International Journal on Science and Technology (IJSAT)
E-ISSN: 2229-7677 ● Website: www.ijsat.org ● Email: editor@ijsat.org

From Contaminants to Cleanse: The Role of Gut Microbes in Heavy Metal Bioremediation

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Abstract
Heavy metal contamination remains one of the most pressing environmental and public health challenges of the 21st century. Exposure to toxic metals such as lead (Pb), cadmium (Cd), mercury (Hg), and arsenic (As) can disrupt essential biological processes, induce oxidative stress, and damage vital organs. However, emerging evidence highlights the gut microbiome as a dynamic defense system capable of mitigating heavy metal toxicity through microbial bioremediation. This process involves the ability of specific gut microbial taxa to bind, transform, sequester, or eliminate metal ions via biosorption, bioaccumulation, methylation, reduction, and precipitation pathways. These interactions not only reduce the bioavailability of toxic metals but also help restore intestinal barrier integrity and immune homeostasis. This paper explores the intricate mechanisms by which gut microbes participate in heavy metal detoxification, emphasizing their roles in metal–microbe interactions, enzymatic transformations, and symbiotic relationships with host physiology. It also discusses how environmental exposure, diet, and antibiotics shape the composition and resilience of the gut microbiome in the face of metal stress. Furthermore, the potential application of probiotic-based bioremediation and microbiome engineering is examined as a promising strategy to enhance detoxification capacity and promote gut health. Understanding these mechanisms offers new perspectives for developing microbiome-driven therapeutics aimed at reducing heavy-metal-induced toxicity, preventing neurodevelopmental disorders, and supporting precision environmental medicine.
Keywords: Gut microbiome, Heavy metals, Microbial bioremediation

1.0 Introduction

Heavy metals (HMs) may contribute to the evolution of many metabolic illnesses, with HM-induced alterations to the gut microbiota serving as an important factor in their genesis and development [1]. The gut microbiota acts as the primary barrier against the harmful effects of heavy metals, exhibiting a dynamic, bidirectional interaction with them [2]. Heavy metals can significantly alter the structure, abundance, and diversity of gut microbial populations, while concurrently affecting their metabolic characteristics [3]. Conversely, the gut microbiota significantly influences the bioavailability of heavy

metals (HMs), affecting their absorption and metabolism via modifying intestinal pH, redox potential, and the expression of genes associated with detoxification processes [4-6]. The microbiota can promote the excretion of heavy metals by bioaccumulation, binding, and enzymatic modification, hence providing protective benefits for the host [7, 8]. Moreover, exposure to heavy metals, especially toxic varieties, might disrupt the gut flora, thereby compromising metabolic and physiological functioning [9]. This disruption may facilitate the initiation or advancement of various ailments, including cardiovascular diseases, neurological disorders, ulcerative colitis, cirrhosis, allergies, diabetes, autism, and other inflammatory diseases [10, 11].

The complex relationships between gut bacteria and heavy metals have gained growing acknowledgment. Cadmium (Cd)-induced dysbiosis in the gut microbiota has been demonstrated to aggravate liver damage by enhancing intestinal permeability in mouse models [1]. Research consistently indicates that HM exposure results in notable alterations in microbial composition, characterized by a reduction in Proteobacteria and Firmicutes and an elevation in Bacteroidetes at the phylum level [12]. Probiotics, characterized as mono- or mixed cultures of live microorganisms that enhance health by restoring intestinal microbial equilibrium, have demonstrated potential in alleviating heavy metal-induced dysbiosis. Common probiotic genera are Bifidobacterium, Lactobacillus, Streptococcus, Enterococcus, Clostridium, Bacillus, and Escherichia coli. Certain strains, specifically those from Lactobacillus, Bacillus, Bifidobacterium, and Clostridium species, have exhibited significant resistance to heavy metals. This resistance is attained by modifying physiological circumstances or by expressing heavy metal-binding peptides/proteins or detoxifying enzymes implicated in heavy metal biotransformation. These procedures not only rectify heavy metal-induced dysbiosis but also confer protection against heavy metal toxicity [12].

