Report

3D Printing: Health Effects of Ultrafine Particle Emissions

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Health Effects of Ultrafine Particle Emissions

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Introduction

The rapidly expanding field of 3D printing has recently boomed with the development of low-cost, compact, and user-friendly printing devices designed for personal use in the office, classroom, and at home. These devices can be used to manufacture simple objects on a small scale, and do so by utilizing a technique known as molten polymer deposition (MPD). During this process, a thermoplastic filament is forced through a heated extrusion nozzle, which melts the feedstock and deposits the extruded plastic onto a baseplate in thin layers, eventually forming a three dimensional solid shape (Bumgarner 2013).

There are various types of thermoplastic feedstock in use today, including acrylonitrile butadiene styrene (ABS), polylactic acid (PLA), polyvinyl alcohol (PVA), polycarbonate (PC), and high density polyethylene (HDPE), with ABS and PLA being the most commonly used(Stephens, Azimi et al. 2013). The primary variances between printers of different feedstock types are nozzle and baseplate operating temperatures. For example, PLA, a bio-degradable, corn-based plastic, requires a nozzle temperature of around 180°C and a baseplate temperature near room temperature. ABS, however, requires a higher nozzle temperature (~220°C) and a baseplate temperature of around 80°C (Weinhoffer 2012).

High temperature processing of thermoplastics has been shown to emit both gases and particles (Contos, Holdren et al. 1995, Unwin, Coldwell et al. 2013). The effect of toxic gases (such as carbon monoxide and hydrogen cyanide) on the human body are well understood, but the effect of inhaled particulates, specifically ultrafine particles (UFPs: particles with a diameter less than 100nm), are of great concern, and are continually being studied. UFPs have unique aerodynamic and biochemical properties that, when inhaled, allow them to reach the deepest part of the lungs (Oberdorster, Oberdorster et al. 2005), where they can then enter tissues and cells, interact on a molecular level, and directly (and indirectly) modify the immune response mechanisms (Chang 2010). This can lead to serious adverse cardiovascular and pulmonary effects in humans (Warheit, Sayes et al. 2008).

Physiology and Toxicology of UFP in the Respiratory and Other Biological Systems.

Several factors determine the toxicity of UFPs, including particle size distribution, surface area, surface structure and reactivity, and mass concentration (Delfino, Sioutas et al. 2005, Sioutas, Delfino et al. 2005, Warheit, Sayes et al. 2008). For example, data from toxicity studies in rats demonstrate that exposure to UFPs (<100nm) is more toxic than exposure to bulk particle-types (>100nm) of similar chemical composition(Donaldson, Brown et al. 2002). Such differences in physiochemical characteristics may alter the potency of the particle, resulting in a broad range of cytotoxic and inflammatory

endpoints. When compared to larger particles, UFPs have order of magnitudes higher particle number concentration and larger concentrations of adsorbed or condensed toxic air pollutants per unit mass, i.e. mass is proportional to volume, while contact is proportional to surface area (Oberdörster 2001). The structure of the particle affects the surface area, which in turn, dictates its ability to adsorb or condense with other air pollutants. For instance, agglomerate structures, which are irregular in shape, have higher surface areas than spherical particles of equal diameter (Sioutas, Delfino et al. 2005), increasing its ability to deposit adsorbed pollutants into the lungs.

