

Understanding the Risk of Microplastic Dermal Absorption

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ABSTRACT

Microplastics are a growing public health concern globally, posing an unprecedented risk both to the environment around us and to our bodies. The major focus of research and policy so far has been on inhaled and ingested microplastics which contribute greatly to the physiological burden of microplastics. However, despite the continued growth of the skincare and cosmetics industry, there remains a significant research gap in the mechanisms and risks of dermal absorption compared to other routes. The aim of this review is to first outline how the skin functions as a barrier and then how microplastics and secondary particles may enter the skin, leading to damaging effects. Critical factors that may influence dermal absorption are discussed including chemical additives in cosmetic products, particle morphology and existing barrier damage. Understanding how particular environmental and health conditions may influence the absorption of microplastics will help drive regulation and reform around microplastic usage in skincare products. This review highlights the need for more rigorous research to better characterise the risk of microplastic dermal absorption and the mechanisms by which microplastics can bypass the skin barrier to mitigate against them. Through the development of more physiologically relevant models and reproducible experimental designs, understanding of microplastics will continue to improve and hopefully lead to better regulation to limit the health and environmental impacts.

Keywords: *Microplastics, nanoplastics, skin, cosmetics, chemical additives, dermal absorption, particulates, barrier damage*

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INTRODUCTION

Every day, humans consume and absorb one credit card worth (5 grams) of microplastic particulates from the air, food and water weekly (Pletz, 2022). They are found in almost everything around us, from the food we eat to the air we breathe (Yakovenko *et al.*, 2025). Furthermore, they are resistant to biodegradation, so this leads to the bioaccumulation of microplastics in food webs, from planktons all the way to humans, due to trophic transfer, which contributes to the polluting effects on organisms (Provencher *et al.*, 2019).

There are many different routes through which microplastics enter the body, the most common being oral consumption (Cox *et al.*, 2019). Given that microplastics are incredibly resistant to biodegradation, they can persist throughout the food chain, leading to biomagnification in higher trophic levels (Provencher *et al.*, 2019). Given the rise in plastic usage, there is a consequential increase in airborne microplastic particulates as 12% of the plastic waste is incinerated and 79% lie in landfills each year (Rhodes, 2018). Airborne microplastics are generated from landfills due to consistent weather exposure leading to direct dispersion into the air or leeching into water sources followed by evaporation. Microplastics are also produced and released into the atmosphere by many actions performed daily like using a heating drying, wearing car tyres and burning candles (Torres-Agullo *et al.*, 2022). Increased microplastic presence in the atmosphere and throughout our ecosystems has led to growing concerns regarding the effects of inhaled and ingested microplastics. Once within the body, microplastics do not remain static but can translocate, via the circulatory system, to distant regions, bringing about deleterious effects (Gałęcka and Całka, 2024). Whilst current understanding of these effects is in its infancy, the recent advances in understanding the impact of microplastics on human physiology will be discussed in detail within this review.

Research has largely focused on oral and nasal routes of administration given the presence of microplastics in our atmosphere, water and food. However, a crucially underappreciated route is the skin, the body's largest organ. Covering more than 15-16% of our body weight, it is constantly exposed to the world around us and acts as an essential barrier (Tobin, 2006). Therefore, the skin is our "brain on the outside", a complex and semi-autonomous system, continuously interfacing with the surrounding environment, and communicating crucial homeostatic signals to the rest of our body (Tobin, 2006). This is why caring for our skin, particularly making sure it is not damaged, is crucial to protect the barrier function of skin. The growth of the skincare industry, driven by shifting mentalities around healthcare and longevity, and in parallel with rising plastic pollution, poses an unprecedented risk of microplastic exposure (Erat and Addor, 2025). Skincare products may not only play a role in helping protect the skin and reinforce barrier function but could also provide a further route for microplastics to enter the body.

