Review

From properties to toxicity: Comparing microplastics to other airborne microparticles

Simon Wieland [a,b], Aylin Balmes [c], Julian Bender [d], Jonas Kitzinger [e], Felix Meyer [f], Anja FRM Ramsperger [a,b], Franz Roeder [g], Caroline Tengelmann [h,i], Benedikt H. Wimmer [j], Christian Laforsch [b,*], Holger Kress [a,*]

[a] Biological Physics, University of Bayreuth, Bayreuth, Germany
[b] Animal Ecology I and BayCEER, University of Bayreuth, Bayreuth, Germany
[c] Institute of Applied Physics, University of Tübingen, Tübingen, Germany
[d] Institute for Biochemistry and Biotechnology, Interdisciplinary Research Center HALOmem, Martin Luther University Halle-Wittenberg, Halle, Germany
[e] Department of Physics, Humboldt University of Berlin, Berlin, Germany
[f] Institute of Clinical Microbiology and Hygiene, University Hospital Regensburg, Regensburg, Germany
[g] Institute of Optics and Quantum Electronics, Friedrich-Schiller-University Jena, Jena, Germany
[h] Medical Faculty, University of Würzburg, Würzburg, Germany
[i] Department of Anesthesiology, Intensive Care Medicine and Pain Therapy, University Hospital of Würzburg, Würzburg, Germany
[j] Faculty of Science, University of Zurich, Zurich, Switzerland

HIGHLIGHTS

- Airborne microplastic (MP) is a potential hazard for human health.
- Size, shape, and surface charge are proposed as possible drivers for MP toxicity.
- Comparisons with other dusts allow identification of putative toxicity mechanisms.
- Mechanistic knowledge of airborne MP toxicity is crucial for hazard evaluation.

GRAPHICAL ABSTRACT
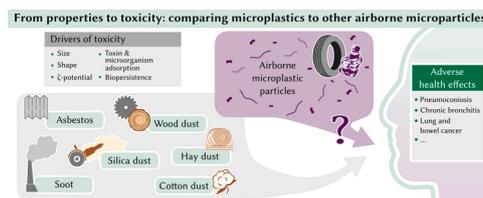
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ABSTRACT

Microplastic (MP) debris is considered as a potentially hazardous material. It is omnipresent in our environment, and evidence that MP is also abundant in the atmosphere is increasing. Consequently, the inhalation of these particles is a significant exposure route to humans. Concerns about potential effects of airborne MP on human health are rising. However, currently, there are not enough studies on the putative toxicity of airborne MP to adequately assess its impact on human health. Therefore, we examined potential drivers of airborne MP toxicity. Physicochemical properties like size, shape, ζ-potential, adsorbed molecules and pathogens, and the MP's bio-persistence have been proposed as possible drivers of MP toxicity. Since their role in MP toxicity is largely unknown, we reviewed the literature on toxicologically well-studied non-plastic airborne microparticles (asbestos, silica, soot, wood, cotton, hay). We aimed to link the observed health effects and toxicology of these microparticles to the abovementioned properties. By comparing this information with studies on the effects of airborne MP, we analyzed possible mechanisms of airborne MP toxicity. Thus, we provide a basis for a

* Corresponding authors.
  E-mail addresses: christian.laforsch@uni-bayreuth.de (C. Laforsch), holger.kress@uni-bayreuth.de (H. Kress).

mechanistic understanding of airborne MP toxicity. This may enable the assessment of risks associated with airborne MP pollution, facilitating effective policymaking and product design.

1. Introduction

Since the 1950 s, plastics have become an essential part of our daily lives and have enabled many technical and medical innovations (Andrady and Neal, 2009; Thompson et al., 2009). The resulting global adoption of plastics and its often inappropriate disposal has been accompanied by a rapidly accumulating amount of waste in the environment, as most commodity plastics are very resistant against both physical and biological degradation (Geyer et al., 2017; Andrady, 2015). The disintegration of such improperly disposed plastic, processes like tire wear, and abrasion from synthetic textiles produce many microscopic plastic particles (microplastics, MP) (Brahney et al., 2021; Mbachu et al., 2020; Zhang et al., 2021). These particles formed by degradation or abrasion processes are classified as secondary MP. In contrast, primary MP is manufactured in a small size, often finding use in cosmetics, such as peelings or cleaning abrasives. It usually reaches the environment via the wastewater stream (Brahney et al., 2021; Syberg et al., 2015). The term MP usually refers to plastic particles smaller than 5 mm (Arthur et al., 2009). However, there is still no uniform definition of the lower size limit of MP. Most commonly, the lower size limit of MP is set between 0.1 and 100 µm (EFSA, 2016; Frias and Nash, 2019). In this review, we use the term MP to describe particles derived from synthetic polymers with sizes between 0.1 µm and 5 mm.

Reports about MP in the environment first focused on marine ecosystems (Thompson et al., 2004; Ng and Obbard, 2006; Imhof et al., 2017). Soon after these reports, MP pollution of other environmental compartments gained increasing attention: MP was also detected in freshwater and terrestrial ecosystems worldwide (Schell et al., 2020; Frei et al., 2019; Piehl et al., 2018; Imhof et al., 2018, 2013; Dris et al., 2015). Even in the most remote areas, MP pollution is detectable (Imhof et al., 2017; Gonzaandacute; lez-Pleiter et al., 2021; Teichert et al., 2021). Especially atmospheric transport of MP is an important driver of MP transfer around the globe (Zhang et al., 2019). As a consequence, attention to this airborne MP is continuously increasing (Brahney et al., 2021; Zhang et al., 2020).

Due to the omnipresence of MP, concerns about possible effects on environmental and human health are rising. It is well established that aquatic organisms interact with MP and consume it together with their food. For example, the ingestion of MP particles has been reported for various organisms, including zooplankton, bivalves, and vertebrates (Teichert et al., 2021; Desforges et al., 2015; Browne et al., 2008; Von Moos et al., 2012; Lu et al., 2016; Vinay Kumar et al., 2021). Upon ingestion, particles have been shown to translocate into the surrounding tissues and the circulatory system of the aquatic organisms (Browne et al., 2008; Von Moos et al., 2012; Lu et al., 2016). Therefore, the exposure of aquatic organisms to MP raised concerns about adverse effects of the particles on these organisms and aquatic ecosystems (Wright et al., 2013; Guzzetti et al., 2018). Also, potential human consumption of MP with contaminated food, e.g., seafood, gained increasing attention (Vinay Kumar et al., 2021; Smith et al., 2018; Cox et al., 2019). Consequently, more and more research focused on possible health issues associated with ingested MP (Carbery et al., 2018; De-la-Torre, 2020).

Likewise, due to the widespread presence of MP in the atmosphere, awareness of breathable MP pollution and potentially harmful effects on human health increased over the last years (Prata, 2018; Amato-Lourenço et al., 2020; Huang et al., 2020; Chen et al., 2020). Parameters like size, shape, surface charge, molecules and pathogens adsorbed to the MP particles, and the MP particles' bio-persistence may contribute to airborne MP toxicity (Prata, 2018; Wright and Kelly, 2017). Yet, the understanding of the role of these parameters concerning potentially adverse health effects of airborne MP is still limited; only very few studies cover this issue. However, some non-plastic microparticles that are well-known drivers of diseases have already been studied in this respect. Therefore, to shed light on the potential effects of airborne MP on human health, it has been proposed to compare MP toxicity to other well-studied airborne particulate matter (Vethaak and Legler, 2021).

In general, human exposure to airborne microparticles depends on their size. For example, the inhalation of microparticles usually is determined by their aerodynamic equivalent diameter (AED, the diameter of a sphere with density 1 g cm^{-3} with the same settling velocity as the microparticle) rather than by their geometrical size (Chen and Fryrear, 2001; Reponen et al., 2001). Commonly, microparticles are categorized according to their AED into particles > 10 µm, particles < 10 µm (PM10), particles < 2.5 µm (PM2.5), and ultrafine particles < 0.1 µm (Kelly and Fussell, 2012). The large particles > 10 µm are assumed to collide with the upper airways upon respiration, whereas PM10 can enter the bronchioles, and PM2.5 and ultrafine particles may even penetrate the alveoli (Prata, 2018; Kelly and Fussell, 2012). Besides determining the exposure pathways, the size of microparticles can affect their toxicity. Because of their large surface-to-volume ratio, smaller particles are potentially more prone to interact with cells and tissues, leading to stronger responses at exposure sites (Schmid and Stoeger, 2016).

Also, the shape of a microparticle influences its toxicity by modifying interactions with cells and tissues. For example, microfibers interact with cells and tissues differently than microspheres, fragments, or films (Allegri et al., 2016). These altered interactions can lead to shape-specific toxicity of different microparticles.

Moreover, the surface charge of microparticles can affect their toxicity. A typical property to quantify the surface charge of microparticles is the particles' ζ-potential (Peltonen and Hirvonen, 2008). The ζ-potential can be a proxy for the electrostatic interactions of microparticles with cells and tissues, determining the adhesion of the microparticles (Silva et al., 2014). The adhesion strength of microparticles could potentially affect their bioavailability and consequently the toxicity of these particles.

Adsorption of molecules and microorganisms following the microparticles' environmental exposure can additionally modify their toxicity. In addition to biomolecules that may increase the bioavailability of the microparticles, the microparticles can also carry adsorbed toxins or pathogenic bacteria, which might enlarge their potential to impact human health (Prata, 2018; Ramsperger et al., 2020; Kirstein et al., 2016).

Lastly, all these potential modes of toxicity may be altered by the microparticles' bio-persistence. Therefore, bio-persistence has been discussed as an essential parameter for the microparticles' interactions with tissues because it potentially alters long-term exposure and associated chronic effects (Oberdorster et al., 1994).

Here, we aim to better comprehend the impact of the abovementioned parameters (size, shape, ζ-potential, adsorbed molecules and organisms, bio-persistence) on MP toxicity by comparing different well-studied non-plastic microparticles to MP. We reviewed the literature on six microparticle pollutants often associated with occupational diseases: Asbestos, silica dust, soot, wood dust, cotton dust, and hay dust. We set our focus on occupational safety and the pathogenesis of microparticle-associated diseases. By giving an overview of the diseases and the pathogenesis associated with each type of microparticle with respect to the abovementioned parameters, we aim to determine to what extent these parameters are the drivers of the microparticle's toxicity. We try to link our results to the existing knowledge on the properties of airborne MP particles, identifying possible mechanisms of their toxicity. Overall, this approach can contribute to understanding airborne MPs' potential

impact on human health and outline possible directions for future MP research.

2. Non-plastic airborne microparticles

2.1. Asbestos

Asbestos unites a group of naturally occurring hydrated silicates that form long and thin crystalline fibers (Pira et al., 2018; Solbes and Harper, 2018). Many beneficial characteristics of asbestos, like high flexibility, low thermal conductivity, and a very high mechanical and chemical resistance, led to a massive increase in its use throughout the 20th century for industrial purposes, such as brake linings, asbestos cement, asphalt concrete, and insulating material. Asbestos was also used in various products of everyday life, for example, artificial snow, shoes, and cigarette filters (Pira et al., 2018; Noonan, 2017). Even though asbestos has been prohibited in many countries, human exposure still occurs during repair and removal of older buildings (Kameda et al., 2014; Hagemeyer et al., 2006). Workplace concentrations of asbestos strongly depend on the materials, processing techniques, and engineering controls implemented to mitigate the asbestos release. During abatement of asbestos insulation, usually, concentrations between 0.02 and 0.2 fibers per cubic centimeter (f cm^{-3}) have been reported, with maximum concentrations up to 100 f cm^{-3} (Williams et al., 2007). Next to occupational exposure, pollution of the ambient air, e. g. in areas with naturally occurring asbestos, increases the risk of asbestos-related diseases (Metintas et al., 2002). Also, some adverse health effects can already occur after contact with very low concentrations of asbestos (Hodgson and Darnton, 2000; Goodman et al., 1999). Therefore, it is common practice in most developed countries to avoid asbestos exposure as far as possible. For example, the US National Institute for Occupational Safety and Health (NIOSH) recommends that workers should not be exposed to more than one f cm^{-3} for 30 min. When working with asbestos, a self-contained breathing apparatus with a full facepiece is recommended by the NIOSH (Barsan, 2007).

