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Nutritional Modulation of Epigenetic Changes Induced by Mycotoxins: A Biochemical Perspective for At-Risk Populations in Africa

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ABSTRACT

Mycotoxins, toxic secondary metabolites produced by fungi, are widespread contaminants of staple foods in Sub-Saharan Africa, contributing to acute toxicity and chronic diseases such as cancer, stunting, and immune dysfunction. Recent evidence has illuminated their capacity to induce epigenetic changes, including alterations in DNA methylation, histone modifications, and noncoding RNA expression, without modifying the DNA sequence. These disruptions, particularly when combined with nutritional deficiencies, can have long-lasting effects on gene regulation and disease susceptibility. This review synthesizes evidence from peer-reviewed studies published between 2007 and 2025, exploring how key nutrients modulate the epigenetic toxicity of common mycotoxins like aflatoxin B1, ochratoxin A, and fumonisin B1. A systematic literature search identified studies evaluating the biochemical and epigenetic responses to mycotoxin exposure and the mitigating roles of dietary factors such as folate, vitamin B12, choline, polyphenols, selenium, and short-chain fatty acids. These nutrients support critical pathways, including one-carbon metabolism, antioxidant defense, and gut microbiota modulation, which intersect with epigenetic regulation. Findings highlight that methyl donors can restore DNA methylation balance, antioxidants can prevent oxidative stress-induced histone and DNA damage, and fiber-rich diets promote microbial metabolites that act as epigenetic modulators. Additionally, exposure to multiple mycotoxins exerts synergistic effects that amplify epigenetic damage, especially in nutritionally vulnerable populations. This review also discusses emerging intersections between mycotoxins and biochemical domains such as neuroepigenetics, mitochondrial signaling, and microbiome-host interactions. Nutritional interventions ranging from diet diversification to bioactive-rich functional foods and probiotic supplementation emerge as promising, culturally adaptable strategies for mitigating these epigenetic threats. An integrative framework is proposed, linking food safety, public health policy, and personalized nutrition to strengthen resilience in affected communities. In conclusion, nutrition-sensitive interventions targeting epigenetic mechanisms offer a feasible and impactful approach to reducing the long-term health burden of mycotoxins in Africa. Future research should focus on biomarker discovery, field-based dietary trials, and AI-driven predictive models to inform context-specific mitigation strategies.

KEYWORDS

Mycotoxins, epigenetics, nutritional modulation, DNA methylation, histone modifications, noncoding RNAs, oxidative stress, gut microbiota, Sub-Saharan Africa

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INTRODUCTION

Mycotoxins are toxic secondary metabolites produced by fungi that contaminate a wide range of agricultural commodities, including staple foods such as maize, groundnuts, and cassava, which are dietary mainstays for many populations in Africa[1]. The widespread presence of mycotoxins poses a significant threat to human and animal health, leading to various acute and chronic diseases[2]. Beyond their direct toxic effects, accumulating evidence suggests that mycotoxins can induce epigenetic modifications, altering gene expression without changing the underlying DNA sequence[3]. These epigenetic changes, including DNA methylation, histone modification, and non-coding RNA regulation, can have long-lasting impacts on cellular function and disease susceptibility, potentially contributing to developmental abnormalities, immune dysfunction, and increased cancer risk[4,5].

Populations in Africa are particularly vulnerable to mycotoxin exposure due to several factors. These include suboptimal pre- and post-harvest storage practices that favor fungal growth and mycotoxin production, limited access to effective detoxification technologies, and often monotonous diets heavily reliant on susceptible crops[6]. Furthermore, co-exposure to multiple mycotoxins is common, potentially leading to synergistic or additive adverse health effects[7]. The intersection of high mycotoxin exposure and pre-existing nutritional deficiencies in many African communities creates a complex scenario where the impact of mycotoxin-induced epigenetic changes may be exacerbated[8].

Nutrition plays a crucial role in maintaining cellular homeostasis and influencing epigenetic processes[9]. Dietary components, such as methyl donors (e.g., folate, vitamin B12, choline), antioxidants (e.g., vitamins A, C, E, selenium), and other bioactive compounds (e.g., polyphenols), can modulate the activity of enzymes involved in epigenetic regulation[10]. Therefore, understanding how specific nutritional factors can counteract or mitigate the epigenetic alterations induced by mycotoxins is of paramount importance for developing effective intervention strategies in at-risk African populations. This review aims to provide a biochemical perspective on the nutritional modulation of mycotoxin-induced epigenetic changes, highlighting potential dietary interventions that could offer protection against the long-term health consequences of mycotoxin exposure in Africa.

Figure 1 contrasts contaminated food with a nutrient-rich alternative. On the left, moldy corn kernels illustrate visible fungal spoilage and mycotoxin risk. On the right, a colorful plant-based meal features vegetables, legumes, grains, and fruits. The comparison emphasizes the importance of food safety and dietary quality in health protection.

SYSTEMATIC REVIEW APPROACH

This section outlines the systematic approach used to review the interplay between mycotoxins, epigenetics, and nutrition in African populations. Relevant literature was identified using electronic databases and specific keywords. Studies were selected based on their focus on epigenetic effects and nutritional interventions. Data extraction summarized key mycotoxins, epigenetic markers, study designs, and findings. A narrative synthesis grouped studies by mycotoxin type and dietary influence. Ethical considerations and research limitations were also addressed.

This review employed a systematic approach to identify and synthesize relevant literature on the interplay between mycotoxins, epigenetics, and nutrition, with a specific focus on the African context. Electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar, were searched using a combination of keywords such as "Mycotoxins", "Aflatoxin", "Ochratoxin", "Fumonisin", "Zearalenone", "Epigenetics", "DNA methylation", "Histone modification", "Non-coding RNA", "Nutrition", "Diet", "Vitamins", "Minerals", "Antioxidants", and "Africa". The search was limited to peer-reviewed articles and relevant reports published between 2007 and 2025 to ensure the inclusion of the most recent findings[11].