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WHAT ARE MICROPLASTICS

According to Frias and Nash, microplastics can be defined as “a synthetic solid particle or polymeric matrix, with regular or irregular shape and with size ranging from 1 μm to 5 mm, of either primary or secondary manufacturing origin, which are insoluble in water” (Frias and Nash, 2019, p. 2). Exposure of these microplastics to heat and fluid can lead to shedding which drives the formation of irregularly shaped secondary microplastics and nanoplastics, the latter of which are particles on the scale of 1 μm or less (Belioka and Achilias, 2024). Microplastics consist of common synthetic polymers including polyethylene, polypropylene, polystyrene, polyvinyl chloride, polyethylene terephthalate and nylon, all of which can be found in products used in daily life (Frias and Nash, 2019). These common polymers can form a polymeric matrix which often contain non-plastic components such as chemical additives that give a plastic the desired properties like flame retardants and UV stabilisers. However, these matrices can also absorb environmental contaminants such as heavy metals and organic pollutants, trapping them within the structure and acting as a carrier (Frias and Nash, 2019).

The microplastics differ largely by the plastic that constitutes them, and there are many different types. Firstly, polyolefins are the most common plastic, known for their hydrophobic nature and buoyancy. These consist of low density polyethylene (used in plastic bags, food bags and squeeze bottles) and polypropylene (used in bottle caps, food containers and disposable cutlery) (Giaganini *et al.*, 2023). Secondly, there are polyesters that are used in synthetic textile fibres, single use bottles and food containers (Thakur *et al.*, 2023). Thirdly, polystyrene microplastics are typically rigid but can also be foamed. When foamed, the expanded polystyrene is used in packaging materials found on coffee cups for insulation (Giaganini *et al.*, 2023). Fourthly, there are polyamides, more commonly known as nylon, and they are known for their strength and durability (used in textiles, fabrics, fishing nets and strings) (Giaganini *et al.*, 2023). Lastly, there are biodegradable polymers like polylactic acid (found in compostable bags), polyhydroxyalkanoates and polyhydroxybutyrate (found in biodegradable packaging and medical implants), and polycaprolactone (found in biodegradable polyurethane foams and biomedical applications) (Thakur *et al.*, 2023).

However, a crucial element of microplastics mentioned earlier was the non-plastic substances absorbed onto the polymeric matrix (Frias and Nash, 2019). Microplastics are a complex mixture of the plastic polymer itself and a cocktail of added or adsorbed chemicals, which has the potential to deeply affect the body on a physiological level (Ali *et al.*, 2025). Furthermore, the matrix-structure carries not only environmental pollutants but may also act as a vector for potential pathogens. Microplastics may therefore act as a trojan horse which, coupled with the effects of microplastics and environmental contaminants, makes them a growing health concern to be further investigated (Fabra *et al.*, 2021).

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THE SKIN AS A BARRIER

The skin is the body's primary barrier and largest sensory organ, accounting for more than 10% of body mass (Walters and Roberts, 2002). It comprises the epidermis, dermis, and hypodermis. The outermost layer is the Stratum Corneum, the primary function of which is to provide a semi-permeable barrier that prevents excessive water loss and blocks the entry of pathogens, allergens, and environmental contaminants (Benson and Watkinson, 2011). It is approximately 0-20 micrometers thick and built from flattened, dead keratinocytes called corneocytes, embedded in a lipid-rich extracellular matrix. The lipid matrix in particular is important for regulating permeability and creating the main transport pathway for substances whilst the corneocytes act as the structural "bricks" of the barrier. These protein-dense, keratin-filled cells provide mechanical strength and resilience of the skin (Benson and Watkinson, 2011). Beneath the Stratum Corneum lie the viable epidermis and dermal layer which are essential for barrier formation, immunity and structural support. Within the epidermis, enzymes are continually secreted by keratinocytes which facilitate the breakdown of cellular components and lipids during the process of cornification, which is crucial for forming the protective barrier (Benson and Watkinson, 2011). The Langerhans cells, which are the surveillance cells of the upper layers, serve to detect pathogenic antigens and initiate immune responses. Supporting the epidermis, the dermal layer is separated by a thin papillary layer and provides the immunological, nutritional and neurological network necessary for maintaining the functions of the skin (Walters and Roberts, 2002). Capillaries and nerves are embedded in the dermal papillae that ensure close proximity to the epidermis, allowing for delivery of nutrients and oxygen, removal of waste, and precise detection of touch, temperature, and pain (Walters and Roberts, 2002). This integration is fundamental for the skin's function as both a dynamic barrier and a sophisticated sensory organ (Walters and Roberts, 2002). This system does not merely supply nutrients, it is the primary route for transporting hormonal signals, immune cells, and signaling molecules to coordinate the functions of distant organs (Benson and Watkinson, 2011). Consequently, anything that enters the skin, whether a vital nutrient, a therapeutic drug, or a foreign particle, gains the potential to be rapidly spread throughout the entire body (Benson and Watkinson, 2011).

