



# A Probiotic-Based Platform for Simultaneous Mycotoxin Neutralization, Heavy Metal Biosorption, and Biofilm Displacement

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## Abstract

**Background and aim:** Mycotoxins, heavy metals, and pathogenic biofilms frequently co-occur in food, feed, and environmental matrices; however, they are almost always investigated and controlled in isolation. The present review examines whether probiotics can instead act as a single, integrated line of defense against all three classes of contaminant, with a focus on lactic acid bacteria of the genera *Lactobacillus* and *Bifidobacterium* and on the yeast *Saccharomyces cerevisiae* var. *boulardii*.

**Method:** A narrative review was conducted, in which structured searches across the major scholarly databases were combined with the targeted screening of specialized journals, and three mechanistically distinct but potentially complementary axes were examined: mycotoxin neutralization through electrostatic and hydrophobic adsorption to the cell envelope and, in certain strains, enzymatic cleavage; heavy metal capture at peptidoglycan-associated carboxyl, hydroxyl, amino, and phosphoryl groups by ion exchange and surface complexation; and biofilm displacement driven by competitive exclusion and the secretion of bacteriocins and organic acids.

**Results and conclusion:** Across these axes, the available evidence is substantial but uneven, and several constraints recur, including a scarcity of long-term human clinical trials, few designs that assess the three functions simultaneously, and persistent difficulties in standardization and real-world translation. Closing these gaps will require more than incremental single-contaminant studies; it will demand integrated experimental designs, standardized efficacy benchmarks, and trials conducted in the populations that carry the heaviest contamination burden. Framed in this way, probiotics emerge not merely as modulators of gut health but as a biologically sustainable, multi-target strategy for the management of complex, co-occurring contamination.

## What is “already known”:

- Lactic acid bacteria and *Saccharomyces cerevisiae* var. *boulardii* bind mycotoxins
- Components of the cell envelope bind the mycotoxin through non-covalent interactions
- Complex is reversible and strongly strain-dependent;
- Envelope sequesters heavy metals at carboxyl, hydroxyl, amino and phosphoryl groups by ion exchange and surface complexation, a process that does not require viable cells
- Probiotics restrict pathogenic biofilms through competitive exclusion, quorum-sensing quenching and the secretion of bacteriocins and organic acids.

## What this article adds:

- One decontamination function may constrain or enhance the others
- Mycotoxin work has reached animal models and real food matrices
- Heavy metal work has reached human participants in a single pilot trial
- Biofilm work has not yet left in vitro study.
- Integrated designs is required to test 3 functions together, standardized efficacy benchmarks, and trials conducted in the populations carrying the heaviest contamination burden.

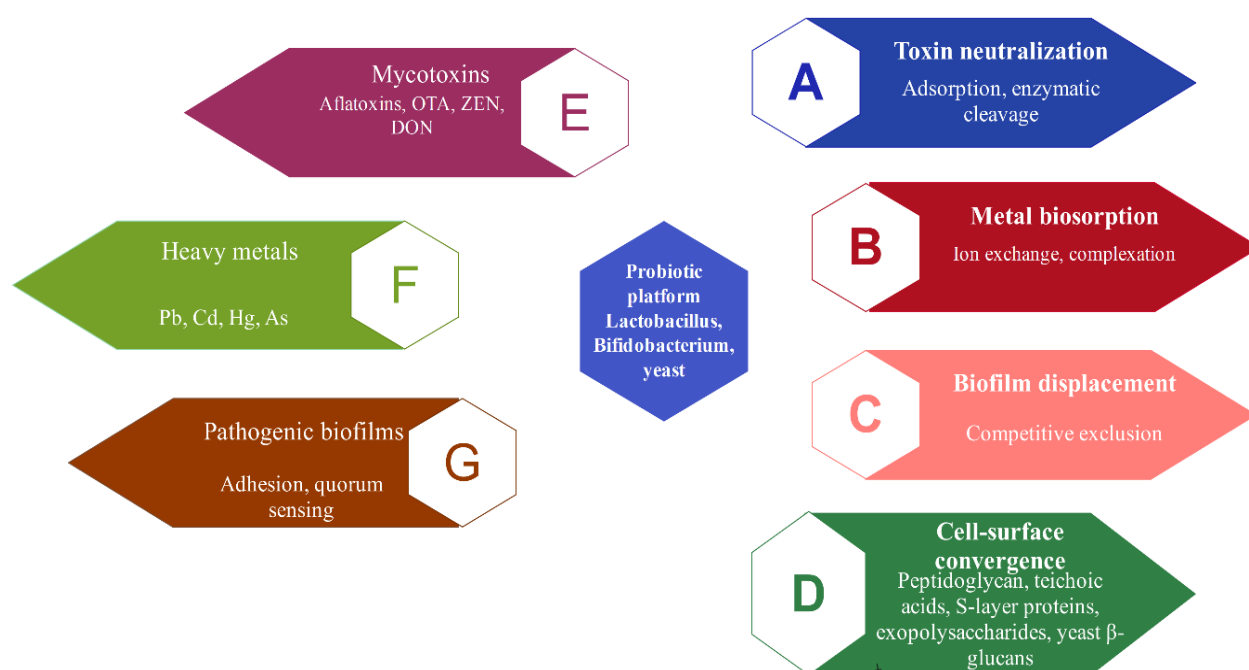
## 1. INTRODUCTION

The strained relationship between human health and the environment is driven, in large part, by toxic contaminants. Among the biological answers to this challenge, probiotics have attracted particular attention for their ability to degrade or modify mycotoxins, reducing their toxicity enzymatically. Mycotoxins rank high on the list of concerns addressed in this review. In livestock production, five stand out above the rest — aflatoxins, trichothecenes, zearalenone, ochratoxins, and fumonisins — and their sharply different chemical structures translate into an equally wide array of acute effects in animals. For years, clay minerals mixed into feed have served as binders, yet their reach is narrow: they capture aflatoxins reasonably well but leave the other toxins largely untouched. This limitation opened the door to a different tactic altogether enlisting microorganisms that break mycotoxins down into harmless metabolites inside the gut, ahead of absorption. The rumen bacterium *Eubacterium* BBSH 797 illustrates the point, neutralizing trichothecenes by reducing their epoxide ring, whereas the yeast *Trichosporon mycotoxinivorans* takes apart both ochratoxin A and zearalenone [1].

The value of *Saccharomyces cerevisiae* var. *boulardii* (*S. cerevisiae* var. *boulardii*) as a detoxifying agent against mycotoxins, heavy metals, and biofilm-forming bacteria has already been shown [2-4]. All three contaminants are common in farmland, water sources, and human environments, where they drive chronic illness, developmental disorders, and the growing problem of antimicrobial resistance. Exposure is more or less continuous, and it falls hardest on developing countries, where monitoring is limited [5]. The relationship runs in both directions: dietary mycotoxins reshape the gut

microbiota, and the microbiota in turn changes what happens to the toxins [6-10]. Probiotics are best known for the way they modulate gut flora and immunity. More recently, they have been examined as broad-spectrum biological agents that bind toxins, take them up by biosorption, and crowd out harmful bacteria [11]. The field sits at the meeting point of microbiology, toxicology, and biotechnology, and it offers an environmentally friendly alternative to chemical treatments, which often bring hazards of their own. Probiotics may even help raise crop yields, quite apart from their benefits to personal health [12]. Progress has been considerable since the mid-twentieth century. Early work looked mainly at the fermentation and antimicrobial properties of lactic acid bacteria; later advances in genomics, molecular biology, and in vitro testing uncovered strains that can bind, modify, or degrade toxins, opening the way to multi-purpose systems [13, 14]. The last five years have seen the field accelerate, as reflected in systematic reviews of how probiotics, prebiotics, and postbiotics act on mycotoxins and heavy metals [15]. The timing matters. Climate change is pushing up mycotoxin levels in crops, heavy metals keep entering water and food chains through industrial discharge, and biofilm-associated resistance is on the rise [16,17]. Yet the literature still lacks a single evaluation framework able to describe mycotoxin decontamination, heavy metal immobilization, and biofilm inhibition carried out by one probiotic organism. How these processes interact, whether they help or hinder one another, has also had little empirical attention. This review therefore surveys the current state of research on probiotic systems that can perform at least three of these tasks at the same time. Figure 1 illustrates three categories of co-contaminants on a probiotic platform.





**Fig.1. Three co-occurring contaminant classes converge on a single probiotic platform.**

## 2. Conceptual and Theoretical Background

The field has moved a long way from single-target probiotic thinking toward more complex, multi-functional technologies. What began as the everyday use of probiotics in food preservation has turned into the study of simultaneous detoxification, a shift driven by wider awareness of environmental pollution [18]. Ideas first worked out in dairy microbiology now inform how researchers approach complex, multi-pollutant settings, and they have made it possible to build integrated platforms that tackle several linked problems at once [19]. A few complementary principles underlie these platforms. Biofilm displacement is explained by microbial antagonism and competitive exclusion, which act through resource competition and the release of inhibitory metabolites [19]. Mycotoxin neutralization rests mainly on adsorption theory, with electrostatic, hydrophobic, and hydrogen-bonding interactions forming between the toxins and the polysaccharides of bacterial cell walls [20, 21].

Mycotoxins are toxic secondary metabolites made chiefly by *Aspergillus*, *Fusarium*, *Penicillium*, and

*Alternaria* [22]. They arise under varied conditions during the cultivation, harvest, storage, and processing of crops, and they turn up repeatedly in staples such as cereals, fruits, and feedstuffs [1]. Exposure, whether acute or long-term, carries a range of consequences: growth inhibition, immune-suppression, reproductive disorders, and cancer [23]. Their toxicity has a structural basis. The lactone ring of the aflatoxins, the epoxide group of the trichothecenes, and the isocoumarin backbone of the ochratoxins each drive damage through the inhibition of DNA, RNA, and protein synthesis, the disruption of sphingolipid biosynthesis, or estrogenic effects [24-27].

Whether the probiotic cell is alive at the point of action seems to matter less than whether its structure stays intact. The acidic stomach kills off many viable cells, yet heat- and acid-treated, non-viable *L. rhamnosus* GG and LC-705 bind AFB1 as well as their live counterparts, and sometimes better [28]. This has a clear practical payoff: shelf-stable, inactivated preparations can match live cultures in food safety and feed supplementation, without the trouble of



keeping cells alive through processing and storage [29-31]. Even so, strains are not interchangeable, and the same strain behaves differently depending on how it has been treated. Viable *L. rhamnosus* GG held on to roughly 50% of the bound AFB<sub>1</sub> after five washes, and LC-705 to 38%; heat or acid treatment lifted both figures to 66–71%. Only 6–11% of the bound toxin came off across the full gastrointestinal pH range of 2 to 10, which is a level of stability that matters a great deal for any real use in the gut. The binding itself looks non-covalent and hydrophobic: organic solvents stripped off nearly all of the surface-bound AFB<sub>1</sub>, whereas autoclaving and sonication released none [20, 32].

Heavy metal biosorption works through surface complexation and ion exchange, with anionic groups carboxyl, hydroxyl, amino, and phosphoryl doing most of the sequestration [18]. LAB removes metals by two routes that are seldom given equal weight in the literature. The first, biosorption, is fast, needs no metabolism, and depends on electrostatic binding between metal cations and the negatively charged cell wall. The second, bioaccumulation, is slower, depends on metabolism, and only matters once the wall's binding sites are full. The difference is practical: dead cells can still biosorb, but bioaccumulation needs a living, active bacterium [33]. Two pathways account for most biodegradation. One is physical: the contaminant binds straight onto cell-wall components, peptidoglycan and polysaccharides in bacteria and 1,3- $\beta$ -glucan in yeasts. The other is metabolic, with

the cell's enzymes converting the toxic compound into something less harmful. The line between the two is rarely clean. Strain identity, contaminant chemistry, and the surrounding conditions all decide which one leads, and often both run together [14]. Systems-biology methods add to this picture by treating probiotics as active regulators inside larger host-microbiome environment networks [18]. The idea that a well-designed strain can trigger several detoxification mechanisms at once, with synergistic results, is what this review calls the multi-target probiotic hypothesis [34, 35].

Network theory has been used to trace how such strains affect microbiome stability, gut barrier permeability, and toxin availability. Making this work in practice means paying close attention to dose, matrix effects, and host-specific responses [36]. In this review, “probiotic-based platform” means the use of beneficial strains, mostly *Lactobacillus* and *Bifidobacterium*, as multi-functional tools. Such strains can deactivate mycotoxins by binding or biodegradation, biosorb heavy metals by binding and intracellular uptake, and block pathogenic biofilm formation through competitive exclusion and antimicrobial activity [37, 38]. The subject cuts across microbiology, environmental toxicology, and applied biotechnology. It takes in in vitro studies, gut-simulation models, and applications in food safety, animal husbandry, human health, and environmental remediation [18]. Figure 2 clearly illustrates the binding of mycotoxins to the probiotic cell surface.



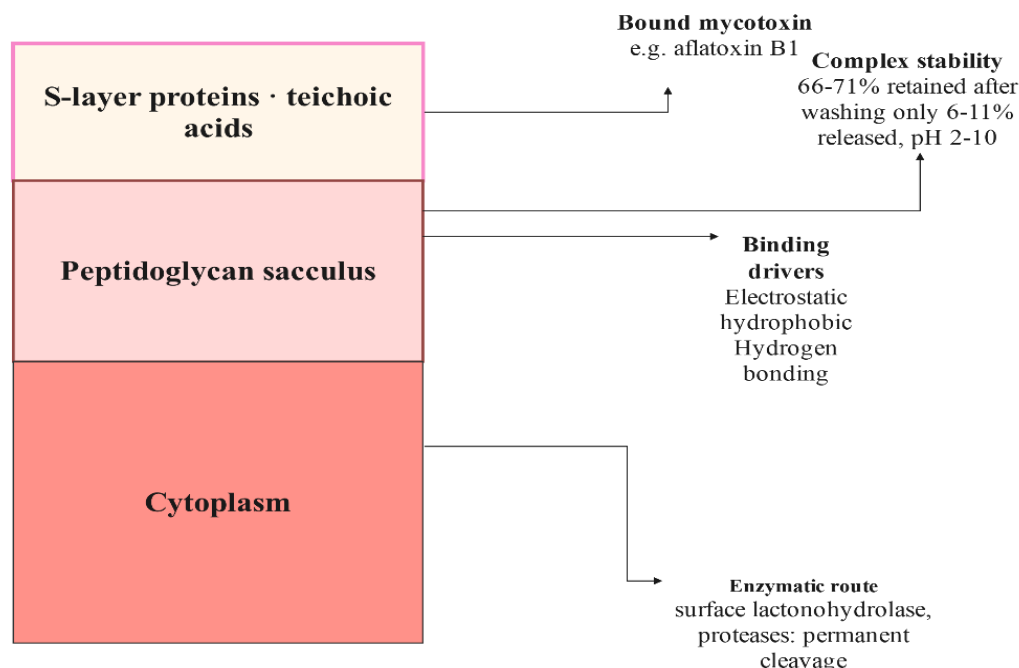


Fig. 2. *Where a mycotoxin binds to the probiotic cell envelope.*

### 3. Historical Development and Evolution

Probiotic detoxification has its roots in the late nineteenth and early twentieth centuries, when lactic acid bacteria were first found in fermented foods. Élie Metchnikoff's study of the link between yogurt and longevity laid the groundwork [39]. Using lactic acid fermentation to make food safer is far from new; it predates microbiology. Long before anyone identified the organisms responsible or understood how they worked, early farming societies fermented cereals, milk, fish, and meat both to preserve them and to enrich them microbially. What has changed is the precision with which those same mechanisms can now be studied, selected for, and aimed at specific contamination problems [40, 41]. When probiotics were first defined, in the mid-twentieth century, the focus was digestive health, and the field grew through the 1980s and 1990s on the back of clinical work on microbiota modulation [42, 43]. In the early 2000s, certain strains of *Lactocaseibacillus rhamnosus* were shown to adsorb mycotoxins such as aflatoxins and

ochratoxin A [20, 44]. Heavy metal biosorption came to the fore in the 2010s as environmental microbiology matured [45], and biofilm modulation developed in parallel within infection control [46]. Over the past decade, the direction has been unmistakable: a turn toward multi-target approaches, carried by advances in genomics and metabolomics [18]. Strains able to handle mycotoxins, heavy metals, and biofilms at once have since been confirmed [47]. Two pressures have sharpened this trend: toxins intensified by climate change, and biofilms that keep developing resistance, both of which point toward ecological bioremediation [14]. Early trial-and-error methods have grown into theory-based, system-level interventions. Years of work on strain specificity, matrix effects, and safety have gradually settled the main doubts and lent these tools real credibility [14, 48]. The upshot is that probiotic approaches drawing on microbiology, toxicology, and engineering now offer workable answers to contamination.

