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REVIEW

Discovering a new paradigm: Gut microbiota as a central modulator of sexual health

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Abstract

The gut microbiota plays a pivotal role in human health, influencing diverse physiological processes, including those related to sexual health. Emerging evidence suggests a bidirectional relationship between the gut microbiota and sexual health, mediated by its impact on systemic inflammation, hormonal regulation, and immune function. A balanced gut microbiota supports optimal levels of sex hormones, such as estrogen and testosterone, which are critical for sexual function and reproductive health. Additionally, gut-derived metabolites such as short-chain fatty acids contribute to maintaining mucosal barrier integrity and regulating immune responses, which are essential for protecting against

infections that may impair sexual health. Conversely, dysbiosis, an imbalance in gut microbial composition, has been linked to conditions such as erectile dysfunction, polycystic ovary syndrome, and reduced libido, emphasizing its role in sexual dysfunction. Lifestyle factors, including diet, stress, and antibiotic use, can modulate the gut microbiota and, consequently, sexual health outcomes. Recent therapeutic approaches, such as probiotics, prebiotics, and fecal microbiota transplantation, offer potential for restoring gut balance and improving sexual health. This review highlights the central role of the gut microbiota in sexual health, emphasizing its importance as a target for therapeutic interventions to enhance overall well-being.

Key Words: Gut microbiota; Sexual health; Mental health; Dysbiosis; Gut brain axis; Short-chain fatty acids; Neurotransmitters; Hormonal regulation; Sexual performance anxiety; Diet; Psychobiotics; Psychological therapies

Core Tip: The gut microbiota is crucial for sexual health, influencing hormonal balance, immune function, and the gut-brain axis. A balanced microbiota supports optimal levels of estrogen and testosterone, while dysbiosis contributes to erectile dysfunction, polycystic ovary syndrome and decreased libido. Microbial metabolites, such as short-chain fatty acids, influence neurotransmitters and mood, affecting sexual desire and function. Therapeutic approaches, including probiotics, prebiotics, and fecal microbiota transplantation, show promise in restoring gut balance and improving sexual health. Recognizing this interplay offers new strategies for managing sexual dysfunction and enhancing overall well-being.

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INTRODUCTION

The human body hosts trillions of microbial cells, primarily located in the gastrointestinal tract, where they collectively form the gut microbiota. This complex and dynamic ecological community plays a vital role in regulating physiological functions and influencing susceptibility to disease through its metabolic activities and interactions with the host organism [1]. The gut microbiota comprises up to 100 trillion microorganisms, including bacteria, archaea, fungi, protozoa, and viruses, located within the intestinal barrier, represents the body's main ecosystem and could even be considered a "separate organ"[2]. This microbial community outnumbers the body's own cells by a factor of 10 and contains a collective genetic repertoire called "microbiome" that is at least 150 times larger than the human genome[3].

Bacterial genera such as *Enterococcus*, *Ruminococcus*, *Bacteroides*, *Bifidobacterium*, and *Akkermansia muciniphila* contribute to immune regulation and essential metabolic processes that support overall well-being. However, other species, such as *Clostridioides difficile*, *Salmonella*, *Helicobacter pylori*, *Escherichia coli O157*, *Bacteroides fragilis*, and *Fusobacterium nucleatum*, are associated with diseases ranging from infections and intestinal inflammation to tumor development[4].

Recent years have seen exponential growth in scientific interest surrounding the microbiota, fueled by discoveries of its impact not only on systemic health but also on sexual health. This relationship extends far beyond physical aspects, deeply influencing the emotional and psychological dimensions of sexuality. Sexual health, defined as a state of complete physical, mental, and social well-being in relation to sexuality, is now understood as a multifaceted component of overall health that interacts with immune, endocrine, and neuropsychological systems[5].

Emerging evidence suggests a bidirectional connection between gut microbiota and sexual health, mediated by its effects on systemic inflammation, hormonal regulation, and immune function[6]. Maintaining a balanced gut microbiota is essential for regulating sex hormones like estrogen and testosterone, which are fundamental for sexual performance and reproductive health. Gut-produced metabolites, such as short-chain fatty acids (SCFAs), also play a vital role in preserving the mucosal barrier and immune system balance, reducing the risk of infections that could affect sexual well-being[7].

The interplay between gut microbiota and sexual health also includes interactions with the central nervous system (CNS). Known as the gut-brain axis, this complex communication pathway influences sexual behavior by modulating neurological and neurochemical processes tied to mood, desire, and sexual response[8]. Recent findings have also linked microbial alterations to changes in neurotransmitter levels, such as serotonin and dopamine, which are known to affect emotional states and sexual motivation[9]. Therefore, the microbiota serves as a crucial mediator between the body and the mind, shaping not only physiological regulation but also emotional and relational well-being.

The composition of the gut microbiota is shaped by various factors, including[10], antibiotics[11], stress[12], age[13], and physical activity[14]. A balanced, fiber-rich diet promotes the growth of beneficial bacteria, whereas antibiotic use and exposure to stress can disrupt microbial balance, leading to alterations in gut microbial composition. Microbial

diversity is further influenced by age and genetic predisposition over the course of life, while regular physical activity has been linked to enhanced gut microbial biodiversity.

