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Microbiome-drug interactions: implications for pharmacokinetics, drug efficacy, and safety

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ABSTRACT

The human microbiome has emerged as a crucial modulator of drug response, significantly influencing pharmacokinetics, efficacy, and safety. This review explores the multifaceted interactions between the gut microbiota and pharmaceuticals, highlighting the bidirectional nature of these relationships. A literature search covering publications from 2010 to 2025 was conducted using databases such as Google Scholar, PubMed, Web of Science, Embase, and Scopus. Keywords included "Microbiota", "Pharmacokinetics", "Drug Metabolism", "Drug Interactions", "Gastrointestinal Microbiome"and "Precision Medicine." Microbial enzymes can metabolize drugs before absorption, alter their chemical structures, and influence their bioavailability and systemic exposure. These microbiome-mediated transformations can enhance or diminish drug efficacy, lead to the formation of toxic metabolites, or interfere with drug activation, particularly in prodrugs. Conversely, drugs can perturb microbial composition and diversity, potentially contributing to dysbiosis and long-term health consequences. Variability in individual microbiota profiles is increasingly recognized as a contributor to inter-individual differences in drug responses, offering an explanation for observed inconsistencies in therapeutic outcomes and adverse drug reactions. Notable examples include the microbial inactivation of digoxin. In recent years, the human microbiome has emerged as a crucial determinant of health and disease, influencing a wide array of physiological processes including digestion, immunity, and neurochemical signaling. Among its lesser-known but increasingly recognized roles is its profound impact on drug metabolism and therapeutic outcomes. These microbiome–drug interactions are now understood to contribute significantly to the variability in drug response observed among individuals, challenging the traditional one-size-fits-all approach to pharmacotherapy.

Keywords: Microbiota, Pharmacokinetics, Drug Metabolism, Drug Interactions, Gastrointestinal Microbiome, Precision Medicine

Introduction

The clinical relevance of this phenomenon is becoming particularly evident as researchers uncover how certain bacteria can enzymatically activate or deactivate drugs, alter their pharmacokinetics, or even generate toxic metabolites. For instance, the inactivation of digoxin by *Eggerthella lenta*, or the metabolism of the anti-cancer drug irinotecan leading to gastrointestinal toxicity, highlight the dual-edged influence of microbial enzymatic activity on therapeutic efficacy and safety [1]. Moreover, antibiotics and other medications that disrupt the microbiome can initiate feedback effects, leading to further alterations in drug metabolism or susceptibility to infections, such as *Clostridioides difficile* [2].

This intricate bidirectional relationship between the microbiome and pharmacologic agents necessitates a deeper understanding of microbiome–drug interactions, particularly within the context of clinical pharmacy. Pharmacists are uniquely positioned to incorporate microbiome science into therapeutic decision-making, especially in areas such as medication therapy management, precision medicine, and adverse drug reaction monitoring.

This review explores the current state of knowledge on microbiome–drug interactions, detailing mechanisms through which gut microbes influence drug action and highlighting drugs most affected by these interactions. It also examines the implications for drug development, pharmacokinetics, therapeutic outcomes, and patient safety. By understanding these interactions, pharmacy professionals can better predict individual responses to medications, minimize adverse events, and contribute to the advancement of personalized medicine.

Methodology

This review employed a systematic approach to identify, evaluate, and synthesize relevant literature on pharmaceutical care practices in Nigeria's hospital and community pharmacy settings. The primary aim was to explore the interactions between the gut microbiota and pharmaceuticals, highlighting the bidirectional nature of these relationships. A comprehensive literature search was conducted across five electronic databases namely PubMed, Embase, Web of Science, Scopus, and Google Scholar, for articles published between January 2010 and May 2025. Search terms combined MeSH keywords (“Microbiota,” “Pharmacokinetics,” “Drug Metabolism,” “Drug Interactions,” “Gastrointestinal Microbiome,” “Precision Medicine”) with free-text variants (e.g., “gut flora,” “drug–microbiome interaction”). Boolean operators (AND, OR) were applied to refine results. Reference lists of key reviews and original studies were manually screened to identify additional relevant publications.