The influence of probiotics on heavy metal removal can be substantiated by sophisticated diagnostic techniques for heavy metals in urine. Numerous technologies have been devised to ascertain the concentration and toxicity of heavy metals, including electrothermal atomic absorption spectrometry, inductively coupled plasma-optical emission spectrometry, and high-pressure liquid chromatography coupled with inductively coupled plasma-mass spectrometry, among others [13-15]. Although high-precision approaches are advantageous, biosensors, point-of-care devices, and analogous technologies are distinguished by their cost-effectiveness, adaptability, and durability, providing quick, non-invasive, and user-friendly solutions that do not necessitate expert staff [16, 17, 18]. Biosensors function by identifying signals produced from interactions between analytes and biological elements, yielding a response that correlates with the quantity of heavy metals [19]. Biosensors, as defined by the International Union of Pure and Applied Chemistry (IUPAC), are self-contained devices that provide quantitative or semi-quantitative data via direct interaction with transducer elements [13]. These methodologies facilitate the evaluation of probiotic therapies and health metrics while also offering insights into additional health variables.

The presence of copper (Cu) in urine may indicate Wilson's disease or urinary tract infections, which have demonstrated inhibitory effects on specific bacteria [20, 21]. This study provides an in-depth examination of the reciprocal relationship between heavy metals and the gut microbiota. It underscores the detrimental impacts of heavy metals on microbial communities and the capacity of probiotics to influence gut

microbiota and facilitate the removal of heavy metals. In contrast to earlier research that examined these topics in isolation, our analysis synthesizes these findings to enhance comprehension of the mechanisms involved in microbial disruption and probiotic-mediated detoxification. This study's primary novel characteristic is the incorporation of modern biosensor technologies for the quick, accurate, and non-invasive detection of heavy metals in urine. These advanced techniques provide real-time monitoring of probiotic therapies and enhance diagnostic capacities for health issues connected to HM. This review seeks to bridge significant gaps by integrating insights on microbial modulation with advanced detection techniques, thereby offering novel views on alleviating heavy metal toxicity and enhancing gut health.

1.1 The Influence of Heavy Metals on Gut Microbial and Host Health

Exposure to heavy metals has been associated with considerable health hazards in both people and animals, mainly due to its effects on the gut flora [22]. About 60% of the ingested heavy metals are absorbed in the intestine, where they may induce oxidative stress and compromise the intestinal barrier, potentially resulting in heightened intestinal inflammation [23, 24, 25]. Although all examined metals induce microbiota dysbiosis, the particular alterations in microbial composition and functional ramifications differ among them.

1.1.1 Arsenic

Chronic exposure to arsenic (As) has been linked to many gastrointestinal problems, including dyspepsia, gastroenteritis, and chronic diarrhea, along with impaired gut barrier integrity [26, 27, 28]. Subchronic exposure to arsenic induces substantial alterations in colonic epithelial architecture and compromises intestinal barrier integrity, chiefly by destruction to the intestinal microvilli [29]. The compromise of the intestinal barrier is associated with elevated production of inflammatory cytokines, including IL-6, IL-8, and TNF-α, as well as the induction of oxidative stress in colonic epithelial cells [30, 31]. Exposure correlates with an increase in pathogenic bacterial populations and a simultaneous decrease in commensal bacteria, explaining its substantial effect on gut microbial balance [32, 33]. Experimental studies on As-exposed mice demonstrated a significant drop in the alpha diversity of the gut microbiota, accompanied by a compositional change characterized by an increase in Bacteroidetes and a reduction in Firmicutes [34].

A study involving 8-week-old mice exposed to arsenic (As) in drinking water at concentrations of 0.5 ppm and 5 ppm revealed that arsenic exposure resulted in intestinal inflammation and a reduction in microbial diversity, especially in the 5 ppm group. In comparison to controls, the 5 ppm arsenic treatment reduced the relative abundance of Bacteroidetes and Firmicutes, but Verrucomicrobia exhibited a considerable increase. Furthermore, there was a decline in the prevalence of Muribaculaceae, Alistipes, Lachnospiraceae, Prevotellaceae, Lactobacillus, and Alloprevotella, whereas the prevalence of Akkermansia, Bacteroides, Bacteroidales, Muribaculum, Ruminococcaceae, Parabacteroides, and Helicobacter rose subsequent to As exposure during the developmental phase [35].

In a separate investigation conducted by Li et al. [36], administration of 50 ppm sodium arsenite in drinking water for two weeks to five-week-old laboratory mice resulted in a considerable reduction in the prevalence of Bacteroidetes and Tenericutes. At the genus level, the arsenite-exposed mice exhibited a

significant increase in the abundance of Acetivibrio, Pelotomaculum, Anaerovorax, Anoxybacillus, Alistipes, Alkalitalea, Chryseobacterium, Bosea, and Curvibacter relative to the control animals. A notable reduction was noted in the prevalence of Clostridium, Syntrophococcus, Fusicatenibacter, Cellulosilyticum, Blautia, Oribacterium, Fastidiosipila, Gemmiger, Intestinimonas, Butyncicoccus, Geosporobacter, Anaerovibrio, Escherichia Shigella, Parasutterella, and Anaeroplasma.