Fig. 1: Comparison of moldy corn kernels and a healthy plant-based meal (self-generated)
This image contrasts food contamination and nutritional intervention. On the left side, corn kernels are visibly covered in mold, indicating spoilage and potential health risks due to mycotoxin contamination. On the right side, a vibrant, nutrient-rich meal is presented, consisting of fresh vegetables, grains, legumes, and fruits such as spinach, broccoli, chickpeas, brown rice, avocado, tomato slices, cucumber slices, and a bowl of mixed berries (strawberries, blueberries, and raspberries). This contrast highlights the importance of food safety and balanced nutrition in preventing health hazards

Table 1: Search strategy and databases used

Database	Keywords used	Years covered	Filter sapplied
PubMed	Mycotoxins, aflatoxin, ochratoxin, epigenetics, DNA methylation, nutrition, Africa	2007-2025	Peer-reviewed, full-text, English language[11-17]
Scopus	Mycotoxins, DNA methylation, histone modification, polyphenols, antioxidants	2007-2025	Original research, African studies, nutritional focus[11-17]
Web of Science	Fumonisin, zearalenone, non-coding RNA, vitamins, minerals, epigenetic markers	2007-2025	Relevance to African populations, nutritional interventions[11-19]
Google Scholar	Mycotoxins, nutritional modulation, food safety, epigenetics, oxidative stress	2007-2025	Recent studies, high citation relevance, human/animal studies[11-19]

The electronic databases (PubMed, Scopus, Web of Science, and Google Scholar) used to identify relevant studies. It includes search keywords related to mycotoxins, nutrition, and epigenetics, along with applied filters such as peer-reviewed articles and African population relevance

Table 1 outlines the systematic approach utilized for literature search, specifying the databases (PubMed, Scopus, Web of Science, Google Scholar), relevant keywords, and filters applied to ensure the inclusion of high-quality, peer-reviewed studies focused on mycotoxins, nutrition, and epigenetics.

Studies were included if they investigated the epigenetic effects of mycotoxins in *in vitro*, *in vivo*, or human population studies, particularly those conducted in or relevant to African populations. Additionally, articles exploring the potential of nutritional interventions to modulate these epigenetic changes were considered. Exclusion criteria included studies focusing solely on the acute toxicological effects of mycotoxins without examining epigenetic mechanisms, studies unrelated to nutrition or epigenetics, and reviews or meta-analyses (although their reference lists were screened for potentially relevant primary research)[12-19].

Table 2 provides an overview of studies examining the effects of mycotoxins on epigenetic mechanisms, including DNA methylation, histone modifications, and non-coding RNA alterations. It categorizes research based on mycotoxin type, study design, and the impact of nutritional interventions on mitigating epigenetic changes.

Table 2: Inclusion and exclusion criteria for literature review

Criteria	Description	Inclusion	Exclusion	Citations
Study type	Type of research evaluated for mycotoxin epigenetic impacts	Original research articles (*in vitro*, *in vivo*, human studies)	Review articles, editorials, or opinion pieces	Watson *et al.*[13] Agriopulou *et al.*[14] Ntsam *et al.*[15] Hattle *et al.*[16] Ezekiel *et al.*[17] Thomposn[18] Ahmad and Yu[19] Liu *et al.*[20]
Focus area	Central subject involving epigenetic mechanisms and nutritional modulation	Studies addressing mycotoxins, epigenetics, and nutrition	Studies focusing solely on toxicity or lacking nutritional/epigenetic relevance	
Geographic relevance	Applicability to African populations and agricultural contexts	Research conducted in or relevant to African dietary and environmental conditions	Studies from unrelated geographical or socio-economic settings	
Publication period	Ensures review includes current trends and up-to-date findings	Studies published from 2007 to 2025	Articles published before 2020 or with an unverifiable date	
Language	Comprehension and extraction of methodological detail	English language studies	Non-English papers without translation	
Data quality	Assessed via transparency, statistical rigor, and proper control	Peer-reviewed with robust methods and appropriate controls	Poorly designed studies lacking reproducibility or statistical support	
Intervention scope	Specific nutritional interventions aimed at countering epigenetic effects	Nutritional studies showing effects on DNA methylation, histone modifications, or ncRNA linked to mycotoxin exposure	Studies lacking epigenetic endpoints or focused solely on acute toxicity	
Accessibility	Availability of full-text data for extraction and evaluation	Freely available or institutionally accessible full texts	Abstract-only or inaccessible studies	
Ethical compliance	Consideration for ethical approval in human/animal studies	Studies indicating IRB or ethical review approval where applicable	Research without ethical documentation or approval details	

That examine the epigenetic effects of mycotoxins, including DNA methylation, histone modifications, and non-coding RNA expression. It highlights experimental models, nutritional interventions, and research conducted in African populations, providing insight into the interplay between toxic exposure and dietary modulation

Data extraction involved summarizing the type of mycotoxin studied, the epigenetic markers investigated, the nutritional interventions explored, the study design, and the key findings. Special attention was paid to studies that directly examined the impact of nutrition on mycotoxin-induced epigenetic alterations. The quality of the included studies was assessed based on established criteria relevant to their design (e.g., appropriate controls, statistical power, clear reporting of methods and results)[14-20]. A narrative synthesis of the findings was conducted, grouping studies based on the specific mycotoxin and nutritional factor investigated[15-20]. Challenges and limitations in the existing literature, as well as potential areas for future research, were also identified[16-20]. The Vancouver citation style was used for referencing[17-20]. Ethical considerations related to research involving human participants and animal models were noted where applicable[18-20]. Efforts were made to identify studies that addressed the specific dietary patterns and nutritional deficiencies prevalent in at-risk populations in Africa[19,20].

NUTRITIONAL MODULATION OF MYCOTOXIN-INDUCED EPIGENETIC CHANGES: MECHANISMS, EVIDENCE, AND IMPLICATIONS FOR AFRICA

This study explores how mycotoxins alter gene regulation through epigenetic modifications. It details how mycotoxins disrupt DNA methylation, histone modifications, and non-coding RNA expression, while highlighting the critical role of nutritional factors like methyl donors and antioxidants in counteracting these effects. The section also discusses the influence of the gut microbiota and the complexities of synergistic effects from multiple mycotoxins. Ultimately, it emphasizes the significant implications for African populations, advocating for nutritional interventions to mitigate mycotoxin-induced health risks.

