, soil dust, residual oil combustion, and secondary sulphates. In Eskişehir, in central Turkey, daily PM₁₀ and PM₁₀ concentrations in 2008/2009 exceeded the EU limit nearly every day and were much higher in the winter due to coal and wood burning for heating (97). High PM₁₀ levels in Kütahya in the western part of Turkey were largely owed to open-cast mines and industrial plants (98).

According to the 2018 Chamber of Environmental Engineers air pollution report (99), the most polluted provinces in terms of PM₁₀ were Amasya, Ankara, Artvin, Bartın, Bilecik, Bolu, Bursa, Çanakkale, Çorum, Edirne, Erzurum, Kocaeli, Ordu, Sakarya, Samsun, Sivas, Tekirdağ, Trabzon, Yalova, and Zonguldak. In terms of PM₁₀, the most polluted were Istanbul, Ankara, Izmir, Adana, Kahramanmaraş – Elbistan, Erzurum – Doğubeyazıt, Ağrı – Taşhan, Iğdır, Bursa, Mersin, Bursa, Niğde, Erzincan, Denizli, Afyon, and Muğla.

Despite the increasing number of air quality studies in Turkey, only a few Turkish studies (100–103) have...
evaluated PM-related health effects and risks in the nation, none of which specifically include CV diseases or mortality. An exception, to some extent, is a small study in 46 Turkish women, which established an association between biomass fuel exposure and high ventricular systolic blood pressure and low myocardial indices, indicating decreased biventricular systolic function (104). Biomass fuels include wood, charcoal, and crop residue, which are burned for cooking and heating and are known to emit PM$_{2.5}$ and increase the risk of CV diseases (105).

However, there is a large multinational prospective cohort study PURE (106), which included participants from Turkey and found that long-term outdoor PM$_{2.5}$ levels were related to increased risk of CV diseases in adults aged 35–70. A 10 µg increase was associated with a 3% increase in the risk of CV deaths, a 5% increase in CV events, a 3% increase in myocardial infarction, and an 8% increase in stroke (107). In addition, the study found a strong association between long-term PM$_{2.5}$ exposure and hypertension (108).

Prevention of particulate matter-related cardiovascular outcomes

Air pollution is controllable, and many of its adverse health effects can be prevented (16). The WHO estimates that reducing PM$_{10}$ pollution from 70 to 20 µg/m$^3$ can lower the related death rate by 15% (1). Laden et al. (109) have shown that a 10 µg/m$^3$ reduction in PM$_{2.5}$ led to a 31% reduction in CV mortality rates, and Clancy et al. (110) stated that the ban on coal burning in Ireland lowered CV mortality by 10.3% and CV morbidity by 7%. In Australia, PM$_{10}$ reduction by 25% annually and by 38% in the winter was reported to have lowered CV morbidity by 17.9% and 19.6%, respectively (111).

National and international regulatory authorities put forward risk assessment strategies, propose and revise PM limits, and intervene on the public level. However, there are also ways of individual prevention with high-efficiency particulate air (HEPA) filter face masks for short periods of acute PM exposure (48) or with exhaust particulate traps fitted on vehicles (112).

In Europe, PM concentrations are expected to drop substantially by 2030 with stricter regulations of car exhaust and residential emissions (43). For instance, in the Czech Republic, residential heating with coal accounted for 70% of the PM$_{10}$ pollution, so the government supported its replacement with natural gas, which resulted in low SO$_2$ and PM$_{10}$ pollution levels and a decline in CV mortality (113). The above mentioned ban on coal burning in Dublin, Ireland and new regulation in Launceston, Australia, both effectively lowered PM$_{10}$ levels and CV and respiratory mortality and morbidity (110, 111).

CONCLUSION

Numerous experimental and epidemiological studies have clearly established the risks of CV morbidity and mortality associated with PM, and future studies of its toxicity mechanisms are required to better understand its direct and indirect effects on the cardiovascular system. Understanding the mechanisms of PM toxicity, especially in vulnerable groups such as children, will help to devise effective air pollution regulations and improve the existing ones to minimise health risks. Interventions on national and personal level have already shown their potential to reduce PM exposure and prevent adverse health effects, but this is a continuous battle, and Turkey has a long way to go in this respect.

Conflict of interests

None to declare.

Acknowledgments

This manuscript was edited for English language by Gazi University Academic Writing Application and Research Center.

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Izloženost lebdećim česticama – kratki pregled s naglaskom na štetne učinke na srce i krvovazilje, djecu i istraživanja provedena u Turskoj

Za izloženost lebdećim česticama (engl. particulate matter, krat. PM) u zraku iz okoliša već se dugo zna da je povezana sa štetnim djelovanjem na zdravlje. Danas se lebdeće čestice posvuda u svijetu smatraju sistemskim toksikantom koji se oštetuje samo pluća. Brojna istraživanja, od onih in vitro do epidemioloških, upućuju na raznovrsne izravne i neizravne mehanizme toksičnosti lebdećih čestica koji su povezani s povećanim rizikom od bolesti srca i krvovazilja, uključujući i neuropatologije, neurokognitivne i neurodegenerativne poremećaje.

IZLOŽENOST LEBDEĆIM ČESTICAMA

Aktuelno ovaj problem postaje još bitniji u svijetu s usporom globalnog topletnja i industrijskog rasta. Osim toga, povećana emisija partikula slobodne atmosferske polutioni je neophodno upozoravati na ove teške posljedice. Osim sto se ove čestice nekompletno oduzimaju kroz kožu, ondje se, nakon oduzimanja, oduzimaju i kroz neke od drugih organa. To se neophodno uzima u obzir kada se zanima o toksičnosti lebdećih čestica.

KLIJUČNE RIJEČI: kardiovaskularna toksičnost; bioaktivnost; oštećenja u strukturi i funkciji srca; neprave plućne poveznice; ranjivosti populacije; regulativna

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