

Sub-statement on the potential risk(s) from exposure to microplastics: Inhalation route

# Microplastics - Inhalation route - Toxicity

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46. The COT have previously reviewed the human data on the toxicity of microplastics via inhalation ([TOX/2019/62](#)). The available toxicity data in humans was based on studies of occupational exposure. As the COT have previously discussed occupational exposure in the [TOX/2019/62](#) statement, it will not be included in this statement.

47. In their discussion of [TOX/2019/62](#) the COT considered that the assessment of microplastics exposure via inhalation could be easier in comparison to the assessment of oral exposure given the availability of occupational data from the synthetic textile industry, however, the context should be considered. The Committee further noted that microplastic concentrations present in food and water were thought to be lower in comparison to airborne exposure.

48. According to Panko et al., (2019), particulate matter (PM) from tyre abrasion may represent between 0.8 and 8.5 % mass fraction of PM10 and 1 to 10% of PM2.5 in the air. However, it is unknown what percentage of the PM2.5 burden consists of microplastics (Zhang et al., 2020).

49. The toxicological properties of microplastics may differ to that of PM2.5 (or other pollutants) due to the additives present and their particular characteristics such as morphology and chemical composition (Zhang et al., 2020).

50. In 2020, the Committee on the Medical Effects of Air Pollutants (COMEAP) considered that the evidence on non-exhaust particles (road surface wear, re-suspended road dust, brake and tyre wear) from road transport and associated health effects should be re-evaluated. COMEAP concluded that, as a whole, the body of published work is small and did not provide a compelling narrative of adverse health effects of exposure to non-exhaust particles. However, as there was strong evidence that exposure to particulate pollutants in ambient air is harmful to health, some health risk associated with exposure to non-exhaust particles was likely.

51. COMEAP concluded that the available evidence is not very informative about which components or sources of particulate air pollution are particularly harmful to health and that evidence relating to non-exhaust emissions from traffic, is limited (COMEAP, 2020; 2022).

## **Inhalation studies (2020-March 2023)**

### **In vitro**

52. When the [Overarching Statement](#) by the COT was published, it included studies up to 2020 and therefore this statement concentrates on those studies published from 2020 to the March 2023.

53. Dong et al., (2020) assessed the pulmonary toxicity of polystyrene microplastics *in vitro* using BEAS-2B lung cells to determine the cytotoxic and inflammatory effects. The polystyrene MPs decreased alpha-1 antitrypsin levels and transepithelial electrical resistance by depleted zonula occludens proteins: these are a type of scaffolding protein. The study results indicated that low levels (10 µg/cm<sup>2</sup>) of polystyrene MPs cause disruption to the protective pulmonary barrier and high levels (1000 µg/cm<sup>2</sup>) may have an adverse effect on human lung health. This is based on testing in a single cell line which is not representative of

*in vivo* exposure. However there are now 3D models available which can provide a more suitable model for examining the effects of airborne microplastics (Winkler et al. 2022).

## **In vivo**

54. There is limited research on *in vivo* exposure to airborne microplastics. Lim et al., 2021 used a modified version of the OECD guideline (TG 412) 28-day inhalation toxicity study using a whole-body system. Sprague-Dawley rats were exposed to three different concentrations (0.75, 1.50 and  $3.00 \times 10^5$  particles/cm<sup>3</sup>) of 0.1  $\mu$ m polystyrene NMPs for 6 h each day, 5 days/week for 2 weeks. There was a lack of dose response and no definitive link between concentration at 14 days exposure, and observed alterations to the physiological, serum biochemical and haematological parameters or markers of respiratory function. However, there was a concentration-dependent increase in the expression of TGF- $\beta$  and TNF- $\alpha$  inflammatory proteins. These authors suggest that sustained exposure to higher concentrations of NMPs may result in alterations at the molecular level, thus a risk to health from inhalation of polystyrene micro/nanoplastics. However, caution must be used when weighting this study as the morphology of the airways in the rat differs markedly from that in humans. In addition, exposure was whole body, rather than inhalation only and therefore may not give a realistic representation of exposure in the human lungs. In addition, it is important to distinguish between normal acute particle clearance mechanisms and more persistent, potentially pathophysiological, responses.