Table 3: Impact of methyl donors on mycotoxin-induced DNA methylation

Nutrient	Mechanism	Study model	Outcome	Citations
Folate	Provides methyl groups via the one-carbon metabolism pathway	Animal and cell models	Modulates DNA methylation in response to mycotoxin exposure	Serefidou *et al.*[25] Gachara *et al.*[26] Amenyah *et al.*[27]
Vitamin B12	Cofactor in methionine synthase for methyl group recycling	*In vivo* models	Protects against global DNA hypomethylation caused by mycotoxins	Kok *et al.*[28] Perlmutter *et al.*[29] Milagro *et al.*[30]
Choline	Donates methyl groups and supports SAM production	*In vitro* hepatocyte studies	Reduces mycotoxin-induced epigenetic gene silencing	
Betaine	Supports remethylation of homocysteine to methionine	Rodent models	Restores altered methylation patterns in liver cells	
Polyphenols	Regulate DNA methyltransferase activity	Various *in vitro* cancer and liver cell models	Modulate methylation of tumor suppressor genes affected by AFB1	

This table summarizes experimental findings on key nutritional methyl donors, folate, Vitamin B12, choline, betaine, and polyphenols and their mechanisms in counteracting DNA methylation disruptions caused by mycotoxin exposure across various biological models

DNA methylation and nutritional modulators: The DNA methylation, the addition of a methyl group to cytosine residues in DNA, is a key epigenetic mark involved in gene silencing and genomic stability[21]. Mycotoxin exposure has been shown to disrupt DNA methylation patterns, leading to both hyper- and hypomethylation of specific genes[22]. Studies have indicated that Aflatoxin B1 (AFB1), a prevalent mycotoxin in Africa, can alter the methylation status of tumor suppressor genes and oncogenes, potentially contributing to hepatocarcinogenesis[23,24].

Several nutritional factors play a critical role in maintaining proper DNA methylation.

Table 3 highlights the protective roles of dietary methyl donors against mycotoxin-induced epigenetic alterations. Folate and B12 maintain methyl group balance; choline and betaine support SAM production, while polyphenols modulate DNMT activity. These nutrients mitigate gene silencing and abnormal methylation patterns in liver and cancer models.

Methyl donors, such as folate, vitamin B12, choline, and betaine, are essential for the one-carbon metabolism pathway, which provides the methyl groups required for DNA methylation reactions[25-30].

Figure 2 visually represents how dietary methyl donors regulate DNA methylation via the one-carbon metabolism pathway. Folate and betaine contribute methyl groups, while vitamin B12 acts as a cofactor in remethylation. These interactions sustain SAM synthesis, essential for DNA methyltransferase activity. Disruption by mycotoxins may impair this epigenetic balance.

Deficiencies in these nutrients, common in many African populations with limited dietary diversity, could exacerbate the effects of mycotoxins on DNA methylation[26-30]. Conversely, adequate intake of these methyl donors may offer some protection against mycotoxin-induced aberrant methylation patterns[27-30]. For instance, supplementation with folate has shown promise in modulating DNA methylation in various contexts[28-30]. Furthermore, dietary polyphenols found in fruits and vegetables, which are often lacking in sufficient quantities in the diets of at-risk populations in Africa, have also been shown to influence DNA methyltransferase (DNMT) activity, the enzymes responsible for DNA methylation[29,30]. Research exploring the direct impact of these nutritional interventions on mycotoxin-induced DNA methylation changes in African populations is crucial.

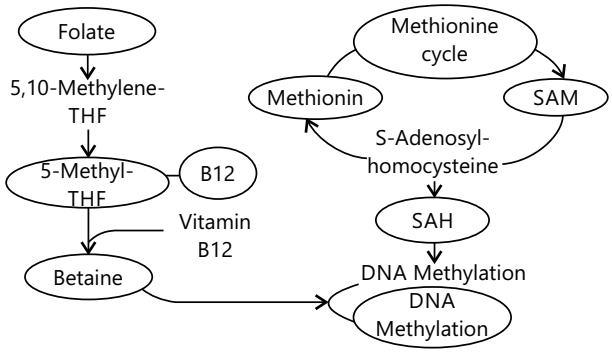

Fig. 2: One-carbon metabolism and nutrient-driven dna methylation pathway (self-generated)

The interlinked roles of folate, Vitamin B12, betaine, and methionine in the methionine cycle and DNA methylation process. Nutrients support the conversion of homocysteine to methionine, facilitating the generation of S-adenosylmethionine (SAM), the universal methyl donor for DNA methylation reactions

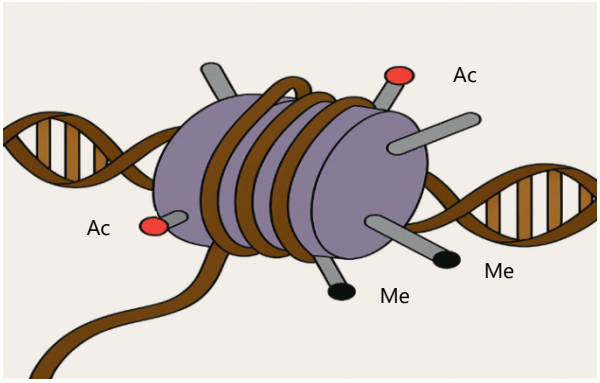

Fig. 3: Epigenetic modulation of dna via histone modifications (self-generated)

Depicts DNA (brown strand) wrapped around a histone core (purple), with modifications acetylation (Ac, red dots) and methylation (Me, black dots) on histone tails (gray rods). These post-translational modifications influence chromatin structure and gene expression

Histone modifications and dietary components: Histone modifications, including acetylation, methylation, phosphorylation, and ubiquitination of histone proteins around which DNA is wrapped, play a crucial role in regulating chromatin structure and gene accessibility[31].

Figure 3 highlights the roles of histone acetylation and methylation in gene regulation. Acetylation typically relaxes chromatin to promote transcription, while methylation may activate or silence genes depending on context. These modifications are targets for dietary influence and are susceptible to disruption by mycotoxins such as ochratoxin A. Nutrients like SCFAs and phytochemicals can restore or modulate these marks to maintain epigenetic balance.

Mycotoxins have been implicated in altering histone modification patterns, thereby affecting gene expression[32]. For example, exposure to ochratoxin A (OTA), another significant mycotoxin contaminant, has been shown to influence histone acetylation and methylation in kidney cells[33]. These alterations can lead to dysregulation of genes involved in cell growth, apoptosis, and inflammation[34].