It has been estimated that globally, about 100,000 people per year die from the consequences of asbestos exposure (Baumann et al., 2013). The main problems caused by asbestos inhalation are asbestosis, interstitial pulmonary fibrosis, pleural disorders and calcified plaques on the lung's outer lining, and several forms of cancer, especially malignant mesothelioma (Goodman et al., 1999; Sporn and Roggli, 2014; Alpert et al., 2020). Even though asbestosis is a nonmalignant disease, it can severely impact the patient's life quality. Eventually, it can lead to hypoxemia and increased lung pressure, potentially affecting the heart muscle in the long term (Sporn and Roggli, 2014).

Although diseases associated with the inhalation of asbestos are very well studied, the exact mechanisms leading to asbestos-related diseases are yet not fully understood (Huang et al., 2011). Accumulation of asbestos fibers is considered one of the main factors inducing a continuous inflammatory response, oxidative stress, and apoptosis (Solbes and Harper, 2018; Craighead and Mossman, 1982). Consequently, surrounding tissues are damaged. Furthermore, ROS (reactive oxygen species) and oxidative stress may also cause DNA damage, increasing the risk of malignant cell proliferation (Solbes and Harper, 2018). Next to inflammatory responses and oxidative stress, the direct influence of asbestos on signaling and transduction pathways is important. This leads to an alteration of various cellular processes governing gene expression, cellular growth and shape, and epithelial permeability (Solbes and Harper, 2018).

The properties of asbestos fibers (Fig. 1A, Table 1) play an essential role in their toxicity. For example, the size of asbestos fibers can vary substantially. In one instance, diameters in the range of 0.01 – 1.5 μm and lengths in the range of 0.01 – 64 μm have been reported (Stanton et al., 1981).

Their thin and elongated fiber shape (Fig. 1A) allows them to deeply penetrate the lung, while at the same time, phagocytosis is prohibited as the fiber length exceeds the size of macrophages (Donaldson et al., 2010). Consequently, primarily long asbestos fibers (length > 5 μm) are the toxicologically most relevant fraction. Nevertheless, short asbestos fibers (length < 5 μm), making up the main part of airborne asbestos fibers, may also contribute to the overall toxicity of asbestos (Boulanger et al., 2014). When used, modified, or inhaled, the asbestos fibers may split up lengthwise and form even thinner fibers, increasing the associated risks (Boulanger et al., 2014; Roggli and Brody, 1984).

The ζ-potential of asbestos can cover a wide range between about − 60 mV and + 55 mV and depends on various parameters, including the mineral variety and the pH value of the medium during the measurement. Interestingly, the magnitude of the fibers' ζ-potential is positively correlated to their hemolytic activity (Light and Wei, 1977). Under conditions similar to the conditions in lung tissue, a negative ζ-potential of asbestos fibers was reported. This negative ζ-potential possibly leads to the formation of reactive oxygen species (ROS) and free radicals, likely increasing cytotoxicity and carcinogenicity of asbestos fibers (Pollastri et al., 2014).

When exposed to the environment, the structure of asbestos fibers may be altered by organic molecules (Holmes et al., 2012). It is not clear whether molecules and biofilms adsorbed during environmental exposure affect the toxicity of asbestos. However, this adsorption may be less relevant compared to other factors since asbestos exposure usually occurs in workplace environments, where the fibers are freshly released into the air.

Overall, the hazardous potential of asbestos fibers is increased by their high bio-persistence in the lung, leading to chronic effects (Donaldson et al., 2010). Although dissolution rates of asbestos depend on the exact mineral variety, the estimated lifetime of asbestos fibers in the lung range from several months to years (Jurinski and Rimstidt, 2001; Hume and Rimstidt, 1992; Oze and Solt, 2010).

2.2. Silica dust

The inhalation of silica (silicon dioxide) dust is a well-known cause of occupational diseases, predominantly silicosis. Silica forms minerals like quartz and is the major component of sand. Depending on its crystal structure, silica dust is classified as amorphous and crystalline silica dust, differing in their physicochemical properties. Crystalline silica dust is thought to be the primary driver of silicosis, although exposure to the amorphous form of silica has potential adverse effects, too (Silicosis Mortality, 2005; Merget et al., 2002). To what extent silica dust is associated with health effects may also depend on the dust's exact crystal structure (Wiessner et al., 1988). In various industrial workspaces, crystalline silica dust can occur at high concentrations. Especially workers in mining, road repair, construction, and brick working face the risk of exposure (Wagner, 1997). However, silica-related diseases are not just recorded in these industries but also among workers involved in processes like denim-jeans sandblasting or jewelry polishing, where the exposure to silica dust may lead to occupational diseases (Barnes et al., 2019). Furthermore, naturally occurring silica dust like Sahara dust is also associated with respiratory diseases and silicosis (Derbyshire, 2007).

Workplace concentrations of silica dust typically depend on the individual working processes. For example, silica dust concentrations of 0.26 mg m^{-3} were reported during wall grinding, whereas during concrete sawing, they were as high as 10.0 mg m^{-3} (Linch, 2002). The NIOSH recommended limit for exposed workers is 0.05 mg m^{-3} for up to ten hours a day. At higher concentrations, protective equipment is required (for D.C and Prevention, 1996). Nevertheless, even at the recommended limit, the risk of developing silicosis and death among lifetime exposed workers is significantly elevated (Mannetje et al., 2002).

Exposure to high concentrations of silica dust potentially leads to silicosis, a fibrotic lung disease. It is the most common type of pneumoconiosis (interstitial lung disease), which caused approximately 13,000 deaths worldwide in 2019 (Institute for Health Metrics and
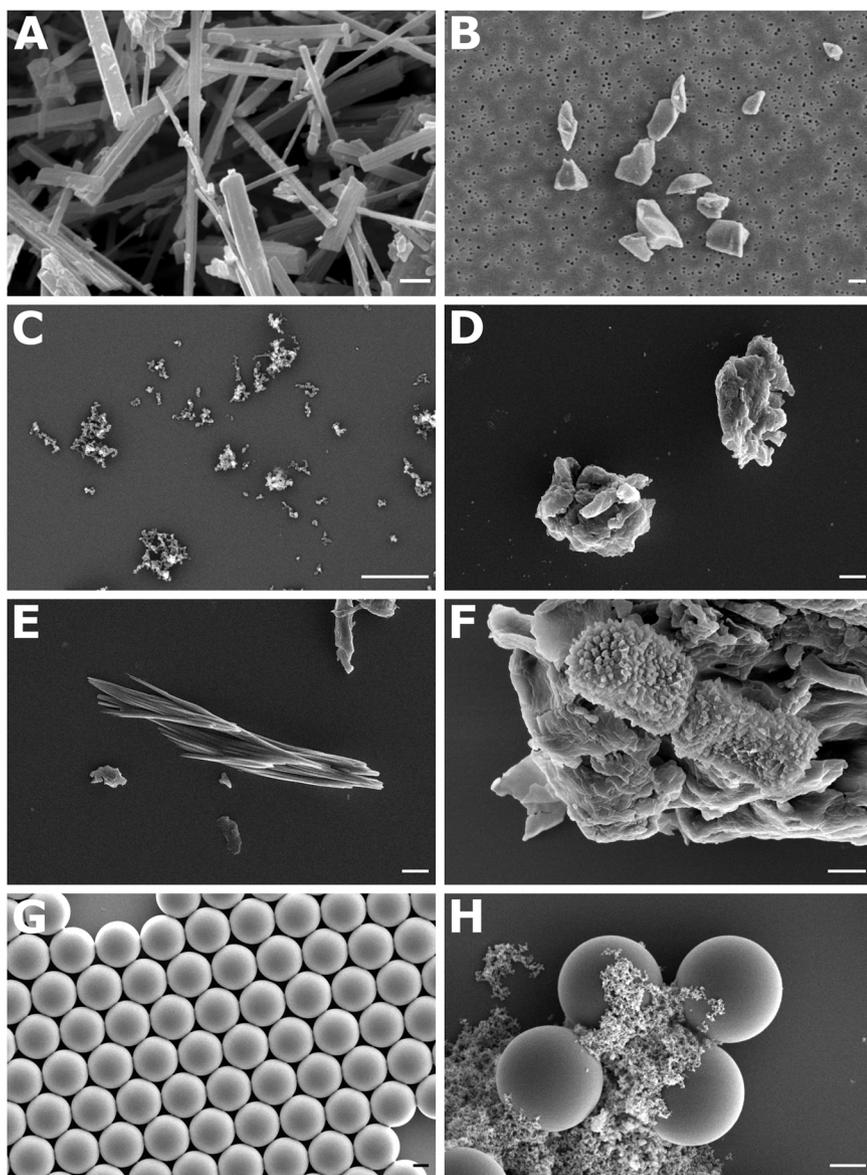
Fig. 1. Scanning electron micrographs of airborne microparticles. A) Amosite asbestos fibers, showing the typical thin and elongated shape. The SEM image was kindly distributed in the public domain by the U.S. Geological Survey (USGS) (Geological Survey, 2007) B) Crystalline silica dust particles, forming irregularly shaped, sharp-edged fragments. The SEM image was taken from S. Mischler et al.: 'Differential activation of RAW 264.7 macrophages by size-segregated crystalline silica' (Mischler et al., 2016) C) Soot particles from diesel exhaust forming grape-like, aciniform structures which are in the size range of several hundred nanometers to micrometers. D) Oak wood particles generated by manual filing (file with 46 cuts per cm) of wood, showing a very irregular surface. E) Cotton fragment formed during cryomilling of raw cotton fibers, showing an elongated shape, which is composed of thin fibers. F) Hay dust particle with adsorbed cells (possibly fungal spores). G) Pristine PS microbeads that are frequently used in MP research. These commercially available MP particles are very monodisperse and have a smooth surface. H) The same PS microbeads after 2 weeks incubation in salt water. Biomolecules adsorbed to their surface, forming an ecocorona. Scale bars: 1 μm. Sample preparation and imaging: Dust samples were manually transferred on a fragment of a silicon wafer, which was mounted on a specimen stub using a carbon adhesive pad. Prior to mounting, the plain and ecocorona-coated PS beads were fixed with Karnovsky's fixative (2% PFA and 2.5% glutaraldehyde in 1x PBS), dehydrated with an ethanol series (30%, 50%, 70%, 80%, 90% for 30 min each; 95% and 100% for 1 h each), and dried in hexamethyldisilazane (HMDS). All samples were coated with 4 nm platinum (Leica EM ACE600 sputter coater). The imaging was performed at 5 kV using an Everhart-Thornley detector (SEM: FEI Apreo VolumeScope, Thermo Fisher Scientific).

Evaluation IHME, 2020). Furthermore, exposure to silica dust is associated with chronic obstructive pulmonary disease (COPD), lung cancer, autoimmune diseases, and renal disease (Leung et al., 2012). There is also a higher prevalence of pulmonary tuberculosis (TeWaterNaude et al., 2006).

