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Sources and legislative control of $PM_{2.5}$ pollution

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Fine particulate matter ($PM_{2.5}$) is a complex mixture of solid and liquid particles suspended in the air. It is produced directly through industrial processes, and indirectly through reactions in the atmosphere. Due to the impacts of $PM_{2.5}$ on human health and the climate, EU legislation currently focuses on reducing the general background concentration of $PM_{2.5}$. The future for legislative control in the UK after Brexit is uncertain.

$PM_{2.5}$ is a blanket term used to describe fine particulate matter with an aerodynamic diameter of 2.5 μm or smaller. The aerodynamic diameter of airborne particulate matter is defined as the diameter of a sphere with a density of 1000 kg/m^3 and the same settling velocity as the particulate of interest (*1*). This property takes into account the often highly irregular shapes of the particulates, which affect their settling velocities. The aerodynamic diameter determines the depth of penetration into the lungs upon inhalation, and therefore the type and severity of any human health impacts.

$PM_{2.5}$ consists of an amalgamation of suspended solid and liquid particles, which have a range of different sources and hence highly variable morphologies and chemical compositions. $PM_{2.5}$ composition is dominated by sulphates and nitrates, formed from gaseous combustion products. $PM_{2.5}$ also contains polyaromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), elemental carbon, trace metals (such as cadmium, lead, and copper), and water, which is adsorbed onto the particulates (*2*).

$PM_{2.5}$ is a much more persistent pollutant than the larger PM_{10} (fine particulates with aerodynamic diameters of 10 μm or smaller). Due to its finer particle size, smaller aerodynamic diameter, and lower settling velocity, it remains suspended in the atmosphere for much longer and can be deposited thousands of kilometres away from the original emission source (*2*). It has an atmospheric half-life of several days or even weeks and can be deposited on buildings, causing damage by catalysing corrosion reactions (*2*). $PM_{2.5}$ also penetrates more readily into indoor environments, where it is more bioavailable to humans (*3*).

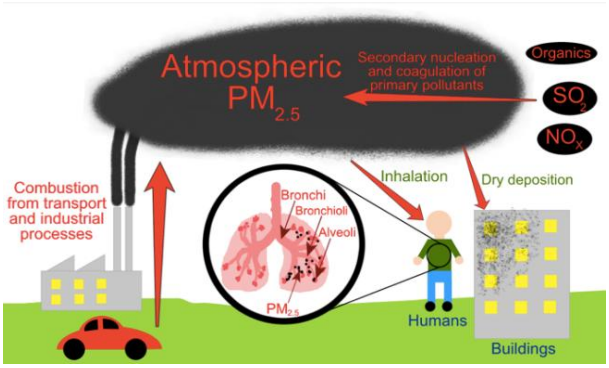

***Figure 1** – A conceptual model for $PM_{2.5}$ shows the main primary and secondary anthropogenic sources in red, the pathways in green, and the receptors in blue.*

Anthropogenic Sources

Direct $PM_{2.5}$ emissions come largely from combustion processes (**Figure 1**). Coal, gasoline, and diesel all release $PM_{2.5}$ during combustion; they are widely used around the world in transportation, energy production, and high temperature industrial processes such as smelting and steel production. Wood burning fires are also an important source. In the UK, non-exhaust vehicle emissions (including brake and tyre wear and road abrasion) are one of the largest growing sources of $PM_{2.5}$. These sources are expected to increase with the number of vehicles on the roads (*1*).

The conversion of primary gaseous pollutants, such as sulphur dioxide, nitrous oxides, and organic compounds, results in the formation of secondary $PM_{2.5}$. These gases react in the atmosphere and undergo nucleation, forming very fine particles, which then coagulate together and increase in size (*2*). Particulates formed in this way are called transformation products and can account for up to 50% of the total $PM_{2.5}$ in some areas (*4*).

Major Impacts

Human Health. Various epidemiological studies have

identified $PM_{2.5}$ pollution as an important environmental risk factor for cardiopulmonary diseases and lung cancer (*5*). When inhaled, the fine particle size allows $PM_{2.5}$ to be deposited in the gas exchange region of the lungs. This region contains the alveoli, which have a more vulnerable and sensitive epithelium than the thicker, mucus-protected bronchioles. Exhalation of $PM_{2.5}$ is slower than for larger particles, allowing them to persist in the lungs and increasing the probability of transfer across the alveolar epithelium into the bloodstream and lymphatic system (*2*).

Climate Change. The interactions of $PM_{2.5}$ with radiative forcing and atmospheric processes generally result in temperature reduction. The secondary aerosols in $PM_{2.5}$ exert a negative radiative forcing effect by directly reflecting insolation and by increasing cloud cover. This is because fine particulates act as condensation nuclei for water molecules, resulting in cloud formation. Clouds formed from $PM_{2.5}$ condensation nuclei have more droplets, but each droplet is smaller. This reduces the likelihood of precipitation; the clouds persist in the atmosphere for longer and reflect more insolation (*1*).

$PM_{2.5}$ with a high elemental carbon content exerts a positive radiative forcing effect because the black carbon absorbs reradiated insolation. However, the magnitude of this effect has not been accurately quantified (*1*). What is clear is that the scale of $PM_{2.5}$ pollution and the associated problems, stress the importance of consistent legislation and cooperation between different countries.

Legislative Control

No threshold has been identified below which $PM_{2.5}$ exposure poses no risk to human health (*4*). As a result, EU legislation emphasises general reduction of $PM_{2.5}$ emissions over large areas. The Directive on Ambient Air Quality and Cleaner Air for Europe (2008/50/EC) introduced average exposure indicator target values to reduce exposure to $PM_{2.5}$ at a population level (*1*). These national exposure reduction targets are relative to the 2010 baseline AEI of the member state (**Table 1**). AEI values are calculated by averaging the three year running mean concentrations, measured at urban background locations across the member state.

Conclusions

Due to the variable composition of $PM_{2.5}$ pollution and the limited research compared to other pollutants, it is unclear which specific constituents cause the impacts to human health discussed above. It is clear, however, that increased $PM_{2.5}$ exposure increases both the number of hospital admissions and the risk of many diseases.

EU legislation rightfully attempts to reduce general public exposure. The UK, although soon no longer a part of the EU, must actively continue to adopt EU directives related to $PM_{2.5}$ into national law. Cooperation between the UK and other European countries is required to control this persistent and transmissible pollutant, as air pollution does not respect international boundaries.

Initial $PM_{2.5}$ Concentration (μg /m³)	Reduction target (%) to be met by 2020
≤ 8.5	0
> 8.5 – < 13	10
= 13 – < 18	15
= 18 – < 22	20
≥ 22	All appropriate measures to achieve 18 μg /m³

***Table 1**. National exposure reduction targets set by the EU (2008/50/EC). From DEFRA, 2012.*

References

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Harrison Frost wrote this Environmental Brief in partial fulfilment of a module in Environmental Pollution, which contributed to his degree in Environmental Science at the University of Reading. He conducted his dissertation project on the modification of biochar with manganese to enhance arsenic, phosphorous and cadmium sorption, which involved a research visit to Universidad Autonoma de Madrid in Spain. Upon graduation, Harrison will stay at the University of Reading to participate in an MSc programme in Environmental Pollution.