Dietary components can influence histone modification enzymes, such as histone acetyltransferases (HATs) and Histone Deacetylases (HDACs)[35]. Short-Chain Fatty Acids (SCFAs), produced by the gut microbiota through the fermentation of dietary fiber, have been shown to inhibit HDAC activity[36]. Diets low in fiber,

Table 4: Dietary compounds influencing histone modification

Compound	Source	Effect on HAT/HDAC	Study outcome	Citations
Curcumin	Turmeric root	Inhibits HDAC activity	Restores histone acetylation balance and reduces mycotoxin-induced gene silencing	Meir *et al.*[31] Tiffon[32] Aricthota *et al.*[33] Ozawa *et al.*[34]
Butyrate	Fermentation of dietary fiber	HDAC inhibitor	Enhances histone acetylation in colon and liver cells	Sasaki and Masaki[35] Franzago *et al.*[36]
Sulforaphane	Cruciferous vegetables (e.g., broccoli)	Modulates HAT and HDAC enzymes	Promotes gene expression through histone acetylation	Fellows *et al.*[37] Martin-Gallausiaux *et al.*[38] Yuille *et al.*[39]
Ochratoxin A	Contaminated grains and coffee	Alters histone methylation and acetylation	Induces DNA damage and epigenetic dysregulation in renal cells	Kaufman-Szymczyk *et al.*[40]
Resveratrol	Red grapes, peanuts	Activates HAT, inhibits HDAC	Regulates histone modifications and restores epigenetic balance	

This table highlights key dietary compounds that modulate histone acetylation or methylation in the context of mycotoxin exposure, as evidenced by cellular and nutritional studies

which is often the case in resource-limited settings in Africa, may compromise the production of SCFAs and thus the potential for HDAC inhibition[37]. Conversely, increased intake of fiber-rich foods could potentially counteract mycotoxin-induced alterations in histone acetylation.

Table 4 presents evidence from *in vitro* and nutritional studies showing how compounds like curcumin, butyrate, sulforaphane, and resveratrol modulate histone-modifying enzymes to influence gene expression[38]. Butyrate and sulforaphane act as HDAC inhibitors, enhancing histone acetylation, while curcumin and resveratrol exhibit dual roles affecting both HAT and HDAC activity. Conversely, ochratoxin A from contaminated grains disrupts histone acetylation and methylation patterns, contributing to epigenetic dysregulation.

Similarly, certain vitamins and minerals, as well as bioactive compounds like butyrate and sulforaphane found in cruciferous vegetables, can modulate histone modification[39]. Investigating the impact of these dietary factors on histone modification changes induced by prevalent mycotoxins in African populations is essential for identifying potential protective nutritional strategies[40].

Non-coding RNAs and nutritional influence: Non-Coding RNAs (ncRNAs), such as microRNAs (miRNAs) and Long Non-Coding RNAs (lncRNAs), are important regulators of gene expression at the post-transcriptional level[41]. Emerging evidence suggests that mycotoxins can affect the expression profiles of various ncRNAs, contributing to their toxicological effects[42]. For instance, AFB1 exposure has been linked to altered expression of miRNAs involved in cell proliferation and apoptosis in liver cells[43,44].

Nutritional factors have also been shown to modulate ncRNA expression[45]. Dietary fatty acids, vitamins, and polyphenols can influence the biogenesis and stability of miRNAs and lncRNAs[46,47].

Figure 4 illustrates the nutrient-sensitive steps in miRNA biogenesis. Nutrients can modulate pri-miRNA transcription, Dicer-mediated processing, and miRNA duplex formation. These interactions collectively influence RISC assembly and gene silencing. Nutritional status therefore, critically impacts miRNA regulatory functions.

For example, omega-3 polyunsaturated fatty acids have been reported to alter miRNA expression in various tissues[48]. Given that the dietary intake of essential fatty acids and other micronutrients may be

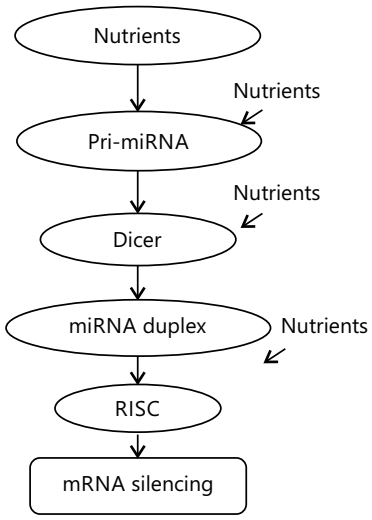

Fig. 4: Nutrient regulation of mirna biogenesis pathway (self-generated)

Schematic representation of how nutrients modulate the biogenesis and activity of microRNAs (miRNAs). Nutrients can influence the transcription of primary miRNA (pri-miRNA), the processing by Dicer, the formation of the miRNA duplex, and the assembly of the RNA-induced silencing complex (RISC), ultimately affecting mRNA silencing

suboptimal in many African communities exposed to mycotoxins, understanding the interplay between nutrition, mycotoxins, and ncRNA regulation is critical[49,50]. Research exploring whether specific dietary interventions can restore or prevent mycotoxin-induced aberrant ncRNA expression in these populations is warranted.

Table 5 summarizes how nutritional components influence the expression of Non-Coding RNAs (ncRNAs) affected by exposure to major mycotoxins such as aflatoxin B1, ochratoxin A, and fumonisin B1. Specific nutrients, including polyphenols, omega-3 fatty acids, and vitamins A and D, have been shown to restore or modulate the expression of dysregulated miRNAs and lncRNAs. These interactions highlight the therapeutic potential of nutritional interventions in counteracting mycotoxin-induced epigenetic disturbances.

Antioxidants and epigenetic modulation: Oxidative stress, characterized by an imbalance between the production of Reactive Oxygen Species (ROS) and the capacity of the antioxidant defense system, is a common consequence of mycotoxin exposure[51]. The ROS can directly damage DNA and also indirectly influence epigenetic machinery[52]. Mycotoxins like AFB1 and OTA can induce oxidative stress, potentially leading to alterations in DNA methylation and histone modifications[53,54].

Dietary antioxidants, including vitamins A, C, and E, selenium, and various phytochemicals, play a crucial role in neutralizing ROS and protecting against oxidative damage[55]. Adequate intake of these antioxidants may help mitigate the oxidative stress induced by mycotoxins and consequently reduce the associated epigenetic perturbations[56].