In the pathogenesis of silica-induced diseases, inflammatory responses and the stimulation of fibroblasts play an essential role (Barnes et al., 2019; Leung et al., 2012). Inhaled silica particles accumulate in the distal airways and are internalized by macrophages, which activate proinflammatory and pro-fibrotic pathways. The uptake by the macrophages leads to the death of the cell, after which silica particles are released and further intensify the inflammatory response. Subsequently, fibroblast growth is stimulated, leading to the formation of onion-like cellular structures around the sites of inflammation. These silicotic nodules have the potential to merge to sizes > 1 cm and form cavities. For example, *Mycobacterium tuberculosis*, the pathogen of tuberculosis, can persist in these cavities. Consequently, phagocytes are less in control of their growth, possibly explaining the higher prevalence of tuberculosis in silica-exposed workers (Barnes et al., 2019; Leung et al., 2012).

The observed adverse health effects of silica dust are related to the particles' properties (Fig. 1B, Table 1). The size of silica dust varies among different sampling sites, working processes, and sampling methods used, making uniform statements about these parameters difficult. For example, in a granite quarry in Vermont, most aerosolized silica particles were inhalable with a size smaller than 10 μm. In the stone finishing areas of the quarry, more than half of the sampled particles were in the size range of 0.5 – 0.7 μm (Sirianni et al., 2008). Similarly, in three different mines in Alaska, Nevada, and South Africa, the average size of the silica particles ranged from 0.5 to 1.7 μm (Chubb and Cauda, 2017).

Dust particles generated during stonework generally have an irregular and fractured shape, which can be seen in crystalline silica dust from mine tailings (Andraos and Gulumian, 2020). This irregular, fractured shape with angular surfaces of dust particles was also observed in crystalline silica-rich ambient particular matter (Dong et al., 2015). Although earlier assumptions that this sharp-edged shape of crystalline silica dust is the primary driver of silicosis have been disproven (Winkler, 1975), mechanically fractured crystalline silica has stronger cytotoxic effects on human macrophages in vitro compared to more regularly-shaped grown crystalline silica (Leinardi et al., 2020).

The amorphous form of silica has a surface covered by hydroxyl groups, leading to hydrophilic properties (Vansant et al., 1995). The

Table 1

Overview of non-plastic microparticle properties and associated health effects. Here, information on shape, size, ζ-potential, and bio-persistence of different non-plastic microparticles is summarized, together with a short synopsis of the health risks. Abbreviations: COPD – chronic obstructive pulmonary disease; AED – aerodynamic equivalent diameter; PAH – polycyclic aromatic hydrocarbon, POP – persistent organic pollutant.

Particle type	Size	Shape	ζ-potential	Adsorbed molecules/ organisms	Bio-persistence	Health risks
Asbestos	Diameter 0.01 – 1.5 μm, length 0.01 – 64 μm (Stanton et al., 1981) Mostly long asbestos fibers > 5 μm toxicologically relevant (Boulanger et al., 2014)	Thin, elongated fibers (Stanton et al., 1981) Fibers longer 5 μm possibly main drivers of asbestos toxicity (Boulanger et al., 2014) In lung, fibers can split up lengthwise into thinner fibers (Boulanger et al., 2014; Roggli and Brody, 1984)	- 60 to + 55 mV, depending on the mineral variety (Light and Wei, 1977) Hemolytic activity correlated to ζ-potential (Light and Wei, 1977), formation of ROS associated with negative ζ-potential (Pollastri et al., 2014)	Environmental exposure alters surface of asbestos fibers (Holmes et al., 2012) However, effect on toxicity not known	Dissolution rates depend on the mineral variety, estimated lifetime of asbestos fibers in lung between several months to years leads to chronic toxicity (Jurinski and Rimstidt, 2001; Hume and Rimstidt, 1992; Oze and Solt, 2010)	Asbestosis, interstitial pulmonary fibrosis, calcified plaques and pleural disorders, cancer (especially mesothelioma) (Goodman et al., 1999; Sporn and Roggli, 2014; Alpert et al., 2020)
Silica dust	Airborne fragments usually in the breathable range < 10 μm, mostly between 0.5 and 2 μm (Sirianni et al., 2008; Chubb and Cauda, 2017)	Irregularly shaped fragments with sharp edges (Andraos and Gulumian, 2020; Dong et al., 2015), mechanically fractured crystalline silica more cytotoxic than regularly shaped grown crystalline silica (Leinardi et al., 2020)	-30 to − 10 mV under physiological pH conditions (Dong et al., 2015; Leinardi et al., 2020). Higher toxicity of freshly fractured quartz due to increased surface radicals (Vallyathan et al., 1995)	Adsorption of PAHs to biogenic amorphous silica (Rabovsky, 1995) However, contribution to overall silica toxicity unclear	Very low rates of dissolution under physiological conditions (Rimstidt and Barnes, 1980), theoretical lifetime of microparticles about 100 – 10000 years (Jurinski and Rimstidt, 2001) contributes to chronic toxicity	Silicosis (Wagner, 1997; Barnes et al., 2019; Derbyshire, 2007; Mannetje et al., 2002) COPD, lung cancer, autoimmune and renal disease (Leung et al., 2012) Increased risk for tuberculosis, due to formation of silicotic nodules (TeWaterNaude et al., 2006)
Soot	Size dependent on combustion process, mostly on the order of several hundreds of nanometers (Kleeman et al., 1999; Torvela et al., 2014), also larger structures up to 100 μm (Watson and Valberg, 2001) Particles grow in size after release due to aggregation and condensation of organic matter (Janssen et al., 2012; Patterson and Kraft, 2007). Size may affect toxicity (Frank et al., 2013), particles with a size of 70 – 110 nm potentially most toxic (Sarkar et al., 2014)	Depending on combustion process formation of grape-like aciniform structures, but also other shapes like xerogels and cenospheres (Watson and Valberg, 2001). Possibly formation of toxic radicals on particles with high curvature (Frank et al., 2013)	-50 to − 30 mV for diesel soot particles under physiological pH and salt conditions (Sarkar et al., 2014; Chen and Huang, 2017). Particles with ζ-potential of − 41 mV to − 37 mV especially bioreactive, causing inflammatory response (Sarkar et al., 2014)	Adsorption of organic compounds, especially PAHs, to soot particles during combustion process and after their release into the environment (Lee, 2010; Watson and Valberg, 2001; Eaves et al., 2017) Adsorbed PAHs play a major role in soot particle toxicity (Barfknecht, 1983; Kirrane et al., 2019)	Clearance from rat lungs decreases with exposure level, possibly leading to chronic effects; half-life of diesel soot particles between 80 and 250 days (Griffis et al., 1983; Wolff et al., 1987)	Occupational exposure: cancer of lung and esophagus (Pukkala et al., 2009), asthma, ischemic heart disease (Li et al., 2008; Hansen, 1983) Ambient exposure: increased risk for cardio-pulmonary diseases, possibly increase in cancer risk (Janssen et al., 2012; Lee, 2010; Stöber and Abel, 1996)
Wood dust	AED of wood dust collected at breathing height of workers between 10 μm and 100 μm (Harper et al., 2002) AED of wood dust directly sampled from sanding mostly < 10 μm, in the breathable range (Määttä et al., 2006; Ojima, 2016; Marková et al., 2018) AED of wood dust directly sampled from milling mostly > 125 μm (Očkajová et al., 2020)	Shape depends on wood type and processing techniques, usually irregularly shaped particles (Mazzoli and Favoni, 2012; Liu et al., 1985) Effect of shape on toxicity not yet studied	Intact spruce wood capillaries: − 10 to + 10 mV at pH 5.6 (Muff et al., 2018) Cellulose nanofibers from cedar wood: − 50 to − 30 mV at pH 6.8–7 (Uetani and Yano, 2012) Effect of ζ-potential on toxicity not known	Chemical treatments (e.g., chromate compounds) potential driver of wood dust toxicity (Klein et al., 2001). Also naturally occurring chemicals (terpenes, polyphenolic compounds) may affect wood dust toxicity (Naarala et al., 2003)	Lignin and cellulose only degradable by enzymes from specialized microorganisms (Slavin et al., 1981; Pérez et al., 2002; Eriksson et al., 1990), therefore likely no degradation of wood in the human lung, promoting chronic toxicity. Wood cellulose fibers persist in rat lungs after 1 year, higher bio-persistence compared to asbestos. Estimated half-life about 3 years (Muhle et al., 1997)	Classified as carcinogenic for humans, especially associated with cancer of nasal cavities and paranasal sinuses (Delzell, 1995). Also associated with lung cancer (Alonso-Sardón et al., 2015; Binazzi et al., 2015; Hancock et al., 2015). Evidence of association with other pulmonary diseases like cryptogenic fibrosing alveolitis and idiopathic pulmonary fibrosis (Hubbard et al., 1996; Gustafson et al., 2007)
Cotton dust	Classified according to size into trash (> 500 μm), dust (50 – 500 μm), micro dust (15 – 50 μm), and breathable dust (< 15 μm) (Dangi and Bhise, 2017). Mostly breathable fraction associated with health effects (Ellakkani et al., 1984)	Fragmented fibers, irregularly shaped particles (Goynes et al., 1986). Effects on toxicity not investigated	Not known	Adsorption of bacterial endotoxins and possibly also pesticides relevant for cotton toxicity (Wang et al., 2003; Rylander, 1987; Solbrig and Obendorf, 1985)	High biodurability of cellulose fibers relevant for chronic effects, degradation only by enzymes of specialized microorganisms (Eriksson et al., 1990). Degradation of cellulose fibers in vivo in mice and rats very slow (Muhle et al., 1997; Ilves et al.,	Asthma, bronchitis, byssinosis (Dangi and Bhise, 2017). Effects possibly also due to bacterial endotoxins and pesticides (Wang et al., 2003; Rylander, 1987; Solbrig and Obendorf, 1985)

Table 1 (*continued*)