The final layer of the skin is the hypodermis which acts as a vital insulator, energy reservoir, and mechanical shock absorber (Benson and Watkinson, 2011). It is a network of fat cells (adipocytes) and connective tissue, composed primarily of collagen and elastin, built into a structure of fibrous chambers which compartmentalize the adipose tissue (Jääskeläinen *et al.*, 2023). The complex environment facilitates and supports vascular and neuronal systems whilst also anchoring the skin to the underlying muscles and bones through the deep fascia (a dense layer of connective tissue) (Jääskeläinen *et al.*, 2023).

The main purpose of the skin is to be the first layer of defense and to protect against chemical, environmental and physical trauma (Dainichi, Hanakawa and Kabashima, 2014). However, it is also a crucial

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sensory organ that helps us understand our external environment and helps to regulate homeostasis, the process of maintaining a constant internal environment (Dainichi, Hanakawa and Kabashima, 2014). Nerves within the dermis project towards the epidermis where free nerve endings receive signals about the environment such as temperature, pressure, humidity (Boulais and Misery, 2008). The dermis is also rich with sweat glands and hair follicles which, in combination, respond to changes in the external environment to maintain homeostasis (Boulais and Misery, 2008). Much like nerve endings, hair follicles can communicate information and act as a conduit for large molecules, serving as an important component of both sensory and barrier function (Corey, 2006).

There are several ways within which substances can move across the skin including transcellular, paracellular, and appendageal routes (McGrath, Eady and Pope, 2004). Larger molecules like proteins can migrate through the hair follicles and sebaceous glands towards the circulatory networks in the dermis (McGrath, Eady and Pope, 2004). Smaller, more hydrophobic molecules like endocrine disruptors and industrial solvents are able to move directly across lipid rich stratum corneum without much resistance, a route that is taken advantage of by topical medications such as nicotine or hormone patches (Shaker *et al.*, 2019). Only very small polar molecules can migrate across cell membranes, unless transported, so transcellular routes are not common (McGrath, Eady and Pope, 2004). However, recent evidence has indicated that neurons can act as sites of retrograde transport, meaning the substance or pathogen moves up along the neurons towards their origin, as shown in the Olfactory bulb and vagus nerve in Parkinson's disease (Santos *et al.*, 2019; Amato-Lourenço *et al.*, 2024). The aforementioned pathways all play a role in the sensory functions of the skin but their dysfunction can impair barrier function as larger molecules will be able move through the skin (Rajkumar *et al.*, 2023). Moreover, increasing skin contact to microplastics, which are often lipid soluble, small and abrasive particles, may serve to increase sensitivity to other environmental risks and diseases (Rajkumar *et al.*, 2023).

MICROPLASTICS IN CONTACT WITH THE SKIN

Both micro- and nanoplastics can enter the body via transdermal routes (Menichetti, Mordini and Montalti, 2024). However, several different factors impact the ease of transfer including size, shape, and water solubility. Smaller particles may use transfollicular or intercellular routes, while larger particles are mostly trapped in the stratum corneum or hair follicles (Menichetti, Mordini and Montalti, 2024). The shape of the particle influences the extent of access as a non-spherical, fibrous structure may align along follicles or lipid channels, aiding deeper penetration (Holmstedt, 2016). Surface chemistry and charge also affect the penetrability. Whether the particle is hydrophobic/hydrophilic will affect how the particle transfers into the body with hydrophobic particles favoring lipid-rich intercellular routes while hydrophilic ones prefer trans-follicular routes (Mikušová and Mikuš, 2021). Cations may interact with negatively charged skin surfaces, enhancing adhesion and penetration, indicating how surface charge could affect penetrability. Surface coatings,

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such as chemical additives, may also reduce aggregation, enhance stability, and potentially alter penetration (Cai *et al.*, 2020).