Table 6 compiles key antioxidants such as vitamins C and E, selenium, polyphenols, and glutathione, and their capacity to reverse or prevent DNA methylation, histone modifications, and miRNA dysregulation. Experimental evidence from *in vivo* and *in vitro* studies underscores their potential in protecting against oxidative epigenetic toxicity caused by AFB1 and OTA exposure.

However, the dietary intake of these antioxidants is often low in at-risk populations in Africa[57-60]. Supplementation or dietary enrichment with antioxidant-rich foods could potentially offer protection

Table 5: Mycotoxin and nutritional modulation of ncrna expression

Mycotoxin	Affected ncRNA	Nutrient modulator	Effect	Citations
Aflatoxin B1	miR-34a	Polyphenols	Downregulation of miR-34a reversed; restored apoptosis balance	Dashwood and Ho[41] Abdelmonem *et al.*[42] Marrone *et al.*[43]
Aflatoxin B1	miR-122, miR-192	Omega-3 fatty acids	Modulated miRNA expression, reduced inflammatory signals	Lizarraga *et al.*[44] Hua *et al.*[45]
Aflatoxin B1	Multiple (miR-24, miR-125b)	Vitamin A, D	Restored disrupted miRNA/lncRNA levels involved in hepatic detoxification	Lee[46] Wyatt *et al.*[47]
Aflatoxin B1	miR-29a, lncRNA H19	Balanced diet (macro/micronutrients)	Improved ncRNA homeostasis and reduced liver injury	Gareev *et al.*[48] Ishihara *et al.*[49]
Ochratoxin A	lncRNA MALAT1, miR-200c	Polyphenols, vitamins	Counteracted oxidative stress and ncRNA misregulation	El-Agamy[50]
Fumonisin B1	miR-21, miR-155	Nutritionally enriched diets	Attenuated pro-inflammatory ncRNA expression	

The interplay between various mycotoxins and their impact on non-coding RNA (ncRNA) expression, with corresponding dietary modulators and their mitigating effects. Nutrients such as polyphenols, omega-3 fatty acids, and vitamins exhibit potential in restoring ncRNA balance disrupted by mycotoxins like aflatoxin B1, ochratoxin A, and fumonisin B1

Table 6: Antioxidants that modulate epigenetic alterations

Antioxidant	Dietary source	Epigenetic effect	Evidence	Citations
Vitamin C and E	Citrus fruits, nuts, seeds	Reduced AFB1-induced DNA methylation and histone modifications	*In vivo* rodent model study showing epigenetic protecton	Sagomba and Ndhlovu[51] Lu *et al.*[52] Alipoor *et al.*[53]
Selenium and Vitamin E	Nuts, cereals, eggs	Combats mycotoxin-triggered oxidative and epigenetic damage	Elemental synergy reduces histone and DNA methylation changes	Jobe *et al.*[54] Surai[55] Albadrani *et al.*[56]
Polyphenols	Fruits, vegetables, tea	Prevented OTA-induced histone modification and DNA methylation	Protective antioxidant pathway activation	Tanvir *et al.*[57] da Silva *et al.*[58] Yang *et al.*[59]
Glutathione	Spinach, avocado, asparagus	Restored DNA methylation altered by AFB1	*In vitro* hepatic protection via methylation correction	Loganathan and Doss[60]
Flavonoids	Berries, dark chocolate	Reversed mycotoxin-induced miRNA dysregulation and chromatin remodeling	Epigenomic modulation through antioxidant action	
General antioxidants	Mixed plant-based diets	Overall reduction of oxidative DNA damage and improved epigenetic stability	Supported across reviews and experimental models	

The role of various dietary antioxidants in mitigating epigenetic alterations caused by mycotoxin-induced oxidative stress. Evidence suggests that nutrients such as vitamins C and E, selenium, polyphenols, and glutathion e can reverse or prevent harmful modifications to DNA methylation and histone structure, thereby offering protective effects. The findings are supported by both *in vivo* and *in vitro* studies across diverse antioxidant sources

against mycotoxin-induced epigenetic changes mediated by oxidative stress[58-60]. Studies investigating the impact of antioxidant interventions on epigenetic markers in individuals exposed to mycotoxins in Africa are needed.

Figure 5 shows how mycotoxin-induced oxidative stress generates ROS that damage DNA and histones, contributing to epigenetic dysregulation[59,60]. These alterations can affect gene expression, potentially leading to adverse health outcomes. Antioxidants such as vitamins C and E, selenium, and polyphenols mitigate this oxidative and epigenetic damage by neutralizing ROS and restoring redox balance[60]. The figure supports the mechanisms described in this sub-section regarding antioxidant intervention in epigenetic modulation under oxidative stress[60].

Gut microbiota and epigenetic interactions: The gut microbiota, a complex community of microorganisms residing in the gastrointestinal tract, plays a significant role in host health, including odulating immune responses and influencing metabolism[61]. Mycotoxins can disrupt the composition and function of the gut microbiota, leading to dysbiosis[62]. Conversely, the gut microbiota can metabolize some mycotoxins, potentially altering their bioavailability and toxicity[63].

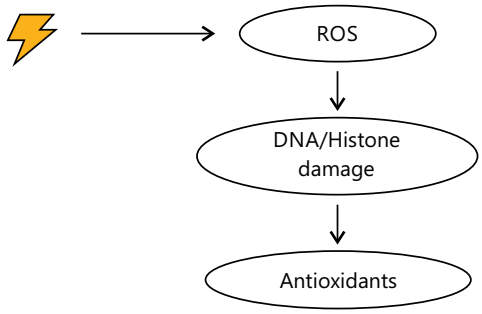

Fig. 5: Oxidative stress pathway linking ros to epigenetic alterations and antioxidant defense (self-generated)

The cascade of events initiated by environmental or cellular stressors leading to the generation of reactive oxygen species (ROS), which in turn cause DNA and histone damage. This triggers antioxidant responses aimed at restoring redox balance and preserving genomic and epigenetic stability

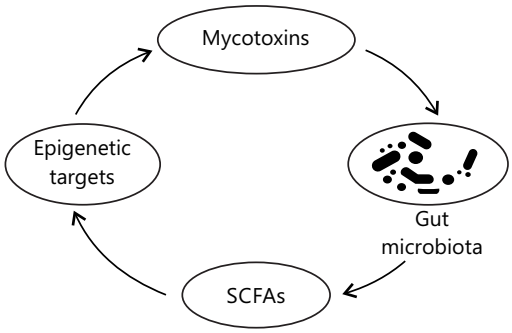

Fig. 6: Gut microbiota mycotoxin epigenetic interaction pathway (self-generated)

The bidirectional relationship between the gut microbiota and epigenetic modifications under the influence of dietary components and mycotoxin exposure. It shows how dysbiosis, SCFA production, and microbial metabolism of toxins modulate host epigenetic responses

Emerging evidence suggests a bidirectional link between the gut microbiota and epigenetics[64].