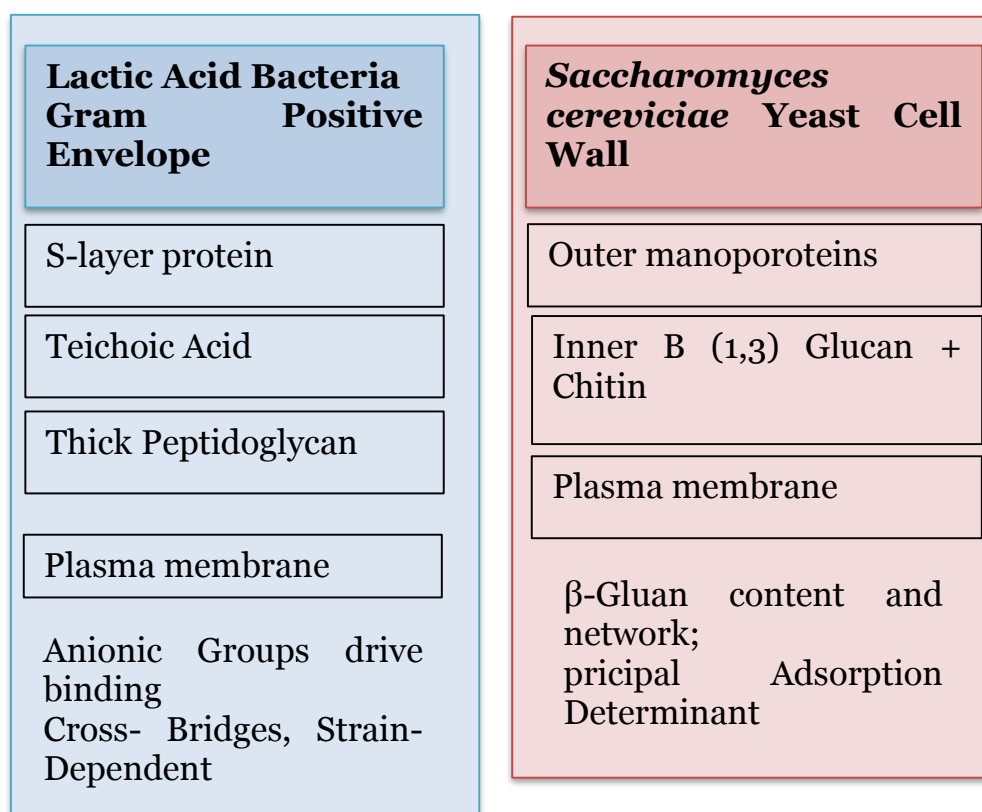


#### 4. Current Trends and Key Issues

Recent advances in probiotic research indicate a clear shift toward multifunctional systems. Researchers are now developing single strains, or carefully selected consortia, capable of integrating several detoxification mechanisms. Real-world contamination, however, rarely arrives one toxin at a time: mycotoxins co-occur with pesticide residues, bacterial toxins share food matrices with antinutrients, and heavy metals accumulate alongside fungal contaminants. In such multi-contaminant scenarios, single-mechanism chemical interventions lose much of their utility. In contrast, LAB operating through simultaneous adsorption, enzymatic hydrolysis, and competitive exclusion of contaminants can address several threats within a single fermentation process while also improving the nutritional profile of the food itself [3]. This approach enables more comprehensive strategies for dealing with the complex reality of concurrent exposure to mycotoxins, heavy metals, and pathogenic biofilms [14, 22]. Mechanistically, microorganisms appear to counter mycotoxin toxicity along three intertwined paths: they can break the toxins down directly in the food before ingestion; they can outcompete pathogens and colonize the gut so that bacterial–toxin complexes form less readily. Absorption is reduced, and they can reshape the intestinal microecology in ways that reinforce the gut barrier and slow toxin penetration [49]. Species including *Lactiplantibacillus plantarum*, *Lactobacillus crispatus*, and *Lactobacillus acidophilus* have attracted considerable attention on account of their

potential to control mixed contaminations in simulated gastrointestinal environments [50]. This research area has been dominated by a few species, with *Lactobacillus*, *Bifidobacterium*, and the yeast *Saccharomyces cerevisiae* occupying a prominent position in these studies [15]. Research in this field has been advanced through sophisticated strain-screening methods and the use of omics approaches. Most of this binding has been attributed to the presence of reactive functional groups in the cell envelope, including the proteins, peptidoglycan, and polysaccharides of bacterial walls, together with the 1,3- $\beta$ -glucan of yeasts [14]. The efficiency of surface binding is governed largely by the architecture and chemistry of the microbial cell wall. In lactic acid bacteria, the Gram-positive envelope is dominated by a thick, multilayered peptidoglycan sacculus decorated with teichoic acids, polysaccharides, and surface-layer (S-layer) proteins; the resulting abundance of anionic functional groups is thought to underlie much of the binding capacity, and differences in the amino-acid composition of the peptidoglycan cross-bridges help to explain why binding is so strongly strain-dependent. In yeasts such as *Saccharomyces cerevisiae*, the wall is instead built from an inner layer of  $\beta$ -glucans and chitin and an outer layer of mannoproteins, with the content and reticular organization of  $\beta$ -(1,3)-D-glucan emerging as a principal determinant of adsorption efficacy. Clarifying these compositional differences provides a rational basis for the selection or engineering of strains with superior binding performance [51–53].





**Fig.3. Cell-wall makeup in bacteria and yeast. The two envelopes expose different binding surfaces, which is why strain choice matters so much for detoxification.**

It is important to note that this property is not confined to living cells; heat-killed or intact biomass, cell-wall fractions such as peptidoglycan, the cytoplasmic fraction, and even extracellular products such as exopolysaccharides and short-chain fatty acids have all been found to possess bioactivity [54-59]. The extent to which a toxin adheres to a microbial surface depends heavily on the composition of that cell wall: some mycotoxins attach non-covalently to the bacterial surface, and Gram-negative strains tend to adsorb more, a behavior that has been attributed to the lipophilic nature of the compounds accumulating within the bacterial pellets [60, 61]. More importantly, beyond toxin removal, these organisms may also play a significant role in reducing inflammation of the intestinal epithelial membrane,

thereby serving as a means of ensuring both food safety and gut health [62, 63]. This growing attention to biological tools in the context of concerns regarding food-chain contamination and environmental pollution worldwide indicates that probiotics are eco-friendly tools capable of replacing conventional

chemicals [64, 65]. The expanding applications of postbiotics and heat-inactivated microorganisms have rendered their use increasingly practical [66]. The detoxification mechanism of *S. cerevisiae* var. *boulardii* operates in two mechanistically separate ways: rapid and reversible adsorption on the constituents of the yeast cell wall, such as  $\beta$ -glucan, mannan, and chitin, and biotransformation by the enzymes secreted by the organism. These mechanisms are not independent, since the type of

contaminant, the matrix properties, and cell viability influence which of them predominates [4]. A key trend is the growing integration of food microbiology, environmental biotechnology, and clinical nutrition; this interdisciplinary collaboration is accelerating the translation of research into real-world applications [67, 68]. Recent studies commonly employ sophisticated in vitro and ex vivo gut models, which reveal strain-specific performance in the simultaneous neutralization of mycotoxins and sequestration of heavy metals [14, 22]. Consortia frequently show synergistic advantages over single strains, particularly in reducing the bioavailability of cadmium, lead, and the aflatoxins [55, 69, 70]. Both in vitro and in vivo evidence indicate that probiotic cultures do more than simply trap toxins; they can also degrade many toxic substances outright, thereby lowering the overall toxicity rather than merely

relocating it [71, 72]. In addition, efforts to control biofilms have gained momentum, as probiotics compete for adhesion sites and produce antibacterial compounds that disrupt unwanted biofilm structures [54, 55, 73-76]. Comparisons of different bacterial strains highlight the connection between the inhibition of quorum sensing and the inhibition of biofilm formation. As shown in Table 1, the efficiency of different lactic acid bacterial strains in inhibiting the AI-2 signaling pathway of *Vibrio parahaemolyticus* was proportional to the inhibition of biofilm formation, whereas strains that enhanced AI-2 signaling did not affect biofilm formation; biofilm inhibition therefore occurred mainly through quorum sensing. Table 1 summarizes representative lactic acid bacterial strains, their reported inhibitory activity, and biofilm formation against *Vibrio parahaemolyticus*.

**Table 1. Representative lactic acid bacterial strains and their reported inhibitory activity against the quorum sensing and biofilm formation of *Vibrio parahaemolyticus*.**

| Strain                                   | AI-2 (quorum-sensing) inhibition      | Biofilm inhibition   | Key probiotic surface traits                                                  | Reference |
|------------------------------------------|---------------------------------------|----------------------|-------------------------------------------------------------------------------|-----------|
| <i>Lactiplantibacillus plantarum</i> A10 | 82.92%                                | 87.70%               | Strong antibacterial spectrum against six pathogens                           | [16]      |
| <i>Lactiacaseibacillus rhamnosus</i> MS1 | 69.54%                                | 77.32%               | Self-aggregation and hydrophobicity both >80%; strong biofilm-forming ability | [16]      |
| Strains B11, L18                         | Promoted AI-2 activity (no quenching) | No inhibitory effect | Illustrates that biofilm suppression tracks with AI-2 quenching               | [16]      |

Beyond the raw inhibition rates, the surface physiology of the strains helps to explain their performance: the most effective isolate combined self-aggregation and hydrophobicity values above 80% with a strong intrinsic biofilm-forming ability. This combination would favor the competitive colonization of adhesion sites and thereby the

displacement of the pathogen. A further strain combined self-aggregation and hydrophobicity values above 80% with a pronounced capacity to form its own biofilm, features that together favor surface colonization [16]. The lactic acid bacterial (LAB) strains that inhibited the quorum sensing (QS) and biofilm formation of *Vibrio parahaemolyticus* were



screened, and their probiotic properties were characterized [16]. Notably, the extent to which individual strains curbed biofilm development varied considerably, and this inhibition tracked closely with their ability to dampen AI-2 signaling activity [16]. Moreover, the delivery of probiotics through fermented food sources has increased their consumer acceptability and sustainability [66]. All of the above trends indicate that a strategy is being developed for the use of the gut microbiome as a natural defense mechanism against environmental toxins [77, 78]. Several issues nonetheless remain. Strain specificity and a high degree of environmental dependence hinder standardization and scale-up [14, 66]. Researchers have debated the relative merits of live and dead cells: although heat-inactivated biomass can bind toxins, sustained detoxification may require active metabolism [79]. Significantly, postbiotics derived from yeast cell walls have shown average absorption efficiencies of around 70% for certain newly identified mycotoxins, which lends further weight to the argument in favor of non-viable formulations [80]. Probiotics owe much of their heavy-metal-reducing capacity to the numerous negatively charged functional groups on the cell wall, which trap the metals; this mechanism acts in concert with changes in the expression of stress-responsive genes following exposure and with the increased fecal excretion of ingested metals [65, 81, 82]. In agriculture and clinical practice, the possible long-term ecological consequences and the hazards of horizontal gene transfer in genetically engineered strains have led to cautious regulatory approaches [14, 66]. The scarcity of human clinical trials, compared with in vitro and animal studies, remains a major translational barrier [14]. That said, the body of quantitative and qualitative evidence remains thin, and further in vivo and longer-term work will be required before these approaches can be confidently applied to functional foods, feeds, or nutraceuticals

[15]. Safety is particularly critical for vulnerable populations with weakened intestinal barriers and high levels of toxin exposure. Although probiotics may be effective in removing infection-causing biofilms, their specificity and their potential effects on beneficial organisms remain to be clarified [83]. Issues of economic sustainability and scalability present a further challenge, especially in developing countries, which are more prone to contamination. Ethical concerns regarding the genetic modification of microorganisms and the appropriate balance between classical and synthetic approaches remain a driving force behind research [66]. Similar problems arise in veterinary medicine and environmental engineering, where optimizing dose and matrix according to specific conditions is crucial [14, 84-87]. These deficiencies should be addressed through additional longitudinal studies and standardized protocols, which would help translate laboratory results into applications [14, 66]. Finally, despite the progress made, deficiencies remain in integrative multi-target research [14, 88]. Studies still tend to examine mycotoxin binding, heavy metal biosorption, and biofilm disruption separately, which restricts insight into their combined effects [89]. Regulatory discussions concerning detoxification claims further reveal the tension between scientific innovation and consumer safety [66]. Even so, multi-target probiotic systems show strong potential for mitigating the combined risks of modern environmental exposures, and future progress will depend on collaborative, cross-disciplinary work that carefully balances effectiveness, safety, and real-world feasibility [14, 66].

## 5. Applications and Practical Implications

### 5.1. Real-world implementation and case examples

Platforms that neutralize mycotoxins, biosorb heavy metals, and displace pathogenic biofilms at once have found a real foothold in the food and feed



industries, where they double as natural preservatives and detoxifiers [62]. In livestock production, selected *Lactobacillus* strains are added to feed to limit aflatoxin and ochratoxin absorption while binding lead and cadmium from contaminated sources [14]. Pilot programs in high-mycotoxin regions have recorded better animal health and less toxin carry-over into meat and dairy [90]. In beverages and dairy, probiotic-fortified fermented products help control biofilms on processing equipment and cut the risk of persistent contamination [66, 91]. *S. cerevisiae* var. *boulardii* brings something bacterial probiotics do not: it competes directly for receptor-binding sites, blunts pathogen virulence, and blocks the filamentation and biofilm formation of *Candida albicans* [92, 93]. It also reduces the adhesion and gut invasion of both *Salmonella enterica* serovar Typhimurium and enteropathogenic *Escherichia coli* while keeping tight-junction proteins intact, which makes it a genuinely multifunctional agent in food-contact and clinical biofilm settings [94-96]. Early food-safety deployments in Southeast Asia and Africa show that these systems can extend shelf life and strengthen safety profiles through natural bioactivity alone, with no artificial additives [14]. Their reach extends past food: probiotic consortia have shown promise in environmental remediation, including wastewater carrying mixed heavy metals and fungal toxins, which speaks to how adaptable they are across settings. In each case, the practical result is a measurable drop in the public-health risk from dietary and environmental toxin exposure [14].