Dysbiosis is closely associated with sexual health conditions, including erectile dysfunction (ED), polycystic ovary syndrome (PCOS) and reduced libido. It impacts sexual function through its effects on systemic inflammation, hormonal regulation, and immune system function.

In ED, dysbiosis aggravates endothelial dysfunction by increasing inflammation and impairing nitric oxide production, which is essential for blood flow[15]. In PCOS, it exacerbates insulin resistance and hormonal imbalances, such as elevated androgen levels, while contributing to chronic inflammation that worsens symptoms and fertility[16]. Dysbiosis also affects libido through the gut-brain axis by altering mood, stress, and sex hormone metabolism, such as testosterone and estrogen, further impairing sexual desire and function[17]. These findings point suggest that the gut microbiota is a novel therapeutic target for addressing sexual dysfunction and improving reproductive and relational health.

This intricate connection between microbiota and sexuality highlights the need for therapeutic interventions to restore microbial balance and enhance sexual health. Emerging strategies, such as probiotics, prebiotics, and fecal microbiota transplantation, offer promising avenues for improving both systemic and sexual well-being[18]. A deeper understanding of this axis could facilitate the development of innovative, personalized treatments that integrate biological and psychological care. Moreover, understanding these dynamics provides new perspectives on the multifaceted nature of sexuality, encompassing physical, emotional, and psychological factors. As the scientific community continues to explore the microbiota's systemic roles, its influence on sexuality emerges as a critical frontier with implications for holistic healthcare. By deepening our knowledge of the gut microbiota's influence on sexual health and its role as a mediator in the gut-brain axis, researchers and clinicians can develop personalized interventions aimed at improving overall health and quality of life. This holistic approach underscores the centrality of the gut microbiota in bridging connections between physical well-being, mental health, and sexual behavior.

HUMAN MICROBIOTA AND VAGINAL MICROBIOME: COMPOSITION, DIVERSITY, AND HEALTH IMPLICATIONS

The human microbiota is a diverse community of microorganisms that colonizes both internal and external surfaces of the human body. The collective genetic material of these microorganisms constitutes the human microbiome. Primarily symbiotic, the microbiota derives benefit from its host. These host-microbe interactions can be neutral (commensalism), beneficial (mutualism), or, under certain circumstances, detrimental (pathogenicity), impacting physiological processes, immune responses, and nutrient acquisition[19]. While the microbiome begins to establish itself at birth, it remains dynamic, with its composition subject to modulation by various exogenous factors, including antibiotic administration.

The human vaginal microbiota (VMB) constitutes a complex and dynamic ecosystem of beneficial microorganisms and opportunistic pathogens that coexist within the vaginal environment[20,21]. Within this ecosystem, a cooperative relationship between the host and its microbial inhabitants provides a primary defense against the proliferation of opportunistic pathogens. This stable, health-promoting microbial equilibrium is termed eubiosis. Conversely, when opportunistic pathogens overwhelm beneficial bacteria, this balance is disrupted, resulting in dysbiosis - a state frequently associated with inflammation and heightened vulnerability to infections. The interplay between the VMB and female reproductive physiology is bidirectional: physiological changes across the lifespan, from birth through post-menopause, influence the VMB, while the VMB itself can in turn affect reproductive health[22].

COMPOSITION AND CLASSIFICATION OF THE VMB

The composition and structure of the VMB have been extensively characterized, utilizing methods ranging from traditional microscopy to advanced high-throughput sequencing technologies[19]. Historically, the VMB of healthy, reproductive-age women has been characterized by *Lactobacillus* dominance, attributed to the production of lactic acid, which maintains a vaginal pH below 4.5[23]. However, the advent of molecular techniques has facilitated the identification of previously undetectable bacterial species, leading to the classification of distinct microbial community state types (CSTs).

Five major microbial CSTs have been identified among reproductive-age women based on the relative abundance and composition of vaginal bacterial species[21,24]. Specifically, CST-I is characterized by the dominance of *Lactobacillus crispatus*, CST-II by *L. gasseri*, CST-III by *L. iners*, and CST-V by *L. jensenii*. In contrast, CST-IV exhibits a diverse collection of facultative anaerobic bacteria alongside reduced *Lactobacillus* levels. CST-IV has been further categorized into two subtypes: CST IV-A, which encompasses species from the genera *Anaerococcus*, *Peptoniphilus*, *Corynebacterium*, *Prevotella*, *Finegoldia*, and *Streptococcus*; and CST IV-B, which includes *Atopobium*, *Gardnerella*, *Sneathia*, *Mobiluncus*, *Megasphaera*, and other taxa within the *Clostridiales order*[21,24].

CST-IV has been strongly associated with vaginal dysbiosis, particularly bacterial vaginosis (BV), according to the Nugent scoring system. However, research has demonstrated the prevalence of CST-IV in asymptomatic individuals, notably among Black and Hispanic women, where it constitutes approximately 40% of cases[24]. This observation raises the question of whether CST-IV should be categorized as a dysbiotic condition or an asymptomatic variation of BV, highlighting the need for a more precise differentiation between a genuinely healthy state and an asymptomatic microbial profile.