A frequent way in which microplastics come into contact with the skin are skincare and cosmetic goods, particularly facial products. Many such products contain microplastics called microbeads, which are typically used in scrubs and facial cleansers (Giustra *et al.*, 2024). These microbeads are derived from common polymers discussed previously, like polyethylene, and are found at an average concentration of 9.6 mg of microbeads per gram of product (Habib *et al.*, 2020). Particularly in facial scrubs, a single use of these products could release between 9,000 to over 70,000 microbead particles into the wastewater system (Habib *et al.*, 2020). There are some products marketed as "environmentally-friendly" that still contain plastic microbeads, revealing potential issues with misleading marketing and the lack of clear, legal labeling standards. Another type of product are facial cleansers, which are one of the most polluted products, containing over 2.8 million microbeads in a single 150ml bottle (Habib *et al.*, 2020). Some of the particulates are shaped non-spherically meaning there is an increased surface area for absorbing pollutants and potentially leading to abrasion (Habib *et al.*, 2020). These microbeads act as a sink for pollutants by concentrating it on their surfaces and a single microbead could concentrate phenanthrene (an aromatic hydrocarbon found as a waste product of incomplete combustion and pollution) at levels over 100,000 times greater than in the surrounding water (Habib *et al.*, 2020). This builds upon the risk of microplastics acting as vectors for disease and environmental toxins, transporting concentrated doses across the skin and into the body (Bour *et al.*, 2020).

Additional microplastics can be found in cosmetic products, including liquid, soluble and semi-synthetic polymers like acrylates, copolymers, polyquaterniums, and silicones (Giustra *et al.*, 2024). These are used for film forming, conditioning, and thickening, particularly in hair products. Once exposed to increased water temperatures and shear stresses, they are highly likely to break down into smaller microplastics (Giustra *et al.*, 2024). Fragrance encapsulates, either micro- or nanocapsules, are also used to prolong the scent in detergents or fragrances (Giustra *et al.*, 2024). A similar risk of micro and nanoplastic shedding is seen here alongside the continuous exposure of the skin to the encapsulated chemicals.

The microplastics found in cosmetics are extremely complex, and there are interactions that take place between microplastics and other ingredients such as surfactants, preservatives and heavy metals (Giustra *et al.*, 2024). These can all alter the toxicity, transport and environmental impact. More importantly, microplastics are released at every stage of the process: during manufacture, during consumer usage, and during disposal (plastic packaging). Containers themselves degrade and shed microscopic fragments, especially when squeezed, crushed, or exposed to heat and sunlight. This shedding is a direct source of smaller microplastics and nanoplastics, which enter wastewater systems when packaging is rinsed or landfilled, adding a significant and continuous stream of particulate pollution independent of the product's formula. This creates a dual exposure pathway:

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environmental pollution and potential direct dermal absorption, through the water coming in contact with the skin (Menichetti, Mordini and Montalti, 2024).

Currently, there is a paucity of research investigating the passage of microplastics through the skin and it is particularly hard to study in humans as microplastics enter the body through many different sources, making disentangling the origin challenging. In one of the only human studies, microplastics were only assessed after being washed off from different body parts to understand retention (Abbasi and Turner, 2021). Whilst it showed the scalp retained the most microplastics due to larger hair surface area of hair and greater electrostatic forces, there is limited contribution to understanding how microplastics can get through the skin. Alternate approaches like *in vitro* models, systems that use cells and tissues to test hypotheses outside the body, help minimise confounding factors with the drawback of lower complexity (Menichetti, Mordini and Montalti, 2024; Han and Kim, 2025). With this approach, researchers have demonstrated that particles of different sizes are able to permeate the skin barrier to varying degrees and there are several key factors that influence this process outlined in the following section.

FACTORS IMPACTING DERMAL ABSORPTION

There are many factors that may impact dermal absorption, including particle size and surface area, additives, skin condition, temperature and duration of exposure (Larese Filon *et al.*, 2016).