## 5.2. Relevance to Professionals, educators, and policymakers

The principles behind probiotic platforms reach a wide set of stakeholders. In clinical gastroenterology and nutrition in particular, they serve as useful adjuncts for managing toxin-associated disorders and restoring gut balance in patients under chronic, low-level exposure [97]. Clinicians can recommend

selected strains to support the gut barrier and offset the effects of mycotoxins and heavy metals in high-risk patients or regions [14]. For teachers in microbiology, biotechnology, and public health, the platforms make strong case studies: they show bioremediation and microbiome science working together, and they push students in sustainable agriculture and environmental health to think across disciplines [98]. Policymakers can lean on the growing evidence to shape rules on food-safety standards, probiotic health claims, and environmental detoxification [99, 100]. International bodies working on global contamination might use the same insights to fold probiotic technologies into food-security policy and One Health frameworks [2, 101]. The field, in short, links current research to real decisions and supports biologically sustainable answers to pressing social problems [102].

## 5.3. Challenges in translating theory into practice

Turning these platforms into routine practice is not straightforward. Strain-specific efficacy shifts with host microbiome, diet, and environment, which makes it hard to build standardized products that behave the same across populations [103]. Regulation is a second barrier: authorities usually want fuller evidence for detoxification claims than for ordinary probiotic health benefits, and that slows approval and commercialization [35]. Cost and logistics: steep production costs and hard distribution in low-resource areas further restrict access exactly where contamination is worst [104]. Other obstacles include fitting the products to existing manufacturing lines, holding stability through storage and transport, and answering public unease about live or genetically modified organisms in food [105]. Stability problems are themselves strain-dependent, and both viability and detoxifying activity can fall during storage, processing, and gut transit [106]. Differences in baseline microbiome add still more variation, shaping



both detoxification efficiency and host response [107, 108].

## 6. Critical Reflections and Gaps in the Literature

### 6.1. Strengths and Contributions of the Current Literature

The expanding body of research on probiotic-based platforms for the simultaneous neutralization of mycotoxins, biosorption of heavy metals, and displacement of pathogenic biofilms exhibits several notable strengths. These studies have convincingly established the multifunctional capacity of selected lactic acid bacterial strains and their ability to address co-occurring contaminants through complementary mechanisms [14,66]. A key strength is the interdisciplinary character of the field, which effectively bridges microbiology, toxicology, and environmental biotechnology; this integration has generated valuable practical insights into sustainable detoxification strategies that extend well beyond traditional single-target interventions [14,109]. Recent work has further clarified strain-specific performance and the benefits of probiotic consortia, providing a more robust understanding of synergistic effects in complex, multi-contaminant environments [110, 111]. The literature also stands out for its increasing emphasis on translational relevance, linking laboratory results to potential applications in food safety, animal husbandry, and human health, while advocating biologically based alternatives to chemical remediation; such efforts align closely with global sustainability priorities [14]. Collectively, these contributions have reinforced theoretical frameworks and stimulated innovation in strain selection, formulation design, and delivery systems [66].

### 6.2. Gaps, Inconsistencies, and Under-Researched Areas

Real gaps remain. Most studies rest on short-term in vitro tests or acute animal models, so the long-term

behavior of these platforms, especially under chronic exposure, is largely unknown [14]. How the interventions interact with diverse human microbiomes is also poorly understood, above all in vulnerable groups such as children, older adults, and the immunocompromised [112]. Regional context is another blind spot: few studies allow for local contaminant profiles, diets, or conditions, which leaves high-burden regions in low- and middle-income countries without tailored data [14]. Results often diverge between clean laboratory conditions and the messy real-world matrices of mixed toxins and shifting pH, a persistent translational gap. The three core functions, mycotoxin neutralization, heavy metal biosorption, and biofilm displacement, are seldom studied together in one design, and that fragmentation hides both their combined effects and any synergistic toxicity [37, 113].

Even cell-wall adsorption, the best-studied mechanism, carries a caveat: binding is mostly reversible, highly strain-specific, and sensitive to pH and temperature in ways that differ sharply between the laboratory and food matrices [114]. For enzymatic degradation and conversion to less toxic derivatives, the ground is shakier still, promising in principle, but not yet backed by the systematic mechanistic evidence the field needs before such pathways can be built into formulations with confidence [3, 114]. The stability of probiotic–AFB<sub>1</sub> complexes under physiological conditions looks encouraging, with under 11% of the bound toxin coming off across the gut pH range; laboratory stability, though, is not the same as clinical efficacy. The binding is reversible, extracellular, and sensitive to strain, treatment history, and matrix. Whether these complexes survive full human digestion, lower actual AFB<sub>1</sub> bioavailability, and reduce the mutagenic load in target tissues is a question that in vitro washing cannot settle, and the longitudinal human trials built around those endpoints are simply missing [20]. That



said, the human evidence is not quite empty. A randomized pilot study in Tanzanian pregnant women and schoolchildren under chronic environmental exposure found that regular consumption of yogurt containing a concentrated *Lactocaseibacillus rhamnosus* culture prevented further rises in blood mercury and arsenic. However, it did not lower the levels already present [3, 115]. The distinction matters: what such work supports is a protective, exposure-limiting role rather than active depuration of an existing body burden. One small trial cannot carry the field. Still, it does mark out the endpoint that future studies should be built around — change in measured body burden under continuing exposure, rather than removal percentages in a beaker. Two further areas are thinly covered: possible interference with the uptake of essential nutrients, and the wider ecological fallout of using probiotics at scale in the environment [116]. Multi-omics is starting to map the relevant pathways, but genuinely comprehensive integrative studies are still rare [66, 117]. The safety of biodegradation itself has had too little scrutiny. Because enzymatic transformation permanently alters toxin structure, it can in principle create new derivatives whose toxicity is unknown; few studies actually follow the fate and activity of these breakdown products, even though calling a toxin “detoxified” because the parent has vanished may conceal equally dangerous metabolites. A parallel problem arises even when no transformation occurs. In fermented maize meal, lactic acid bacteria cut fumonisin B1 by nearly three-quarters. Yet, the fermented product was not significantly less toxic to a human esophageal carcinoma cell line — because binding lowers the measured concentration without necessarily altering how much toxin the body can take up [3]. Removal percentages and toxicological benefit are therefore not interchangeable, and reviews that treat them as equivalent overstate what the evidence supports. Routine monitoring of degradation by-

products should be treated as a precondition for moving enzymatic detoxification into food and feed [22].

### 6.3. Results from Existing Human Studies

Most evidence on the neutralization of mycotoxins and the biosorption of heavy metals by probiotics comes from laboratory and animal models; however, a small but growing number of clinical and human trials—particularly for heavy metal detoxification—provide preliminary support. The most notable is a randomized open-label pilot study in Tanzanian pregnant women and schoolchildren chronically exposed to environmental toxins. Participants consumed yogurt containing *Lactocaseibacillus rhamnosus* GR-1 ( $\approx 10^{10}$  CFU/250 g serving). In pregnant women, probiotic yogurt exerted a protective effect, significantly attenuating further increases in blood mercury (by 3.2 nmol/L,  $P = 0.035$ ) and arsenic (by 2.3 nmol/L,  $P = 0.011$ ) levels compared to controls. However, it did not reduce pre-existing burdens. Effects were less pronounced or non-significant in children [118]. The most notable recent clinical evidence comes from a randomized, double-masked trial involving 152 Chinese workers exposed to heavy metals at a metallurgical plant in Jinchang, Gansu. For 12 weeks, participants consumed 250 g daily of probiotic yogurt containing *Lactobacillus bulgaricus*, *Streptococcus thermophilus*, and the copper-resistant strain *Pediococcus acidilactici* GR-1 ( $\approx 10^{10}$  CFU/g). Compared to conventional yogurt, this regimen resulted in a significantly greater reduction in blood copper levels, alongside favorable changes in gut microbiota and metabolomic profiles, supporting a microbiome-mediated mechanism for heavy metal detoxification [119]. Regarding mycotoxins, direct evidence from human studies remains very limited. A small randomized controlled trial using a combination of *L. rhamnosus* LC705 and *Propionibacterium freudenreichii* demonstrated a



reduction in aflatoxin exposure biomarkers and a potential decrease in liver cancer risk after five weeks; however, larger confirmatory trials are lacking. Systematic reviews of clinical trials confirm moderate benefits concerning heavy metal levels—particularly in highly exposed populations—but highlight inconsistent effects on blood parameters and a scarcity of data on human health outcomes associated with various other contaminants or biofilms [120,121]. Regulatory hurdles and substantiation: Claims regarding detoxification benefits require stronger evidence than those concerning the general benefits of probiotics. Demonstrating reduced bioavailability, altered toxicity, or clinical benefits is challenging and costly. Postbiotic or heat-killed products may mitigate viability-related issues but still face rigorous scrutiny [122]. Mechanical and ecological niches, potential interference with essential nutrient uptake, impacts on native microbiota, the fate of degradation by-products, and the long-term ecological effects of large-scale application have been insufficiently studied. Synergistic or antagonistic interactions among the three target functions—mycotoxin binding, metal biosorption, and biofilm displacement—are rarely tested in integrated experimental designs [123].

#### 6.4. Ongoing Debates and Unresolved Issues

Several debates continue to influence both research priorities and practical implementation in this field [124,125]. One central question is the relative value of viable versus non-viable cells. While live probiotics may provide additional metabolic benefits, inactivated biomass often demonstrates strong biosorption capacity, raising practical questions about efficacy, safety, and shelf life [14, 66]. Safety concerns surrounding genetically enhanced strains represent another unresolved issue. Safety questions are not hypothetical. The FAO has formally identified four risk categories tied to probiotic use in medically vulnerable populations: systemic infec-

tions, harmful metabolic byproducts, excessive immune activation, and gene transfer, specifically the possibility that antibiotic resistance genes carried by probiotic cells migrate to resident gut pathogens. None of these risks disqualifies probiotics as a therapeutic strategy, but each one demands strain-level safety data before clinical or large-scale food applications can move forward with confidence [110,126]. Discussions focus on the risks of horizontal gene transfer and unintended ecological impacts. Standardization and regulatory frameworks also remain contentious, as marked variability in strain performance makes it difficult to establish universal guidelines for detoxification [127, 128]. Other debates address the selectivity of biofilm-displacement mechanisms and the possibility that broad-spectrum activity could disrupt beneficial microbial communities in the gut or environment [14]. Questions of cost-effectiveness, scalability, and equitable access in industrial and clinical settings further complicate the path forward. Finally, opinions differ on whether the future lies in personalized, exposure-tailored probiotics or in broadly applicable consortia [129, 130]. These unresolved questions highlight the need for continued rigorous research to refine theoretical models and better connect laboratory promise with real-world outcomes [131].

#### 7. Materials and Methods

This narrative review examines the emerging idea of probiotic platforms that can contribute to mycotoxin neutralization, heavy metal biosorption, and biofilm displacement [132]. Structured searches were run in PubMed, Scopus, Web of Science, and Google Scholar and were last updated in June 2026. Three groups of terms were combined with AND, and the terms within each group with OR: organism terms (probiotic, lactic acid bacteria, *Lactobacillus*, *Bifidobacterium*, *Saccharomyces boulardii*, postbiotic); contaminant terms (mycotoxin, aflatoxin, ochratoxin, zearalenone, heavy metal, cadmium, lead, biofilm,



quorum sensing); and mechanism terms (detoxification, biosorption, adsorption, binding, biodegradation, competitive exclusion, bacteriocin). These searches were complemented by targeted screening of specialized journals in microbiology, applied biotechnology, environmental toxicology, and food science, and by hand-searching the reference lists of the retrieved articles. Authoritative books, review articles, and reports from bodies such as the World Health Organization and the Food and Agriculture Organization were consulted as well, so that the review would rest on both current evidence and established conceptual frameworks [13,133]. Peer-reviewed publications in English were included when they examined probiotic mechanisms or applications bearing on at least one of the three focal domains: mycotoxin detoxification, heavy metal binding or biosorption, and biofilm-related effects. Records were excluded when they were not peer-reviewed, when no full text was available, or when they dealt with probiotics without reference to any of the three contaminant classes. Work published between 2010 and 2026 received the most weight, though earlier landmark papers were retained where they supplied essential historical or theoretical context. Titles and abstracts were screened first, and the remaining full texts were assessed against these criteria; where sources overlapped, preference went to studies on strain-specific activity, mechanistic detail, synergy, or translation, rather than to narrow single-contaminant reports [14, 66]. The authors carried out screening, and disagreements were resolved by discussion. Because the three literatures rest on very different designs, each study was also noted as *in vitro*, simulated gastrointestinal or *ex vivo*, animal *in vivo*, or human clinical, so that results from simple buffer systems would not be read as equivalent to those from complex matrices or living hosts. Where the original authors stated that their own design could not establish bioavailability or clinical effect,

that caution was carried forward here rather than dropped [20]. Because the aim was an integrative, interpretive synthesis and not a pooled effect estimate, a narrative approach was chosen. It let the literature be organized by theme (conceptual, historical, applied, and critical) which supported a coherent account of mechanisms, applications, and gaps. It left room to weave together experimental studies, theory, and practical concerns from different fields [134, 135]. The approach has its limits, among them the risk of selection bias and the lack of a quantitative meta-analysis, both recognized constraints of narrative review [134].

## 8. Synthesis of Findings

The studies reviewed here point to this very fact: selected lactic acid bacteria and yeasts act as multifunctional agents, targeting all three categories of contaminants through the mechanisms outlined in Section 2 [14,20]. Mycotoxin neutralization runs mainly through surface adsorption, though some strains also achieve partial degradation. Heavy metal biosorption turns on interactions with wall components — peptidoglycans, exopolysaccharides, and anionic groups such as carboxyl, hydroxyl, amino, and phosphoryl [136]. Biofilm displacement relies on competitive exclusion, antimicrobial activity, and the occupation of niches that would otherwise support pathogen growth [38]. Two further findings cut across all three axes. First, the negative charge of the LAB wall gives it a natural pull on heavy metal cations,  $\text{Hg}^{2+}$ ,  $\text{Cd}^{2+}$ , and  $\text{Pb}^{2+}$  among them [81], and strains that make exopolysaccharides push this further, exposing extra carboxyl, hydroxyl, and phosphate groups that act as additional binding sites; EPS-producers are therefore not just better binders but bind across a wider range of metals and conditions [137]. Two constraints govern how far this capacity extends. Binding is sharply pH-dependent: adsorption of cadmium and lead is negligible below



pH 3 and climbs to a maximum between pH 4 and 6, which means gastric conditions work against the very mechanism being relied upon [138]. Selectivity also varies by element, with LAB showing a preference order of  $\text{Hg}^{2+} > \text{Cd}^{2+} > \text{Pb}^{2+} = \text{As}^{3+}$ , so that arsenic — the metalloid of greatest concern in several high-burden regions — is bound least readily of all [3, 139].