DISCOVERY OF NEW VAGINAL BACTERIAL SPECIES

Recent research has identified a novel bacterial genus, *Vaginella massiliensis*, isolated from vaginal samples of healthy women. This rod-shaped, non-motile, non-spore-forming, obligate aerobe, Gram-negative bacterium belongs to the *Flavobacteriaceae* family within the *Bacteroidetes* phylum. The type strain of this genus is Marseille P2517[25]. The discovery of *Vaginella massiliensis* further emphasizes the complexity and diversity of the vaginal microbiome, highlighting the potential role of previously unrecognized bacterial species in vaginal health.

FACTORS CONTRIBUTING TO *LACTOBACILLUS* DOMINANCE IN A HEALTHY VMB

The predominance of *Lactobacillus* species in a balanced VMB is largely attributed to their capacity to produce antimicrobial compounds[26]. These include lactic acid, which contributes to maintaining an acidic vaginal environment that inhibits the proliferation of pathogenic bacteria; bacteriocins, narrow-spectrum antimicrobial peptides that selectively target harmful bacteria; and hydrogen peroxide (H_2O_2), which plays a crucial role in host defense against infections. These antimicrobial substances collectively contribute to the stability and resilience of the VMB, underscoring the importance of *Lactobacillus* dominance in maintaining vaginal health. This dominance helps prevent vaginal infections, can reduce the risk of sexually transmitted infections, and promotes better reproductive health and response to gynecological treatments [27]. This balance is essential for vaginal ecosystem stability and resilience, reinforcing the importance of an adequate presence of *Lactobacillus* in maintaining sexual health and protecting against infections[28,29]. An imbalance and decrease of *Lactobacillus* species can compromise these protective mechanisms and increase the risk of infections and other complications[30].

Lactic acid

Lactobacilli produce lactic acid through the fermentation of carbohydrates in the vaginal epithelium of women of reproductive age, primarily glycogen. This acidic milieu provides a protective barrier against infection, inhibiting the colonization of potential vaginal pathogens. Studies have demonstrated that *Lactobacillus* abundance contributes to vaginal acidification, resulting in an average pH of 3.5 ± 0.2, largely due to lactic acid accumulation[31]. The protective levels of vaginal lactic acid are primarily dependent on the VMB, as the host's epithelial cells contribute only 4%-30% of the total lactic acid produced[32].

Lactic acid within the vagina exists in two isomeric forms, D (–) and L (+). It has been proposed that glycogen availability in the vaginal lumen increases due to the exfoliation of glycogen-rich epithelial cells, a process mediated by hyaluronidase-1 and matrix metalloproteinase activity. This, in turn, leads to the degradation of available glycogen by α-amylase, subsequently converted into D (–) lactic acid by *Lactobacilli*.

Recent studies suggest that lactic acid, particularly the L (+) isomer, can inhibit Human immunodeficiency virus (HIV) 1 infection independently of pH reduction[33]. Furthermore, both D (–) and L (+) lactic acid stimulate an anti-inflammatory response in human cervicovaginal epithelial cells against HIV[34]. Both isomers have been shown to inhibit histone deacetylases, thereby enhancing DNA repair through transcriptional regulation of related genes[35].

Lactic acid has also been shown to inhibit a range of infections, including *Chlamydia trachomatis*, herpes simplex virus type 2, HIV-1, and various BV-associated microorganisms[33,36-38]. Lactic acid modulates the host's immune response through various mechanisms. It increases production of the anti-inflammatory mediator interleukin (IL)-1 receptor antagonist in vaginal epithelial cells, while inhibiting pro-inflammatory mediators such as IL-6, IL-8, tumor necrosis factor α, regulated upon activation, normal T cell expressed and secreted (RANTES/CCL5), and macrophage inflammatory protein-3[34]. It also induces the release of transforming growth factor β, which stimulates antiviral responses[39]; activates the Th17 lymphocyte pathway *via* IL-23 production in response to bacterial lipopolysaccharides (LPSs)[40]; and increases cytosolic lactic acid, which inhibits cAMP production, thereby promoting autophagy in epithelial cells for the degradation of intracellular microbes and the maintenance of homeostasis[41]. These studies collectively demonstrate the diverse defensive properties of lactic acid, which vary depending on its isomeric form, and which individually or synergistically influence host susceptibility and host-microbiota interactions.

Bacteriocins

Bacteriocins, such as *Bacteriocin IIa*, *IIc*, J46, *Acidocin LF221A*, *Gassericin T*, and *type-A Lantibiotic*, are proteinaceous substances with bactericidal activity. They are produced by *Lactobacilli*, particularly *L. crispatus* and *L. gasseri*[42]. These molecules disrupt the cell membrane of non-indigenous pathogenic organisms, such as *S. aureus*, *Klebsiella spp.*, *E. faecalis*, and *E. coli*, playing a critical role in preventing their growth[42].