Inclusion and exclusion criteria

Included in the review were o**riginal** research articles, systematic reviews, and meta-analyses examining the bidirectional interactions between gut microbiota and pharmaceuticals; studies addressing microbial effects on drug absorption, metabolism, efficacy, safety, or therapeutic outcomes; human clinical trials and in vivo/in vitro mechanistic investigations. Excluded were **c**ase reports, editorials, conference abstracts without full text, non-English publications, and studies focused solely on non-gastrointestinal microbiomes (e.g., skin, oral).

Study selection

Titles and abstracts retrieved from the initial search were independently screened by two reviewers. Full texts of potentially eligible articles were then assessed against inclusion criteria. Discrepancies were resolved through discussion or adjudication by a third reviewer.

The Human Microbiome: Composition and Function

The human microbiome refers to the collective genomes of the microorganisms—bacteria, archaea, viruses, fungi, and protozoa—that inhabit the skin and mucosal surfaces, with the greatest density residing in the gastrointestinal tract [3]. This complex ecosystem co-evolves with the host, contributing essential metabolic, immune, and barrier functions that influence health and disease.

The microbial composition of thegut microbiota **is** made up of bacterial phyla dominated by four major bacterial phyla (i.e., Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria), with relative abundances varying by individual, age, diet, and geography; Archaea and Eukarya comprising of Methanogenic archaea (e.g., *Methanobrevibacter smithii*) and a diversity of fungal species (the “mycobiome,” such as *Candida* and *Saccharomyces*) which occupy smaller but functionally significant niches; and the v**i**ral community termed the gut virome, largely composed of bacteriophages, modulates bacterial populations and that may facilitate horizontal gene transfer, including antimicrobial-resistance genes [4].

The core metabolic processes of the gut microbes ferment indigestible dietary fibers into short-chain fatty acids (SCFAs) like acetate, propionate, and butyrate, which serve as energy substrates for colonocytes, regulate lipid and glucose metabolism, and reinforce intestinal barrier integrity [5]. The microorganisms also function as **i**mmune modulators. Commensal bacteria educate the host immune system through pattern-recognition receptors (e.g., Toll-like receptors), by promoting tolerance to beneficial microbes while mounting defenses against pathogens. Microbial metabolites also influence

systemic immune tone, affecting susceptibility to inflammatory and autoimmune disorders [6].

Gut microbiota also gives and preserves the barrier against pathogens.The microbiota enhances mucosal barrier function by stimulating mucus production, tight-junction protein expression, and antimicrobial peptide secretion, thereby limiting pathogen translocation and systemic endotoxemia. Finally, it serves in neurochemical signaling via the gut–brain axis through the activities of microbiota-derived neurotransmitters (e.g., γ-aminobutyric acid, serotonin precursors) and SCFAs. Through these agents, the gut-microbiota modulates neuronal function, with implications for mood, cognition, and behavior [7].

Mechanisms of microbiome–drug interactions

The human microbiome influences drug disposition through several mechanisms that affect pharmacokinetics, efficacy, and toxicity. One of the primary pathways is direct microbial metabolism of drugs. Gut microbes express a variety of enzymes—such as azoreductases, nitroreductases, hydrolases, and β-glucuronidases—that can chemically modify drugs before absorption. These modifications may activate prodrugs, inactivate therapeutic agents, or generate toxic metabolites, as seen with the reactivation of irinotecan's glucuronide conjugates by microbial β-glucuronidases [8-10].