Figure 6 maps out the interplay between gut microbiota and host epigenetics in the context of mycotoxin exposure. It shows how mycotoxins disrupt microbial balance, while beneficial microbes produce metabolites like Short-Chain Fatty Acids (SCFAs) that modulate DNA methylation and histone acetylation. This interaction is diet-sensitive, highlighting the importance of fiber and prebiotics in regulating both microbial and epigenetic health.

Microbial metabolites, such as SCFAs, can influence histone modifications in the host[65]. Furthermore, the gut microbiota can indirectly affect DNA methylation through the production of metabolites involved in one-carbon metabolism[66]. Nutritional factors, particularly dietary fiber and prebiotics, can significantly impact the composition and function of the gut microbiota[67]. Diets rich in these components, which may be lacking in some African populations, can promote the growth of beneficial bacteria and the production of health-promoting metabolites[68].

Table 7 outlines the role of microbial-derived metabolites such as butyrate, acetate, and propionate in regulating host epigenetic mechanisms. These metabolites influence histone acetylation and DNA methylation by acting as HDAC inhibitors or providing substrates for methylation. Their production is modulated by dietary intake of fibers and prebiotics, supporting gut-epigenome homeostasis.

Table 7: Microbial metabolites involved in epigenetic regulation

Metabolite	Microbial source	Epigenetic target	Dietary driver	Citations
Butyrate	*Faecalibacterium prausnitzii* and *Roseburia* spp.	Histone deacetylases (HDACs)	Dietary fiber (prebiotics)	Jalouli *et al.*[61] Rudrapal *et al.*[62] Owumi *et al.*[63]
Acetate	*Bacteroides* spp. and *Akkermansia muciniphila*	DNA methyltransferases (DNMTs), HDACs	Resistant starch, oligosaccharides	Afzaal *et al.*[64] Choi *et al.*[65] Guerre[66]
Propionate	*Bacteroides* spp. and *Veillonella* spp.	HDAC inhibition, miRNA regulation	Whole grains, legumes	Zhang *et al.*[67] Tian and Chen[68]
Indole derivatives	*Clostridium* spp. and *Lactobacillus* spp.	Aryl hydrocarbon receptor (AhR) modulation	Tryptophan-rich diets	Holmes *et al.*[69]
Secondary bile acids	*Bacteroides* spp. and *Clostridium* spp.	Histone acetylation, nuclear receptor signaling	High-fat diet, bile salt metabolism	
Lactate	*Lactobacillus* spp.	HDAC inhibition, histone modification	Fermented foods, probiotics	
Phenylacetic acid	*Clostridium* spp. and *Bacteroides* spp.	Histone methylation	Protein-rich diets	
Aflatoxin-degrading enzymes	*Lactobacillus plantarum*	Modulates toxin-induced epigenetic stress	Probiotic supplementation	

This table summarizes key microbial metabolites produced in the gut that influence host epigenetic mechanisms. It highlights the microbial taxa responsible for generating each metabolite, their respective epigenetic targets (including HDACs, DNMTs, histone modifications, and miRNAs), and the dietary components that drive their production. The interplay of these factors underpins a dynamic microbe-diet-epigenome axis relevant to health and disease

Investigating how dietary interventions aimed at modulating the gut microbiota can influence mycotoxin-induced epigenetic changes in at-risk African populations represents a promising area of research[68,69].

Synergistic effects of multiple mycotoxins and nutrition: In real-world scenarios, particularly in Africa, exposure to multiple mycotoxins is common[70]. These co-exposures can lead to synergistic or additive toxic effects, which may also extend to epigenetic modifications[71]. For example, combined exposure to AFB1 and fumonisins has been shown to have more severe health consequences than exposure to either mycotoxin alone[72].

Figure 7 visualizes the enhanced toxicity of mycotoxin mixtures (e.g., AFB1+fumonisin) and their epigenetic impacts[73]. Nutritional interventions (polyphenols, probiotics, micronutrients) disrupt synergistic damage by mitigating oxidative stress and genotoxicity. The model highlights how nutrient deficiencies exacerbate vulnerability in co-exposure scenarios, while dietary diversification offers protection. It contextualizes Table 8 data within real-world exposure contexts in Africa.

The nutritional status of individuals can also influence the combined effects of multiple mycotoxins on epigenetic regulation[74]. Deficiencies in essential nutrients may render individuals more susceptible to the adverse effects of co-exposure. Conversely, adequate or enhanced intake of specific nutrients may offer broader protection against the epigenetic disruption caused by multiple mycotoxins[75].

Table 8 catalyses evidence on mycotoxin co-exposure synergism (e.g., aflatoxin+fumonisin nephrotoxicity) and nutritional countermeasures[75,76]. Deficiencies in selenium/zinc exacerbate epigenetic dysregulation, while antioxidant-rich diets reduce DNA damage[76,77]. African field studies confirm growth retardation from mixed exposures in maize-dependent populations[77]. Data advocate integrative nutrition (e.g., probiotic/isolavone supplementation) to break toxicity cycles[78].