These data collectively explain the significant and complex influence of As on the gut flora. As-induced dysbiosis affects microbial diversity and abundance, exacerbates intestinal inflammation and oxidative stress, and contributes to larger systemic consequences.

1.1.2 Cadmium

Cadmium (Cd) exposure is linked to considerable impairments in intestinal barrier integrity and alterations in gut microbiota composition. Cadmium exposure has been demonstrated to diminish the expression and induce aberrant location of essential cell adhesion proteins, including ZO-1, ZO-2, junctional adhesion molecule A (JAM-A), occludin, and claudin-1, in intestinal epithelial cells. This disturbance results in heightened intestinal permeability, hence undermining gut integrity and function [8, 37]. Significantly, Pb exposure parallels certain effects of As and Cd by diminishing microbial diversity and fostering pathogenic bacteria, nevertheless, its impact on the gut microbiota is more uniform across several investigations. Li et al. [36] illustrated that the administration of 50 ppm Cd chloride in drinking water to laboratory mice for two weeks significantly diminished bacterial diversity in the intestinal microbiota, relative to controls, with a notable reduction in the abundance of Bacteroidetes and Proteobacteria.

At the genus level, cadmium exposure resulted in an increase in the abundance of specific bacteria, including Anaerosporobacter, Acidaminobacter, Prevotella, Barnesiella, Parabacteroides, and Alistipes, while the abundance of bacteria such as Syntrophococcus, Clostridium, Eisenbergiella, Coprococcus, Blautia, Anaerostipes, Hespellia, Cellulosilyticum, Ruminococcus, Intestinimonas, Gemmiger, Caloramator, Alkaliphilus, Thermotalea, Filifactor, Gracilibacter, Anaerovorax, Peptococcus, Kandleria, Anaerovibrio, Desulfovibrio, Klebsiella, Parasutterella, Brevundimonas, Acinetobacter, Mucispirillum, Anaeroplasma, Arthrobacter, and Clostridium XIVb, III, IV, XI, and XVIII was significantly diminished.

Yang et al. [38] conducted a further investigation into the effects of different dosages of cadmium exposure in rats. They discovered that cadmium exposure resulted in a change of microbiota composition, characterized by large reductions in the number of Prevotella and Lachnoclostridium, whereas populations of Escherichia coli–Shigella increased. A 2023 investigation [39] corroborated these findings, demonstrating analogous alterations in the intestinal microbiota composition, characterized by elevated numbers of pathogenic bacteria such as Helicobacter and Campylobacter in a mouse model subjected to 3.6 mg/L oral Cd for 23 weeks. Notably, in contrast to arsenic, which predominantly alters microbial communities in favor of Bacteroidetes, cadmium exposure seems to reduce beneficial microorganisms across several phyla while enhancing opportunistic pathogens. The alteration of the gut microbiota by Cd underscores the compound's contribution to intestinal inflammation, microbial dysbiosis, and the impairment of gut barrier integrity. These findings emphasize the necessity for additional study on alleviating Cd toxicity and investigating methods to restore gut microbial equilibrium post-exposure.

1.1.3 Mercury

Mercury (Hg) exposure dramatically undermines intestinal barrier integrity by diminishing the expression of critical intercellular junction proteins, resulting in heightened intestinal permeability and promoting the absorption of other hazardous metals [9]. Consequently, Hg exposure markedly reduces the expression of claudin 1, occludin, ZO-1, and JAM1 in colon epithelial cells. Furthermore, mercury exposure augments the volume of intestinal cells and membrane permeability without compromising cell viability, hence facilitating the absorption of other hazardous metals under analogous physiological settings [40]. Research indicates that the administration of mercury chloride (HgCl2) in mice leads to substantial alterations in gut microbiota composition. A notable rise in the prevalence of Butyricimonas, Dehalobacterium, Coprococcus, Oscillospira, and Bilophila was observed, whereas the prevalence of Sporosarcina, Jeotgailcoccus, Staphylococcus, and Acinetobacter was greatly diminished [41].