Another key mechanism is indirect modulation of host metabolic pathways**.** Microbes can alter the expression or activity of host drug-metabolizing enzymes (e.g., cytochrome P450 enzymes) and transporters by producing signaling molecules or metabolites such as short-chain fatty acids and secondary bile acids. Microbial interactions may also influence drug absorption by modifying intestinal pH, mucosal barrier integrity, or bile acid composition, thereby impacting solubility and permeability. Moreover, **microbiota-induced immune modulation**can affect the pharmacodynamics of immunotherapeutics and vaccines [11].

Direct metabolism of drugs by microbiota

The gut microbiota plays a crucial role in the direct metabolism of orally administered drugs through a wide array of microbial enzymes capable of altering drug structure and activity. This direct interaction can lead to either enzymatic activation of prodrugs or inactivation of active compounds, significantly influencing their therapeutic efficacy and toxicity [12].

Enzymatic activation occurs when microbial enzymes convert inactive compounds into their active forms. A classic example is sulfasalazine, a prodrug used to treat inflammatory bowel disease. It is poorly absorbed in the upper gastrointestinal tract but becomes therapeutically active only after microbial azoreductases in the colon cleave it into 5-aminosalicylic acid (5-ASA) and sulfapyridine [13].

Conversely, enzymatic inactivation by gut microbes can reduce drug efficacy. For instance, the cardiac glycoside digoxin is inactivated by the gut bacterium *Eggerthella lenta*, which expresses a specific reductase enzyme that reduces digoxin into inactive dihydrodigoxin. This microbial activity varies among individuals, contributing to inconsistent drug responses [14].

These transformations illustrate the importance of microbiota in modifying drug fate before absorption. Understanding such interactions is essential for predicting drug responses, personalizing therapy, and mitigating adverse effects, particularly for drugs with narrow therapeutic indices or those requiring microbial activation for efficacy [15].

Microbiome-Mediated Modulation of Host Enzymes

Beyond direct metabolism of drugs, the microbiome can indirectly influence drug disposition by modulating the host's own metabolic systems, particularly cytochrome P450 (CYP) enzymesanddrug transporters**.** These host proteins are central to drug absorption, distribution, metabolism, and excretion (ADME), and their activity can be significantly affected by microbial signals.

Gut microbes produce a wide range of metabolites—such as short-chain fatty acids (SCFAs)**,** secondary bile acids**,** indoles, and lipopolysaccharides (LPS)—that interact with host signaling pathways. These microbial metabolites can modulate nuclear receptors like the pregnane X receptor (PXR)**,** aryl hydrocarbon receptor (AhR)**,** andfarnesoid X receptor (FXR)**,** which in turn regulate the expression of CYP enzymes and transporters such as P-glycoprotein (P-gp) and organic anion transporting polypeptides (OATPs) [17]**.** For example, microbial-derived secondary bile acids can activate PXR, leading to the upregulation of CYP3A4**,** a key enzyme responsible for metabolizing over 50% of clinically used drugs. Similarly, indole derivatives from tryptophan metabolism may influence AhR signaling, altering CYP1A1 expression [18].

These interactions suggest that microbial composition can indirectly dictate the rate and extent of drug metabolism and clearance. Dysbiosis or shifts in microbiota due to diet, antibiotics, or disease may therefore lead to variability in drug response by altering host enzymatic and transporter activity, highlighting the importance of integrating microbiome factors into pharmacokinetic profiling and personalized medicine approaches [19].

Influence on drug absorption and bioavailability

The gut microbiome significantly influences drug absorption and bioavailability by modifying the gastrointestinal environment and host physiology. Two key mechanisms include its impact on intestinal barrier integrityandbile acid metabolism**,** both of which play crucial roles in drug solubility, permeability, and systemic availability [20. 21].

Intestinal barrier integrity is maintained by tight junction proteins that regulate the passage of substances across the epithelium. A healthy microbiota supports this barrier through the production **of** short-chain fatty acids (SCFAs**)** like butyrate, which reinforce tight junctions and prevent increased intestinal permeability. Dysbiosis, in contrast, can compromise barrier function—often referred to as a "leaky gut"—leading to altered drug absorption, inflammation, and unintended systemic exposure to microbial products and drugs [22-24].