Particle type	Size	Shape	ζ-potential	Adsorbed molecules/ organisms	Bio-persistence	Health risks
Hay dust	Ca. 95% of hay dust particles smaller than 5 µm, therefore breathable (Séguin et al., 2010; O'Connor et al., 2013)	Mostly spherical, but also irregularly shaped and rod-like particles (O'Connor et al., 2013) Effects of shape on toxicity not known	Not known	Dust contains significant amounts of bacterial and fungal spores, adsorbed endo- and mycotoxins (Séguin et al., 2010) These are likely the main causes of adverse health effects (Reboux et al., 2007; Cano-Jiménez et al., 2016)	2018) Wood cellulose fibers half-life ~ 3 years in rat lungs, half-life of cellulose fibers from recycled newspapers 72 days in rat lungs (Muhle et al., 1997) No significant degradation of cellulose in vitro in lung airway lining fluid and phagolysosomal fluid after up to 9 months (Stefaniak et al., 2014)	Farmer's lung disease (allergic alveolitis) (Gregory and Lacey, 1963; Siegel et al., 1991), likely caused by bacterial endotoxins and mycotoxins (Reboux et al., 2007; Cano-Jiménez et al., 2016)
Microplastics	Measured size distributions are likely influenced by sampling & detection limits (Zhang et al., 2020) Measured sizes for atmospheric MP fibers between 20 µm and 5 mm (Cai et al., 2017; Li et al., 2020; Szewc et al., 2021), predominantly 100–700 µm (Allen et al., 2019; Cai et al., 2017); fragments between 5 µm and 750 µm (Szewc et al., 2021); films between 10 µm and 1520 µm (Szewc et al., 2021). Studies with particularly low detection limits measured 70% of MP fallout particles < 63 µm (Klein and Fischer, 2019); at human respiratory height 80% of MP < 20 µm (Li et al., 2020)	Many reports of fibers (Liu et al., 2019; Abbasi et al., 2019; Cai et al., 2017; Dris et al., 2016; Szewc et al., 2021), but also fragments and films (Allen et al., 2019; Szewc et al., 2021). Higher prevalence of fibers indoors than outdoors (Liu et al., 2019; Dris et al., 2017). However, due to focus of MP research on spherical particles (Lim, 2021) role of shape in MP toxicity not known	Surface functionalization of plastics is a common method to adjust material properties (Johansson, 2017), furthermore functional surface groups induced by weathering and photodegradation of MP (Meides et al., 2021; Fechine et al., 2004; Fernando et al., 2007; Decker and Zahouily, 1999); therefore, ζ-potential of MP likely varies in a large range. Even supposedly identical model MP particles have substantially different ζ-potentials, affecting their interactions with cells (Ramsperger et al., 2021)	Adsorbed POPs and heavy metals (Rochman et al., 2014; Hirai et al., 2011; liang Liao and yan Yang, 2020) have a controversial role in MP toxicity (Koelmans et al., 2021); formation of ecocoronas and biofilms (Ramsperger et al., 2020; Galloway et al., 2017) enhances cellular internalization (Ramsperger et al., 2020); films possibly carry dangerous pathogens (Kirstein et al., 2016; Imran et al., 2019; Gkoutselis et al., 2021)	Extraordinarily high bio-persistence of conventional polymers (Andrady, 2015); therefore likely very high bio-persistence of MP particles in organisms. Biodegradation of biodegradable polymers usually by specialized microorganisms and conditions limited to industrial composting plants (Millican and Agarwal, 2021; Bagheri et al., 2017), therefore likely high bio-persistence in the lung. Due to bio-persistence accumulation of MP in the organism, leading to chronic exposure (Mohamed Nor et al., 2021)	Mainly diseases due to occupational exposure (Prata, 2018): Pneumoconiosis (Ng et al., 1991; Studnicka et al., 1995), interstitial lung disease (Cortez Pimentel et al., 1975; Kern et al., 1998; Eschenbacher et al., 1999), chronic bronchitis (Cortez Pimentel et al., 1975; Miller et al., 1975), cough and dyspnea (Valic and Zuskin, 1977; Zuskin et al., 1998; Lougheed et al., 1995; Kern et al., 2000), allergic and asthmatic reactions (Cortez Pimentel et al., 1975; Muittari and Veneskoski, 1978), lung cancer (Mastrangelo et al., 2002; Hours et al., 2007; Kern et al., 2011). Cancer in digestive system, large bowel, stomach, and esophagus (Mastrangelo et al., 2002; Vobecky et al., 1978; Gallagher et al., 2015)

ζ-potential of crystalline silica dust is pH-dependent. At physiological conditions, values of about $-30 - -10$ mV have been reported (Dong et al., 2015; Leinardi et al., 2020). Interestingly, freshly fractured crystalline silica is linked to a more intense inflammatory response, potentially due to increased surface radicals (Vallyathan et al., 1995).

There is evidence that biogenic amorphous silica (e. g. from the shells of diatoms) has the potential to adsorb toxic chemicals like polycyclic aromatic hydrocarbons (PAHs), acting as a vector for those chemicals into the body (Rabovsky, 1995). However, it is unclear in how far this contributes to the overall toxicity of silica dust, as the main part of silica-related health effects is caused by crystalline silica dust that is freshly generated during the processing of stone (Silicosis Mortality, 2005).

Due to the high bio-persistence of silica dust, chronic effects play a significant role in silica toxicity (Leung et al., 2012). Because of their low dissolution rates (Rimstidt and Barnes, 1980), crystalline silica particles are extraordinarily durable under physiological conditions. Under conditions similar to the lung, a lifetime of about 100 – 10,000 years has been estimated for crystalline silica microparticles (Jurinski and Rimstidt, 2001).

2.3. Soot

Soot is formed by the incomplete combustion of organic materials such as wood, coal, fossil fuels, and plastics (IARC, 2012). Its physical properties and chemical composition heavily depend on the type of burned material and the specific combustion conditions(Kleeman et al.,

1999). The dominant constituent is elemental carbon, accounting for about 60% of soot mass, followed by inorganic and organic matter, including PAHs (IARC, 2012).

For example, chimney sweeps are subjected occupationally to inhalable soot dust (Pukkala et al., 2009; Hogstedt et al., 2013). The median exposure level of Swedish chimney sweeps to inhalable soot dust was found to be 3.8 mg m^{-3} (Hogstedt et al., 2013). Other occupations with potentially increased soot exposure are industrial, maintenance, or service professions performed near fossil fuel combustion engines, such as truck drivers and miners (Donaldson et al., 2005).

Besides occupational exposure, contamination of the ambient air by environmental soot in particulate matter air pollution is also prevalent. Typical ambient exposure levels to soot particles < 10 µm close to a moderately frequented urban street lie in the range of 20–60 µg m^{-3} (Boogaard et al., 2010).

Chimney soot is classified as carcinogenic by the International Agency for Research on Cancer (IARC) (IARC, 2012). The discovery of high incidences of scrotal cancer in chimney sweeps during the 18th century was the first documented case of occupational cancer (Pott, 1775; Benmoussa et al., 2019). Although scrotal cancer due to soot exposure is nowadays mostly preventable, increased incidences of cancers of the lung and the esophagus, among other types of cancer, are still reported for chimney sweeps (Pukkala et al., 2009). Additional epidemiological evidence exists for their occupational risk of asthma and ischemic heart disease (Li et al., 2008; Hansen, 1983). Apart from occupational health risks, ambient exposure to airborne particulate matter, including soot, is also a well-known factor associated with

adverse health impacts. Soot-associated diseases include respiratory and cardiac diseases, which can increase mortality in exposed cohorts (Janssen et al., 2012; Lee, 2010). It is also discussed whether ambient exposure to soot particles is associated with an increase in cancer. For example, diesel exhaust is classified as carcinogenic by the IARC (IARC, 2014). However, whether ambient exposure to diesel exhaust particles (DEPs) is associated with diseases like lung cancer is still controversial (Stöber and Abel, 1996; Hesterberg et al., 2012). One difficulty in assessing the risk of ambient soot exposure is disentangling the specific health impacts of soot and other constituents of airborne particulate matter. Nevertheless, some evidence points towards the fraction of black carbon in the overall particulate matter mass being a robust indicator for adverse health effects (Janssen et al., 2011).

The toxicological pathways leading to the observed health effects of soot likely depend on the soot particle types. Usually, oxidative stress and inflammatory responses are involved (Donaldson et al., 2005). For example, upon exposure to DEPs, proinflammatory signaling pathways increase cytokine expression in vitro *(*Terada et al., 1999*)* and in human lungs in vivo *(*Salvi et al., 2000*)*. Apart from inflammatory responses, ROS generated by soot particles has a mutagenic potential, possibly leading to cancer upon prolonged exposure (Watson and Valberg, 2001; Barfknecht, 1983).

The physicochemical properties of soot particles (Fig. 1C, Table 1) depend on the source material and the combustion conditions (Torvela et al., 2014). Size distributions of particles from wood smoke, for example, were found to peak at 0.1–0.2 μm in diameter. Particles from cigarette smoke displayed a distribution peak between 0.3 and 0.4 μm (Kleeman et al., 1999). Soot particles from highly efficient combustion processes, including modern diesel-fueled engines, have sizes in the order of tens of nanometers, which agglomerate to structures with a typical size of several hundred nanometers (Fig. 1C) (Watson and Valberg, 2001; Omidvarborna et al., 2015). During the combustion of heavy oil fuels, particles with an average size of 10–100 μm are formed. In domestic chimney soot, particles > 1 μm up to 1 mm are found (Watson and Valberg, 2001). After their release into the atmosphere, particle aggregation and the condensation of organic matter increases the soot particles' size (Janssen et al., 2012; Watson and Valberg, 2001; Patterson and Kraft, 2007). There is evidence that the soot particles' size affects their toxicity (Frank et al., 2013). In one study, the bioreactivity of soot in the size range of 70–110 nm was increased compared to smaller and larger particles, leading to an increased expression of cytokines (Sarkar et al., 2014).

The shapes of soot particles are similarly diverse. Particles formed from highly efficient combustion processes aggregate to form aciniform, grape-like structures (Fig. 1C) (Watson and Valberg, 2001; Omidvarborna et al., 2015). During the combustion of heavy fuel oils, cenospheres (hollow spheres) can be formed. In domestic chimney soot resulting from wood or coal burning, xerogels and irregular pieces of coke and char are prevalent. Xerogels are porous, carbonaceous fragments with an extraordinarily high surface area (Watson and Valberg, 2001). Potentially, the shape of soot particles can affect their toxicity. For example, by activating molecular oxygen on their surface, an increasing curvature might lead to a stronger reactivity of the soot particles (Frank et al., 2013).

The ζ-potential of DEPs lies mainly between − 50 to − 30 mV under physiological conditions (Sarkar et al., 2014; Chen and Huang, 2017). It was found that DEPs in the zeta-potential range between − 41 mV and − 37 mV showed bioreactivity in immune cells. This leads to an increase in cytokine expression and inflammatory responses (Sarkar et al., 2014).

An essential parameter in soot toxicity is adsorbed PAHs, such as benzo(a)pyrene (Barfknecht, 1983; Kirrane et al., 2019). These organic compounds condensate on soot particles during the combustion process and after their release into the environment (Lee, 2010; Watson and Valberg, 2001; Eaves et al., 2017). Consequently, soot particles are a well-known exposure route to PAHs, known for their carcinogenicity (Lee, 2010). The fraction of PAHs and other organic compounds is

associated with the mutagenicity of soot particles (Medalia et al., 1983).

The bio-persistence of soot particles might contribute to their toxicity. From experiments with DEPs in lung cells of rats, the half-life time of persistence in the lung is known to lie between 80 and 250 days, while the clearance efficiency decreases with the exposure level (Griffis et al., 1983; Wolff et al., 1987).

2.4. Wood dust

The main components of wood particles are cellulose and lignin. Most commonly, wood dust is generated during the processing of wood. Typical exposition levels for woodworkers are (0.2 ± 2.6) mg m^{-3} for wood dust particles with a size < 4 μm, (0.3 ± 2.8) mg m^{-3} for particles in the size range 4 – 10 μm, and (1.5 ± 2.7) mg m^{-3} for dust in the size range 10 – 100 μm (Glindmeyer et al., 2008).

According to the IARC, wood dust is classified as carcinogenic to humans. In their report, the IARC concludes that hardwood is associated with the formation of adenocarcinoma of the nasal cavities and para-nasal sinuses, whereas, at the time of the study, the correlation was less clear after exposure to softwood dust (Delzell, 1995). Since then, multiple meta-analyses have been published, strengthening the conclusion of the IARC of a strong association between lung cancer and wood dust (Alonso-Sardón et al., 2015; Binazzi et al., 2015; Hancock et al., 2015).

Another significant risk associated with wood dust are pulmonary diseases. For example, an increased risk for cryptogenic fibrosing alveolitis, a disease characterized by severe inflammation and fibrosis of the deep respiratory tract, was found in patients exposed to wood dust (Hubbard et al., 1996). Furthermore, idiopathic pulmonary fibrosis, which leads to the thickening of lung tissue, is significantly associated with exposure to both soft- and hardwood dust (Gustafson et al., 2007).

The pathogenesis of diseases associated with wood dust exposure includes inflammatory responses and the generation of ROS. For example, the carcinogenicity of particulate matter, including wood dust, is potentially linked to an immune response and the subsequent generation of ROS (Knaapen et al., 2004). Furthermore, although the exact mechanisms leading to idiopathic pulmonary fibrosis are still unclear, inflammation is likely involved (Gustafson et al., 2007). The role of ROS in wood dust toxicity was investigated in several in vitro studies, exposing mouse macrophages and human leukocytes to different hard- and softwood dust (Naarala et al., 2003; Klein et al., 2001).