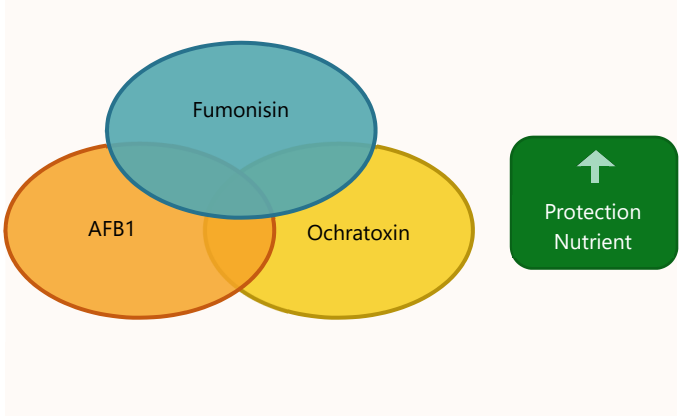

Fig. 7: Interplay of co-occurring mycotoxins and nutritional interventions (self-generated)

Synergistic toxicity from mycotoxin combinations (AFB1, fumonisin, ochratoxin) and protective nutrient-mediated pathways. Overlapping exposure areas denote amplified health risks, while nutritional modulators (antioxidants, micronutrients) counter epigenetic disruption and organ damage

Table 8: Combined effects of mycotoxins and nutritional status

Mycotoxin combo	Effect	Nutritional modulator	Study design	Citations
Aflatoxin+Fumonisin	Synergistic intestinal and liver toxicity	Antioxidant-rich diets	*In vitro* and animal models	Hoffmann *et al.*[71] Paulo *et al.*[72]
Multiple mycotoxins (AFB1, DON, ZEN)	Enhanced epithelial damage and immune suppression	Micronutrients (Se, Zn, Vit A)	*In vitro* and human exposure data	Li *et al.*[73] Pastor *et al.*[74] Divella *et al.*[75]
Aflatoxin+Zearalenone	Endocrine disruption and genotoxicity	Isoflavones and probiotics	Animal experiments	Sagy *et al.*[76] Kamala *et al.*[77]
Co-occurring fumonisin, aflatoxin, ochratoxin	Growth retardation, gut permeability alteration	Dietary diversity and protein adequacy	Field studies in children	Gruber-Dorninger *et al.*[78] Apeh *et al.*[79]
Mixed mycotoxin exposure in maize	Cumulative dietary toxicity	Food processing (fermentation, nixtamalization)	Dietary surveys and food analysis	
Aflatoxin+Ochratoxin A	Nephrotoxicity and oxidative stress	Polyphenols and flavonoids	Animal models and mechanistic studies	
Multiple mycotoxins in infants	Compromised immunity and micronutrient deficiencies	Infant complementary feeding strategies	Cross-sectional and cohort studies	
General co-exposure	Synergistic toxicity and malnutrition interaction	Nutritional counselling and interventions	Review and meta-analysis	

The interactive effects of combined mycotoxin exposures and nutritional status. It details specific mycotoxin combinations, their toxicological outcomes, modulating nutritional factors, and the nature of supporting studies. These findings underscore the importance of integrative nutritional strategies to mitigate mycotoxin-related health risks, especially in vulnerable populations across Sub-Saharan Africa

Research specifically examining the nutritional modulation of epigenetic changes induced by mixtures of mycotoxins, relevant to the African context, is crucial for developing effective risk mitigation strategies[79]. This research should consider the common co-occurrence of mycotoxins in staple foods and the prevalent nutritional deficiencies in these populations.

IMPLICATIONS FOR AT-RISK POPULATIONS IN AFRICA

The findings discussed in this review have significant implications for at-risk populations in Africa who face chronic exposure to mycotoxins and often experience nutritional deficiencies[80]. Understanding how nutrition can modulate mycotoxin-induced epigenetic changes opens avenues for developing targeted dietary interventions to mitigate the long-term health risks associated with mycotoxin exposure[81].

Fig. 8: Multidimensional approach to mycotoxin risk mitigation (self-generated)

Four-pillar framework for reducing mycotoxin-related health risks: Food Safety (contamination control), Nutrition Education (dietary awareness), Community Action (local implementation), and Research (evidence generation). Arrows denote interdependencies between pillars

Table 9: Recommendations for nutritional interventions

Strategy	Nutrient focus	Implementation approach	Target population	Citations
Dietary diversification	Vitamins, minerals, antioxidants	Promote varied diets including cereals, legumes, fruits, and vegetables	Rural and urban low-income households	Xu *et al.*[81] Chen *et al.*[82] Cheng *et al.*[83]
Functional food promotion	Anti-epigenetic bioactives	Encourage consumption of biofortified and functional foods	Populations at risk of aflatoxin exposure	Turner *et al.*[84] Nji *et al.*[85] Visser *et al.*[86]
Nutrition-sensitive food safety education	Mycotoxin awareness, micronutrients	Integrate food safety into nutrition education programs	Women, caregivers, and school-age children	Kimanya *et al.*[87] Ingenbleek *et al.*[88] Wild and Gong[89]
Community-based hygiene interventions	Sanitation-linked nutrient retention	Local programs addressing water, sanitation, and hygiene (WASH)	Underserved rural communities	
Policy-driven public health campaigns	Nutrient-enabling environments	National aflatoxin mitigation strategies and food system reforms	Policymakers, educators, and food vendors	
Risk communication and systems approach	Environmental nutrition interaction	Holistic assessment of environmental and dietary toxins	General African populations in hotspot regions	

This table presents strategic nutritional interventions to mitigate mycotoxin exposure risks across African communities. Dietary diversification, functional food intake, food safety education and hygiene-centered programs are recommended for vulnerable populations. Policy campaigns and systems-based communication target broader public health infrastructure and environmental factors

Figure 8 proposes an integrated strategy to address mycotoxin exposure in African populations. Food safety practices reduce contamination in staples[81], while nutrition education promotes protective diets rich in methyl donors and antioxidants[82]. Community-driven hygiene interventions enhance nutrient retention[82,83], and research identifies epigenetic biomarkers for intervention efficacy[83]. The model aligns with Table 9 evidence-based recommendations for vulnerable groups.

Promoting dietary diversity, increasing the intake of fruits, vegetables, and whole grains rich in methyl donors, antioxidants, and fiber, and addressing micronutrient deficiencies are crucial steps[83].