The particle size is a critical factor as smaller particles, like nanoplastics can reach deeper layers, while larger microplastics are mostly limited to the skin surface or hair follicles. Smaller microplastics also cannot penetrate the stratum corneum completely, but can be driven deeper into hair follicles and sweat glands creating a "reservoir effect", allowing for potential interaction with follicular and immune cells (Larese Filon *et al.*, 2016). Nanoplastics on the other hand, pose a greater threat for the body. Since they are significantly smaller than microplastics, they can translocate through the stratum corneum and the lipid matrix, and enter directly into the circulatory and nervous systems. There is a "goldilocks zone" for the optimal size of particles for cellular uptake and dermal absorption, roughly 50nm (Menichetti, Mordini and Montalti, 2024). Particles smaller than this (smaller than 10 nm) may penetrate easily but are also likely to be cleared more rapidly. Particles around 50-100 nm are efficiently taken up by cells through endocytosis and may have a higher chance of persisting and causing biological effects (Menichetti, Mordini and Montalti, 2024).

The shape of the microplastic is also an important factor in determining dermal absorption and there are two elements that influence this: the aspect ratio and the specific shape itself (Menichetti, Mordini and Montalti, 2024). The aspect ratio is the ratio of a particle's length to its width and this influences how flexible the particle is. During absorption, particles can become mechanically embedded, where they can act like tiny

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splinters, anchoring themselves into the stratum corneum or follicular pores and resisting removal (Menichetti, Mordini and Montalti, 2024). Stiffer particles or matrices can activate immune cells via impaired phagocytosis which can in turn trigger inflammation whereas flexible fibers may tangle or fold. Stiff particles are likely to remain in the stratum corneum, while flexible particles are more likely to penetrate deeper layers of the skin (Menichetti, Mordini and Montalti, 2024). Regarding the specific shape, there are three different categories: spheres, plates/2D sheets, and irregular fragments. Most primary microplastics are spherical in shape, with the smallest surface area to volume ratio for any given size and symmetry that allows for relatively predictable diffusion (Filella, 2015). An alternative shape, plates have a very high surface area, maximizing contact and adhesion with lipids or cell membranes (Filella, 2015). They may sit parallel to the skin, decreasing penetration and potentially reducing the absorption of other particles. Finally, due to degradation, the most common shape for secondary microplastics are the irregular fragments (Filella, 2015). They typically have ragged edges with very rough surfaces and will typically cause irritation due to buildup in follicles (Filella, 2015). These fragments create focal points for stress, and mechanical abrasion due to their roughness can physically disrupt the skin's protective lipid barrier, increasing permeability and the risk of inflammation (Filella, 2015).

Another aspect that might affect the ease of dermal absorption are additives commonly found on the microplastics (Akpojevwe Abafe, Harrad and Abou-Elwafa Abdallah, 2024). This not only affects the particle, but it will also alter the skin barrier. Plasticizers for example affect how the particle interacts with the lipid-rich stratum corneum of the skin by producing a surface layer that is more or less hydrophobic than the basic microplastic (Andrady *et al.*, 2023). A more compatible, lipophilic surface can enhance adhesion and penetration. Plasticizers can also make the microplastics more flexible and soft which encourages deeper penetration into the skin, as they can deform and navigate the skin's barrier pathways (McClements, 2010). However, the most crucial issue associated with additives is the leaching of chemicals. This may disrupt the skin's lipid organization, temporarily compromising the Stratum Corneum and making it more permeable for both the chemical itself and the now altered particles (Andrady *et al.*, 2023). Hydrophobic additives dissolving into the skin's lipids can for example create an easier route that micro- or nanoplastics can use, ultimately increasing the pathways through which they can permeate the skin barrier (Aristizabal *et al.*, 2024).

Any break in the epidermis creates a direct physical portal for particles of all sizes to bypass the stratum corneum and enter the deeper dermis (Aristizabal *et al.*, 2024). Chronic wounds like diabetic ulcers and pressure sores may contribute to and propagate microplastic exposure as the wound healing could be further impaired as a result of a contaminated microenvironment (Aristizabal *et al.*, 2024; Menichetti, Mordini and Montalti, 2024). A more common and detrimental form of skin damage would be dermatitis and eczema as these conditions often feature a defective skin barrier, due to genetic mutations in proteins and chronic inflammation (Aristizabal *et al.*, 2024). This will lead to increased transepidermal water loss, a key indicator of barrier dysfunction, widened gaps between keratinocytes, allowing for enhanced paracellular penetration of nanosized

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particles and a pro-inflammatory state that can be aggravated by foreign particles (Aristizabal *et al.*, 2024). Factors such as UV exposure and humidity are also likely to impact the rate of dermal absorption (Egambaram, Kesavan Pillai and Ray, 2020). Changes in humidity and UV will alter the hydration and therefore the permeability of the skin. They may also degrade the skin and help break down the microplastics on the upper layers, leading to the production of more secondary microplastics which are shown to penetrate deeper (Menichetti, Mordini and Montalti, 2024).