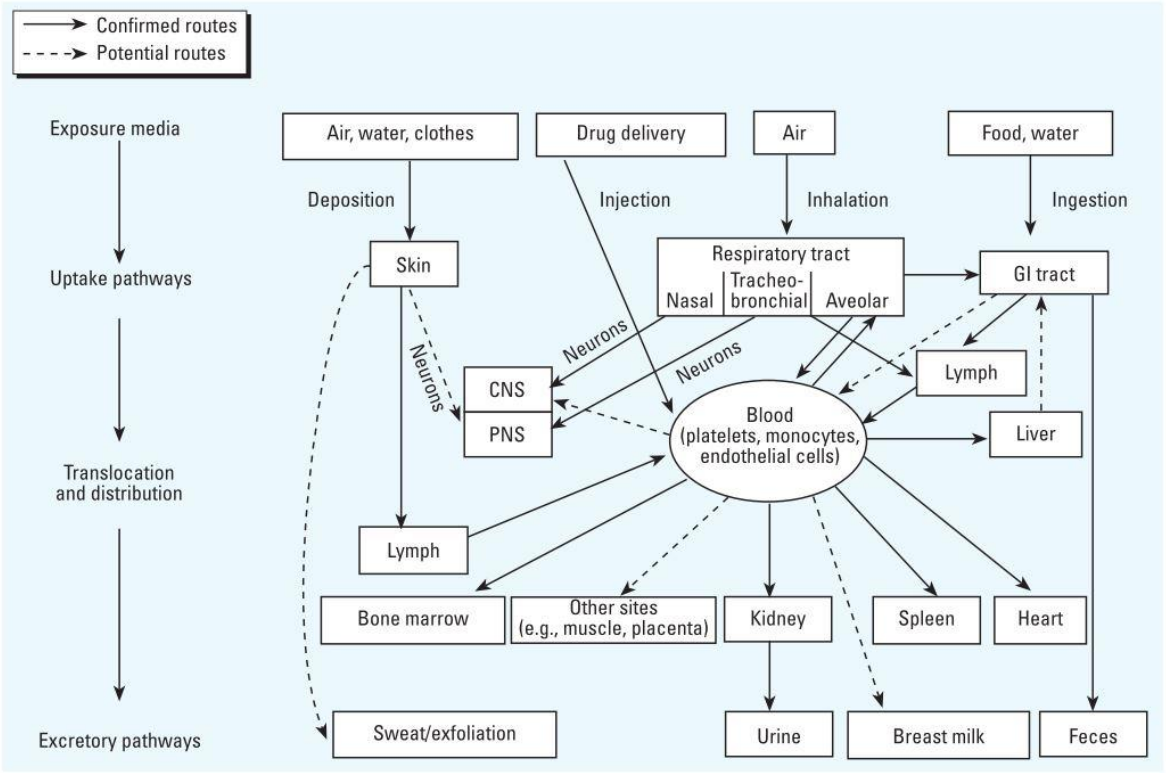

Figure 1. Biokinetics of UFPs. Uptake, translocation, accumulation, and retention rates, as well as potential adverse effects, largely depend on physicochemical properties of the particle. Source: Oberdorster, 2005. Focusing on the "air" and "respiratory tract" pathway, it is clear that exposure via inhalation can affect multiple systems in the body, depending upon the actual exposure site. Nasal and tracheo-bronchial exposures can affect nervous system function, while deposition into the alveolar region (in addition to the trachea-bronchial region) can affect the circulatory system, including blood cells and lymph, which in turn affect vital organs and bone marrow.

It is important to understand the physical and chemical properties of UFPs in order to understand how they interact with tissues and cells in a living organism. While the small size and large surface area are critical factors, biological activity is dependent upon the presentation of surface moieties on the particle. Upon examining the mechanisms in more details, it has been determined that typically, larger

particles that deposit into the respiratory tract are either phagocytized by macrophages or cleared by mucus. On the other hand, UFPs, because of their small size, can penetrate the airway epithelium and enter cells by endocytosis, where they localize inside mitochondria. Here they are capable of activating pro-inflammatory responses in the immune system, resulting in immune system derangement that can lead to higher incidences of autoimmune, allergic and even neoplastic diseases (Chang 2010). Animal studies using rats have also shown that UFPs induce both apoptosis and proliferation in lung epithelial cells (Sydlik, Bierhals et al. 2006). The primary mechanism behind this response is thought to be the formation of reactive oxygen species (ROS) and free radicals, induced by chemicals (organics and metals) adsorbed onto the surface of the particle (Li, Sioutas et al. 2002, Sioutas, Delfino et al. 2005). While very little is known about the chemical composition of 3D printer emissions, many of the polymers used are organic, and it is quite possible that metals (especially colored transition metals) are used to affect the polymer color, which would produce the harmful vapors that adsorb to particle surfaces. In addition to causing inflammation as a result of the immune response to ROS, macrophage accumulation in alveolar spaces can cause lung tissue thickening and lead to the development of pulmonary fibrosis (Warheit, Sayes et al. 2008). It is also possible for UFPs that translocate from the alveoli to the pulmonary tissues to translocate further into the blood circulation via lymphatic channels or directly via the endothelium. Once in the blood, they can translocate into vital organs, such as the liver (Nemmar 2002, Oberdorster, Sharp et al. 2002). Because UFPs are capable of forming free radicals in the blood when ROS are present, they affect levels of various enzymes and proteins responsible for the regulation of basic cardiovascular functions such as vasoconstriction, smooth muscle relaxation, and red cell adhesion through altered gene expression, which in turn affect cardiac rhythm and blood pressure, and can lead to problems such as inflammation, thrombosis, ischemia, arrhythmia, high blood pressure, decreased heart rate variability, atherosclerosis, and even myocardial infarction (Delfino, Sioutas et al. 2005). An epidemiologic study in Denmark found that exposure to UFPs (likely from traffic-generated pollution) was associated with higher morbidity due to stroke, specifically from mild ischemic stroke of non-cardiac (likely thrombotic) origin (Andersen, Olsen et al. 2010).