Additionally, increased cyto- and chemokines expression was observed upon exposure to wood dust, indicating an inflammatory response (Long et al., 2004; Määttä et al., 2005, 2006). Interestingly, this was independent of the individual wood types, with similar inflammatory responses for soft- and hardwood dust (Määttä et al., 2006). An inflammatory response to wood dust was also observed in vivo in mice. In these studies, eosinophil cell counts were increased upon wood dust instillation, and lymphocytes and neutrophils migrated into the lung of the mice. Furthermore, the presence of these immune cells was associated with an increase in proinflammatory chemokines. Strikingly, no such effects were observed in the same experimental setup upon exposure to titanium dioxide particles of the same size, indicating that these immune system responses were wood-specific (Määttä et al., 2006).

Physical properties of wood dust (Fig. 1D, Table 1) likely depend on the wood type and processing technique. For example, when sampling directly at the milling of wood, the share of particles smaller than 125 μm is only between 1% and 15%, while larger particles make up most of the wood dust (Očkajová et al., 2020). However, the aerodynamic diameters of wood dust particles collected at breathing height at working places measure between 10 μm and 100 μm (Harper et al., 2002). Wood dust samples directly generated by sanding of hard and softwood shows a smaller aerodynamic diameter in the breathable range of < 10 μm (Määttä et al., 2006; Ojima, 2016; Marková et al., 2018), and particles of similar size were generated by manual filing (Fig. 1D).

Like the wood particles' size, also their shape depends on wood type and processing technique. Usually, the shape of wood dust particles is

complex and irregular, with a rough surface (Fig. 1D) (Mazzoli and Favoni, 2012; Liu et al., 1985). How the wood dust particles' shape affects their toxicity, has not been studied yet.

The ζ-potential of intact spruce wood capillaries was between − 10–+10 mV at pH 5.6 (Muff et al., 2018). Cellulose nanofibers produced from cedar wood powder at pH 6.8–7 showed a ζ-potential of − 50 - − 30 mV (Uetani and Yano, 2012). However, the effect of the ζ-potential on wood dust particle toxicity is not known.

The ROS production strongly depended on the individual wood types, possibly linked to different natural chemical compounds (e.g., terpenes in softwoods and polyphenolic compounds in hardwoods) (Naarala et al., 2003). However, chemicals used for wood treatment (e. g., chromate compounds) are also discussed as potential drivers of wood dust toxicity (Klein et al., 2001).

The very high bio-persistence of wood likely enhances wood dust toxicity in the lung. Biodegradation of lignin and cellulose requires enzymes found in specialized microorganisms (Slavin et al., 1981; Pérez et al., 2002; Eriksson et al., 1990). There is evidence that wood cellulose fibers persist in rat lungs even a year after exposure and that their estimated half-life is about three years (Muhle et al., 1997).

2.5. Cotton dust

A natural organic airborne pollutant is cotton dust, which is generated during the processing of cotton. It consists mainly of cotton fibers and contains bacteria, fungi, and other plant materials such as cotton stems and leaves (Ayer and Mackison, 1974). Cotton dust exposure in textile mills varies between different processes and mills; total dust concentrations in the range of about 0.1–10 mg m^{-3} were observed in a study investigating five textile mills and fourteen different processes (Hammad et al., 1981).

Increased exposure of cotton industry workers to cotton dust is associated with diseases such as asthma, bronchitis, byssinosis (a disease associated with breathing difficulties and chest tightness), and unspecific respiratory problems (Castranova et al., 1996; Dangi and Bhise, 2017). A 15-year follow-up observation showed that the cumulative incidence of byssinosis was 24% among cotton textile workers. In addition, chronic bronchitis and cough were more common and persistent than in the control group (Wang et al., 2003).

Several parameters (Fig. 1E, Table 1) affect the toxicity of cotton dust. The particles in cotton dust are grouped according to their size into 'trash' (> 500 μm), 'dust' (50–500 μm), 'micro dust' (15–50 μm) and 'breathable dust' (< 15 μm) (Dangi and Bhise, 2017). Usually, the breathable fraction of cotton dust is linked to adverse effects on animal and human health (Ellakkani et al., 1984).

Mill-collected cotton dust contains a significant fraction of fragmented fibers (Fig. 1E) and large pieces of plant material. The individual cotton dust particles are often irregularly shaped (Goynes et al., 1986).

To date, there have been no investigations of the ζ-potential of breathable cotton dust.

The diseases related to cotton dust exposure are likely not directly caused by the cotton particles but primarily by bacterial endotoxins or residual pesticides adsorbed to the cotton dust, possibly releasing mediators inducing inflammations (Wang et al., 2003; Rylander, 1987; Solbrig and Obendorf, 1985). Consistently, after exposing guinea pigs to breathable cotton dust, all treated animals showed a respiratory response, whereas no response was observed when the animals were exposed to pristine cellulose powder with the same particle size distribution (Ellakkani et al., 1984).

Cotton is likely very bio-persistent, possibly contributing to its chronic toxicity, because it contains a large amount of cellulose. As already mentioned in the section on wood dust, the degradation of cellulose requires enzymes that are only present in specialized microorganisms (Eriksson et al., 1990). In rat and mouse lungs in vivo, cellulose fibers were very persistent and had an estimated half-life of up to 3 years (Muhle et al., 1997; Ilves et al., 2018). Consistently, in lung

airway lining fluid and phagolysosomal fluid in vitro, there were no significant signs of degradation of cellulose after up to 9 months of exposure (Stefaniak et al., 2014).

2.6. Hay dust

Exposure to hay dust has been linked to occupational diseases of the lung. Apart from organic debris, pollen and toxins such as endotoxins and mycotoxins are significant contaminants in hay dust (Séguin et al., 2010). The exposure levels to hay dust vary broadly. For example, during the operation of a bedding chopper, the total dust level was found to be in the range of about 10–70 mg m^{-3} (Olenchock et al., 1990). Farmer's increased exposure to hay dust can lead to various respiratory symptoms; most prominently, the inhalation of dust from moldy hay can lead to the so-called farmer's lung disease (Gregory and Lacey, 1963; Siegel et al., 1991). Farmer's lung disease is the most common form of extrinsic allergic alveolitis; it is classified into an acute, subacute, and chronic stage (Reboux et al., 2007). Interestingly, also farm animals like dairy cows exposed to hay dust displayed asthma-like symptoms (Siegel et al., 1991).

The physicochemical properties of hay dust (Fig. 1F, Table 1) may contribute to its toxicity. For example, about 95% of hay dust particles are smaller than 5 μm, enabling them to enter the bronchioles upon inhalation (Séguin et al., 2010; O'Connor et al., 2013). Their shape is primarily spherical, although irregularly shaped and rod-like particles also occur in hay dust(O'Connor et al., 2013). It is not known whether their shape affects the hay dust particles' toxicity. To date, there have been no investigations of the ζ-potential of hay dust particles.

The most prominent parameter affecting hay dust toxicity are microorganisms, endotoxins, and mycotoxins associated with the hay dust (Séguin et al., 2010). Usually, these are the cause of the farmer's lung disease (Reboux et al., 2007; Cano-Jiménez et al., 2016). Since not the hay itself but inhaled microorganisms cause farmers' lung disease, studies focus on identifying relevant antigens present in the hay dust. The primary treatment of the disease is avoiding the antigens (Cano-Jiménez et al., 2016).

Moreover, like cotton, hay mainly consists of cellulose, likely making it very bio-persistent (Eriksson et al., 1990; Muhle et al., 1997; Ilves et al., 2018; Stefaniak et al., 2014).

2.7. Comparability of non-plastic microparticles and MP

In this section, we focused on different non-plastic microparticles of various materials that potentially determine their physicochemical properties. Some of these non-plastic microparticles are chemically more related to MP than others. For example, wood, cotton, and hay (Sections 2.4–2.6) consist mainly of cellulose, an organic polymer, and are therefore more similar to MP than asbestos or silica, which are inorganic crystals (Sections 2.1 and 2.2). MP, usually consisting of synthetic polymers, is a diverse group of contaminants with a wide range of physical and chemical properties (Rochman et al., 2019). However, although chemically very different, the physicochemical properties governing the different non-plastic and plastic microparticles' interactions with cells and tissues might still be comparable (Table 1). Similarities of particles in size, shape, ζ-potential, adsorbed molecules and microorganisms, and bio-persistence may explain similarities in their toxicity and the underlying toxicological mechanisms. Therefore, a comparison of the properties and toxicology of non-plastic particles with MP can improve the understanding of the role of physicochemical properties in MP toxicity.

3. Airborne MP

3.1. Occurrence of airborne MP pollution

Since the topic of MP pollution is relatively new and research mainly

focused first on aquatic and then terrestrial ecosystems, the number of literature on airborne MP is still very small (Enyoh et al., 2019). Nevertheless, first attempts to monitor airborne plastic pollution have been made.

There are different methodological approaches to identify MP in the atmosphere. For example, some studies investigated the MP concentration in dust samples either by directly collecting the dust (Liu et al., 2019; Abbasi et al., 2019; Yukioka et al., 2020) or by investigating the content of a vacuum cleaner bag (Dris et al., 2017). Furthermore, the atmospheric fallout has been measured by dry and wet sample collectors with different opening diameters (Allen et al., 2019; Cai et al., 2017; Dris et al., 2016), by collecting plant leaves with deposited particles on their surfaces (Liu et al., 2020), through direct filtering of the air (Dris et al., 2017; Li et al., 2020) or by using a human breathing thermal manikin (Vianello et al., 2019). However, the data's comparability is limited by the differences in sampling techniques and inconsistencies in data presentation, often quantifying particle fallout per area, particle count per volume, or particle mass per volume. Several earlier reviews focused on these issues (Mbachu et al., 2020; Amato-Lourenço et al., 2020; Huang et al., 2020; Enyoh et al., 2019; Can-Güven, 2021; Chen et al., 2020).

Airborne MP particles, including fibers, fragments, and films, have been found in various environments. Many reports focused on the occurrence of MP in urbanized areas. For example, Abbasi et al. monitored the MP pollution in street dust and the atmosphere (Abbasi et al., 2019). The dust samples mainly consisted of a heterogeneous mixture of MP shapes, whereas fibers were the predominant type of shape in atmospheric samples. This study is in concordance with numerous other studies, showing that the predominant type of MP in atmospheric samples are fibers (Liu et al., 2019; Cai et al., 2017; Dris et al., 2016; Szewc et al., 2021), with a higher amount found in indoor than in outdoor samples (Liu et al., 2019; Dris et al., 2017).

However, MP within the atmosphere is not restricted to urbanized areas. For example, Allen et al. sampled the atmospheric fallout in remote areas of the Pyrenean mountains and found MP fibers, films, and fragments in all samples (Allen et al., 2019). In contrast to other, more urbanized sampling locations, MP fragments (< 50 μm) were the predominant type. Sahara dust in these samples possibly indicates that MP fragments in the atmosphere can be transported similarly (Allen et al., 2019). Furthermore, airborne MP were also detected in the remote marine atmosphere of the Atlantic (Trainic et al., 2020) and the South China Sea as well as the East Indian Ocean (Wang et al., 2020) and were discussed to be able to travel hundreds to thousands of kilometers due to their irregular and elongated shapes (Trainic et al., 2020; Wang et al., 2020).