Table 9 synthesizes intervention strategies to disrupt mycotoxin-induced epigenetic damage in Africa. Dietary diversification combats micronutrient deficiencies linked to susceptibility[83,84], while functional foods

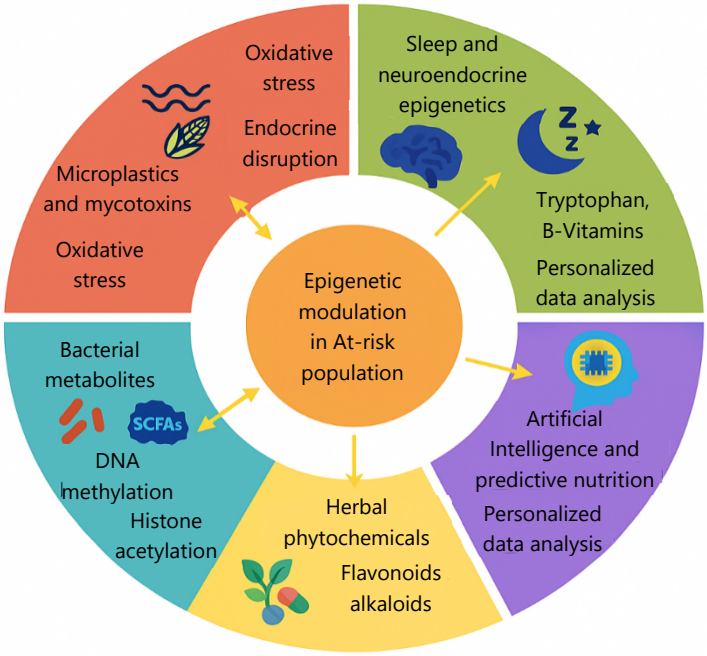

Fig. 9: Integrated nutritional-epigenetic response model to environmental pollutants (self-generated)
Five major domains influencing epigenetic responses to environmental toxins. Nutritional components and emerging technologies offer protective modulation. This integrative model underscores the complexity of diet-environment-gene interactions in vulnerable populations

(e.g., biofortified crops) deliver anti-epigenetic bioactives[84]. Policy-driven food safety campaigns and WASH programs address environmental determinants[85], and systems-based risk communication enables holistic responses[86].

Furthermore, research is needed to identify specific nutritional biomarkers that can indicate susceptibility to mycotoxin-induced epigenetic damage and to monitor the effectiveness of dietary interventions[87].

Culturally appropriate and sustainable food-based strategies should be prioritized[88]. Public health initiatives aimed at improving food safety practices, reducing mycotoxin contamination in staple crops, and promoting nutrition education are essential components of a comprehensive approach to protect the health of these vulnerable populations[89]. Future research should focus on conducting well-designed intervention studies in African settings to evaluate the efficacy of specific nutritional strategies in modulating mycotoxin-related epigenetic alterations and improving health outcomes.

INTEGRATIVE PERSPECTIVES ON EMERGING BIOCHEMICAL INTERACTIONS AND NUTRITIONAL EPIGENETICS

Recent advances in nutritional epigenetics have expanded our understanding of how biochemical signals interact with environmental toxins to modulate gene expression. Mycotoxins are increasingly being considered alongside other emerging pollutants such as microplastics, which may similarly disrupt epigenetic homeostasis through oxidative stress, mitochondrial dysfunction, and interference with hormone signaling pathways[90]. These pollutants, including mycotoxins, can influence systemic biochemical networks, with cumulative effects on metabolic, immune, and neuroendocrine systems.

A growing body of evidence indicates that neurobiological rhythms, particularly those governing sleep and circadian cycles, are under both nutritional and e pigenetic regulation[91]. Mycotoxins have been associated with altered sleep architecture and endocrine function, potentially via modulation of melatonin pathways and DNA methylation enzymes. Dietary modulation through polyphenols, methyl donors, and specific amino acids may provide a corrective mechanism to restore disrupted neuroepigenetic circuits.

Moreover, bacterial-derived metabolites are emerging as potent biochemical mediators that influence host immunity, neurotransmitter regulation, and gene expression through epigenetic remodeling[92]. These findings emphasize the potential of microbiome-targeted nutritional strategies such as the use of prebiotics, probiotics, and postbiotics to counteract mycotoxin-induced epigenetic alterations.

Parallel insights from herbal biochemistry offer another promising intervention pathway. Certain phytochemicals from traditional herbal medicines possess epigenetic activity, including inhibition of Histone Deacetylases (HDACs) and DNA Methyltransferases (DNMTs), thereby modulating gene expression in a manner that can mitigate mycotoxin toxicity[93]. The integration of nutraceuticals with known molecular targets could strengthen community-based dietary interventions.

Finally, Artificial Intelligence (AI) is beginning to play a pivotal role in biochemical research, offering computational models to predict nutrient toxin epigenome interactions[94]. The AI-based platforms can help identify population-specific dietary strategies that optimally counteract the epigenetic burden of mycotoxins, accounting for genetic polymorphisms, metabolic profiles, and environmental exposures. Future research in African populations should incorporate these novel perspectives to enhance the effectiveness of nutrition-sensitive interventions.

Figure 9 illustrates how diverse biochemical and environmental factors influence epigenetic modulation in at-risk populations. Key contributors include microplastics, gut microbiota, sleep regulation, herbal phytochemicals, and AI-guided nutrition. Arrows represent directional influences on DNA methylation, histone acetylation, and overall epigenetic stability.

CONCLUSION

Mycotoxin exposure poses a significant epigenetic and public health challenge in Sub-Saharan Africa, especially among nutritionally vulnerable populations. This review highlights the pivotal role of nutrition in modulating mycotoxin-induced alterations in DNA methylation, histone modifications, and noncoding RNA expression. Key dietary components, including methyl donors, antioxidants, and fiber, offer promising protective mechanisms against these epigenetic disruptions. Integrating nutrition-sensitive strategies with food safety interventions can mitigate long-term health risks. Culturally appropriate dietary interventions and public health education are essential for sustainable impact. Future research should prioritize biomarker discovery and targeted trials to inform evidence-based policies.

SIGNIFICANCE STATEMENT

This manuscript highlights a critical yet often overlooked dimension of public health in Sub-Saharan Africa, the intersection between chronic mycotoxin exposure and nutrition-driven epigenetic regulation. By synthesizing current evidence on how key dietary components such as methyl donors, antioxidants, and microbiota modulators influence gene expression and mitigate toxin-induced epigenetic disruptions, the study presents a compelling framework for preventive health. It emphasizes the potential of nutrition as a cost-effective, culturally appropriate strategy to reduce the long-term health burden associated with mycotoxins. The findings offer valuable insights for integrating nutritional interventions into food safety policies and population health programs.

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