Moreover, exposure to 0.4 μg/mL inorganic mercury (IHg) has demonstrated a phylum-level augmentation of Tenericutes and a reduction of Verrucomicrobia, signifying substantial modifications in microbiota relative to controls. This exposure resulted in a notable augmentation in the abundance of intestinal pathogenic bacteria at the genus and species levels, encompassing Streptococcus, Enterococcus faecalis, Peptostreptococcus, Staphylococcus aureus, Pneumococcus, Erysipelas, Bacillus anthracis, Tetanus, Spirochetes, Actinomycetes, and Tuberculosis, alongside an increase in sulfate-reducing bacteria relative to controls [42].

Ultimately, the exposure of rats to methylmercury, a more lethal variant of Hg (0.4 μg/mL, corresponding to 10 μg/kg body weight), resulted in substantial alterations in the microbiota composition. A decline was observed in the populations of Lactobacillaceae, Bacteroidaceae, Streptococcaceae, and Sutterellaceae, while a rise was noted in Desulfovibrionaceae, Helicobacteraceae, Peptococcaceae, and Rhodospirillaceae[43].

These findings explain the significant effect of Hg on intestinal homeostasis, with extensive implications for metabolic, immunological, and inflammatory systems. Hg exposure, due to its capacity to alter gut microbiota composition, may increase the risk of gastrointestinal and systemic inflammatory disorders.

1.1.4 Lead

Lead (Pb) exposure significantly reduces the expression of the MUC2 gene and tight junction proteins (ZO-1, claudin 1, and occludin) in intercellular junctions, resulting in heightened intestinal permeability in both mice subjected to low doses over extended periods and those exposed to high doses over brief intervals [44]. A study investigating the impact of 10 ppm lead chloride in drinking water (equal to 2 mg/kg body weight/day) on mice over 13 weeks revealed a decrease in the prevalence of Ruminococcus, Clostridiales, and Oscillospira after only 4 weeks of exposure. At the conclusion of the 13-week exposure period, a substantial reduction in the prevalence of Lachnospiraceae, Blautia, Coprococcus, and Ruminococcus was noted [4].

Xia et al. [45] investigated the impact of 15 weeks of exposure to 0.1 mg/L lead in drinking water on laboratory mice. Through quantitative real-time PCR analysis of cecal contents, they demonstrated a

decrease in the prevalence of the Bacteroidetes and Firmicutes phyla. Additionally, 16S rRNA gene sequencing revealed a reduction in Firmicutes and an elevation in Bacteroidetes and Proteobacteria phyla. At the genus level, lead exposure was associated with an elevation in Parabacteroides and a reduction in Dehalobacterium.

A further study demonstrated that mice exposed to 1.83 g/L lead acetate ((CH3COO)2Pb·3H2O) in their drinking water experienced intestinal dysbiosis, marked by a reduction in beneficial species including Akkermansia muciniphila, Faecalibacterium prausnitzii, and Oscillibacter ruminantium [46]. Population-based human investigations corroborate these findings.

A cross-sectional study with 696 participants investigated the relationship between urine lead concentrations and gut microbiota makeup. The findings indicated a significant association between heightened urine Pb levels and a greater prevalence of Desulfovibrio, Eubacterium, and Ruminococcus. Conversely, there was a significant reduction in Clostridium, Coprococcus, and Pediococcus [47]. These findings collectively indicate that Pb exposure disturbs gut microbial equilibrium, promoting the growth of pro-inflammatory and pathogenic bacteria while diminishing beneficial commensal species. Dysbiosis may contribute to systemic toxicity generated by lead, resulting in inflammatory and metabolic abnormalities.

Heavy metals are recognized for their disruption of signaling pathways that influence numerous cellular activities, such as growth, proliferation, survival, metabolism, and apoptosis. Bioaccumulation can result in various toxic effects on body tissues and organ systems, primarily by disrupting antioxidant defense mechanisms, inducing oxidative stress, and substituting essential metals in biological processes, which leads to cellular damage and an elevated risk of diseases such as cancer, neurodegenerative disorders, and cardiovascular complications.

2.0 The role of Gut Microbes in reducing Heavy Metals

Heavy metals, once collected in the body, can induce different chronic illnesses and elevate the risk of cancer. These metals, owing to their enduring presence in the environment, pollute the soil and infiltrate the food chain, impacting human health and ecosystems. While conventional detoxification treatments are available, they frequently entail significant expenses and may produce adverse side effects. A promising and minimally intrusive alternative is the utilization of probiotics, live bacteria that, when provided in suitable dosages, confer health advantages to the host by facilitating the excretion of metals from the body [48, 49].