H_2O_2

H_2O_2 is another antimicrobial compound produced *in vitro* by many *Lactobacillus* species in the presence of oxygen (O_2) [43]. However, the low O_2 levels within the vagina create an anaerobic environment, raising questions regarding whether H_2O_2 can reach concentrations sufficient to exert toxicity against vaginal pathogens. This notion was further supported by O'Hanlon *et al*[36], who demonstrated that under low O_2 conditions characteristic of the vagina, H_2O_2 does not significantly affect BV-associated bacteria. Furthermore, they found that high H_2O_2 levels were more detrimental to the *Lactobacilli* responsible for maintaining eubiosis than to the bacteria associated with dysbiosis[36].

Conversely, earlier studies suggested that hydrogen peroxide-producing vaginal *Lactobacilli* confer greater protection against BV and inhibit *Candida* growth, preventing invasive hyphal formation[44]. Collectively, these findings suggest that lactic acid and bacteriocin production constitute the primary protective mechanisms of *Lactobacilli* within the VMB, while the role of H_2O_2 in VMB protection remains debated, potentially serving as a marker for other yet identified physiological factors.

CANDIDA AND ITS ROLE IN THE VMB

The VMB of healthy, reproductive-age women includes a fungal component, commonly termed the vaginal mycobiota, and its corresponding genomic profile is known as the vaginal mycobiome[45]. Early culture-based studies detected vaginal fungi in approximately 20% of asymptomatic women, with *Candida albicans* as the predominant species (72%-91%), followed by non-*albicans Candida* species such as *C. glabrata*, *C. tropicalis*, and *C. parapsilosis*[46]. However, the full complexity of the vaginal mycobiota has been underappreciated due to limitations of these conventional techniques.

Recent advancements in high-throughput sequencing have provided deeper insights into the diversity of vaginal fungal communities. A landmark study published in 2013 utilized pyrosequencing to analyze the vaginal mycobiota of asymptomatic women in Estonia, revealing *Candida* species in 64.5% of participants, a significantly higher prevalence than the 20% previously reported using culture-based methods[45,46]. Consistent with prior research, *C. albicans* was the most prevalent species (82%), while non-*albicans Candida* species, including *C. dubliniensis*, *C. parapsilosis*, *C. krusei*, and an unclassified strain (*Candida* sp. VI04616), were also detected. Notably, 38% of the operational taxonomic units identified in this study lacked proper taxonomic classification, highlighting the challenges inherent to molecular-based fungal taxonomy[45].

While recent studies have revealed a more diverse vaginal mycobiota than previously recognized, *C. albicans* remains the dominant species, underscoring its ecological significance[45,47]. Despite its common presence in healthy women, *Candida* is still classified as an opportunistic pathogen due to its high prevalence (85%-95%) in cases of vulvovaginal candidiasis (VVC), the second most common vaginal dysbiosis after BV[48]. This raises the critical question of whether the presence of *Candida* contributes to a stable microbiota (eubiosis) or predisposes the host to dysbiosis and disease. The answer may reside in *Candida*'s capacity for dimorphic transition, a key factor in its dual lifestyle. This transition allows *Candida* to switch between a yeast form and a filamentous hyphal form, enabling it to exist as both a harmless commensal and a virulent pathogen. The yeast form is typically observed in healthy, asymptomatic women, whereas the hyphal form is strongly associated with severe VVC, reinforcing the link between yeast morphology and commensalism and between hyphal growth and pathogenicity[49].

Multiple factors influence *Candida*'s ability to persist as a commensal organism, with nutrition playing a crucial role. Studies indicate that different carbon sources affect *Candida*'s cell wall structure, which, in turn, influences its virulence and interactions with the host immune system[50,51]. This fungus exhibits remarkable metabolic flexibility, utilizing diverse carbon sources, including glycogen, its metabolic byproducts, and even lactic acid, allowing it to adapt to fluctuations within the vaginal environment[52].

A recent study suggests that when *Candida* utilizes lactate as its sole carbon source, it modulates immune responses by reducing phagocytosis, increasing IL-10 production and decreasing IL-17 levels[51]. This modulation decreases its susceptibility to immune attack, facilitating persistence within the VMB without eliciting robust inflammatory responses [51]. Further studies have corroborated this observation, suggesting that *Candida* actively suppresses immune responses to promote long-term commensalism[53,54].

VMB in recurrent vulvovaginal infections

Disruptions in the VMB can lead to dysbiosis, a state where competing microbial communities favor those adapted to less favorable conditions. This process may result in a decrease in beneficial *Lactobacillus* species, increasing host susceptibility to opportunistic pathogens, including those naturally present at low levels in the vaginal environment and those that are sexually acquired. Persistent imbalance can exacerbate dysbiosis, potentially leading to chronic inflammation and persistent vaginal infections, such as vaginitis or other vulvovaginal infections. These infections are typically classified according to the dominant microbial community or the responsible pathogen and contribute to symptoms such as excessive vaginal discharge (with or without malodor), vulvar pruritus, fissures, pain, erythema, edema, dysuria, dyspareunia, and skin lesions. Of these, abnormal vaginal discharge is the most common sign of infection[55-58].