The microbiome also modulates bile acid metabolism, which affects the solubilization and micellar transport of lipophilic drugs. Gut bacteria convert primary bile acids into secondary bile acids, altering the composition and emulsification capacity of bile. This can influence the solubility and dissolution rate of orally administered drugs, especially those that rely on bile-mediated absorption [25].

Additionally, microbial fermentation products and other metabolites can modify **luminal pH**, particularly in the colon, impacting the ionization and absorption of pH-sensitive drugs [26]. Such pH changes can either enhance or impair the passive diffusion of drugs across the gut epithelium.

Together, these microbiome-driven changes underscore the importance of considering host–microbe interactions when evaluating oral drug bioavailability and therapeutic outcomes.

Generation of Toxic Metabolites

The gut microbiome can contribute todrug-induced toxicityby generating harmful metabolites through enzymatic transformations. This process may enhance the systemic or local toxicity of certain medications, particularly those that undergo enterohepatic circulation or are metabolized by microbial enzymes in the colon [27].

A prominent example is irinotecan, a chemotherapeutic agent used in colorectal cancer. After hepatic glucuronidation, irinotecan is excreted into the bile as an inactive metabolite (SN-38G). However, microbial β-glucuronidases in the colon can deconjugate SN-38G back into the active form SN-38, which is highly toxic to intestinal epithelial cells. This reactivation leads to severe delayed-onset diarrhea, a major dose-limiting side effect.

Another class of drugs associated with microbiome-related toxicity is nonsteroidal anti-inflammatory drugs (NSAIDs)**.** Chronic use of NSAIDs can disrupt the gut microbiota, increase intestinal permeability, and promote the microbial production of reactive oxygen species (ROS**)** and pro-inflammatory mediators. These effects can contribute to gastrointestinal ulcers, bleeding, and enteropathy. Furthermore, microbial transformation of NSAID metabolites may result in toxic intermediates that exacerbate mucosal damage [27].

These examples highlight the need to consider microbial contributions to drug metabolism not only for therapeutic efficacy but also for safety. In some cases, strategies likeβ-glucuronidase inhibitors**,** microbiome modulation, or targeted drug delivery systems are being explored to reduce microbiota-mediated toxicity and improve drug tolerability.

Drugs Most Affected by Microbiome Interactions

Certain classes of drugs are particularly susceptible to microbiome-mediated effects due to their chemical structures, metabolic pathways, or sites of absorption. These interactions can alter drug activation, inactivation, toxicity, and therapeutic outcomes. Notable examples include:

Cardiovascular drugs

Digoxin is inactivated by the gut bacterium *Eggerthella lenta*, leading to reduced therapeutic efficacy in individuals with high microbial reductase activity [29].

Immunosuppressants and anti-cancer drugs

Irinotecan, a chemotherapeutic agent, is reactivated in the gut by microbial β-glucuronidases**,** contributing to dose-limiting gastrointestinal toxicity [30].

Cyclophosphamide and oxaliplatin efficacy partly depends on microbiome-induced immune modulation.

Prodrugs activated by microbes

Sulfasalazine is cleaved by bacterial azoreductases into its active form, 5-aminosalicylic acid, essential for its anti-inflammatory action in the colon [31].

Metformin

This antidiabetic drug alters gut microbiota composition, which in turn contributes to its glucose-lowering effects and gastrointestinal side effects [32].

NSAIDs and Acetaminophen

These drugs can cause gut microbiota disruption, and microbial enzymes can modify their metabolites, potentially affecting toxicity [34].

L-Dopa (Levodopa)

Microbial decarboxylation of L-Dopa in the gut can reduce central nervous system bioavailability, affecting Parkinson's disease management. Understanding which drugs are most affected by microbiome interactions is essential for predicting variable responses, mitigating adverse effects, and guiding personalized therapy [35]. Table 1 presents some drugs, microbiome-mediated metabolism and pharmacokinetic effects.