The arsenic resistance genes of specific bacterial species, including Bacteroides, Clostridium, Alistipes, and Bilophila, facilitate their chemical modification of arsenic. These bacteria convert arsenic into a less poisonous form via methylation, thereby assuring their survival and safeguarding the host organism from the detrimental effects of this element [50, 51]. The findings from a study on the management of arsenic poisoning with probiotics indicated that they mitigated the adverse effects of arsenic on oxidative stress and reproductive dysfunction in female rats. The probiotic formulation, comprising 137.14 million cells of each Lactobacillus acidophilus, Lactobacillus rhamnosus, Bifidobacterium longum, and Bifidobacterium bifidum, along with 28.57 million cells of Saccharomyces boulardii, resulted in the

restoration of antioxidant activities (superoxide dismutase (SOD), catalase, and NPSH), a decrease in malondialdehyde (MDA) and cadmium (Cd) levels, and enhanced vitamin B12 and estradiol levels.

Furthermore, they reduced the expression of inflammatory markers and tissue lesions, offering protection against DNA damage and uterine–ovarian necrosis [52]. A separate study examining the impact of probiotics on arsenic poisoning in Wistar rats revealed that arsenic elevated levels of malondialdehyde (MDA), myeloperoxidase (MPO), and inflammatory cytokines, while diminishing serum antioxidant defense markers. Probiotics comprising Lactobacillus and Bifidobacterium lactis exhibited a notable protective effect, diminishing genotoxicity and enhancing SOD levels, TNF-α, and interferon-gamma (IFN-γ). The advantageous results were ascribed to the probiotics' capacity to diminish the generation of reactive oxygen species (ROS) and alleviate intestinal inflammation [53].

The preventive benefits of probiotics against cadmium toxicity have been thoroughly investigated. A study demonstrated that Wistar rats exposed to cadmium (Cd) showed considerable metal accumulation in the liver and kidneys, heightened oxidative stress (indicated by increased malondialdehyde [MDA] and 8-hydroxy-2′-deoxyguanosine [8-OHdG]), and modified levels of glutathione (GSH) and metallothionein. Treatment utilizing Lactobacillus- and Bifidobacterium-based probiotics, together with nano-probiotics, markedly mitigated these effects, with the nano-formulation demonstrating superior efficiency in decreasing GSH and 8-OHdG levels [54]. Djurasevic et al. [55] revealed that the administration of Lactobacillus rhamnosus Rosell-11, Lactobacillus acidophilus Rosell-52, and Bifidobacterium longum Rosell-175 markedly enhanced cadmium excretion via feces, thereby diminishing its concentrations in the blood, liver, and kidneys, while simultaneously ameliorating liver function.

Lactobacillus plantarum CCFM8610, a strain characterized by its substantial Cd-binding ability and antioxidant capabilities, exhibited a marked decrease in Cd accumulation in tissues, mitigation of inflammation, and enhancement of gut barrier integrity. Pediococcus pentosaceus GS4 effectively diminished Cd accumulation in the liver and intestines, enhanced membrane integrity, and alleviated Cd-induced intestinal lesions [56]. A comparative analysis of 13 probiotic strains regarding their cadmium detoxification capabilities revealed that Streptococcus thermophilus demonstrated the greatest resistance to cadmium and exhibited strong antioxidant activities, markedly decreasing cadmium levels in the bloodstream [57].

Majlesi et al. [58] examined the impact of Lactobacillus plantarum and Bacillus coagulans on mercury-induced toxicity in a rat model. The treatment of probiotics significantly mitigated Hg toxicity by lowering Hg concentrations in the liver and kidneys and preserving glutathione peroxidase (GPx) and superoxide dismutase (SOD) levels. Probiotics resulted in a notable decrease in creatinine, urea, bilirubin, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) levels. In a separate investigation, Lactobacillus brevis 23017 safeguarded the intestinal barrier, mitigating weight loss and intestinal lesions induced by mercury. It also regulated tight junction proteins and diminished inflammation and oxidative stress via the mitogen-activated protein kinase (MAPK) and nuclear factor kappa-light-chain-enhancer of activated B cell (NF-κB) pathways [59]. Exposure to mercury chloride induced oxidative stress and renal impairment, including tubular necrosis, in laboratory mice. Probiotic supplementation with Lactobacillus plantarum, Lactobacillus delbrueckii spp. Bulgaricus PXN39, Lactobacillus acidophilus, Lactobacillus

rhamnosus, Bifidobacterium bifidum, Streptococcus salivarius spp. Thermophilus, and Enterococcus faecium significantly altered MDA levels and antioxidant enzyme activities, positively influencing the histopathological lesions caused by mercury toxicity [60].