The three most common types of vulvovaginal infections are BV, VVC, and trichomoniasis[55-58]. Recurrent vulvovaginal infections, characterized by repeated episodes of these conditions, are an increasing area of research interest. For example, BV is considered recurrent when recurrence rates reach 30%-50% within three months, while recurrent VVC is diagnosed after at least four episodes within a 12-month period[59]. Similarly, recurrent trichomoniasis has been reported, with recurrence rates between 5%-8% within two months of initial diagnosis[60].

BV-VMB composition

BV is a dysbiotic condition characterized by a decrease in lactic acid-producing bacteria and a concomitant increase in anaerobic bacterial diversity. This diverse community includes species from genera such as *Anaerococcus*, *Atopobium*, *Bacteroides*, as well as BV-associated bacteria (*BVAB1*, *BVAB2*, and *BVAB3*), and species such as *Gardnerella*, *Leptotrichia*, *Mobiluncus*, *Mycoplasma*, *Peptostreptococcus*, *Peptoniphilus*, *Prevotella*, and *Sneathia*.

Advances in molecular techniques have continually expanded the list of potential BV-associated agents, including unculturable microorganisms, a limitation of older culture-based methods[61]. For example, three novel strains - *Olegusella massiliensis* (strain KHD7T), *Ezakiella massiliensis* (strain Marseille P2951T), and *Corynebacterium fournierii* (strain Marseille P2948T) - have recently been isolated from the VMB of a woman with BV[62-64]. Furthermore, women with BV have decreased levels of both lactic acid isomers, resulting in a vaginal pH over 4.5[65].

VVC

VVC is a dysbiotic condition characterized by *Candida* species overgrowth, with the transition to a mycelial form being a critical early step in its pathogenesis[66]. Symptoms such as vulvar pruritus, burning, and thick, white vaginal discharge resembling cottage cheese can significantly impact quality of life, often leading to discomfort in social and intimate relationships[67]. Furthermore, VVC has been associated with an increased risk of reproductive and obstetric complications, including intra-amniotic infections, preterm birth, and low birth weight in newborns[68].

Similar to BV, VVC pathogenesis involves three primary stages: Adherence to epithelial cells, invasion, and biofilm formation, followed by the secretion of virulence factors. Two key proteins, Als3 and Ssa1, function in the "invasion" process[69,70].

Studies have shown that environmental factors, such as nutrient depletion, neutral pH, a temperature of 37 °C, and low *Candida* cell densities (< 10^7 cells/mL), collectively influence *Candida* gene expression, triggering hyphal formation. This then facilitates adherence to vaginal epithelial cells and further progression of infection[71]. These genes encode proteins including agglutinin-like sequence protein (Als3), secreted aspartic proteases (Sap 4 to 6), hyphal wall protein, and hypha-associated proteins (Hgc1, Ece1, and Hyr1)[72]. Among these, Als3 and hyphal wall protein function as "adhesins", promoting *C. albicans* attachment to host epithelial cells[73]. Following adhesion, *Candida* invades host cells, initiating further pathogenic processes.

The primary treatment for VVC remains antifungal medications, although their effectiveness is increasingly challenged by high recurrence rates and the emergence of drug-resistant *Candida* strains[74]. Probiotics are among the most extensively studied alternative therapies and may offer improved outcomes when used in conjunction with prebiotics or traditional antifungal treatments. Their immunomodulatory, antibiofilm, and antifungal properties, coupled with their capacity to restore a healthy vaginal microbiome, make them promising candidates for VVC management[74]. VMB transplantation also holds considerable promise for restoring balance to the vaginal ecosystem. However, research in this area remains in its early stages, and the limited availability of clinical and *in vivo* data restricts its current application as a standard treatment[74].

GUT MICROBIOTA

The gut microbiota exerts diverse effects on the intestinal environment, influencing distant organs and systems, and is now recognized as a functional endocrine organ. It plays a pivotal role in the female endocrine reproductive system across the lifespan, interacting with hormones such as estrogens, androgens, and insulin, among others. Dysbiosis is associated with numerous diseases and conditions, including pregnancy complications, PCOS, endometriosis, and cancer. However, the mechanisms underlying these interactions remain incompletely understood.

It is widely recognized that the number of microorganisms in the human body is roughly equivalent to the number of human cells, with the genetic content of these microbes exceeding the human genome by at least 150-fold[75]. A growing body of evidence suggests that the microbiome functions as a supplementary organ, actively contributing to the regulation and maintenance of physiological processes. Numerous host and environmental factors, including diet, host genetics, and hormonal regulation, influence the microbiome. Sex hormones, such as estradiol, progesterone, and testosterone, play key roles in host-microbiota communication, affecting critical physiological functions including reproduction, cellular differentiation, apoptosis, inflammation, metabolism, and brain function[76]. In women, the microbiome influences every phase of reproduction, encompassing follicle maturation, oocyte development, fertilization, embryo migration, implantation, pregnancy, and childbirth. Notably, linear relationships have been established between the gut microbiota and serum hormone levels, giving rise to the emerging concept of the "microgenderome"[77].