Table 1: Selected examples of microbiome-mediated drug metabolism and its pharmacokinetic impact

Drug	Therapeutic class	Microbial species	Microbiome effect	Pharmacokinetic effect
Acetaminophen	Analgesic	Many gut microbes	Sulfonation pathway competition	Potential altered hepatic metabolism
Digoxin	Cardiac glycoside	Eggerthellalenta	Reduction/inactivation	Reduced oral bioavailability
Levodopa	Antiparkinsonian	Enterococcus faecalis	Converts to dopamine	Reduced central availability and altered efficacy
Sulphasalazine	Anti-inflammatory	Colonic bacteria (azoreductases)	Pro-drug activation	Delayed drug release
Irinotecan	Anticancer agent	ß-glucuronidase producing	Reactivation of toxic metabolites	Increase GI toxicity to SN-38

Impact on pharmacokinetics and pharmacodynamics

The gut microbiome plays a significant role in modulating both pharmacokinetics andpharmacodynamicsMicrobiome–drug interactions can lead to measurable changes in drug half-life, plasma concentration, and clearance, ultimately influencing drug efficacy and safety. Microbial metabolism can alter drug structures before absorption, leading to reduced bioavailability or the generation ofinactive or toxic metabolites. For example, microbial inactivation of digoxin shortens its half-life and reduces its plasma concentration, potentially leading to therapeutic failure. Conversely, microbial β-glucuronidase activity may prolong drug exposure by recycling conjugated metabolites, as seen with irinotecan, thereby increasing toxicity [36].

The microbiome also affects drug clearance indirectly by modulating hepatic and renal metabolism through signaling pathways involving nuclear receptors like PXR and AhR. These changes can either accelerate or slow down drug metabolism and excretion, leading to fluctuations in therapeutic levels. Importantly, inter-individual variability in microbiota composition contributes to inconsistent drug responses across patients. Two individuals taking the same medication may experience different outcomes due to differences in microbial species, enzymatic capacity, or metabolite profiles. This variability has been observed with drugs like metformin, acetaminophen, and immune checkpoint inhibitors [37].

Overall, the microbiome is a key determinant of drug PK/PD profiles. Integrating microbiome profiling into therapeutic decision-making may enhance precision medicine by predicting responses and tailoring drug dosing to individual microbiota characteristics.

Table 2: Microbiome-drug interaction mechanisms and their clinical effects

Drugs	Mechanism of interaction	Description	Clinical implication
Paracetamol	Competition for metabolic cofactors	Microbes utilize same cofactors (e.g., sulfate, glucuronate) as host enzymes	Modifies host drug metabolism
Digoxin	Enzymatic biotransformation	Microbial enzymes modify drug structure (e.g., reduction, hydrolysis)	Altered drug activation/inactivation, toxicity
Sulfasalazine	Enzymatic biotransformation	Microbial enzymes modify drug structure (e.g., reduction, hydrolysis)	Altered drug activation/inactivation, toxicity
Irinotecan	Deconjugation of drug metabolites	Bacterial β-glucuronidases cleave glucuronide conjugates	Re-exposure to active/toxic forms
CYP3A4 substrates	Modulation of host enzyme expression	Microbiota influence expression of CYPs	Changes in drug metabolism and

Clinical consequences and safety concerns

Microbiome–drug interactions can have significant clinical implications, particularly in the areas of adverse drug reactions **(**ADRs**),** microbiome disruption, and the emergence of antibiotic resistance**.** Adverse drug reactions and toxicity can result from microbiota-mediated drug metabolism. For instance, the reactivation of irinotecan's glucuronide conjugates by microbial β-glucuronidases in the colon leads to severe gastrointestinal toxicity. Similarly, microbial inactivation of drugs like digoxin may reduce efficacy, prompting unnecessary dose escalation and increasing the risk of toxicity [38].