Second, how much a strain achieves varies so widely that strain identity, rather than species or genus, is the most reliable predictor of performance in the published record. Table 2 examines specific probiotic strains and their detoxification functions, considering various contaminant types, different matrices, and mechanistic pathways.

**Table 2. Selected probiotic strains and their documented detoxification performance across contaminant classes, matrices, and mechanistic pathways**

| Probiotic Type                                                | Contaminant Class                   | Mechanism                                                                        | Key Finding                                                                                                                                                                                                                                                                               | Reference |
|---------------------------------------------------------------|-------------------------------------|----------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------|
| <i>Lactobacillus acidophilus</i> EMCC1324                     | Aflatoxins (AFB1, AFB2, AFG1, AFG2) | Cell-surface binding and passive absorption                                      | Up to 95.59% removal across combined in vitro and in vivo rat models                                                                                                                                                                                                                      | [137]     |
| <i>Limosilactobacillus reuteri</i> CGMCC1.3264                | Zearalenone (ZEN)                   | Enzymatic hydrolysis via surface-displayed lactonohydrolase                      | Complete (100%) hydrolysis in contaminated maize kernels                                                                                                                                                                                                                                  | [141]     |
| <i>Lactobacillus delbrueckii subsp. bulgaricus</i> KLDS1.0207 | Lead (Pb)                           | Cell wall binding and enhanced fecal excretion                                   | Measurable increase in fecal Pb elimination in BALB/c mice at 50 mg/kg/day exposure<br>Viable cells retained 38–50% of initially bound AFB1 after five aqueous washes, rising to 66–71% after heat or acid treatment; only 6–11% was released across the gastrointestinal pH range (2–10) | [142]     |
| <i>Lactocaseibacillus rhamnosus</i> GG / LC-705               | Aflatoxin B1 (AFB1)                 | Extracellular, noncovalent surface adsorption driven by hydrophobic interactions | Up to 96.88% AFM1 removal in reconstituted low-fat milk within 90 min at 37 °C<br>79.5–86.7% removal for encapsulated preparations versus 33–40% for other formulations, under simulated gastrointestinal                                                                                 | [20]      |
| <i>Saccharomyces cerevisiae</i> var. <i>boulardii</i>         | Aflatoxin M1 (AFM1)                 | Rapid cell wall adsorption via $\beta$ -glucans, mannans, and chitin             | Up to 96.88% AFM1 removal in reconstituted low-fat milk within 90 min at 37 °C                                                                                                                                                                                                            | [143]     |
| <i>Saccharomyces cerevisiae</i> var. <i>boulardii</i> RCo09   | Aflatoxin B1 (AFB1)                 | Cell wall adsorption, enhanced by encapsulation                                  | 79.5–86.7% removal for encapsulated preparations versus 33–40% for other formulations, under simulated gastrointestinal                                                                                                                                                                   | [144]     |



|                                                                                   |                                              |                                                         |                                                                                                                        |       |
|-----------------------------------------------------------------------------------|----------------------------------------------|---------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------|-------|
|                                                                                   |                                              |                                                         | conditions (50 ng/mL, 10 <sup>9</sup> CFU/mL, 4 h)                                                                     |       |
| <i>Saccharomyces cerevisiae</i> var. <i>boulardii</i> CNCM I-1079                 | Deoxynivalenol (DON)                         | NF-κB and p38 MAPK pathway attenuation; host modulation | Histological restoration and metabolic normalization in the intestine, liver, and kidney of male piglets after 28 days | [145] |
| <i>Saccharomyces cerevisiae</i> var. <i>boulardii</i> + <i>Lactobacillus</i> spp. | Phthalates (DEHP, DBP) and Bisphenol A (BPA) | Direct binding to cell wall components                  | 29.25% (DEHP), 29.04% (DBP), and 41.75% (BPA) binding within 4 h under in vitro conditions                             | [146] |

*Lactiplantibacillus plantarum*, *Lactobacillus crispatus*, and *Lactobacillus acidophilus* perform especially well in simulated gut models and complex matrices [14,136]. Consortia frequently outdo single strains, with synergistic drops in the bioavailability of major contaminants aflatoxins, ochratoxins, cadmium, and lead [14,147, 148] the numbers for *S. cerevisiae* var. The numbers for *S. cerevisiae* var. *boulardii* can be striking. Still, they are strongly dose-dependent: in reconstituted low-fat milk at 37 °C and a 90-minute contact time, removal of aflatoxin M1 rose from 45.93% at 10<sup>7</sup> CFU/mL to 96.88% at 10<sup>9</sup> CFU/mL, the latter ahead of both *Lacticaseibacillus casei* and *Lactobacillus acidophilus* tested under the same conditions; shortening contact time to 30 min lowered the figure to 91.5% [4]. Reported efficacy therefore reflects the assay conditions as much as the organism, and headline percentages should be read against the dose, contact time, and matrix that produced them. Enzymes add a dimension that

adsorption cannot reach on its own. A 54 kDa serine protease (ysp3) from *S. cerevisiae* var. *boulardii* hydrolyses and neutralizes *Clostridioides difficile* toxins A and B [149, 150], and a 63 kDa phosphatase (pho8) targets *E. coli* endotoxin. These are not passive binding events but active catalytic conversions that permanently change toxin structure, which raises the prospect that enzymatic detoxification could complement adsorption or replace it where reversible binding is a worry [150]. Exopolysaccharide production, paired with modern omics, has been central to spotting strains with strong binding and several detoxification pathways [14, 136]. Put together, the evidence makes these probiotics versatile tools for food safety, animal feed, and environmental remediation [14,66]. Table 3 summarizes evidence on probiotic-mediated detoxification across three contaminant categories, detailing representative strains, mechanisms, experimental models, and the resulting level of evidence.



**Table 3. Evidence map for probiotic-mediated detoxification across the three contaminant classes, showing representative strains, mechanisms, experimental models, and the level of evidence attained.**

| Contaminant class              | Representative strain                                                | Principal mechanism                                          | Experimental model                            | Level of evidence           | Ref.     |
|--------------------------------|----------------------------------------------------------------------|--------------------------------------------------------------|-----------------------------------------------|-----------------------------|----------|
| <b>Mycotoxins</b>              |                                                                      |                                                              |                                               |                             |          |
| Aflatoxins (B1, B2, G1, G2)    | <i>Lactobacillus acidophilus</i> EMCC1324                            | Cell-surface binding and passive absorption                  | Infant cereal, followed by albino rats        | In vitro and animal in vivo | [140]    |
| Aflatoxin B1                   | <i>Lactocaseibacillus rhamnosus</i> GG and LC-705                    | Non-covalent adsorption at the extracellular surface         | Phosphate-buffered saline                     | In vitro, buffer only       | [20]     |
| Aflatoxin M1                   | <i>S. cerevisiae</i> var. <i>boulardii</i>                           | Wall adsorption via $\beta$ -glucans, mannans, chitin        | Reconstituted low-fat milk                    | In vitro, food matrix       | [143]    |
| Aflatoxin B1                   | <i>S. cerevisiae</i> var. <i>boulardii</i> RCo09                     | Wall adsorption, improved by encapsulation                   | Simulated gastrointestinal tract              | Simulated gastrointestinal  | [144]    |
| Zearalenone                    | <i>Limosilactobacillus reuteri</i> CGMCC1.3264                       | Enzymatic hydrolysis by surface-displayed lactonohydrolase   | Contaminated maize kernels                    | In vitro, food matrix       | [141]    |
| Deoxynivalenol                 | <i>S. cerevisiae</i> var. <i>boulardii</i> CNCM I-1079               | Attenuation of NF- $\kappa$ B and p38 MAPK signaling         | Male piglets over 28 days                     | Animal in vivo              | [145]    |
| <b>Heavy metals</b>            |                                                                      |                                                              |                                               |                             |          |
| Lead                           | <i>Lactobacillus delbrueckii</i> subsp. <i>bulgaricus</i> KLDS1.0207 | Wall binding with increased fecal excretion                  | BALB/c mice                                   | Animal in vivo              | [142]    |
| Cadmium and lead               | Lactic acid bacteria, various strains                                | Ion exchange and surface complexation                        | Aqueous solution                              | In vitro, buffer only       | [3]      |
| Mercury and arsenic            | <i>Lactocaseibacillus rhamnosus</i> GR-1                             | Biosorption limiting further uptake                          | Pregnant women and schoolchildren in Tanzania | Human clinical, pilot trial | [3, 126] |
| <b>Pathogenic biofilms</b>     |                                                                      |                                                              |                                               |                             |          |
| <i>Vibrio parahaemolyticus</i> | <i>Lactiplantibacillus plantarum</i> A10                             | Quenching of AI-2 quorum sensing                             | Biofilm assay                                 | In vitro                    | [16]     |
| <i>Vibrio parahaemolyticus</i> | <i>Lactocaseibacillus rhamnosus</i> MS1                              | Competitive exclusion through aggregation and hydrophobicity | Biofilm assay                                 | In vitro                    | [16]     |



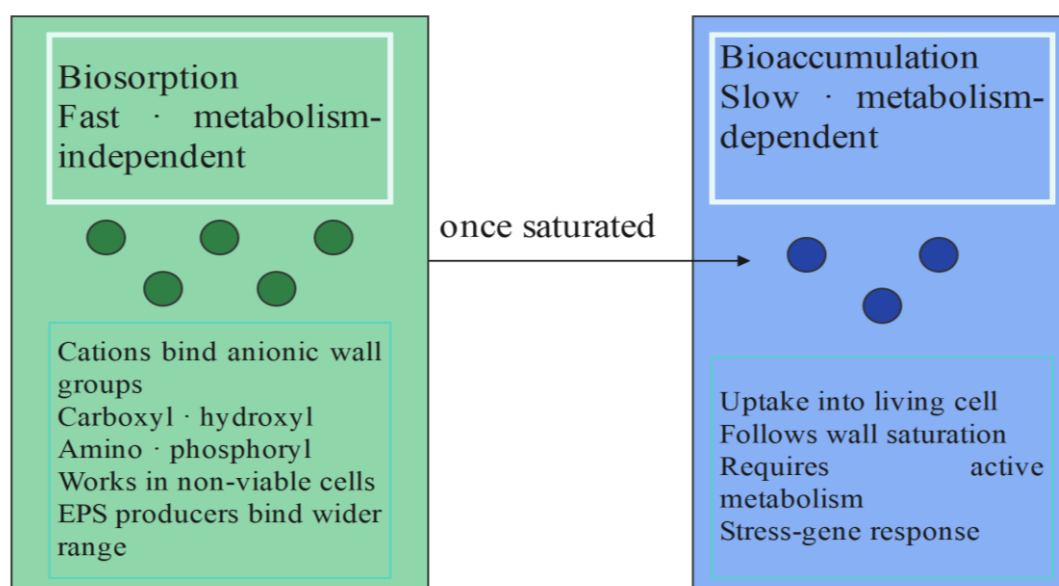
|                                                    |                                                                           |                                                              |                                               |                             |          |
|----------------------------------------------------|---------------------------------------------------------------------------|--------------------------------------------------------------|-----------------------------------------------|-----------------------------|----------|
| <i>E. coli</i> , <i>S. Typhimurium</i>             | <i>Levilactobacillus brevis</i> DFO1, crude bacteriocin                   | Bacteriocin-mediated blocking of biofilm establishment       | Biofilm assay                                 | In vitro                    | [157]    |
| <i>Candida albicans</i> , <i>Salmonella</i> , EPEC | <i>S. cerevisiae</i> var. <i>boulardii</i>                                | Competition for receptor sites and suppression of virulence  | T84 intestinal cell monolayers                | In vitro, cell culture      | [93–96]  |
| <b>Mixed contaminants</b>                          |                                                                           |                                                              |                                               |                             |          |
| Phthalates (DEHP, DBP) and bisphenol A             | <i>S. cerevisiae</i> var. <i>boulardii</i> with <i>Lactobacillus</i> spp. | Direct binding to wall components                            | Binding assay                                 | In vitro                    | [146]    |
| <b>Mycotoxins</b>                                  |                                                                           |                                                              |                                               |                             |          |
| Aflatoxins (B1, B2, G1, G2)                        | <i>Lactobacillus acidophilus</i> EMCC1324                                 | Cell-surface binding and passive absorption                  | Infant cereal, followed by albino rats        | In vitro and animal in vivo | [140]    |
| Aflatoxin B1                                       | <i>Lactocaseibacillus rhamnosus</i> GG and LC-705                         | Non-covalent adsorption at the extracellular surface         | Phosphate-buffered saline                     | In vitro, buffer only       | [20]     |
| Aflatoxin M1                                       | <i>S. cerevisiae</i> var. <i>boulardii</i>                                | Wall adsorption via $\beta$ -glucans, mannans, chitin        | Reconstituted low-fat milk                    | In vitro, food matrix       | [143]    |
| Aflatoxin B1                                       | <i>S. cerevisiae</i> var. <i>boulardii</i> RCO09                          | Wall adsorption, improved by encapsulation                   | Simulated gastrointestinal tract              | Simulated gastrointestinal  | [144]    |
| Zearalenone                                        | <i>Limosilactobacillus reuteri</i> CGMCC1.3264                            | Enzymatic hydrolysis by surface-displayed lactonohydrolase   | Contaminated maize kernels                    | In vitro, food matrix       | [141]    |
| Deoxynivalenol                                     | <i>S. cerevisiae</i> var. <i>boulardii</i> CNCM I-1079                    | Attenuation of NF- $\kappa$ B and p38 MAPK signaling         | Male piglets over 28 days                     | Animal in vivo              | [145]    |
| <b>Heavy metals</b>                                |                                                                           |                                                              |                                               |                             |          |
| Lead                                               | <i>Lactobacillus delbrueckii</i> subsp. <i>bulgaricus</i> KLDS1.0207      | Wall binding with increased fecal excretion                  | BALB/c mice                                   | Animal in vivo              | [142]    |
| Cadmium and lead                                   | Lactic acid bacteria, various strains                                     | Ion exchange and surface complexation                        | Aqueous solution                              | In vitro, buffer only       | [3]      |
| Mercury and arsenic                                | <i>Lactocaseibacillus rhamnosus</i> GR-1                                  | Biosorption limiting further uptake                          | Pregnant women and schoolchildren in Tanzania | Human clinical, pilot trial | [3, 126] |
| <b>Pathogenic biofilms</b>                         |                                                                           |                                                              |                                               |                             |          |
| <i>Vibrio parahaemolyticus</i>                     | <i>Lactiplantibacillus plantarum</i> A10                                  | Quenching of AI-2 quorum sensing                             | Biofilm assay                                 | In vitro                    | [16]     |
| <i>Vibrio parahaemolyticus</i>                     | <i>Lactocaseibacillus rhamnosus</i> MS1                                   | Competitive exclusion through aggregation and hydrophobicity | Biofilm assay                                 | In vitro                    | [16]     |
| <i>E. coli</i> , <i>S. Typhimurium</i>             | <i>Levilactobacillus brevis</i> DFO1, crude bacteriocin                   | Bacteriocin-mediated blocking of biofilm establishment       | Biofilm assay                                 | In vitro                    | [157]    |



|                                                       |                                                                                 |                                                                      |                                   |                           |             |
|-------------------------------------------------------|---------------------------------------------------------------------------------|----------------------------------------------------------------------|-----------------------------------|---------------------------|-------------|
| <i>Candida albicans</i> ,<br><i>Salmonella</i> , EPEC | <i>S. cerevisiae</i> var.<br><i>boulardii</i>                                   | Competition for<br>receptor sites and<br>suppression of<br>virulence | T84 intestinal<br>cell monolayers | In vitro, cell<br>culture | [93–<br>96] |
| <b>Mixed contaminants</b>                             |                                                                                 |                                                                      |                                   |                           |             |
| Phthalates (DEHP,<br>DBP) and<br>bisphenol A          | <i>S. cerevisiae</i> var.<br><i>boulardii</i> with<br><i>Lactobacillus</i> spp. | Direct binding to<br>wall components                                 | Binding assay                     | In vitro                  | [146]       |