Probiotic therapies have demonstrated potential in alleviating lead poisoning. Zhai et al. [61] demonstrated that a supplement comprising grape seed extract, tea polyphenols, and Lactobacillus plantarum CCFM8661, as well as another supplement containing vitamin C, calcium carbonate, zinc acetate, and L. plantarum CCFM8661, effectively diminished Pb levels, safeguarded antioxidant enzyme activities, and ameliorated oxidative stress markers and cognitive impairments in mice. These supplements showed enhanced protection relative to chelator therapies, and given during lead exposure conferred a markedly larger protective effect than post-exposure administration. Lactobacillus plantarum CCFM8661 mitigated Pb poisoning by reinstating antioxidant enzyme activity and diminishing Pb concentrations in blood and tissues [62].

The treatment's efficiency was markedly enhanced when the probiotic was taken during the entire period of Pb exposure. A different probiotic examined for Pb toxicity in mice was Lactobacillus delbrueckii subsp. bulgaricus KLDS1.0207, which demonstrated a reduction in mortality, an increase in Pb excretion via feces, a prevention of Pb buildup in tissues, and an enhancement of antioxidant activities in the liver and kidneys [63]. Moreover, bioleaching systems employ efflux pumps to discharge hazardous metals into the intestinal lumen. Finally, metal solubilization transpires via the bacterial release of lactic and acetic acids, which affect metal bioavailability. These systems jointly facilitate the regulation of metal toxicity and diminish heavy metal absorption in the human body.

Conclusion and Future Perspective

HMs significantly affect the makeup and function of gut microbiota, leading to dysbiosis and a series of detrimental health consequences. The reciprocal interaction between gut microbiota and heavy metals highlights the necessity of preserving microbial equilibrium to reduce toxicity. Probiotic therapies have arisen as a viable approach to mitigate heavy metal-induced dysbiosis by reinstating microbial equilibrium, augmenting detoxifying mechanisms, and fortifying gut barrier integrity. These findings underscore the therapeutic potential of probiotics in HM detoxification and in larger contexts including metabolic and inflammatory illnesses. The incorporation of modern biosensing technology represents a notable advancement in real-time heavy metal detection and exposure monitoring, in addition to microbiome modification. Biosensors offer a non-invasive, quick, and extremely sensitive method for detecting heavy metals in urine, facilitating continuous monitoring of exposure levels and the efficacy of detoxification measures. Utilizing biosensors, healthcare professionals and researchers may evaluate the immediate effects of probiotic and dietary interventions, enabling precision medicine strategies customized to individual detoxification requirements.

Future research should concentrate on the creation of targeted probiotic medicines particularly engineered to modify the gut microbiota in reaction to heavy metal exposure. Identifying bacterial strains with superior heavy metal-binding properties will be essential for enhancing probiotic compositions. Furthermore, advancing research on synbiotics, which are combinations of probiotics and prebiotics, may augment the detoxification efficacy of microbial interventions by supplying substrates that foster the proliferation of beneficial bacteria engaged in metal chelation and excretion. A crucial domain for progress is the enhancement and miniaturization of biosensor technologies. The incorporation of graphene-based

nanomaterials, molecularly imprinted polymers, and artificial intelligence-driven data processing into biosensor platforms would enhance sensitivity, precision, and user-friendliness. The advancement of wearable or point-of-care biosensors has the potential to transform real-time exposure monitoring, enabling patients to monitor heavy metal levels in their urine and modify detoxification procedures accordingly. The integration of microbiome characterization with biosensor data presents a possible avenue for the development of tailored detoxification strategies. By customizing probiotic therapies, dietary guidelines, and lifestyle alterations according to an individual's gut microbiota profile and heavy metal exposure levels, healthcare professionals can employ precision medicine approaches that enhance detoxification results.

Moreover, broadening clinical research on the long-term health consequences of chronic low-dose heavy metal exposure and microbiota-targeted therapies would yield greater understanding of preventive and therapeutic strategies. Cooperative endeavors among microbiologists, toxicologists, bioengineers, and clinicians will be crucial for converting laboratory discoveries into applicable solutions for environmental health, public policy, and customized medicine. The amalgamation of microbiota modification and biosensor-based monitoring is a potent and novel strategy for regulating heavy metal exposure. Progress in these domains has the capacity to markedly diminish health hazards associated with HM poisoning, enhance patient outcomes, and facilitate the development of next-generation detoxification techniques customized to individual requirements.

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