Estrogen and gut microbiota interaction

The relationship between estrogens and the gut microbiota is bidirectional: Estrogens influence the microbiota, and the microbiota actively modulates estrogen levels. Estrogens play a crucial role in regulating gut microbiota composition, with the microbial group capable of metabolizing estrogens often referred to as the "estrobolome"[78]. Estrogen receptor β expression and serum steroid hormone concentrations, particularly estradiol, fluctuate throughout life, emphasizing the importance of estrogen regulation for overall female health. The gut microbiota contributes to estrogen metabolism; for example, antibiotic use has been shown to decrease estrogen levels[79].

Gut microbes secrete β-glucuronidase, an enzyme that converts conjugated estrogens into their active deconjugated forms[78]. Dysbiosis and reduced gut microbiota diversity decrease β-glucuronidase activity, leading to decreased estrogen and phytoestrogen deconjugation, thus reducing their circulating active forms. Lower circulating estrogen levels can disrupt estrogen receptor activation, potentially contributing to hypoestrogenic conditions such as obesity, metabolic syndrome, cardiovascular disease, and cognitive decline[78,80]. Conversely, an overabundance of β-glucuronidase-producing bacteria can increase circulating estrogen levels, potentially promoting conditions such as endometriosis and certain cancers. Additionally, estrogen levels are implicated in the pathogenesis of diseases like PCOS, endometrial hyperplasia, and fertility disorders[81].

Research has demonstrated that specific bacterial orders, such as *Lactobacillales*, and phyla including *Proteobacteria*, *Bacteroidetes*, and *Firmicutes*, vary depending on estrogen receptor β status in mice, suggesting that steroid receptor activity and dietary composition may influence gut microbiota diversity[82]. The negative correlation between gut microbiota alpha diversity and estradiol concentrations warrants further investigation. It is likely that microbiota composition participates in sex hormone regulation, while, conversely, sex hormones may influence microbial diversity [83].

Recent studies have also highlighted the role of the gut microbiota in mediating the protective effects of 17β-estradiol against metabolic endotoxemia and chronic low-grade inflammation. In experiments, male mice treated with 17β-estradiol and ovariectomized female mice exhibited reduced levels of *Proteobacteria* and LPS biosynthesis, consistent with results observed in normal female mice. Estrogen or estrogen-like compounds can reduce LPS production and intestinal permeability, thus decreasing the risk of metabolic endotoxemia[84]. Furthermore, estrogen can enhance intestinal epithelial barrier integrity, with females exhibiting greater resistance to intestinal damage compared to males[85].

Estrogens are also associated with several hormone-dependent cancers, including those of the endometrium, cervix, ovary, prostate, and breast. Alterations in gut microbiota composition have been observed in many of these cancers, suggesting a potential role for the microbiota in cancer initiation and progression[86]. For instance, it has been proposed that high-fat diet-associated steroid hormones may influence the gut microbiome, introducing carcinogens that could potentially affect breast tissue or estrogen metabolism, contributing to tumor growth.

The gut microbiota influences various aspects of female health, including fertility, obesity, diabetes, and cancer. Estrogens not only regulate the gut microbiota but also modulate estrogen metabolism and levels, with significant implications for various pathological conditions. For example, decreased ratios of estrogen metabolites to parent compounds, along with reduced fecal microbiota diversity, have been linked to an increased risk of breast cancer in postmenopausal women[87].

In postmenopausal women, increased gut microbiota diversity positively correlates with a higher ratio of estrogen metabolites in urine[88]. Furthermore, total fat mass and abdominal fat, key factors in the development of insulin resistance and type 2 diabetes, are elevated in postmenopausal women compared to premenopausal women[89-91]. This suggests that the gut microbiota may play an essential role in regulating estrogen levels and metabolism during menopause.

Furthermore, the gut microbiota can metabolize estrogen-like compounds from foods, such as soy isoflavones, which, in turn, promotes the growth of beneficial bacteria[92]. Soy isoflavone supplementation can increase *Bifidobacterium* concentrations and suppress the growth of unclassified *Clostridiaceae* in postmenopausal women[92,93]. In this context, *Bifidobacterium* enhances nutrient absorption, supports immune function, and prevents infections, while *Clostridiaceae* is associated with inflammation and obesity. These findings suggest that the complex interactions between the host, microbiota, and estrogens influence a wide range of pathways impacting female health and disease.

Estrogens and the gut microbiota may work synergistically to shape various aspects of women's health, including fertility, obesity, diabetes, and cancer. A deeper understanding of the interactions between estrogens and the gut microbiota will provide valuable insights, potentially leading to novel strategies for reducing the risk of endocrine diseases in women.