Read across the rows rather than down them; Table 3 exposes something the removal percentages alone conceal. Work on mycotoxins has reached animal models and, in several cases, genuine food matrices. Work on heavy metals has reached human participants exactly once, in a single pilot trial, and that trial showed prevention of further accumulation rather than clearance of an existing burden [3, 132]. Work on biofilms has not yet left the in vitro stage at all. The three axes this review treats as a single platform therefore rest on foundations of markedly unequal depth, and claims about integrated multi-target detoxification should be qualified accordingly. The gap is not evenly distributed either: it is widest precisely where translation would matter most, in the populations carrying the heaviest contamination

burden. Set against the historical development traced in Section 3 and the applications described in Section 5, these mechanistic findings complete a consistent picture: the same surface chemistry that governs binding in vitro also underlies the reported gains in food, feed, and environmental settings, and postbiotic or heat-killed preparations extend that chemistry to formats with better stability and easier regulatory acceptance [151-153]. What the evidence keeps returning to, across all three axes, is variation — driven by strain, matrix, and individual host factors [14, 154]. Figure 4 illustrates two pathways for heavy metal removal: adsorption, which operates initially and does not require living cells, and bioaccumulation, which occurs after the cell



**Fig. 4. Two routes remove heavy metals. Surface biosorption acts first and needs no living cell; bioaccumulation follows once wall sites fill and depends on active metabolism.**



## 9. Discussion

What gives this field its coherence is that three separate theoretical traditions — adsorption theory, surface complexation and ion exchange, and microbial antagonism — turn out to describe events on the same physical structure [12]. That is the substantive claim behind the multi-target probiotic hypothesis. It is stronger than the weaker observation that probiotics happen to do several useful things at once: because the three axes share a site of action, selecting for one may constrain or enhance the others, which single-function methods can neither exploit nor predict [14, 18, 66]. Systems biology extends the argument by casting probiotics as active regulators inside host–microbe–environment networks rather than as passive sorbents [14, 155, 156]. Why strain identity should matter so much is best explained by these same variables: dose, matrix, and host together determine which mechanism dominates at any moment, so a wall chemistry that binds well under

one set of conditions may underperform under another. On the same logic, a defined consortium spreads that risk across several surface chemistries at once, which is the most plausible reason mixed cultures hold up better than single strains in multi-contaminant settings [14, 66, 157].

### 9.1. Consistencies, Contradictions, and Methodological Considerations

The literature reveals both consistent patterns and notable contradictions. Many studies report strong simultaneous activity in simulated gastrointestinal and in vitro models; however, the results often diverge about the relative contributions of viable versus non-viable cells, since some contexts emphasize active metabolic processes while others demonstrate effective passive biosorption by inactivated biomass [14, 66]. These differences likely arise from variations in experimental matrices, pH levels, exposure

duration, and the balance between adsorption and enzymatic mechanisms. Translational outcomes also show inconsistencies across regional contaminant profiles and individual microbiomes, which stem largely from the predominance of short-term studies and the limited inclusion of vulnerable populations or high-burden settings [14, 158]. The metal literature shows how tangled this disagreement really is. Across sixteen LAB strains tested against copper, every one bound more as living cells than as dead biomass [158]. Yet *Lactiplantibacillus plantarum* PTCC 1896 bound more cadmium after heat-killing and less lead, while *Weissella viridescens* MY 205 lost its capacity for cadmium and lead but gained it for mercury [160–162]. No single rule survives these results. What they suggest instead is that inactivation alters the cell surface in ways whose consequences depend jointly on the strain and on the metal in question — which is precisely why blanket claims about viable versus non-viable preparations should be treated with suspicion. Such discrepancies illustrate the difficulty of generalizing findings from controlled laboratory conditions to real-world food chains and clinical settings, in which synergistic toxicities and complex ecological interactions add layers of difficulty that are not captured in single-contaminant designs [66, 162].

### 9.2. Strengths, Limitations, and Implications

The main strength of the field is its interdisciplinary base in microbiology, toxicology, and biotechnology. That base has produced usable insight into sustainable detoxification with direct bearing on food safety, animal husbandry, and public-health policy [89, 163]. By reaching past single-target thinking, the work supports holistic platforms that align with sustainability goals as environmental pressure mounts. The limits are just as clear. The three core functions are rarely studied together in one fully integrated design, and long-term ecological effects and possible interference with essential-nutrient uptake have had too little attention [14, 66]. These gaps,



together with the built-in variability of narrative review methods, cap how far the current conclusions can generalize and argue for more standardization and longitudinal work [20, 42]. On balance, the findings sharpen the theory of microbial detoxification networks and give practical direction for managing contamination, feeding more nuanced frameworks for complex environmental-health problems while keeping the emphasis on context-aware, evidence-based use [14, 22]. One pattern keeps recurring: lactic acid bacteria and their bacteriocins can block pathogen biofilms from forming even when they cannot break down mature, pre-formed structures — a distinction with clear practical weight [164].

### 9.3. Future research perspectives

Where should the field go next? Four routes stand out, and each addresses a weakness identified above rather than simply extending current practice. Targeted strain improvement is the most direct. CRISPR-Cas9 has now been adapted to probiotic yeasts, which makes rational redesign feasible for the first time. The obvious target is the cell wall itself: if flocculins and other wall adhesins can be engineered so that contaminants bind more tightly and release less readily, that would address reversibility — the single most persistent weakness of adsorption-based detoxification [165-168]. Beyond surface modification, CRISPR can also knock out genes responsible for undesirable metabolites or insert entirely new catabolic pathways that degrade mycotoxins enzymatically, offering a permanent rather than reversible mode of action. However, strains free of antibiotic-resistance markers will be a precondition for any realistic food application, and regulatory pathways for genetically modified probiotics remain to be clearly defined. A second route runs through enzymes rather than surfaces. Endogenous proteases and phosphatases could in principle be amplified, or entirely new catabolic activities introduced, widening the range of compounds a strain can degrade beyond

anything surface binding alone can reach [169, 170]. Synthetic biology offers tools that go beyond single-gene edits. Refactored genetic circuits could enable probiotics to sense a contaminant and respond by upregulating specific binding or degradation pathways only when needed. Engineering consortia with complementary, division-of-labor strategies (e.g., one strain degrades mycotoxins while another biosorbs heavy metals) could outperform any single organism in multi-contaminant matrices. The successful surface display of lactonohydrolase on *Limosilactobacillus reuteri* for zearalenone hydrolysis already proves this concept is viable; scaling it to other toxins is the logical next step. The third concerns mechanism. Metabolomics, proteomics, and transcriptomics have mostly been applied under clean laboratory conditions; applying them within actual food matrices would show which metabolites, wall components, and secreted proteins really govern binding and how stable that binding is. That shift would move the field past descriptive removal percentages toward mechanistic explanation [4]. Integrated multi-omics metagenomics, transcriptomics, proteomics, and metabolomics used together can also identify biomarkers of detoxification efficacy in human trials, which is essential for regulatory approval and personalized applications. This approach would help us understand host-microbe interactions at a systems level and predict how different formulations will perform in different populations. The fourth is consortium design. Pairing yeasts with lactic acid bacteria produces complementary modes of action. It may improve adsorption through co-aggregation, while combining living cells with prebiotic or postbiotic components could limit desorption during processing and storage [171]. One concrete approach already demonstrated is immobilizing yeast cell-wall fractions on nano-silica entrapped in alginate, which reduces desorption and allows the adsorbent to be reused [172]. Artificial intelligence and machine



learning can accelerate strain discovery beyond current screening limits. Instead of testing hundreds of strains empirically, AI models trained on cell-wall composition, surface hydrophobicity, and functional-group data could predict multi-target binding efficiency *in silico*. Coupled with high-throughput phenotypic screening, this approach would prioritize the most promising candidates for validation, saving time and resources. AI could also optimize consortium compositions by predicting synergistic or antagonistic interactions between strains across different contamination profiles. Precision microbiome interventions represent the clinical frontier beyond consortia design. Rather than a one-size-fits-all probiotic, future formulations could be tailored to an individual's baseline microbiome composition, genetic background, and specific exposure profile (e.g., high aflatoxin in one region, high cadmium in another). This requires large-scale human cohort studies that integrate exposure monitoring, microbiome sequencing, and clinical outcomes — a challenging but necessary step for real translational impact. Two cautions belong alongside this optimism. Engineered strains raise regulatory questions that conventional probiotics do not, and strains free of antibiotic-resistance markers will be a precondition for any realistic food application [166]. Nor does any of this remove the need for the standardized protocols and reporting conventions called for earlier. Public perception of genetically modified organisms in food remains a hurdle, and responsible innovation will require transparent safety assessment alongside technological progress.

## 10. CONCLUSION

This review synthesizes existing evidence into a novel conceptual framework, presenting probiotic systems not merely as individual agents but as multifunctional detoxification platforms with three primary objectives: mycotoxin neutralization, heavy metal biosorption, and biofilm displacement. What

distinguishes this framework from previous reviews is its emphasis on functional convergence at the cell wall level—enabling the design of hybrid systems. In contrast, past research has examined these functions in isolation. Evidence indicates that strains of *Lactobacillus*, *Bifidobacterium*, and *Saccharomyces boulardii* perform all three functions, although the feasibility of executing them simultaneously requires further investigation. While the primary mechanisms—adsorption to polysaccharides, surface complexation, and competitive inhibition—are well understood, the fact that they all converge at the microbial surface offers a promising basis for engineering synergistic systems. However, the path to clinical application faces challenges such as strain stability within the gastrointestinal tract, interaction with the indigenous microbiome, determination of effective dosages, and long-term safety; consequently, fewer than 5 percent of studies have reached the stage of human trials. Therefore, future research priorities should focus on conducting long-term trials involving at-risk populations, evaluating concurrent effects within complex food and biological matrices, and establishing standardized protocols. Modern perspectives in this field transcend traditional approaches, encompassing areas such as CRISPR-based strain engineering, the design of engineered probiotics, AI-assisted discovery of superior strains, and the application of multi-omics approaches to elucidate host-microbe interactions. Furthermore, personalized formulations—tailored to an individual's microbiome characteristics and exposure patterns—combined with optimized consortia, offer a promising pathway for realizing this multifaceted framework in real-world settings.

## 11. DECLARATIONS

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### 11.2. Conflict of Interest

The authors declare no conflict of interest.

### 11.3. Authors' Contributions

**Tanaz Tarahi:** Conceptualization, Investigation, Writing the first draft, **Shima Jahanfar:** Supervision, Resources, Data curation, Edit and Review of manuscript, **Maryam Soheili:** Software, Edit and review of manuscript.

### 11.4. Using Artificial Intelligence chatbots

Grok and Claude AI were used to translate and rewrite the articles, and Grammarly was used to review the text; however, the authors subsequently reviewed these sections.

## REFERENCES

- Schatzmayr G, Zehner F, Täubel M, Schatzmayr D, Klimitsch A, Loibner AP, et al. Microbiologicals for deactivating mycotoxins. *Molecular nutrition & food research*. 2006;50(6):543–51.  
<https://doi.org/10.1002/mnfr.200500181>
- Średnicka P, Juszczuk-Kubiak E, Wójcicki M, Akimowicz M, Roszko M. Probiotics as a biological detoxification tool of food chemical contamination: A review. *Food and Chemical Toxicology*. 2021;153:112306.  
<https://doi.org/10.1016/j.fct.2021.112306>
- Petrova P, Arsov A, Tsvetanova F, Parvanova-Mancheva T, Vasileva E, Tsigoriyna L, et al. The complex role of lactic acid bacteria in food detoxification. *Nutrients*. 2022;14(10):2038.  
<https://doi.org/10.3390/nu14102038>
- Pereira KN, de Oliveira ACD, de Souza HF, Ullah S, Nasir U, Ali S, et al. Application of *Saccharomyces cerevisiae* var. *boulardii* for Biological Detoxification of Chemical Contaminants in Foods: A Comprehensive Review. *Foods*. 2025;14(24):4260.  
<https://doi.org/10.3390/foods14244260>
- Zahir A, Ge Z, Khan IA. Public health risks associated with food process contaminants—A review. *Journal of Food Protection*. 2025;88(2):100426.  
<https://doi.org/10.1016/j.jfp.2024.100426>
- Guerre P. Mycotoxin and gut microbiota interactions. *Toxins*. 2020;12(12):769.  
<https://doi.org/10.3390/toxins12120769>
- Zhang J, Liu X, Su Y, Li T. An update on T2-toxins: Metabolism, immunotoxicity mechanism and human exposure assessment of intestinal microbiota. *Heliyon*. 2022;8(8).  
<https://doi.org/10.1016/j.heliyon.2022.e10012>
- Shabani M, Ghoshehy A, Mottaghi AM, Chegini Z, Kerami A, Shariati A, et al. The relationship between gut microbiome and human diseases: mechanisms, predisposing factors, and potential intervention. *Frontiers in Cellular and Infection Microbiology*. 2025;15:1516010.  
<https://doi.org/10.3389/fcimb.2025.1516010>
- Abou Izzeddine N, Ahmad K, Bacha C, Jabbour M, Najjar M, Salhab S, et al. The microbial guardians: Unveiling the role of gut microbiota in shaping neurodegenerative disease. *IBRO Neuroscience Reports*. 2025;19:17–37.  
<https://doi.org/10.1016/j.ibneur.2025.05.014>
- Anchidin-Norocel L, Iatcu OC, Lobiuc A, Covasa M. Heavy metal–gut microbiota interactions: probiotics modulation and biosensors detection. *Biosensors*. 2025;15(3):188.  
<https://doi.org/10.3390/bios15030188>
- Zoghi A, Khosravi-Darani K, Sohrabvandi S. Surface binding of toxins and heavy metals by probiotics. *Mini reviews in medicinal chemistry*. 2014;14(1):84–98.  
<https://doi.org/10.2174/1389557513666131211105554>
- Massoud R, Zoghi A. Potential probiotic strains with heavy metals and mycotoxins bioremoval capacity for application in foodstuffs. *Journal of Applied Microbiology*. 2022;133(3):1288–307.  
<https://doi.org/10.1111/jam.15685>
- Abdel-Megeed RM. Probiotics: a promising generation of heavy metal detoxification.