3.2. Properties of airborne MP

MP contamination of the atmosphere includes particles of all sizes (Table 1). Different studies report MP fibers ranging from smaller than 20 μm to 5 mm in length (Cai et al., 2017; Li et al., 2020; Szewc et al., 2021), with a predominant length of 100–700 μm (Allen et al., 2019; Cai et al., 2017). Detected MP fragments ranged from 5 μm to 750 μm (Szewc et al., 2021) and films from 10 μm to 1520 μm (Szewc et al., 2021). However, the reported size ranges are heavily influenced by sampling and detection limits, often making it challenging to quantify contamination of the atmosphere with microscopic particles (Zhang et al., 2020). To assess potential risks associated with airborne MP pollution, improved tools for the study of very small MP < 10 μm will be required (Vethaak and Legler, 2021).

A study on atmospheric fallout of MP particles in Paris mentions a count of 118 fallout particles per square meter per day (p m^{-2} d^{-1}), ranging from 29 p m^{-2} d^{-1} to 280 p m^{-2} d^{-1} (Dris et al., 2015). Consistently, at different urban and suburban sites, fallouts of fibers of (110 ± 96) p m^{-2} d^{-1} and (53 ± 38) p m^{-2} d^{-1} respectively were reported (Dris et al., 2016). Similarly, fallouts of 175–313 p m^{-2} d^{-1} occurred in an urban area in China (Cai et al., 2017). The corresponding MP fiber

concentrations at human respiratory height were reported to be between 0.3 and 20 particles per cubic meter (p m^{-3}) indoors and 0.1–0.5 p m^{-3} outdoors, the most abundant fraction of fibers being in the size class of 50–250 μm, which was the lowest size reported in this study (Dris et al., 2017). Furthermore, a study conducted in the catchment of the River Weser in Germany reported a mean concentration of 46 p m^{-3} of airborne MP only in the size range between 4 and 10 μm (Kernchen et al., 2021). All studies reported a large variability of observed particle numbers, possibly correlated with the amount of rainfall, time of year, and sampling site (Dris et al., 2017, 2016, 2015). Similarly, up to 4.18 p m^{-3} of suspended atmospheric MP particles were reported in Shanghai (Liu et al., 2019).

3.3. Inhalation and ingestion of airborne MP

Inhalation and ingestion are the main exposure routes of airborne MP to humans (Fig. 2A). Based on the results above, and assuming an average breathing volume of 6 l min^{-1}, it was estimated that humans might be exposed to 26–130 airborne MP of different sizes per day (Prata, 2018). A study using a human breathing thermal mannequin to simulate human exposure to indoor airborne MP contamination found even higher numbers since they measured 272 p d^{-1} of inhaled MP (Vianello et al., 2019). A critical shortcoming of these reports is their limited detection of MP smaller than 50–100 μm, likely leading to a gross underestimation of reported MP contamination. For example, in a study with a lower detection limit of 5–13 μm, about 70% of the detected MP fallout particles were smaller than 63 μm. Overall, a fallout of 136–512 p m^{-2} d^{-1} was reported (Klein and Fischer, 2019). More recently, a study with a lower detection limit of 0.8 μm reported a MP fiber concentration of about 5600 p m^{-3} at human respiratory height. More than 80% of all detected airborne particles were smaller than 20 μm (Li et al., 2020). Consequently, assuming an average breathing volume of 6 l min^{-1}, humans might be exposed to more than 48,000 MP p d^{-1} by inhalation. These findings reinforce the concerns about airborne MP contamination raised by earlier studies (Prata, 2018; Vianello et al., 2019). Furthermore, ingestion of airborne MP mainly occurs due to the fallout of atmospheric MP during meal preparation and consumption. Approximately, humans consume 40 – 190 p d^{-1} in this way (Catarino et al., 2018).

3.4. MP in the lung

Once inhaled, MP can infiltrate the lung depending on its aerodynamic diameter (Fig. 2B). Especially MP with an AED < 10 μm has the potential to penetrate the lower respiratory tract (Prata, 2018; Martens and Jacobi, 1973; Foord et al., 1978; Williams et al., 2011; Lippmann et al., 1980). There is evidence that plastic fibers can penetrate the human deep lung (Pauly et al., 1998; Amato-Lourenço et al., 2021), and plastic fibers were present in the lower respiratory tract after inhalation by rats (Porter et al., 1999; Warheit et al., 2003). In addition, it has been shown that MP particles can penetrate the lungs' surface lining layer and are taken up by endothelial cells (Geiser et al., 2003, 2005; Deville et al., 2015; Goodman et al., 2021). Clearance (clearance from the respective organ, not necessarily the organism) of MP from the lung occurs via two main pathways: via the mucociliary escalator and via an internalization by pulmonary macrophages, the first enabling transport of inhaled MP into the gastrointestinal tract (Lippmann et al., 1980; Geiser et al., 2005).

3.5. MP in the gastrointestinal tract

Airborne MP may enter the gastrointestinal tract via inhalation and the mucociliary escalator (Fig. 2B) and by settling onto food during preparation and consumption (Catarino et al., 2018). Particles > 50 μm are likely excreted from the gastrointestinal tract. Schwabl et al. (2019) reported 20 MP particles larger than 50 μm per 10 g in human stool samples (Schwabl et al., 2019). To which extent smaller MP are excreted from the gastrointestinal tract is not yet investigated. Other than
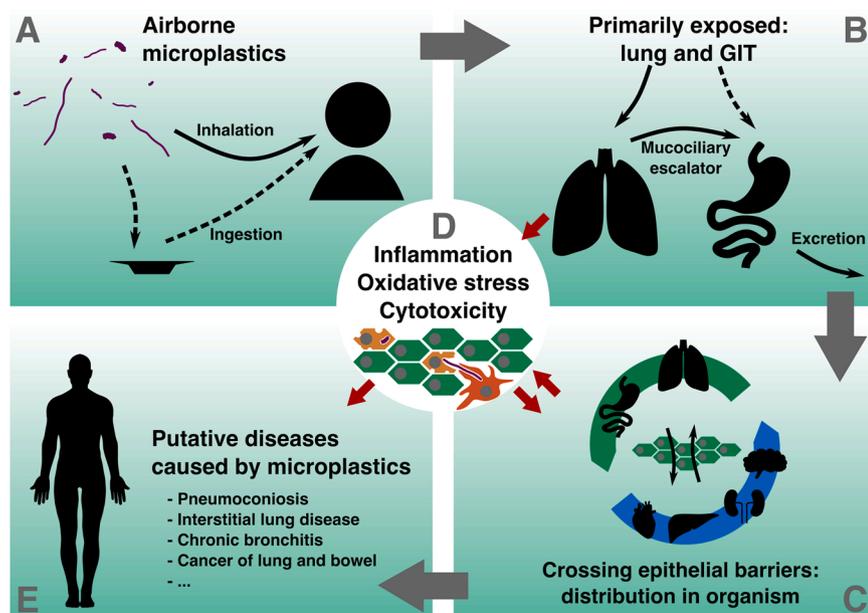
Fig. 2. Potential pathways of MP toxicity. A) Airborne MP can enter the human organism either by inhalation or ingestion, primarily exposing the lung and gastrointestinal tract (GIT) (Prata, 2018; Catarino et al., 2018). B) MP particles in the upper airways can be cleared via the mucociliary escalator, translocating these particles into the GIT, from which a share of the particles will be excreted (Lippmann et al., 1980; Schwabl et al., 2019). However, some of the MP particles are translocated deeper into the tissues of the lung and GIT(Geiser et al., 2005; Volkheimer, 1975; Carr et al., 2012; Sanders and Ashworth, 1961; Lefevre et al., 1989; Jani et al., 1989; Des Rieux et al., 2005). C) By crossing epithelial barriers, MP can also enter the lymphatic system and bloodstream (Volkheimer, 1975; Jani et al., 1989; Eyles et al., 2001). From there, MP particles can distribute in the whole organism, ultimately reaching different secondarily exposed organs (Volkheimer, 1975; Jani et al., 1989, 1990; Urban et al., 2000; Eyles et al., 2001; Deng et al., 2017; Wick et al., 2010; Ragusa et al., 2021). D) In exposed tissues, MP particles may cause inflammatory responses and oxidative stress, eventually leading to cytotoxicity (Geiser et al., 2005; Goodman et al., 2021; Mahadevan and Valiyaveettil, 2021). These effects reduce the epithelial barrier resistance, increase MP's mobility in the organism (Hamoir et al., 2003; Yacobi et al., 2008; Di Dong et al., 2020), and may potentially cause additional adverse effects on human health. E) Among others, diseases like pneumoconiosis (Ng et al., 1991; Studnicka et al., 1995), interstitial lung disease (Cortez Pimentel et al., 1975; Kern et al., 1998; Eschenbacher et al., 1999), chronic bronchitis (Cortez Pimentel et al., 1975; Miller et al., 1975), and cancer of the lung and bowel might be associated with MP (Mastrangelo et al., 2002; Hours et al., 2007; Kern et al., 2011; Vobecky et al., 1978; Gallagher et al., 2015). These diseases are prevalent in exposed workers of the flocking and VC/PVC industry (Prata, 2018).

excretion, important clearance mechanisms from the gastrointestinal tract are para- and transcellular internalization of MP into the surrounding tissues. It has been shown that MP can pass the epithelial layer between enterocytes in a paracellular manner (Volkheimer, 1975). For the transcellular translocation of microparticles, including MP, the uptake by M-cells into the Peyer's patches (lymphoid follicles in the small intestine) plays a significant role (Carr et al., 2012; Sanders and Ashworth, 1961; Lefevre et al., 1989; Jani et al., 1989; Des Rieux et al., 2005). Furthermore, it was shown that the translocation efficiency strongly depends on particles size (Carr et al., 2012; Jani et al., 1990). For example, the total uptake efficiency into rat gastrointestinal mucosa of polystyrene (PS) nanoparticles with a size of 50 nm and 100 nm was around 33% and 26%, respectively, much higher compared to 1 µm sized MP, of which only 4.5% were taken up (Jani et al., 1990). Nevertheless, studies report varying magnitudes of translocation efficiency, ranging from 0.04% to 4.5% for particles in the micrometer size range in rodent models (Carr et al., 2012; Jani et al., 1990).

3.6. Distribution of MP in the organism

MP that was displaced into tissues can become internalized by resident tissue macrophages (Geiser et al., 2005). Macrophages as professional phagocytes do not only internalize micrometer-sized bacteria (Underhill and Goodridge, 2012), but also microparticles with different surface properties (Ramsperger et al., 2020; Desjardins and Griffiths, 2003; Irmscher et al., 2013; Kress et al., 2007). Especially the uptake into Peyer's patches in the interstitium by M-cells enables interactions of MP with various immune cells and the subsequent transport to the lymphatic system (Jani et al., 1989). Also, paracellular transport allows MP to move from tissues into the lymphatic system (Volkheimer, 1975). Macrophages not only transport microparticles intracellularly (Keller et al., 2017), but they can also act as 'transporters' for MP inside the organism, distributing the MP through the lymphatic system to different organs (Walker and Bullough, 1973; Urban et al., 2000). Moreover, particles that have been displaced into the lymphatic system can enter the bloodstream (Eyles et al., 2001). There is also evidence for MP being

directly transferred from primarily exposed organs into the bloodstream (Volkheimer, 1975). Once in the blood, MP distributes in the whole organism and can be found in various secondarily exposed organs (Fig. 2C).

Many studies report evidence of MP migrating to various organs in humans and different model species, including mice, rats, and dogs. MP have been found in the liver (Volkheimer, 1975; Jani et al., 1990; Deng et al., 2017), spleen (Jani et al., 1990; Urban et al., 2000; Eyles et al., 2001), kidneys and urine (Volkheimer, 1975; Deng et al., 2017), bone marrow (Jani et al., 1990), brain and cerebrospinal fluid (Volkheimer, 1975), and the placenta (Wick et al., 2010; Ragusa et al., 2021). Interestingly, inflammatory responses at sites of MP exposure can reduce the integrity of epithelial barriers and activate macrophages. In this way, the mobility of MP in the body might be increased (Hamoir et al., 2003; Yacobi et al., 2008; Di Dong et al., 2020). Finally, the MP particles can irreversibly accumulate in tissues over the human lifetime (Mohamed Nor et al., 2021).