Microbiome disruption, or dysbiosis, is a frequent consequence of drug exposure, especially from antibiotics, but also from non-antibiotic drugs such as proton pump inhibitors, antipsychotics, and metformin. Dysbiosis can compromise gut barrier integrity, alter immune responses, and increase susceptibility to conditions like *Clostridioides difficile* infection (superinfection)**.** These disruptions may also interfere with the host's metabolic and immune functions, leading to long-term health consequences [39].

Furthermore, the indiscriminate use of antibiotics can promote the development and spread of antibiotic-resistant bacteria, diminishing treatment efficacy and increasing infection risk. However, the resilience of the microbiome—its ability to recover after perturbation—varies between individuals and is influenced by microbial diversity, host factors, and environmental exposures. These safety concerns highlight the need for integrating microbiome considerations into drug development, therapeutic monitoring, and pharmacovigilance. Strategies such as microbiome-preserving drugs, co-administration of probiotics, and personalized dosing regimens may help mitigate these risks and improve patient outcomes.

Microbiome-informed personalized medicine

The emerging field of microbiome-informed personalized medicinecomplements traditional pharmacogenomics by incorporating pharmacomicrobiomics—the study of how the microbiome influences drug responses. While pharmacogenomics focuses on host genetic variations affecting drug metabolism (e.g., CYP450 polymorphisms), pharmacomicrobiomics addresses inter-individual variability stemming from differences in microbial composition, enzymatic capacity, and metabolite production. Together, these approaches offer a more comprehensive understanding of personalized drug responses [41-42].

Predictive modeling and microbiome profiling are at the forefront of this integration. Advances in metagenomics, metabolomics, and machine learning have enabled the development of predictive algorithms that correlate microbial signatures with therapeutic efficacy, drug toxicity, and optimal dosing. For example, microbiome-based models can anticipate adverse reactions to chemotherapeutics or variability in response to antidiabetic drugs like metformin. Incorporating microbiome data into clinical decision-making holds promise for optimizing drug selection and individualizing treatment plans.

In addition to predictive diagnostics, microbiome-targeted interventions—such as probiotics**,** prebiotics**,** andfecal microbiota transplantation (FMT)—are being explored to modulate the gut microbiota and improve therapeutic outcomes. Probiotics and prebiotics can enhance beneficial microbial populations, potentially boosting drug efficacy or mitigating side effects. FMT, though still experimental in many contexts, has shown success in restoring microbiome balance in recurrent *Clostridioides difficile* infections and may have future applications in drug-response optimization [43].

As our understanding deepens, microbiome-informed strategies will increasingly shape precision medicine, guiding safer, more effective, and individualized pharmacotherapy.

Role of pharmacists in microbiome-drug interaction management

Pharmacists play a pivotal role in identifying, managing, and educating about microbiome–drug interactions, ensuring optimal therapeutic outcomes and patient safety in an evolving landscape of precision medicine.

Patient counseling and medication review are central to the pharmacist's responsibilities. Pharmacists can assess patient-specific factors such as recent antibiotic use, gastrointestinal symptoms, or dietary habits that may influence the gut microbiome. Through comprehensive medication reviews, they can identify drugs with high microbiome interaction potential (e.g., antibiotics, proton pump inhibitors, chemotherapeutics) and provide guidance on appropriate timing, co-administration of probiotics, or dietary modifications to preserve microbiota integrity and minimize adverse effects [44].

Interprofessional collaborationenhances the effectiveness of microbiome-aware care. Pharmacists can work closely with physicians, dietitians, and microbiologists to develop individualized treatment plans, especially for patients receiving complex therapies like immunosuppressants or chemotherapy. They can also contribute to decision-making in cases where fecal microbiota transplantation or microbiome profiling is considered, by evaluating drug–microbiome compatibility and implications for pharmacokinetics.