INTESTINAL MICROBIOTA, FEMALE REPRODUCTIVE TRACT, AND EMBRYO DEVELOPMENT

The intestinal microbiota and its metabolites significantly influence various stages of embryonic development, from gamete formation to processes such as fertilization, implantation, placentation, miscarriage, and birth. Numerous studies have demonstrated the presence of gut bacteria in the female reproductive system[94-96]. Women experiencing preterm labor often exhibit lower levels of *Lactobacillus crispatus* and higher levels of *Sneathia amnii* in their VMB. These shifts are associated with increased pro-inflammatory cytokines in vaginal fluid[97]. A meta-analysis of five independent studies encompassing 3201 samples revealed substantial differences in microbiota composition between women that delivered prematurely and those that carried to term. Furthermore, the maternal vaginal microbiome can influence the neonatal gut microbiome. Neonates born *via* cesarean section exhibit significantly reduced microbial diversity and altered microbiota compared to those born vaginally[98].

Recent molecular analyses have confirmed that the uterine cavity is not sterile, demonstrating that endometrial communities are derived from specific macroflora, with notable associations to gastrointestinal microbiota, particularly the *Bacteroidetes* and *Proteobacteria* phyla[99]. While some researchers suggest that uterine microbiota may represent transient or invading bacteria rather than a stable population, its potential contribution to uterine health and homeostasis remains a topic of debate[100]. Among women undergoing *in vitro* fertilization, the presence of certain bacterial species within the uterine cavity has been linked to lower implantation and pregnancy success rates[101]. Specifically, an endometrial microbiota not dominated by *Lactobacillus* is associated with decreased rates of implantation, pregnancy, and live birth, although the specific mechanisms by which bacteria interfere with embryo implantation are not yet fully understood. The intestinal microbiota and its metabolites can also influence endometrial and uterine immunity during implantation and placentation, acting as mediators in local microbiota interactions[102]

For instance, Th1 cells are a significant component of the immune system within the human endometrium, and a balanced Th1/Th2 ratio is essential for proper implantation. Dysbiosis of the intestinal microbiota can provide immune-stimulatory signals that activate both innate and adaptive immune responses. In mice with altered gut microbiota, the differentiation of various T cell subtypes is impaired[103,104]. Given that T cells predominantly reside in the deeper layers of the endometrium, they are thought to play an important role in early placenta formation after implantation.

Placental integrity and function are crucial for fetal growth and development. One study demonstrated that the placenta harbors a metabolite-rich microbiota, and variations in the placental microbiota have been linked to a history of antenatal infections, potentially influencing intrauterine infections and preterm birth[96]. Recent research has found that *Escherichia* species are abundant in meconium and are a significant contributor to early-onset sepsis in neonates with extremely low birth weight, with the placenta acting as a likely source of *Escherichia* in neonatal meconium[105].

Disruptions in the gut microbiota can also lead to ovarian dysfunction, including impaired oocyte development and abnormal ovulation. Studies in *Drosophila* have shown that changes in gut bacteria can affect the germline. The absence of certain gut species, such as *Acetobacter*, has been found to repress oogenesis and alter phenotypic traits[106]. Antibiotic-induced dysbiosis in mice leads to changes in estrous cycles, with reductions in oocyte area and increased zona pellucida thickness in dysbiotic groups[107]. Fecal microbiota transplants from women with ovarian disorders to mice result in ovarian dysfunction and impaired fertility[108], whereas treatment with *Lactobacillus* or fecal microbiota from healthy rats restores estrous cycles and normalizes ovarian morphology[109]. Additionally, obesity-related dysbiosis plays a significant role in ovarian dysfunction associated with metabolic syndrome[110]. Obesity negatively impacts ovarian function and oocyte quality, but bariatric surgery in obese women has been shown to normalize ovulatory patterns, improve fertility, and enhance fetal health. These improvements may be attributed to alterations in the gut microbiome [111].

The microbiota in follicular fluid has been shown to influence *in vitro* fertilization outcomes. Specifically, certain microorganisms, particularly *Lactobacillus spp*., are associated with better embryo quality and higher rates of embryo transfer and pregnancy success[112]. Recent studies suggest that lower levels of trimethylamine N-oxide and its intestinal precursor, gamma-butyrobetaine, in follicular fluid are associated with the development of high-quality embryos, indicating that microbiota-dependent metabolites may serve as valuable biomarkers for predicting embryonic development[113]. In conclusion, the intestinal microbiota is closely linked to the health of the female reproductive system. Future research should focus on identifying specific microbiota-related factors that can be targeted therapeutically to enhance fertility in women with reproductive disorders.

SCFAS AND THE GUT-BRAIN-SEX CONNECTION: A NEUROBIOLOGICAL PERSPECTIVE

The gut-brain axis has emerged as a key regulator of mood and overall well-being, with direct implications for sexual health. The gut microbiota plays a fundamental role in host physiology, influencing neurochemical and endocrine processes that modulate behavior and emotional responses. Dysbiosis can impair this bidirectional communication, contributing to mood disorders and affecting key aspects of sexuality, including motivation and function[114].