- Biological trace element research*. 2021;199(6):2406.  
<https://doi.org/10.1007/s12011-020-02350-1>
14. Pop OL, Suharoschi R, Gabbianelli R. Biodetoxification and protective properties of probiotics. *Microorganisms*. 2022;10(7):1278.  
<https://doi.org/10.3390/microorganisms10071278>
  15. Lázaro Á, Vila-Donat P, Manyes L. Emerging mycotoxins and preventive strategies related to gut microbiota changes: probiotics, prebiotics, and postbiotics—a systematic review. *Food & Function*. 2024;15(18):8998–9023.  
<https://doi.org/10.1039/d4fo01705f>
  16. PENG J, SHANGGUAN W, ZHANG M, ZHONG Q. Probiotic properties of lactic acid bacteria quenching the quorum-sensing of *Vibrio parahaemolyticus*. *Science and Technology of the Food Industry*. 2023;44(8):180–6.  
<https://doi.org/10.13386/j.issn1002-0306.2022070186>
  17. Cui Y, Wang D, Zhang L, Qu X. Research progress on the regulatory mechanism of biofilm formation in probiotic lactic acid bacteria. *Critical Reviews in Food Science and Nutrition*. 2025;65 (25): 4869–83.  
<https://doi.org/10.1080/10408398.2024.2400593>
  18. Pane M, De Prisco A, Amoroso A, Deusebio G, Marzorati M, Corazzari M, et al. Probiotic detoxification of heavy metals: functional assessment in simulated intestinal and ex vivo models. *Frontiers in Microbiology*. 2026; 17:1821114.  
<https://doi.org/10.3389/fmicb.2026.1821114>
  19. García-Reyes RA, García-Cancino A, Arrevillaga-Boni G, Espinoza-Monje M, Gutiérrez-Zamorano C, Arrizon J, et al. Identification and characterization of probiotic *Lactiplantibacillus plantarum* BI-59.1 isolated from tejuino and its capacity to produce biofilms. *Current Microbiology*. 2023;80(7):220.  
<https://doi.org/10.1007/s00284-023-03319-8>
  20. Haskard CA, El-Nezami HS, Kankaanpää PE, Salminen S, Ahokas JT. Surface binding of aflatoxin B1 by lactic acid bacteria. *Applied and environmental microbiology*. 2001;67 (7): 3086–91. <https://doi.org/10.1128/aem.67.7.3086-3091.2001>
  21. c Ooi VE, Liu F. Immunomodulation and anti-cancer activity of polysaccharide-protein complexes. *Current Medicinal Chemistry*. 2000;7(7):715–29.  
<https://doi.org/10.2174/0929867003374705>
  22. Liu L, Xie M, Wei D. Biological detoxification of mycotoxins: current status and future advances. *International Journal of Molecular Sciences*. 2022;23(3):1064.  
<https://doi.org/10.3390/ijms23031064>
  23. Taheur FB, Kouidhi B, Al Qurashi YMA, Salah-Abbès JB, Chaieb K. Biotechnology of mycotoxins detoxification using microorganisms and enzymes. *Toxicon*. 2019;160:12–22.  
<https://doi.org/10.1016/j.toxicon.2019.02.001>
  24. Marin S, Ramos A, Cano-Sancho G, Sanchis V. Mycotoxins: Occurrence, toxicology, and exposure assessment. *Food and chemical toxicology*. 2013;60:218–37.  
<https://doi.org/10.1016/j.fct.2013.07.047>
  25. Khan GI, Akbar A, Anwar M, Shafee M, Sadia H, Razzaq A, et al. 01. Eco-friendly bacterial biodegradation of mycotoxins. *Pure and Applied Biology (PAB)*. 2020;9(3):1708–17.  
<http://dx.doi.org/10.19045/bspab.2020.90181>
  26. Loi M, Fanelli F, Liuzzi VC, Logrieco AF, Mulè G. Mycotoxin biotransformation by native and commercial enzymes: Present and future perspectives. *Toxins*. 2017;9(4):111.  
<https://doi.org/10.3390/toxins9040111>
  27. Gil-Serna J, Vázquez C, Patiño B. Mycotoxins in functional beverages: A review. *Beverages*. 2020;6(3):52.  
<https://www.mdpi.com/2306-5710/6/3/52>



- 28.El-Nezami H, Kankaanpää P, Salminen S, Ahokas J. Physicochemical alterations enhance the ability of dairy strains of lactic acid bacteria to remove aflatoxin from contaminated media. *Journal of Food Protection*. 1998;61(4):466–8.  
<https://doi.org/10.4315/0362-028x-61.4.466>
- 29.Orrhage K, Sillerström E, Gustafsson J-Å, Nord C, Rafter J. Binding of mutagenic heterocyclic amines by intestinal and lactic acid bacteria. *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis*. 1994;311(2):239–48.  
[https://doi.org/10.1016/0027-5107\(94\)90182-1](https://doi.org/10.1016/0027-5107(94)90182-1)
- 30.Thyagaraja N, Hosono A. Binding properties of lactic acid bacteria from 'Idly'towards food-borne mutagens. *Food and chemical toxicology*. 1994;32(9):805–9. [https://doi.org/10.1016/0278-6915\(94\)90156-2](https://doi.org/10.1016/0278-6915(94)90156-2)
- 31.Zhang XB, Ohta Y, Hosono A. Antimutagenicity and binding of lactic acid bacteria from a Chinese cheese to mutagenic pyrolyzates. *Journal of Dairy Science*. 1990;73(10):2702–10.  
[https://doi.org/10.3168/jds.s0022-0302\(90\)78955-2](https://doi.org/10.3168/jds.s0022-0302(90)78955-2)
- 32.Haskard C, Binnion C, Ahokas J. Factors affecting the sequestration of aflatoxin by *Lactobacillus rhamnosus* strain GG. *Chemico-biological interactions*. 2000;128(1):39–49.  
[https://doi.org/10.1016/S0009-2797\(00\)00186-1](https://doi.org/10.1016/S0009-2797(00)00186-1)
- 33.Mrvčić J, Stanzer D, Šolić E, Stehlik-Tomas V. Interaction of lactic acid bacteria with metal ions: opportunities for improving food safety and quality. *World Journal of Microbiology and Biotechnology*. 2012;28(9):2771–82.  
<https://doi.org/10.1007/s11274-012-1094-2>
- 34.Chen L, Garmaeva S, Zhernakova A, Fu J, Wijmenga C. A systems biology perspective on environment–host–microbe interactions. *Human Molecular Genetics*. 2018;27(R2):R187–R94.  
<https://doi.org/10.1093/hmg/ddy137>
- 35.Van Niel EW, Larsson CU, Lohmeier-Vogel EM, Rådström P. The potential of biodegradation activity as a probiotic property of *Lactobacillus reuteri*. *International journal of food microbiology*. 2012;152(3):206–10.  
<https://doi.org/10.1016/j.ijfoodmicro.2011.10.007>
- 36.Langelier C. A proactive charcoal approach shields the gut microbiome from systemic antibiotics. *Science Translational Medicine*. 2018;10 (430): eaas8966.  
<https://doi.org/10.1126/scitranslmed.aas8966>
- 37.Halttunen T, Collado M, El-Nezami H, Meriluoto J, Salminen S. Combining strains of lactic acid bacteria may reduce their toxin and heavy metal removal efficiency from aqueous solution. *Letters in applied microbiology*. 2008;46(2):160–5.  
<https://doi.org/10.1111/j.1472-765x.2007.02276.x>
- 38.Woo J, Ahn J. Probiotic-mediated competition, exclusion and displacement in biofilm formation by food-borne pathogens. *Letters in applied microbiology*. 2013;56(4):307–13.  
<https://doi.org/10.1111/lam.12051>
- 39.Zommiti M, Feuilloley MG, Connil N. Update of probiotics in the human world: a nonstop source of benefactions till the end of time. *Microorganisms*. 2020;8(12):1907.  
<https://doi.org/10.3390/microorganisms8121907>
- 40.Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food. *FAO Plant Production and Protection Paper*. 2017;232.
- 41.Petrova P, Ivanov I, Tsigoriyna L, Valcheva N, Vasileva E, Parvanova-Mancheva T, et al. Traditional Bulgarian dairy products: ethnic foods with health benefits. *Microorganisms*. 2021;9(3):480.  
<https://doi.org/10.3390/microorganisms9030480>
- 42.Fuller R. Probiotics in man and animals. *The Journal of Applied Bacteriology*. 1989; 66 (5): 365–78.
- 43.Hill C, Guarner F, Reid G, Gibson G, Merenstein D, Pot B, et al. Expert consensus document. The International Scientific Association for Probiotics



- and Prebiotics consensus statement on the scope and appropriate use of the term probiotic. *Nature reviews Gastroenterology & hepatology*. 2014;11 (8):506–14.  
<https://doi.org/10.1038/nrgastro.2014.66>
44. Peltonen K, El-Nezami H, Haskard C, Ahokas J, Salminen S. Aflatoxin B1 binding by dairy strains of lactic acid bacteria and bifidobacteria. *Journal of Dairy Science*. 2001;84(10):2152–6.  
[https://doi.org/10.3168/jds.s0022-0302\(01\)74660-7](https://doi.org/10.3168/jds.s0022-0302(01)74660-7)
  45. Kratochvil D, Volesky B. Advances in the biosorption of heavy metals. *Trends in biotechnology*. 1998;16(7):291–300.  
[https://doi.org/10.1016/S0167-7799\(98\)01218-9](https://doi.org/10.1016/S0167-7799(98)01218-9)
  46. Fooks L, Gibson GR. Probiotics as modulators of the gut flora. *British journal of nutrition*. 2002;88(S1):s39–s49.  
<https://doi.org/10.1079/bjn2002628>
  47. Shetty PH, Jespersen L. *Saccharomyces cerevisiae* and lactic acid bacteria as potential mycotoxin decontaminating agents. *Trends in food science & technology*. 2006;17(2):48–55.  
<https://doi.org/10.1016/j.tifs.2005.10.004>
  48. Woloszynek S, Pastor S, Mell J, Nandi N, Sokhansanj B, Rosen G. Engineering human microbiota: influencing cellular and community dynamics for therapeutic applications. *International review of cell and molecular biology*. 324: Elsevier; 2016. p. 67–124.  
<https://doi.org/10.1016/bs.ircmb.2016.01.003>
  49. Xia D, Mo Q, Yang L, Wang W. Crosstalk between mycotoxins and intestinal microbiota and the alleviation approach via microorganisms. *Toxins*. 2022;14(12):859.  
<https://doi.org/10.3390/toxins14120859>
  50. Fernández MF, Boris S, Barbes C. Probiotic properties of human lactobacilli strains to be used in the gastrointestinal tract. *Journal of applied microbiology*. 2003;94(3):449–55.  
<https://doi.org/10.1046/j.1365-2672.2003.01850.x>
  51. Chapot-Chartier M-P, Kulakauskas S. Cell wall structure and function in lactic acid bacteria. *Microbial cell factories*. 2014;13(Suppl 1):S9.  
<https://doi.org/10.1186/1475-2859-13-s1-s9>
  52. Jouany J, Yiannikouris A, Bertin G. The chemical bonds between mycotoxins and cell wall components of *Saccharomyces cerevisiae* have been identified. *Arch Zootech*. 2005;8(4).
  53. Yiannikouris A, Poughon L, Cameleyre X, Dussap C-G, François J, Bertin G, et al. A novel technique to evaluate interactions between *Saccharomyces cerevisiae* cell wall and mycotoxins: application to zearalenone. *Biotechnology letters*. 2003;25 (10): 783–9.  
<https://doi.org/10.1023/a:1023576520932>
  54. Lee E-S, Song E-J, Nam Y-D, Lee S-Y. Probiotics in human health and disease: from nutraceuticals to pharmabiotics. *Journal of Microbiology*. 2018;56(11):773–82.  
<https://doi.org/10.1007/s12275-018-8293-y>
  55. Azad MAK, Sarker M, Wan D. Immunomodulatory effects of probiotics on cytokine profiles. *BioMed research international*. 2018;2018(1):8063647.  
<https://doi.org/10.1155/2018/8063647>
  56. Chang C-J, Lin T-L, Tsai Y-L, Wu T-R, Lai W-F, Lu C-C, et al. Next-generation probiotics in disease amelioration. *Journal of food and drug analysis*. 2019;27(3):615–22.  
<https://doi.org/10.1016/j.jfda.2018.12.011>
  57. Asha MZ, Khalil SF. Efficacy and Safety of Probiotics, Prebiotics and Synbiotics in the Treatment of Irritable Bowel Syndrome: A systematic review and meta-analysis. *Sultan Qaboos University Medical Journal*. 2020;20(1):e13.  
<https://doi.org/10.18295/squmj.2020.20.01.003>
  58. Islam SU. Clinical uses of probiotics. *Medicine*. 2016;95(5):e2658.  
<https://doi.org/10.1097/md.0000000000002658>



59. Bustamante M, Oomah BD, Oliveira WP, Burgos-Díaz C, Rubilar M, Shene C. Probiotics and prebiotics potential for the care of skin, female urogenital tract, and respiratory tract. *Folia microbiologica*. 2020; 65(2) :245–64.  
<https://doi.org/10.1007/s12223-019-00759-3>
60. Lemke A, Burkhardt B, Bunzel D, Pfeiffer E, Metzler M, Huch M, et al. Alternaria toxins of the alternariol type are not metabolised by human faecal microbiota. *World Mycotoxin Journal*. 2016;9(1):41–50.  
<https://doi.org/10.3920/WMJ2014.1875>
61. Crudo F, Aichinger G, Mihajlovic J, Varga E, Dellaflora L, Warth B, et al. In vitro interactions of Alternaria mycotoxins, an emerging class of food contaminants, with the gut microbiota: a bidirectional relationship. *Archives of Toxicology*. 2021;95(7):2533–49.  
<https://doi.org/10.1007/s00204-021-03043-x>
62. Vinderola CG, Ritieni A. Role of probiotics against mycotoxins and their deleterious effects. 2014.  
<https://doi.org/10.5539/jfr.v4n1p10>
63. Corbo MR, Campaniello D, Speranza B, Altieri C, Sinigaglia M, Bevilacqua A. Neutralisation of toxins by probiotics during the transit into the gut: challenges and perspectives. *International Journal of Food Science and Technology*. 2018;53(6):1339–51.  
<https://doi.org/10.1111/ijfs.13745>
64. Yao Y, Long M. The biological detoxification of deoxynivalenol: A review. *Food and Chemical Toxicology*. 2020;145:111649.  
<https://doi.org/10.1016/j.fct.2020.111649>
65. Abdel-Megeed RM. Probiotics: a promising generation of heavy metal detoxification. *Biological trace element research*. 2021;199(6): 2406–13.  
<https://doi.org/10.1007/s12011-020-02350-1>
66. Sionek B, Szydłowska A, Jaworska D, Kołożyn-Krajewska D. Benefits of probiotics—biodetoxification. *Applied Sciences*. 2025;15 (10): 5297. <https://doi.org/10.3390/app15105297>
67. Ferrocino I, Coccolin L. Current perspectives in food-based studies exploiting multi-omics approaches. *Current Opinion in Food Science*. 2017;13:10–5.  
<https://dx.doi.org/10.1016/j.cofs.2017.01.002>
68. Teng TS, Chin YL, Chai KF, Chen WN. Fermentation for future food systems: Precision fermentation can complement the scope and applications of traditional fermentation. *The EMBO Reports*. 2021;22(5):EMBR202152680.  
<https://doi.org/10.15252/embr.202152680>
69. Jabbari V, Mokarram RR, Khiabani MS, Askari F, Ahmadi E, Hassanzadeh A, et al. Molecular Identification of Lactobacillus acidophilus as a probiotic from traditional doogh samples and evaluation of their antimicrobial activity against some pathogenic bacteria. *Biomed Res*. 2017;28(4):1458–63.
70. Hashemi SMB, Gholamhosseinpour A. Fermentation of table cream by Lactobacillus plantarum strains: effect on fungal growth, aflatoxin M1 and ochratoxin A. *International Journal of Food Science and Technology*. 2019;54(2):347–53.  
<https://doi.org/10.1111/ijfs.13943>
71. Hedman R, Pettersson H. Transformation of nivalenol by gastrointestinal microbes. *Archives of animal nutrition*. 1997;50(4):321–9.  
<https://doi.org/10.1080/17450399709386142>
72. Baralić K, Živančević K, Božić D, Đukić-Ćosić D. Probiotic cultures as a potential protective strategy against the toxicity of environmentally relevant chemicals: State-of-the-art knowledge. *Food and Chemical Toxicology*. 2023; 172:113582.  
<https://doi.org/10.1016/j.fct.2022.113582>