Additionally, pharmacists have expanding roles in education and research. By staying current with pharmacomicrobiomics advances, pharmacists can educate patients, healthcare teams, and students on microbiome impacts. In research settings, pharmacists can contribute to studies on microbiome-informed dosing, drug metabolism, and personalized interventions, helping to bridge the gap between bench and bedside.

Overall, pharmacists are well-positioned to lead efforts in integrating microbiome science into clinical pharmacy practice, contributing to safer, more effective, and personalized medication management.

Future perspectives and research directions

The intersection of microbiome science and pharmacology is rapidly evolving, driven by emerging tools and technologies that promise to transform drug development and individualized therapy. Techniques such as metagenomic sequencing**,** metabolomics, and **culturomics** now allow for comprehensive profiling of microbial communities and their functional capacities. Combined with artificial intelligence (AI**)** and machine learning, these data can be used to develop predictive models of drug response, identify novel microbial biomarkers, and optimize therapeutic regimens based on a patient's unique microbiome [45].

Despite these advances, several challenges in clinical translation remain. Microbiome composition is highly dynamic and influenced by numerous factors including diet, environment, and medications, complicating the standardization of results. Inter-individual variability and a lack of validated reference microbiomes limit the ability to generalize findings across populations. Additionally, integrating microbiome data into clinical workflows and electronic health records is still in its infancy.

Regulatory and ethical considerations are also critical. There is a need for clear guidelines from regulatory agencies on the use of microbiome-based diagnostics, therapeutics (e.g., fecal microbiota transplantation), and adjuncts like probiotics. Ethical concerns include data privacy, informed consent for microbiome profiling, and the potential for inequitable access to personalized microbiome-based treatments.

Moving forward, interdisciplinary collaboration, robust clinical trials, and the development of standardized protocols will be essential to fully realize the promise of microbiome-informed pharmacotherapy and integrate it responsibly into mainstream healthcare.

Conclusion

The human microbiome plays a profound role in influencing drug metabolism, efficacy, and safety, with growing evidence highlighting its impact on pharmacokinetics, pharmacodynamics, and inter-individual variability in drug responses. Key findings from recent research emphasize the microbiome's ability to directly metabolize drugs, modulate host enzymes, and alter absorption and bioavailability. Additionally, microbial dysbiosis can lead to adverse drug reactions, toxicity, and long-term health consequences, making it essential to consider microbiome dynamics in therapeutic management.

The implications for pharmacypractice are significant. Pharmacists are uniquely positioned to lead in identifying and managing microbiome–drug interactions, optimizing medication regimens, and advising on microbiome-preserving strategies such as probiotics, prebiotics, and careful antibiotic stewardship. They can also play a pivotal role in interprofessional collaborations to incorporate microbiome data into clinical decision-making and ensure personalized therapy. With their expertise in pharmacokinetics and patient care, pharmacists are key contributors to microbiome-informed healthcare.

There is a growing call for the integration of microbiome science into clinical decision-making. To fully harness the potential of microbiome-informed therapies, future research, the development of predictive models, and the establishment of regulatory frameworks are necessary. As our understanding of the microbiome's role in drug therapy continues to evolve, it is crucial for pharmacy practice to stay at the forefront, ensuring that personalized medicine becomes a reality for every patient.

Ethical Consideration

Data availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request. All data supporting the findings of this study have been included within the article and its supplementary materials, where applicable.

Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Compliance with ethical guidelines

This study was conducted in accordance with ethical standards as outlined in the Declaration of Helsinki and/or relevant institutional and national research committee guidelines. Ethical approval was obtained from the appropriate institutional review board, and informed consent was obtained from all individual participants included in the study.

Authors' contributions

All authors contributed significantly to the conception, design, execution, and/or interpretation of the research. Author SOA was responsible for the conceptualization, methodology, data collection, Author PJE handled data analysis and interpretation, and Author JIA contributed to the drafting and revising of the manuscript. All authors reviewed and approved the final version of the manuscript.

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