3.7. MP-associated diseases

There are some reports of MP-associated diseases (Fig. 2E, Table 1), most of which focus on people working in the textile, flocking, and VC/PVC (vinyl chloride/polyvinyl chloride) industries. In these industries, the concentration of airborne MP can reach extraordinarily high levels, up to 7–40 mg m^{-3} or 10^6 particles m^{-3} (Bahners et al., 1994; Burkhart et al., 1999). Due to these high levels of exposure to MP, it was possible to link occupational diseases to MP pollution. Many of the reported effects occurred in the lung and the gastrointestinal tract, the primarily exposed organs. A recent review discusses occupational diseases resulting from MP exposure (Prata, 2018). Briefly, there were reports about pneumoconiosis (Ng et al., 1991; Studnicka et al., 1995), interstitial lung disease (Cortez Pimentel et al., 1975; Kern et al., 1998; Eschenbacher et al., 1999), chronic bronchitis (Cortez Pimentel et al., 1975; Miller et al., 1975), and various other acute and chronic symptoms, including cough and dyspnea (Valic and Zuskin, 1977; Zuskin et al., 1998; Lougheed et al., 1995; Kern et al., 2000). Apart from that,

occupational exposure to MP has been correlated to allergic and asthmatic reactions (Cortez Pimentel et al., 1975; Muittari and Veneskoski, 1978). Additionally, studies reported a significantly increased risk of these workers for lung cancer (Mastrangelo et al., 2002; Hours et al., 2007; Kern et al., 2011).

Because of their high exposure to MP, workers in the textile and flocking industry are also subject to gastrointestinal tract diseases. Cases of cancer in the digestive system, large bowel, stomach, and esophagus that were assumed to be due to their high exposure were also reported (Mastrangelo et al., 2002; Vobecky et al., 1978; Gallagher et al., 2015). In addition, more recent in vivo studies on mice found decreased intestinal mucus and microbiome dysbiosis (a disruption of the gut microbiome) after exposure to MP (Lu et al., 2018; Jin et al., 2019).

There are also MP-associated diseases reported in secondary organs. Interactions of human red blood cells with PS MP led to blood cell aggregation and attachment to endothelial cells in vitro (Barshtein et al., 2016). In rats, injection of PS microspheres led to vascular occlusions, inflammation, and pulmonary embolism (Zagorski et al., 2003; Jones et al., 2003). *In vitro*, it was shown that exposure of sheep blood to MP induced hemolysis (Hwang et al., 2019). In lymph nodes of patients with total hip arthroplasty, granular histiocytosis caused by wear particles from the artificial joints, including MP, was observed (Hicks et al., 1996). In mice, inflammation of the liver and disturbed hepatic lipid metabolism were observed upon exposure to MP (Deng et al., 2017).

3.8. Toxicology of MP

Inflammation and oxidative stress contribute to the toxicity of MP (Fig. 2D). For example, in tissues, MP was observed to cause aggregations of macrophages, granulation tissue, and a foreign body response (Urban et al., 2000; Willert and Semlitsch, 1996; Doorn et al., 1996). Consequently, granulomas with a tendency towards necrosis are formed, leading to fibrosis and scarring of the tissue (Willert and Semlitsch, 1996). Inflammatory responses are regulated and enhanced by cytokine production. Upon exposure to PS particles, increased IL-6 and IL-8 production, inflammation, and oxidative bursts in multiple human and murine monocyte and macrophage cell lines were observed (Di Dong et al., 2020; Brown et al., 2001; Prietl et al., 2014).

Similarly, PE (polyethylene) MP also caused increased interleukin and TNFα production (Green et al., 1998; Nich and Goodman, 2014). Increased cytokine production can activate nearby macrophages, enhancing inflammatory responses in exposed tissues (Hicks et al., 1996; Nich and Goodman, 2014; Morawski et al., 1995; Devane et al., 1995). These inflammatory responses lead to the release of oxidizing species (Sternschuss et al., 2012). Furthermore, MP can carry oxidizing radicals on their surface due to weathering, inducing oxidative stress in cells (White and Turnbull, 1994; Gewert et al., 2015). Oxidative stress can also be enhanced by releasing adsorbed oxidizing chemicals, e.g., metals (Kelly and Fussell, 2012; Valavanidis et al., 2013).

Inflammatory responses and oxidative stress may induce cytotoxicity. For example, in hamster BHK-21 cells, PVC and PMMA (polymethyl methacrylate) MP with a size of 0.12 μm and 0.14 μm, respectively, induced increased concentrations of ROS, leading to reduced cell viability (Mahadevan and Valiyaveettil, 2021). Similarly, reduced viability and striking morphological changes were observed in human lung A459 cells upon exposure to PS MP (Goodman et al., 2021). Moreover, genotoxic effects may result from MP exposure due to mechanic stress and leaching of monomers and additives, again leading to cytotoxicity and possibly cancer (Çobanoğlu et al., 2021).

Apart from that, MP may act as a vehicle for toxic chemicals to enter the organism. Studies showed that the leaching of monomers and additives from MP contribute to their toxicity (Porter et al., 1999; Mastrangelo et al., 2002; Xu et al., 2003). Furthermore, chemical compounds, including persistent organic pollutants and heavy metals, can adsorb to MP and can be released in the organism once ingested (Rochman et al., 2014; Hirai et al., 2011; liang Liao and yan Yang,

2020). These chemical pollutants may not only be cancerogenic (Chen et al., 2019), but also act as endocrine disruptors in organisms (Chen et al., 2019). Endocrine-disrupting chemicals can threaten fertility and reproductive health and affect the regulation of many vital functions (D'Angelo and Meccariello, 2021). For example, upon exposure to PS MP in mice, changes in serum neurotransmitters and increased acetylcholinesterase activity have been reported, potentially leading to neurotoxicity (Deng et al., 2017).

4. Potential drivers of MP toxicity

4.1. Size

A common way of classifying ambient microparticles is to group them according to their size (Kelly and Fussell, 2012). Due to their potential to enter the lower respiratory tract, especially particles with an AED < 10 μm are toxicologically relevant (Prata, 2018; Kelly and Fussell, 2012). This is the case for all microparticle types covered by this review article, including MP (Section 3). Furthermore, the transport of microparticles inside the organism takes place more readily for smaller micro- and nanoparticles (Kelly and Fussell, 2012). These fine and ultrafine particles increase the health risk due to their higher probability of passing epithelial barriers and because their reactivity with cells and tissues is increased (Kelly and Fussell, 2012; Geiser et al., 2005). In epidemiological studies about particulate pollution associated with road traffic (among others containing soot particles, Section 2.3), it was reported that fine PM2.5 and ultrafine particle pollution has stronger adverse health effects than larger particles (Schwartz et al., 1996; Dominici et al., 2006; Mirowsky et al., 2013). Although ultrafine particles substantially contribute to the toxicity of ambient particulate matter, it is not sufficiently well understood to what extent coarser particles are associated with adverse health effects (Kelly and Fussell, 2012). For MP particles, it has been shown in vivo in rats and in vitro in Mono Mac 6 cells that smaller particles at the lower size limit of MP and nanoparticles have stronger proinflammatory effects than larger particles (Brown et al., 2001). Their increased biological activity might be associated with the larger total surface area of smaller microparticles, leading to enhanced interactions between microparticles and cells (Kelly and Fussell, 2012; Schmid and Stoeger, 2016; Brown et al., 2001). Thus, the small PM10 and ultrafine MP particles are potentially important drivers of adverse effects on human health. Since their small size also enables them to enter the respiratory tract, we suggest for future studies quantifying airborne MP to focus on particles with an AED < 10 μm. This will be important to assess the risk for human health caused by airborne MP.

4.2. Shape

Interactions of microparticles with cells and tissues also depend on the microparticles' shape. Some pollutants, including asbestos and natural textile fibers like cotton (Sections 2.1 and 2.5), come in elongated fiber or rod shapes. Irregular shapes are prevalent in other airborne particles, like silica dust and soot particles (Sections 2.2 and 2.3). MP particles are found various shapes (Section 3): elongated fibers, for example, released by textile abrasion; irregularly shaped fragments, among others resulting from the natural degradation and fragmentation of larger plastic items; and well-defined spherical particles, often added as primary MP to cosmetic products. Various MP shapes were detected so far in the atmosphere, with fibers being the most common type. However, it has to be noted that this could be caused by the sampling and analytical methods used (Cai et al., 2017; Dris et al., 2016; Szewc et al., 2021).

Of all particle types, asbestos is a very prominent example of how fiber-shaped particles induce toxicity. The thin, long, inorganic fibers can enter the lung, where resident tissue macrophages try to clear them by phagocytosis. Due to the fiber length exceeding the typical

macrophages' size, the internalization process can often not be completed, which can cause chronic inflammation and cancer (Section 2.1) (Donaldson et al., 2010; Padmore et al., 2017). This 'frustrated phagocytosis' is a well-studied mechanism relevant to the toxicity of various fibers from chemically inert materials. For glass and titanium dioxide fibers, it was shown that frustrated phagocytosis leads to increased toxicity once a critical fiber length of 12–15 µm is exceeded (Padmore et al., 2017; Hamilton et al., 2009). There is evidence that fiber-shaped particles might have increased toxicity for titanium dioxide compared to spherical particles (Allegri et al., 2016). Possibly, next to the fibers' length, their bending stiffness might be determining their toxicity, affecting whether they can be crumpled and consequently phagocytosed (Lehmann et al., 2019). Since frustrated phagocytosis mostly depends on the particle geometry, this might be a highly relevant toxicological mechanism for all fiber-shaped microparticles, including MP fibers.

Workers in the textile and flocking industry exposed to high levels of MP fibers exhibit conditions that may be caused by fiber toxicity and frustrated phagocytosis, like pneumoconiosis and lung cancer (Section 3.7). However, to what extent frustrated phagocytosis is associated with these occupational diseases remains to be studied. Furthermore, to date, a large proportion of in vitro research on MP toxicity focuses on spherical PS particles, neglecting MP fiber pollution and other types of polymers (Lim, 2021). Therefore, to get a clearer view of the risks associated with MP fibers, we propose to increase the focus on MP fibers in future in vitro toxicity studies. Here, especially breathable MP fibers with a diameter up to 3 µm (AED < 10 µm) should be considered (Lippmann, 1990; Morgan, 1995). Furthermore, it will be crucial for risk assessment to identify the breathable portion of MP fiber pollution in indoor environments. Here, a high level of textile abrasion is expected, and a significant fraction of the world population spends most of their time indoors (Schweizer et al., 2007).

4.3. ζ-Potential

Another important physicochemical property for the assessment of MP toxicity may be the particles' ζ-potential. The ζ-potential of microparticles depends amongst others on their surface charge, resulting, for example, from functional surface groups. In the fabrication of plastic products, surface functionalization is a common method to adjust material properties (Johansson, 2017). Thus, MP particles most probably cover a broad range of ζ-potentials. It was recently shown that even supposedly identical model MP particles have substantially different ζ-potentials (Ramsperger et al., 2021). Also, once exposed to the environment, functional surface groups might be generated on MP particles due to photodegradation of the particles (Meides et al., 2021). For example, carboxylic groups can result from UV exposure of PET (polyethylene terephthalate), PE, PP (polypropylene), and acrylate polymers (Fechine et al., 2004; Fernando et al., 2007; Decker and Zahouily, 1999). Thus, weathering might alter the ζ-potential of MP particles by introducing negatively charged surface groups. It has been shown that the interaction of microparticles with cells changes with the particles' ζ-potential, indicating that hydrophobic interactions and electrostatic forces may play a crucial role in cell-particle interactions (Liu et al., 2013; Shao et al., 2015).