73. Ahtesh FB, Stojanovska L, Apostolopoulos V. Antihypertensive peptides released from milk proteins by probiotics. *Maturitas*. 2018;115:103–9. <https://doi.org/10.1016/j.maturitas.2018.06.016>
74. Banerjee D, Jain T, Bose S, Bhosale V. Importance of probiotics in human health. *Functional Food and Human Health: Springer*; 2018. p. 539–54.
75. Amin N, Boccardi V, Taghizadeh M, Jafarnejad S. Probiotics and bone disorders: the role of RANKL/RANK/OPG pathway. *Aging clinical and experimental research*. 2020;32(3):363–71. <https://doi.org/10.1007/s40520-019-01223-5>
76. Hedin C, Whelan K, Lindsay JO. Evidence for the use of probiotics and prebiotics in inflammatory bowel disease: a review of clinical trials. *Proceedings of the Nutrition Society*. 2007;66(3):307–15. <https://doi.org/10.1017/s0029665107005563>
77. Tu P, Chi L, Bodnar W, Zhang Z, Gao B, Bian X, et al. Gut microbiome toxicity: connecting the environment and gut microbiome-associated diseases. *Toxics*. 2020;8(1):19. <https://doi.org/10.3390/toxics8010019>
78. Chi L, Tu P, Ru H, Lu K. Studies of xenobiotic-induced gut microbiota dysbiosis: from correlation to mechanisms. *Gut Microbes*. 2021;13(1):1921912. <https://doi.org/10.1080/19490976.2021.1921912>
79. Ibrahim F, Halttunen T, Tahvonen R, Salminen S. Probiotic bacteria as potential detoxification tools: assessing their heavy metal binding isotherms. *Canadian journal of microbiology*. 2006;52(9):877–85. <https://doi.org/10.1139/w06-043>
80. Xu R, Yiannikouris A, Shandilya UK, Karrow NA. Comparative assessment of different yeast cell wall-based mycotoxin adsorbents using a model- and bioassay-based in vitro approach. *Toxins*. 2023;15(2):104. <https://doi.org/10.3390/toxins15020104>
81. Halttunen T, Finell M, Salminen S. Arsenic removal by native and chemically modified lactic acid bacteria. *International journal of food microbiology*. 2007;120(1-2):173–8. <https://doi.org/10.1016/j.ijfoodmicro.2007.06.002>
82. Zhai Q, Tian F, Zhao J, Zhang H, Narbad A, Chen W. Oral administration of probiotics inhibits absorption of the heavy metal cadmium by protecting the intestinal barrier. *Applied and environmental microbiology*. 2016;82(14):4429–40. <https://doi.org/10.1128/AEM.00695-16>
83. Mack DR. Safety Issues of Probiotic Ingestion. *Probiotics in Pediatric Medicine: Springer*; 2009. p. 69–80.
84. Afshar P, Shokrzadeh M, Raeisi SN, Ghorbani-HasanSaraei A, Nasiraii LR. Aflatoxins biotransformation strategies based on probiotic bacteria. *Toxicon*. 2020;178:50–8. <https://doi.org/10.1016/j.toxicon.2020.02.007>
85. Tajik H, Sayadi M. Effects of probiotic bacteria *Lactobacillus acidophilus* and *Lactobacillus casei* on aflatoxin B1 detoxification within a simulated gastrointestinal tract model. *Toxin Reviews*. 2022;41(1):92–9. <https://doi.org/10.6084/m9.figshare.13213909>
86. Arab M, Sohrabvandi S, Mortazavian A, Mohammadi R, Tavirani MR. Reduction of aflatoxin in fermented milks during production and storage. *Toxin Reviews*. 2012;31(3-4):44–53. <https://doi.org/10.3109/15569543.2012.738350>
87. Sarlak Z, Rouhi M, Mohammadi R, Khaksar R, Mortazavian AM, Sohrabvandi S, et al. Probiotic biological strategies to decontaminate aflatoxin M1 in a traditional Iranian fermented milk drink (Doogh). *Food control*. 2017;71:152–9. <http://dx.doi.org/10.1016/j.foodcont.2016.06.037>
88. Bansal M, Yang J, Karan C, Menden MP, Costello JC, Tang H, et al. A community computational challenge to predict the activity of pairs of



- compounds. *Nature biotechnology*. 2014;32(12): 1213–22. <https://doi.org/10.1038/nbt.3052>
89. Styriak I, Conková E. Microbial binding and biodegradation of mycotoxins. *Veterinary and human toxicology*. 2002;44(6):358–61.
90. Smith T, Girish C. Prevention and control of animal feed contamination by mycotoxins and reduction of their adverse effects in livestock. *Animal Feed Contamination: Elsevier*; 2012. p. 326–51.
91. Pino A, Bartolo E, Caggia C, Cianci A, Randazzo CL. Detection of vaginal lactobacilli as probiotic candidates. *Scientific reports*. 2019;9(1):3355. <https://doi.org/10.1038/s41598-019-40304-3>
92. Karimi R, Hosseinzadeh D. Probiotics and Gastro-Intestinal disorders: Augmentation, Enhancement, and strengthening of epithelial lining. *Probiotics: CRC Press*; 2024. p. 188–215. <https://doi.org/10.1201/9781003452249-9>
93. Krasowska A, Murzyn A, Dyjankiewicz A, Łukaszewicz M, Dziadkowiec D. The antagonistic effect of *Saccharomyces boulardii* on *Candida albicans* filamentation, adhesion and biofilm formation. *FEMS yeast research*. 2009;9 (8): 1312–21. <https://doi.org/10.1111/j.15671364.2009.00559.x>
94. Czerucka D, Dahan S, Mograbi B, Rossi B, Rampal P. *Saccharomyces boulardii* preserves the barrier function and modulates the signal transduction pathway induced in enteropathogenic *Escherichia coli*-infected T84 cells. *Infection and immunity*. 2000;68(10):5998–6004. <https://doi.org/10.1128/iai.68.10.5998-6004.2000>
95. Martins FS, Dalmaso G, Arantes RM, Doye A, Lemichez E, Lagadec P, et al. Interaction of *Saccharomyces boulardii* with *Salmonella enterica* serovar Typhimurium protects mice and modifies T84 cell response to the infection. *PLoS One*. 2010; 5 (1):e8925. <https://doi.org/10.1371/journal.pone.0008925>
96. Pontier-Bres R, Munro P, Boyer L, Anty R, Imbert V, Terciolo C, et al. *Saccharomyces boulardii* modifies *Salmonella typhimurium* traffic and host immune responses along the intestinal tract. *PLoS One*. 2014;9(8):e103069. <https://doi.org/10.1371/journal.pone.0103069>
97. Collado MC, Isolauri E, Salminen S, Sanz Y. The impact of probiotic on gut health. *Current drug metabolism*. 2009;10 (1): 68–78. <https://doi.org/10.2174/138920009787048437>
98. Muth TR, Caplan AJ. Microbiomes for all. *Frontiers in Microbiology*. 2020;11:593472. <https://doi.org/10.3389/fmicb.2020.593472>
99. Xu X, He S, Zhang X. New food safety concerns associated with gut microbiota. *Trends in food science & technology*. 2013;34(1):62–6. <https://doi.org/10.1080/19490976.2024.2410476>
100. Chen L. Gut microbiota manipulation to mitigate the detrimental effects of environmental pollutants. *MDPI*; 2021. p. 127. <https://doi.org/10.3390/toxics9060127>
101. Joint F. Probiotics in food. Health and nutritional properties and guidelines for evaluation. 2006.
102. Leonard P. The role of biological research in supporting policy needs. *Marine environmental research*. 2002;54(3-5):209–13. <https://doi.org/10.13140/RG.2.1.4320.6165>
103. Sanders ME, Klaenhammer TR, Ouwehand AC, Pot B, Johansen E, Heimbach JT, et al. Effects of genetic, processing, or product formulation changes on efficacy and safety of probiotics. *Annals of the New York Academy of Sciences*. 2014; 1309(1):1–18. <https://doi.org/10.1111/nyas.12363>
104. Tajabadi Ebrahimi M, Jafarvand E. Probiotics industry: review of current challenges and the future roadmap. *Payesh (Health Monitor)*. 2021;20(3):263–73. <http://dx.doi.org/10.52547/payesh.20.3.263>
105. Jankovic I, Sybesma W, Phothirath P, Ananta E, Mercenier A. Application of probiotics in food



- products—challenges and new approaches. *Current Opinion in Biotechnology*. 2010;21 (2):175–81.  
<https://doi.org/10.1016/j.copbio.2010.03.009>
106. Mafe AN, Büsselberg D. Microbiome–mycotoxin interactions and probiotic strategies: implications for gut health and cancer. *Frontiers in Nutrition*. 2026;13:1783295.  
<https://doi.org/10.3389/fnut.2026.1783295>
107. Razavi S, Janfaza S, Tasnim N, Gibson DL, Hoorfar M. Nanomaterial-based encapsulation for controlled gastrointestinal delivery of viable probiotic bacteria. *Nanoscale advances*. 2021;3 (10):2699–709.  
<https://doi.org/10.1039/dona00952k>
108. Silva PBVd, Rosales TKO, Fabi JP. Nanoencapsulation of biotics: Feasibility to enhance stability and delivery for improved gut health. *Pharmaceutics*. 2025;17(9):1180.  
<https://doi.org/10.3390/pharmaceutics17091180>
109. Ohtake H, Silver S, Rasul Chaudhry G. Biological degradation and bioremediation of toxic chemicals. *Chapman & Hall, Portland*; 1994
110. Nybom SM, Salminen SJ, Meriluoto JA. Specific strains of probiotic bacteria are efficient in the removal of several different cyanobacterial toxins from solution. *Toxicon*. 2008;52(2):214–20.  
<https://doi.org/10.1016/j.toxicon.2008.04.169>
111. Tejero-Sariñena S, Barlow J, Costabile A, Gibson GR, Rowland I. Antipathogenic activity of probiotics against *Salmonella Typhimurium* and *Clostridium difficile* in anaerobic batch culture systems: is it due to synergies in probiotic mixtures or the specificity of single strains? *Anaerobe*. 2013;24:60–5.  
<https://doi.org/10.1016/j.anaerobe.2013.09.011>
112. Relman DA, Lipsitch M. Microbiome as a tool and a target in the effort to address antimicrobial resistance. *Proceedings of the National Academy of Sciences*. 2018;115(51):12902–10.  
<https://doi.org/10.1073/pnas.1717163115>
113. Gallo A, Masoero F. In vitro models to evaluate the capacity of different sequestering agents to adsorb aflatoxins. *Italian Journal of animal science*. 2010; 9 (1):e21. <https://doi.org/10.4081/ijas.2010.e21>
114. Muhialdin BJ, Saari N, Meor Hussin AS. Review on the biological detoxification of mycotoxins using lactic acid bacteria to enhance the sustainability of foods supply. *Molecules*. 2020;25(11):2655.  
<https://doi.org/10.3390/molecules25112655>
115. Bisanz, J.E.; Enos, M.K.; Mwanga, J.R.; Changalucha, J.; Burton, J.P.; Gloor, G.B.; Reid, G. Randomized open-label pilot study of the influence of probiotics and the gut microbiome on toxic metal levels in Tanzanian pregnant women and school children. *mBio* 2014, 5, e01580-14  
<https://doi.org/10.1128/mbio.01580-14>
116. Marco ML, Tachon S. Environmental factors influencing the efficacy of probiotic bacteria. *Current opinion in biotechnology*. 2013;24 (2): 207–13. <https://doi.org/10.1016/j.copbio.2012.10.002>
117. Palsson B, Zengler K. The challenges of integrating multi-omic data sets. *Nature chemical biology*. 2010;6(11):787–9.  
<https://doi.org/10.1038/nchembio.462>
118. Bisanz JE, Enos MK, Mwanga JR, Changalucha J, Burton JP, Gloor GB, Reid G. Randomized open-label pilot study of the influence of probiotics and the gut microbiome on toxic metal levels in Tanzanian pregnant women and school children. *mBio*. 2014 Oct 7;5(5):e01580-14.  
<https://doi.org/10.1128/mBio.01580-14>
119. Feng, P., Yang, J., Zhao, S., Ling, Z., Han, R., Wu, Y., Salama, E.S., Kakade, A., Khan, A., Jin, W. and Zhang, W., 2022. Human supplementation with *Pediococcus acidilactici* GR-1 decreases heavy metals levels through modifying the gut microbiota and metabolome. *npj Biofilms and Microbiomes*, 8(1), p.63.  
<https://doi.org/10.1038/s41522-022-00326-8>