Considering all the aforementioned factors, skincare and cosmetics products provide a source of not only irregularly shaped microplastics but also an abundance of potentially harmful chemical additives. The effects of these are not limited to just the skin but also other physiological systems within the body as is discussed in the next section.

HEALTH RISKS ASSOCIATED WITH MICROPLASTICS

An abundance of research in cell-based and animal models has demonstrated that microplastics, both directly and indirectly, lead to cell death and inflammation (Liu and You, 2023; Han and Kim, 2025). Whilst a detailed description of the effects are not in the scope of the review, a broad summary is outlined below with a focus on the dermal route of absorption (Liu and You, 2023; Baroni *et al.*, 2025).

With improvements in detection methods, micro- and nanoplastics have been found throughout the body including the liver, heart, kidneys, brain and placenta (Enyoh *et al.*, 2023). Ingested or inhaled particles damage not only the gut and lungs but can often translocate into the systemic circulation. Particularly, in the gut which is a key interface with the external world, microplastics have a broad spectrum of effects which are somewhat dependent on size, shape and type (Liu and You, 2023). They include changes in the microbial flora, intestinal epithelium and barrier integrity, all of which contribute to and are exacerbated by inflammation (Schneider *et al.*, 2024). From the gut and mouth, oral administration of microplastics has been demonstrated to impact neuronal function in the brain, including memory, social behaviour and motor function (Liu and You, 2023; Baroni *et al.*, 2025). There is also evidence of oral and nasal administration contributing to Parkinson's disease (Liang *et al.*, 2022, 2025). Microplastics, however, are not the sole health concern as they also act as vectors for potentially harmful chemicals. As highlighted previously, heavy metals and chemical additives like phthalates can be found in many different products and can be absorbed to or encapsulated within microplastics. Aside from the common risks of cytotoxicity (cell death), there are growing concerns as to the endocrine disrupting functions of microplastics and such chemicals (Ullah *et al.*, 2023). Due to the shape and composition of some particles, they are able to cross membranes like the blood brain barrier which protects the brain from foreign molecules. Within the brain, they can access centres of hormonal regulation like the hypothalamus and pituitary gland which are crucial to many homeostatic functions (Graceli *et al.*, 2020). In line

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with the presence of microplastics in the placenta, endocrine disruption can lead to developmental abnormalities amongst many deleterious effects (Ragusa *et al.*, 2021). The impact of microplastics alone and as vectors are far reaching, potentially across generations, and the influence on human mood and behaviour has only recently come to light (Ragusa *et al.*, 2021).

The skin, despite being the largest organ of the body, is not rigorously studied in the context of microplastic toxicity. However, there is *in vitro* data from skin models and immune cells that shed light on the interactions taking place within the skin (Menichetti, Mordini and Montalti, 2024; Han and Kim, 2025). Outlined here are several of the ways in which microplastics can lead to and propagate barrier dysfunction and the consequences of that.

As mentioned, some particles are able to pass directly through the outer layer of the skin which is partly aided by chemicals either within solution or absorbed to the particle surface (Menichetti, Mordini and Montalti, 2024; Baroni *et al.*, 2025). Smaller plastics, like those below 1 μm , are able to cross the cell membrane of keratinocytes and fibroblasts found in the deeper layers of the skin. Within these cells, a cascade of processes occurs beginning with disruption of mitochondrial function. This organelle plays a fundamental role in regulating energy homeostasis within the cell and a series of different studies have highlighted the capacity of micro- and nanoplastics to induce oxidative stress via the mitochondria (Xu *et al.*, 2019; Wang, Xu and Jiang, 2023). This leads to activation of pro-inflammatory signalling and apoptotic pathways, which in turn results in cell death and recruitment of immune cells (Liu and You, 2023). Alongside the cytotoxic effects, microplastics can also reduce fibroblast proliferation and migration and impact the expression of cell adhesion molecules, leading to overall weaker barrier function (Han and Kim, 2025). Similarly, rougher microplastics and fragments can cause direct damage to cell membranes, leading to rupture and release of inflammatory signals (Gopinath *et al.*, 2021). Immune cells, like dendritic cells, interact directly with plastic particles and with small molecules released from damaged cells (Han and Kim, 2025). Depending on the surface structure, some nanoplastics can mimic damage associated with molecular patterns (DAMPs) that activate immune cells, stimulating the release of cytokines and chemokines to recruit more cells into the microenvironment (Baroni *et al.*, 2025). Macrophages and other myeloid cells infiltrate, propagating inflammatory signals, restructuring blood vessels and releasing proteases like matrix metalloproteases (Han and Kim, 2025). Breakdown of the extracellular matrix and subsequent remodelling by fibroblasts, coupled with increased blood flow may lead to changes in barrier permeability and easier routes for microplastics to reach systemic circulation.