Furthermore, the cytotoxicity of polymeric, metal, and mineral nano- and microparticles has been linked to the particles' ζ-potential (Shao et al., 2015; Kaur and Tikoo, 2013; Motskin et al., 2009). For example, a study focusing on the interaction of *E. coli* and *D. magna* with differently coated silver particles showed that the strength of interaction and particle toxicity depends on the magnitude of the difference in ζ-potential between particle and organism (Silva et al., 2014). Similarly, for asbestos (Section 2.1), with increasing magnitude of the ζ-potential also the hemolytic activity increased (Light and Wei, 1977). In addition, more negatively charged asbestos fibers have been linked to increased ROS production (Pollastri et al., 2014). Due to electrostatic interactions,

similar results may be expected for the interaction of MP particles with mammalian cells. For example, Ramsperger et al. recently reported enhanced interactions between MP particles and cells for particles with a higher $|\zeta|$, leading to an increased cytotoxicity (Ramsperger et al., 2021). Accordingly, the ζ-potential of MP particles might be an essential proxy for their toxicity. We think that it should therefore always be considered in future in vitro and in vivo effect studies with MP particles.

4.4. Adsorbed molecules and organisms

Additionally, microparticle toxicity might be altered by toxins, biomolecules, and pathogens adsorbed to the particles' surface. For example, soot particles (Section 2.3) adsorb organic matter and PAHs during the combustion process and after their release into the environment (Lee, 2010; Watson and Valberg, 2001; Eaves et al., 2017). PAHs are usually classified as hazardous due to their carcinogenicity (Lee, 2010). The inhalation of soot particles present in road dust is a well-known exposure route to PAHs (Kelly and Fussell, 2012; Lee, 2010). Like soot, MP particles can introduce humans to potentially toxic chemicals. This issue has been covered by several earlier reviews (Prata, 2018; Rochman et al., 2014; Prata et al., 2020; Campanale et al., 2020). For example, it is controversially discussed whether PAHs, persistent organic pollutants, and heavy metals may adsorb to MP in the environment, enabling them to enter the organism (Hirai et al., 2011; liang Liao and yan Yang, 2020; Koelmans et al., 2021).

Similarly, adsorbed biomolecules and pathogenic microorganisms can contribute to the toxicity of microparticles. For natural organic microparticles like hay dust, toxicity likely results from bacterial endotoxins and mycotoxins of microorganisms colonizing the hay particles (Section 2.6). Similar observations have been made for MP particles. Upon exposure to the environment, a biofilm or ecocorona may form on the particles' surface (Ramsperger et al., 2020; Ramsperger et al., 2020; Galloway et al., 2017). These films may carry potentially dangerous pathogens, including fungal pathogens and antibiotic-resistant bacteria (Kirstein et al., 2016; Imran et al., 2019; Gkoutselis et al., 2021).

Additionally, biomolecules found in the ecocorona of MP particles increase particle-cell interactions (Ramsperger et al., 2020) and potentially carry endotoxins and mycotoxins that cause an immune response leading to allergies. A similar effect leads to the farmer's lung disease upon exposure to hay dust (Section 2.6). Therefore, ecocorona and biofilm formation upon environmental exposure of MP should be considered when assessing the risk of airborne MP contamination. For future studies, we suggest including biodegradable polymers in this kind of research. MP particles made up from these materials might present an additional carbon source for the colonizing microorganisms, similar to natural organic particles like hay and cotton. Therefore, these particles might be preferable for microorganisms, possibly increasing the toxicity emerging from their environmental exposure.

4.5. Bio-persistence

All the modes of microparticle toxicity mentioned above also depend on the particles' residence time in exposed tissues. Therefore, the bio-persistence of inhaled microparticles has been discussed as a critical parameter for their toxicity (Oberdorster et al., 1994). Microparticles that reside in the lung for several months or even many years, like asbestos or crystalline silica dust (Sections 2.1 and 2.2), are often associated with chronic inflammation, potentially leading to abnormal tissue growth (fibrosis) and cancer (Goodman et al., 1999; Wagner, 1997; Leung et al., 2012). Furthermore, non-degradable particles may accumulate inside the organism when exposed repeatedly, potentially leading to adverse health effects even at low environmental concentrations (Noonan, 2017; Metintas et al., 2002). In addition, certain types of microparticles which are often perceived as biodegradable, such as cellulose-based particles like cotton or wood dust (Sections 2.4 and 2.5), can be very bio-persistent inside mammalian organisms (Harper et al.,

2002; Eriksson et al., 1990). Although specialized fungi and microorganisms can enzymatically decompose these materials, they are not degradable in the lung due to a lack of the necessary enzymes (Eriksson et al., 1990; Stefaniak et al., 2014). Likewise, MP particles are likely very bio-persistent. Conventional carbon-based polymers are not biologically degradable and thus extraordinarily biodurable (Andrady, 2015), besides few exceptions like the enzymatic degradation of PET by the bacterium *Ideonella sakaiensis (*Tanasupawat et al., 2016*)*.

Moreover, nominally biodegradable polymers like PLA (polylactic acid) or PBAT (polybutylene adipate terephthalate) are under environmental conditions often very bio-persistent: Effective degradation of these plastics is usually limited to industrial composting plants (Millican and Agarwal, 2021; Bagheri et al., 2017). Thus, these polymers, like conventional plastic, may not be decomposed in the lung and other exposed tissues. Because of their potential to accumulate in the organism (Mohamed Nor et al., 2021), we want to stress the importance of investigating the magnitude of breathable MP pollution in environments relevant to human exposure. We suggest for future studies to focus especially on indoor areas, where high levels of breathable MP pollution are expected.

4.6. Other possible drivers of toxicity

4.6.1. Surface roughness

Effects of microparticles on cells likely depend on their surface area (Kelly and Fussell, 2012; Schmid and Stoeger, 2016; Brown et al., 2001). However, with increasing surface roughness, the effective surface area of the microparticles also increases, potentially reinforcing interactions between microparticles and cells (Gatoo et al., 2014). For example, for chitosan microspheres, it was shown that cells adhere preferentially to rough particles (Zan et al., 2008). Stronger particle-cell interactions may potentially lead to enhanced particle toxicity (Silva et al., 2014). The different particle types we focused on in this review strongly vary in their surface roughness. Some particles, like PS microspheres readily employed for MP research (Section 3), usually have smooth surfaces, whereas other particle types like silica, soot, or wood particles (Sections 2.2–2.4) are more irregularly shaped and rough (Fig. 1). Since MP consists of various polymers with different properties, the surface roughness likely differs considerably between MP particles. However, to date, there is no well-established uniform quantification of the surface roughness of MP, making it difficult to assess its potential role in MP toxicity. We suggest for future research on MP toxicity that the surface roughness of model particles should be considered as one parameter that potentially impacts health effects. Since the surface area of micro- and nanoparticles has been discussed as the most effective dose metric for their toxicity (Schmid and Stoeger, 2016), it may be insightful to normalize effects to the effective surface area of MP particles, including surface roughness, which may be measured by gas adsorption to the particles using the Brunauer-Emmett-Teller theory (BET surface area) (Schmid and Stoeger, 2016; Ono-Ogasawara and Kohyama, 1999).

4.6.2. Chemical identity, monomers, and additives

Although some of the toxicological impacts of microparticles on human health can be attributed to universal physicochemical properties like size, shape, and ζ-potential, the chemical identity of microparticle pollution should not be neglected. Especially plastic is a highly diverse class of materials, consisting of numerous polymer types frequently used in various applications. It covers a broad spectrum of physical and chemical properties, often tuned by surface functionalization (Johansson, 2017). This complexity likely leads to a large diversity of MP particles containing a mixture of chemicals like unreacted monomers and additives, potentially contributing to their toxicity (Prata, 2018; Rochman et al., 2019; Prata et al., 2020). To date, most research on MP toxicity is based on PS microspheres (Lim, 2021; Jacob et al., 2020). This type of model MP alone might be insufficient to assess the risks associated with airborne MP contamination because different plastic types in

the environment may lead to the formation of different MP particles that contain different monomers and additives. Thus, we think that future studies should take a more realistic suite of model MP particles from different polymers with different additives and surface functionalization into account. This can lead to a more complete understanding of the health risks associated with airborne MP pollution.

5. Conclusions and future perspectives

Identifying the potential drivers of MP toxicity is a crucial step towards better assessing the potential health risks caused by breathable MP pollution. Here, we focused on the potential role of the microparticles' size, shape, ζ-potential, adsorbed molecules and microorganisms, and bio-persistence in the toxicity of airborne MP. By reviewing the adverse effects on human health associated with six different, well-studied airborne non-plastic microparticle pollutants, we aimed to identify the role of these parameters in microparticle toxicity. We then compared our results to already existing knowledge about the putative toxicity of airborne MP, providing a basis for identifying possible ways in which the abovementioned parameters affect MP toxicity. The parameters we considered in this review article are likely relevant for assessing risks associated with airborne MP particles. To better understand their role, we suggest for future studies to consider the following points:

- Smaller MP particles may not only be more readily respired but may also interact stronger with cells and tissues due to their relatively larger surface area, making them toxicologically more relevant. Therefore, it will be essential to quantify the magnitude of breathable (AED < 10 μm) MP pollution in outdoor and indoor environments.
- Their shape can affect how MP particles interact with cells. Especially fibers might have the potential to induce adverse effects by causing frustrated phagocytosis, leading to persisting inflammation. Thus, we propose that in vitro toxicity assays focus not exclusively on spherical microparticles but include particles of different shapes, especially fibers.
- The ζ-potential of MP particles is an easy-to-measure property that might proxy their interactions with cells and tissues and their surface reactivity. Thus, it should always be specified according to a consistent protocol in in vitro and in vivo toxicity studies to ensure comparability between studies.
- Environmental exposure of MP might significantly alter its toxicity due to the adsorption of toxic molecules, ecocoronas, and microorganisms. Therefore, to understand their potential role in MP toxicity, environmentally exposed MP should be included in effect studies.
- Since conventional and biodegradable MP is likely not degradable in the human organism, it can accumulate over the human lifetime, leading to chronic toxicity. Therefore, it will be crucial to assess the MP pollution in all areas that are relevant to human exposure.

However, other parameters than the abovementioned likely also affect the toxicity of airborne MP. For example, the surface roughness of MP might contribute to its toxicity by enlarging its effective surface area. Furthermore, the polymer type of an MP particle might determine its physicochemical properties and likely play a role in its toxicity. In addition, the leaching of potentially toxic monomers and additives can contribute to the toxicity of MP from different polymers.

Overall, understanding how microparticle properties influence adverse health effects may lead to a more fundamental understanding of MP toxicity and support risk assessments of MP pollution. However, since all these parameters likely play a combined role for MP toxicity, and due to the highly diverse nature of MP particles, a broad range of mechanistic knowledge will be required to enable effective policy-making regarding MP pollution.

Declaration of Competing Interest

Author contributions

H.K. initiated the article. All authors contributed to the conceptualization of the article. H.K., C.L., and S.W. supervised the writing process. S.W., C.T., and BH.W. wrote the introduction, C.T. wrote Section 2.1, F. M. Section 2.2, J.K. Section 2.3, J.B. Section 2.4, A.B. Sections 2.5 and 2.6, AFRM.R. and S.W. Section 3. S.W. wrote Section 4 with the help of F.R., and S.W. wrote the conclusions. All authors reviewed the manuscript and contributed to its finalization. J.B. designed the graphical abstract. S.W. designed Figs. 1 and 2 and performed the SEM preparation and imaging.

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