120. LaTouche, L., 2023. Clinical Utility of Probiotics Therapy in Managing Mycotoxin Illness. *Integrative Medicine: A Clinician's Journal*, 22 (4), p.12.
121. Darbandi, A., Navidifar, T., Koupaei, M., Afifirad, R., Nezhad, R.A., Emamie, A., Talebi, M. and Kakanj, M., 2025. The effect of the combination of probiotics and heavy metals from various aspects in humans: a systematic review of clinical trial studies. *Health Science Reports*, 8(3), p.e70521. <https://doi.org/10.1002/hsr2.70521>
122. Abbasi, A., Sheykhsaran, E., Hosseinzadeh, N., Bazdar, M., Hamehjani, M., Aghapour, B. and Shojaee-Aliabadi, S., 2026. Novel approaches in establishing chemical food safety based on the detoxification capacity of probiotics and postbiotics: a critical review. *Probiotics and Antimicrobial Proteins*, 18(1), pp.1641-1681.
123. D'Anna, R., Nutritional supplementation for recurrent urinary tract infections in women. [www.clinicaltrials.gov/show/NCT03597152](http://www.clinicaltrials.gov/show/NCT03597152).
124. Sibanda T, Marole TA, Thomashoff UL, Thantsha MS, Buys EM. Bifidobacterium species viability in dairy-based probiotic foods: challenges and innovative approaches for accurate viability determination and monitoring of probiotic functionality. *Frontiers in Microbiology*. 2024;15:1327010. <https://doi.org/10.3389/fmicb.2024.1327010>
125. Janardana Reddy S. Probiotics in aquaculture: importance, influence and future perspectives. *Int J Bioassays*. 2015;4:3710–8.
126. Organization FaAOaWH. Guidelines for the Evaluation of Probiotics in Food. London, Ontario, Canada: *Joint FAO/WHO Working Group*; 2002.
127. Donohue DC. Safety of probiotics. *Asia Pacific Journal of Clinical Nutrition*. 2006;15(4):563–9. <https://doi.org/10.1186/2045-7022-1-S1-S9>
128. Bamunuarachchige T, Wickramasinghe H, Dissanayaka D, Wickramaratna N. Genetic engineering of probiotic microorganisms. *Probiotics: Biology, Genetics and Health Aspects: Springer*; 2011. p. 109–38. [https://doi.org/10.1007/978-3-642-20838-6\\_5](https://doi.org/10.1007/978-3-642-20838-6_5)
129. Vanderhoof JA. Probiotics: future directions. *The American journal of clinical nutrition*. 2001;73(6):1152S–5S. <https://doi.org/10.1093/ajcn/73.6.1152s>
130. O'Toole PW, Marchesi JR, Hill C. Next-generation probiotics: the spectrum from probiotics to live biotherapeutics. *Nature microbiology*. 2017;2 (5): 17057. <https://doi.org/10.1038/nmicrobiol.2017.57>
131. Steger-Hartmann T, Raschke M. Translating in vitro to in vivo and animal to human. *Current Opinion in Toxicology*. 2020;23:6–10. <https://doi.org/10.1016/j.cotox.2020.02.003>
132. Luo Y, Liu X, Yuan L, Li J. Complicated interactions between bio-adsorbents and mycotoxins during mycotoxin adsorption: Current research and future prospects. *Trends in Food Science & Technology*. 2020;96:127–34. <https://doi.org/10.1016/J.TIFS.2019.12.012>
133. Green BN, Johnson CD, Adams A. Writing narrative literature reviews for peer-reviewed journals: secrets of the trade. *Journal of chiropractic medicine*. 2006;5(3):101–17. [https://doi.org/10.1016/s0899-3467\(07\)60142-6](https://doi.org/10.1016/s0899-3467(07)60142-6)
134. Ferrari R. Writing narrative style literature reviews. *Medical writing*. 2015;24(4):230–5. <https://doi.org/10.1179/2047480615Z.0000000000329>
135. Snyder H. Literature review as a research methodology: An overview and guidelines. *Journal of business research*. 2019;104:333–9. <https://doi.org/10.1016/j.jbusres.2019.07.039>
136. Wang Y, Han J, Ren Q, Liu Z, Zhang X, Wu Z. The involvement of lactic acid bacteria and their exopolysaccharides in the biosorption and detoxication of heavy metals in the gut. *Biological*



- Trace Element Research*. 2024;202(2):671–84.  
<https://doi.org/10.1007/s12011-023-03693-1>
137. Landersjö C, Yang Z, Huttunen E, Widmalm G. Structural Studies of the Exopolysaccharide Produced by *Lactobacillus rhamnosus* strain GG (ATCC 53103). *Biomacromolecules*. 2002;3(4):880–4. <https://doi.org/10.1021/bm020040q>
138. Chen, W.; Zhai, Q. Applications of Lactic Acid Bacteria in Heavy Metal Pollution Environment. In *Lactic Acid Bacteria in Foodborne Hazards Reduction*, 1st ed.; Chen, W., Narbad, A., Eds.; Springer: Singapore, 2018; pp. 213–248.
139. Kinoshita, H.; Sohma, Y.; Ohtake, F.; Ishida, M.; Kawai, Y.; Kitazawa, H.; Saito, T.; Kimura, K. Biosorption of heavy metals by lactic acid bacteria and identification of mercury binding protein. *Res. Microbiol.* 2013, 164, 701–709.  
<https://doi.org/10.1016/j.resmic.2013.04.004>
140. Hamad GM, Taha TH, Hafez EE, Ali SH, El Sohaimy SA. Supplementation of Cerelac baby food with yeast–probiotic cocktail strains induces high potential for aflatoxin detoxification both in vitro and in vivo in mother and baby albino rats. *Journal of the Science of Food and Agriculture*. 2018;98(2):707–18.  
<https://doi.org/10.1002/jsfa.8518>
141. Liu F, Malaphan W, Xing F, Yu B. Bioremediation of fungal mycotoxins zearalenone by engineered probiotic bacterium *Lactobacillus reuteri* with surface-displayed lactonohydrolase. *Applied Microbiology and Biotechnology*. 2019;103(21):8813–24.  
<https://doi.org/10.1007/s00253-019-10153-1>
142. Li B, Jin D, Yu S, Ertareri Evvie S, Muhammad Z, Huo G, et al. In vitro and in vivo evaluation of *Lactobacillus delbrueckii* subsp. *bulgaricus* KLDS1.0207 for the alleviative effect on lead toxicity. *Nutrients*. 2017;9(8):845.  
<https://doi.org/10.3390/nu9080845>
143. Rezasoltani S, Ebrahimi NA, Boroujeni RK, Aghdaei HA, Norouzinia M. Detoxification of aflatoxin M1 by probiotics *Saccharomyces boulardii*, *Lactobacillus casei*, and *Lactobacillus acidophilus* in reconstituted milk. *Gastroenterology and Hepatology from Bed to Bench*. 2022;15(3):263.  
<https://doi.org/10.22037/ghfbb.v15i3.2402>
144. Poloni VL, Bainotti MB, Vergara LD, Escobar F, Montenegro M, Cavaglieri L. Influence of technological procedures on viability, probiotic and anti-mycotoxin properties of *Saccharomyces boulardii* RCo09, and biological safety studies. *Current research in food science*. 2021;4:132–40.  
<https://doi.org/10.1016/j.crfs.2021.02.006>
145. Alassane-Kpembé I, Canlet C, Tremblay-Franco M, Jourdan F, Chalazaviel M, Pinton P, et al. 1H-NMR metabolomics response to a realistic diet contamination with the mycotoxin deoxynivalenol: Effect of probiotics supplementation. *Food and Chemical Toxicology*. 2020;138:111222.  
<https://doi.org/10.1016/j.fct.2020.111222>
146. Baralić K, Pavić A, Javorac D, Živančević K, Božić D, Radaković N, et al. Comprehensive investigation of hepatotoxicity of the mixture containing phthalates and bisphenol A. *Journal of hazardous materials*. 2023;445:130404.  
<https://doi.org/10.1016/j.jhazmat.2022.130404>
147. Gratz S, Mykkänen H, El-Nezami H. Aflatoxin B1 binding by a mixture of *Lactobacillus* and *Propionibacterium*: in vitro versus ex vivo. *Journal of food protection*. 2005;68(11):2470–4.  
<https://doi.org/10.4315/0362-028x-68.11.2470>
148. Pais P, Almeida V, Yilmaz M, Teixeira MC. *Saccharomyces boulardii*: what makes it tick as successful probiotic? *Journal of Fungi*. 2020;6(2):78. <https://doi.org/10.3390/jof6020078>
149. Yilmaz M. Predicting the Mechanisms of Probiotic Activity in *Saccharomyces Boulardii*: A Contribution to the Development of the



- ProBioYeast Database: Universidade de Lisboa Lisbon, Portugal; 2019.  
<https://doi.org/10.1007/s12602-025-10634-y>
150. Tomićić ZM, Čolović RR, Čabarkapa IS, Vukmirović ĐM, Đuragić OM, Tomićić RM. Beneficial properties of probiotic yeast *Saccharomyces boulardii*. *Food and Feed Research*. 2016;43(2):103–10—10.  
<https://doi.org/10.5937/FFR1602103T>
  151. Salminen S, Collado MC, Endo A, Hill C, Lebeer S, Quigley EM, et al. The International Scientific Association of Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of postbiotics. *Nature reviews Gastroenterology & hepatology*. 2021;18(9):649–67.  
<https://doi.org/10.1038/s41575-021-00440-6>
  152. Isaac-Bamgboye FJ, Mgbachidinma CL, Onyeaka H, Isaac-Bamgboye IT, Chukwugozie DC. Exploring the potential of postbiotics for food safety and human health improvement. *Journal of nutrition and metabolism*. 2024;2024(1):1868161.  
<https://doi.org/10.1155/2024/1868161>
  153. Lin C-W, Chen Y-T, Ho H-H, Kuo Y-W, Lin W-Y, Chen J-F, et al. Impact of the food grade heat-killed probiotic and postbiotic oral lozenges in oral hygiene. *Aging (Albany NY)*. 2022;14(5):2221.  
<https://doi.org/10.18632/aging.203923>
  154. Zeise L, Bois FY, Chiu WA, Hattis D, Rusyn I, Guyton KZ. Addressing human variability in next-generation human health risk assessments of environmental chemicals. *Environmental health perspectives*. 2012;121(1):23.  
<https://doi.org/10.1289/ehp.1205687>
  155. Ruas-Madiedo P, Gueimonde M, Margolles A, De Los Reyes-Gavilán CG, Salminen S. Exopolysaccharides produced by probiotic strains modify the adhesion of probiotics and enteropathogens to human intestinal mucus. *Journal of food protection*. 2006;69(8):2011–5.  
<https://doi.org/10.4315/0362-028x-69.8.2011>
  156. Lücke F-K. Lactic acid bacteria involved in food fermentations and their present and future uses in food industry. *Lactic Acid Bacteria: Current Advances in Metabolism, Genetics and Applications: Springer*; 1996. p. 81–99 .  
<https://doi.org/10.1023/A:1002066306013>
  157. Ameen FA, Hamdan AM, El-Naggar MY. Assessment of the heavy metal bioremediation efficiency of the novel marine lactic acid bacterium, *Lactobacillus plantarum* MFO42018. *Scientific reports*. 2020;10(1):314.  
<https://doi.org/10.1038/s41598-019-57210-3>
  158. Clarke G, Sandhu KV, Griffin BT, Dinan TG, Cryan JF, Hyland NP. Gut reactions: breaking down xenobiotic–microbiome interactions. *Pharmacological reviews*. 2019;71(2):198–224.  
<https://doi.org/10.1124/pr.118.015768>
  159. Tian, F.; Xiao, Y.; Li, X.; Zhai, Q.; Wang, G.; Zhang, Q.; Zhang, H.; Chen, W. Protective Effects of *Lactobacillus plantarum* CCFM8246 against Copper Toxicity in Mice. *PLoS ONE* 2015, 10, e0143318.  
<https://doi.org/10.1371/journal.pone.0143318>
  160. Pakdel, M.; Soleimani-Zad, S.; Akbari-Alavijeh, S. Screening of lactic acid bacteria to detect potent biosorbents of lead and cadmium. *Food Control* 2019, 100, 144–150.  
<https://doi.org/10.1016/j.foodcont.2018.12.044>
  161. Kinoshita, H.; Ohtake, F.; Ariga, Y.; Kimura, K. Comparison and characterization of biosorption by *Weissella viridescens* MYU 205 of periodic group 12 metal ions. *Anim. Sci. J.* 2016, 87, 271–276.  
<https://doi.org/10.1111/asj.12425>
  162. Mikó Z, Ujszegi J, Gál Z, Imrei Z, Hettyey A. Choice of experimental venue matters in ecotoxicology studies: comparison of a laboratory-based and an outdoor mesocosm experiment. *Aquatic Toxicology*. 2015;167:20–30.  
<https://doi.org/10.1016/j.aquatox.2015.07.014>



163. Zhu Y, Hassan YI, Lepp D, Shao S, Zhou T. Strategies and methodologies for developing microbial detoxification systems to mitigate mycotoxins. *Toxins*. 2017;9(4):130. <https://doi.org/10.3390/toxins9040130>
164. Kim N-N, Kim WJ, Kang S-S. Anti-biofilm effect of crude bacteriocin derived from *Lactobacillus brevis* DFO1 on *Escherichia coli* and *Salmonella Typhimurium*. *Food control*. 2019;98:274–80. <https://doi.org/10.1016/j.foodcont.2018.11.004>
165. O. Kolawole, J. Meneely, B. O. Greer, O. Chevallier, D. S. Jones, L. Connolly and C. Elliott, Comparative in vitro assessment of a range of commercial feed additives with multiple mycotoxin binding claims, *Toxins*, 2019, 11(11), 659, <https://doi.org/10.3390/toxins11110659>.
166. P. Trinh, J. R. Zaneveld, S. Safranek and P. M. Rabinowitz, One health relationships between human, animal, and environmental microbiomes: A mini-review, *Public Health Front.*, 2018, 6, 235, <https://doi.org/10.3389/fpubh.2018.00235>.
167. C. A. Thaïss, D. Zeevi, M. Levy, E. Segal and E. Elinav, A day in the life of the meta-organism: diurnal rhythms of the intestinal microbiome and its host, *Gut Microbes*, 2015, 6(2), 137–142, <https://doi.org/10.1080/19490976.2015.1016690>.
168. S. Subramanian, H. Thiruvengadamani and M. Sathivelu, Comparison of Human gut Microbiota with other Animals, *Res. J. Pharm. Technol.*, 2022, 5541–5547, <https://doi.org/10.52711/0974-360x.2022.00935>
169. J. S. Ahn, E. Lkhagva, S. Jung, H. J. Kim, H. J. Chung and S. T. Hong, Fecal microbiome does not represent whole gut microbiome, *Cell. Microbiol.*, 2023, 1–14, <https://doi.org/10.1155/2023/686841>.
170. F. F. Campos, P. A. Sales Junior, A. J. Romanha, M.S.S.Araújo, E.P.Siqueira, J.M.Resende, T.M.A.Alves, O. A. Martins-Filho, V. L. Santos, C. A. dos Rosa, C. L. Zani and B. B. Cota, Bioactive endophytic fungi isolated from *Caesalpinia echinata* Lam. (Brazilwood) and identification of beauvericin as a trypanocidal metabolite from *Fusarium* sp., *Mem. Inst. Oswaldo Cruz*, 2015, 110(1), 65–74, <https://doi.org/10.1590/0074-02760140243>
171. J. Wiese, J. F. Imhoff, T. A. M. Gulder, A. Labes and R. Schmaljohann, Marine fungi as producers of benzocoumarins, a new class of inhibitors of glycogen-synthase kinase 3 $\beta$ , *Mar. Drugs*, 2016, 14(11), 200, <https://doi.org/10.3390/md14110200>.
172. F. Crudo, G. Aichinger, J. Mihajlovic, E. Varga, L. Dellafiora, B. Warth, C. Dall'Asta, D. Berry and D. Marko, In vitro interactions of *Alternaria*, mycotoxins, an emerging class of food contaminants, with the gut microbiota: a bidirectional relationship, *Arch. Toxicol.*, 2021, 95(7), 2533–2549, <https://doi.org/10.1007/s00204-021-03043-x>.