Secondary Exposure Routes and Pathways for UFP

While inhalation is the most common route of exposure to UFPs, exposure is not limited to the lungs. In fact, UFPs that are inhaled through the nasal cavity are actually capable of circumventing the blood-brain barrier, and translocate to regions of the brain. (See Figure 1) An experiment in rats showed that up to 20% of UFPs deposited on the olfactory mucosa can translocate to the olfactory bulb along

axons of the olfactory nerve, serving as a portal of entry into the central nervous system (Oberdörster, Sharp et al. 2004). Other routes of exposure include ingestion and dermal uptake. It is well known that people initiate hand-to-mouth contact frequently and, when combined with the knowledge that particles inhaled into the tracheobronchial region are often cleared via the muco-ciliary escalator, it is highly likely that exposure to UFPs occurs in the gastro-intestinal (GI) tract, where they can then translocate to the blood, and are taken up by the liver and spleen (Oberdorster, Sharp et al. 2002). Additionally, UFPs can penetrate the skin and distribute via uptake into lymphatic channels. Experiments have demonstrated that flexing the skin results in penetration of UFPs as small as 1um, and that they are able to enter blood circulation via regional lymph nodes. Skin sensory nerves have the potential to uptake and translocate UFPs as well, similar to what has been observed in the olfactory nerve (Oberdorster, Oberdorster et al. 2005). It is reasonable to assume that ingestion through muco-ciliary clearance, and dermal contact of ultra-fine particles from 3D printing can result in exposure through GI and lymphatic pathways.

UFP Production from 3D Printers

The ability of ultra-fine particles to uptake into the body and cause significant adverse health effects is evident. Several reviews of experimental data examining pulmonary and cardiovascular effects of inhaled UFPs have proposed pathophysiological mechanisms, but presently, no studies have focused specifically on identifying the chemical composition of UFPs released from 3D printing activities. It is of paramount importance that the composition, size distribution, and region of deposition (particularly within the trachea-pulmonary system) of emission from 3D printers be studied further. Analysis of previous epidemiological studies makes it clear that the chemical composition is crucial in determining the body's physiological response to an exposure. A study is currently underway at Georgia Institute of Technology to assess the concentrations, size distributions, and emission rates of particles emitted from 3D printers utilizing various colors of ABS thermoplastic and other feedstocks. By characterizing the emissions from a 3D printer similar to ones that could be used in a classroom or office setting, an estimate of a real-world exposure scenario can be determined.

Several factors help determine the amount of exposure, including ventilation, and particle coagulation, condensation, evaporation, and deposition. From previous studies, it has been shown that coagulation (loss of small particles and gain of large particles due to particle collisions) is a significant aerosol process relating to UFP dynamics and the primary cause for the shift of particle size distribution following an episodic high-concentration UFP release (Rim, Green et al. 2011). This is because smaller

particles have higher mobility, and higher number concentrations than large particles. Deposition is a significant source of particle loss, mainly for larger particles, because as particles coagulate and condense, their size and density increase, making them more subject to the effects of gravity and air flow, causing them to settle onto surfaces, thus being removed from the circulating air. While coagulation is the most significant factor when there is no ventilation, particle deposition loss is just as substantial when there is ventilation through duct work or mechanical systems, because the turbulence of the air flow causes more collisions of particles with walls and ductwork.

Preliminary results of the GT study show that there is a large release of particles at the start of operation, and that over time, the particle emission rate decreases, corroborating what is already known about particle size distribution and evolution. More research is needed to validate the methodology for measuring particles from 3D printers, and to measure differences between types of printers, types of filaments, effects of ventilation and environmental conditions like relative humidity and temperature, as well as to determine the chemical components of 3D printer emissions. These results have implications for using 3D printers, especially in settings where exposure may occur to susceptible populations, such as classrooms, or workplace settings. For those who already own personal, small-scale 3D printers, minimizing exposure to UFPs from emissions is important for preventing adverse health outcomes, and the best way to do that is by only operating under well-ventilated, and where possible, enclosed operation chambers.

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