The effects of microplastics on the body are far reaching and continue to be unveiled as research advances. Of the discussed impacts, a major concern that has arisen is the susceptibility of neuronal cells to microplastics and the associations they may have with neurodegenerative disease and neurodevelopmental conditions (Baroni *et al.*, 2025). Oral and inhaled routes are the main focus in this context as there is a more

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direct pathway for plastic particulates to enter the brain. As an organ directly in contact with the external environment and regularly exposed to high concentrations of microplastics, the skin facilitates uptake of small and potentially detrimental particles that can easily enter circulation. Further research should therefore focus on understanding how dermal absorption of microplastics can contribute to systemic inflammation which predisposes to further diseases.

CONCLUSIONS

In this review, we aimed to highlight the risks of microplastic dermal absorption, an underappreciated route for microplastics to enter the body. Although the focus of ongoing research has been on inhaled and ingested routes, the growing skincare industry paired with increased plastic usage globally may pose a significant and understudied health risk. Whilst the skin barrier is a tightly regulated system, evidence indicates that micro- and nanoplastics can permeate the outer layers and penetrate deep in the epidermis (Menichetti, Mordini and Montalti, 2024). This is facilitated, in part, by the chemicals that are absorbed to the particle surface. Once embedded in the skin, the plastic particulates can perpetuate further infiltration by driving cell death and inflammatory processes that weaken the skin barrier. These effects potentially spread to far-reaching areas given the network of blood vessels and neurons found in the skin and evidence already indicates the presence of microplastics in most major organs (Enyoh *et al.*, 2023). In the context of pre-existing damage and health conditions, or environmental factors like humidity and sun exposure, there is likely to be even more efficient absorption of microplastics across the skin (Menichetti, Mordini and Montalti, 2024). Skincare products may exacerbate this process due to the absorption of and presence of chemicals in solutions with microplastics. With the microplastic particles acting as vectors, they can facilitate delivery of harmful toxins deep into the skin where they will contribute to inflammation and cell death. There is currently a lack of research exploring key questions around the dermal absorption of microplastics despite the growing burden they are likely to exert. Discussed within this review were the impacts of both micro- and nanoplastics, the latter of which are often formed from the degradation and fragmentation of larger plastics. However, most studies investigate uniform particle shapes and sizes which does not accurately capture the real-world impact. Furthermore, research is often aimed at understanding the acute effects by introducing high concentrations of microplastics which potentially dismisses the long-term impacts (Menichetti, Mordini and Montalti, 2024). This also means that long-term exposure is not investigated despite being the major risk factor of microplastics in skincare and cosmetic products. Similarly, factors like humidity and UV exposure are often excluded yet may contribute to the breakdown and absorption of microplastics on the skin. It is incredibly challenging to model the aforementioned challenges for many reasons. Firstly, current technology is restricted to detection of particles down to 200 nm and the images are not resolved sufficiently to study morphology (Baroni *et al.*, 2025). This means that reproducible generation of different particle shapes and sizes is limited until detection methods improve. Secondly, the skin is a complex organ that is challenging to accurately model using *in vitro* systems. Developments in organ-on-a-chip

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methodologies have increased the physiological accuracy of research but cannot yet replace animal or clinical validation. As computational approaches rapidly advance, with the support of artificial intelligence, it may be possible to test hypotheses using *in silico* modeling in tandem with *in vitro* systems.

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