

Mud bath therapy and microplastic elimination from the body of patients affected by rheumatic diseases: a hypothesis

Sir,
Microplastics are plastic particles smaller than 5 mm, including primary microplastics intentionally produced at small sizes and secondary microplastics formed from the breakdown of larger plastic items, originating from sources such as synthetic textiles, personal care products and plastic packaging for food and water. The increasing recognition of microplastics as ubiquitous environmental contaminants has raised concern regarding their potential role in chronic inflammation and immune dysregulation (Table I), including effects relevant to rheumatic diseases (1). Microplastics have been detected in human biological fluids and joint-related compartments (2), indicating systemic distribution following environmental exposure. However, strategies to facilitate the elimination of internalised microplastics remain largely unexplored (3).

Here we propose the hypothesis that heat-based therapies, including hot mud bath therapy, may promote the excretion of certain microplastics or microplastic-associated compounds through thermally induced sweating and enhanced cutaneous clearance mechanisms (Fig. 1). Therapeutic mud baths, long used in rheumatic diseases for pain relief and anti-inflammatory effects, induce sustained hyperthermia, profuse sweating, and increased cutaneous perfusion (4, 5). These responses may influence the mobilisation and elimination of environmental xenobiotics via eccrine sweat glands, which represent a relatively underappreciated excretory pathway for certain substances that are incompletely cleared by renal or hepatic routes, particularly protein-bound or amphipathic compounds. Heat exposure increases dermal blood flow and lymphatic activity, potentially promoting the transport of circulating or tissue-associated microplastics and plastic-derived chemicals toward the skin (3, 6). Preliminary analytical reports suggest that microplastics or plastic additives may be detectable in sweat (7), although

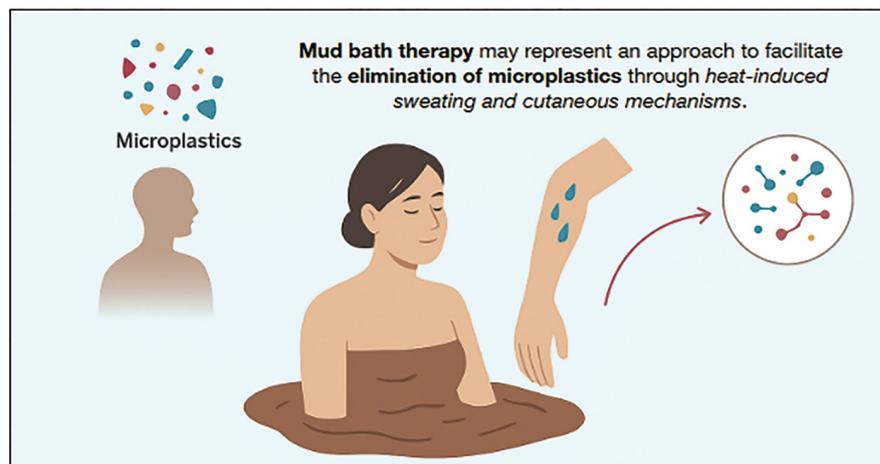


Fig. 1. Mud bath therapy and elimination of microplastics (image created with the assistance of artificial intelligence; all text, scientific content and interpretations were developed and approved by the authors).

systematic data on clearance kinetics are lacking. Mud bath therapy may provide additional mechanisms beyond heat alone: peloids are fine-grained natural materials (such as peat, clay, or marine muds) rich in mineral and organic components and matured in thermal mineral waters. These materials exhibit adsorptive and ion-exchange properties, raising the possibility that mobilised contaminants could be sequestered at the skin surface during prolonged contact (8, 9). Mineral waters used in balneotherapy, which often follows peloid application, may further modulate skin permeability and inflammatory signalling (10). Collectively, these mechanisms support the hypothesis that mud bath therapy could represent a non-pharmacologic pathway for modifying microplastic-associated burden in some rheumatic diseases.

Therapeutic muds themselves may represent a potential source of environmental microplastics, as these particles are ubiquitous in marine, freshwater, and sedimentary environments. Natural peloids can be exposed to microplastics originating from contaminated source waters, sediments, urban runoff, wastewater inputs, or handling and processing procedures. Environmental studies have documented microplastics in Italian coastal and inland sediments, including mud-producing environments (11, 12); however, direct quantitative analyses of microplastics in commonly used therapeutic muds remain scarce. From a health perspec-

tive, dermal exposure is the primary route during mud bath therapy. Intact skin is an effective barrier, and current evidence suggests that systemic absorption of intact microplastic particles is likely minimal, with potential effects expected to be local and largely theoretical. Artificial or formulated therapeutic muds, compared with naturally sourced peloids, are generally produced under more controlled conditions and may allow greater oversight of potential contaminants. Regardless of mud origin, routine analytical monitoring would be useful to ensure safety in the context of increasing environmental plastic pollution.

In conclusion, while this hypothesis remains speculative, it raises several testable questions of relevance to environmental rheumatology:

1. Can microplastics or plastic-associated chemicals be reliably detected and quantified in sweat following thermal therapies?
2. Do repeated heat-based interventions reduce systemic microplastic burden or biomarkers of exposure?
3. Could such interventions modify inflammatory or clinical outcomes in patients with environmentally influenced rheumatic diseases?

We emphasise that controlled clinical and mechanistic studies are required to evaluate efficacy, safety, and reproducibility. Nonetheless, given the established use of hot mud baths in rheumatologic care, investigating

Table I. Selected types of microplastics with potential pro-inflammatory relevance.

Microplastic type	Common sources	Typical form	Proposed pro-inflammatory mechanisms
Polyethylene (PE)	Packaging, plastic bags, films	Fragments	Activation of immune pathways; adsorption of toxins and metals
Polypropylene (PP)	Food containers, medical plastics	Fragments, fibres	Oxidative stress induction; cytokine release in exposed cells
Polystyrene (PS)	Disposable items, insulation	Spheres, fragments	NLRP3 inflammasome activation; macrophage activation (experimental models)
Polyester or nylon	Synthetic textiles	Fibres	Mechanical irritation; carrier for additives and pollutants
Polyvinyl chloride (PVC)	Pipes, flooring, medical devices	Fragments	Plasticizer (e.g., phthalate) release; endocrine and immune modulation

their potential role in mitigating environmental toxicant burden represents a promising and underexplored research avenue. We hope this hypothesis stimulates further discussion and research into non-pharmacologic strategies addressing environmental contributors to rheumatic diseases. For example, future studies could quantify microplastics and plastic-associated additives in sweat, blood, and urine before and after standardised hot mud bath protocols, enabling direct assessment of clearance kinetics, particle characteristics, and correlations with inflammatory biomarkers.

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