A key element in understanding this relationship lies in the fermentation of indigestible fibers by gut bacteria, which leads to the production of SCFAs such as butyrate, acetate, and propionate (Table 1). These SCFAs serve multiple functions within the host, ranging from maintaining gut integrity and modulating the immune system to exerting neuroactive properties essential for brain health[115]. Their role in the gut-brain axis is primarily mediated through two key mechanisms: First, SCFAs act as ligands for specific G-protein-coupled receptors critical for neural communication[116]; second, SCFAs modulate histone deacetylases, which are vital regulators of brain development, neuroplasticity, and neuropsychiatric disorders[116].

Because SCFAs are primarily synthesized in the colon, their ability to cross the blood-brain barrier (BBB) has been a subject of research. Specific transporters in intestinal and endothelial cells facilitate SCFA absorption, allowing these metabolites to reach the brain, where they contribute to BBB integrity, regulate neurotransmitter levels, and influence neuronal activity[117]. Given their role in neurotransmitter homeostasis, SCFAs directly impact mood-related neurochemistry, modulating levels of glutamate, glutamine, gamma-aminobutyric acid (GABA), serotonin, dopamine, norepinephrine, and epinephrine[117]. These neurotransmitters are intimately linked to sexual behavior, affecting desire, arousal, and reward processing. Consequently, alterations in SCFAs production can lead to impaired sexual motivation, pleasure, and overall satisfaction.

The gut microbiota also plays a crucial role in microglial cell homeostasis, which in turn influences synaptic pruning and neuroimmune regulation. Microglia serves as the primary immune cells of the CNS, actively regulating neural connectivity and synaptic function[117]. Dysregulation of microglial activity due to SCFA imbalance may contribute to emotional and social dysfunction, further influencing the individual's sexual experiences. In addition to their effects on neuroinflammation and microglial homeostasis, SCFAs also influence neuroendocrine pathways, particularly through the hypothalamic-pituitary-adrenal (HPA) axis, a central regulator of the body's stress response. Studies have shown that SCFA administration can attenuate cortisol responses to acute psychosocial stress, indicating that SCFAs contribute to the bidirectional communication between the gut and the brain, impacting mood regulation and emotional resilience[118]. While the connection between SCFAs, microglia, and sexual health remains an emerging area of research, it is evident that gut microbial composition significantly impacts neuropsychiatric and behavioral outcomes, thereby affecting various aspects of human sexuality. Beyond their role in SCFA production, some gut bacteria are capable of directly synthesizing neurotransmitters, influencing host physiology and behavior. Notably, *Bifidobacterium spp.* and *Lactobacillus spp.* produce GABA, an inhibitory neurotransmitter involved in stress regulation, whereas *Escherichia spp*., *Streptococcus spp*., *Candida spp*., and *Bacillus spp.* contribute to serotonin and dopamine biosynthesis[119]. These microbial-derived neurotransmitters play a fundamental role in pleasure, motivation, and sexual reward pathways. Although their direct impact on the brain is limited due to the selective permeability of the BBB, they exert indirect effects *via* interactions with the enteric nervous system and vagus nerve signaling. Moreover, certain bacterial strains, such as *Bifidobacterium infantis*, enhance circulating tryptophan levels, leading to increased serotonin availability in the brain, which is associated with improvements in

Table 1 Short-chain fatty acids: Composition, absorption, and circulatory levels

Short-chain fatty acid	Relative abundance (%)	Fecal concentration (g/kg)	Main absorption route	Plasma concentration (μM)	Biological function
Acetate	About 60%	About 60 g/kg	Absorbed *via* MCTs in the colon; partially crosses the BBB	100-200	Primary energy source for peripheral tissues, precursor for lipogenesis, contributes to appetite regulation and gut-brain communication
Propionate	About 20%	About 10-20 g/kg	Mainly used by hepatocytes, with a small fraction entering the circulation	1-15	Plays a crucial role in hepatic gluconeogenesis and cholesterol metabolism, modulates inflammatory pathways
Butyrate	About 20%	About 3.5-32.6 g/kg	Primary energy source for colonocytes; also crosses the BBB	1-15	Essential for maintaining gut barrier integrity, possesses anti-inflammatory and neuroprotective properties

MCTs: Monocarboxylate transporters; BBB: Blood-brain barrier.

mood and sexual motivation[119]. Similarly, microbial contributions to phenylalanine biosynthesis can enhance dopamine and norepinephrine production, further reinforcing libido and sex drive[119].

Inflammation and gut permeability also play significant roles in modulating sexual health. Intestinal inflammation, often associated with dysbiosis, leads to increased production of bacterial endotoxins such as LPSs, which activate immune pathways and contribute to neuroinflammation[120]. These inflammatory cascades involve toll-like receptor 4 activation and pro-inflammatory cytokine release (IL-1β, IL-6, tumor necrosis factor α), ultimately affecting limbic system structures responsible for emotional regulation[120].

Animal models offer compelling support for a link between systemic inflammation and impaired sexual behavior. In rats, neonatal exposure to LPS affects the timing of puberty, disrupts sexual performance in adulthood, and reduces sperm viability. These effects were accompanied by long-lasting alterations in the hypothalamic-pituitary-gonadal axis and hormonal regulation, highlighting how early immune activation can negatively influence reproductive and sexual outcomes later in life[121]. Clinical data from patients with inflammatory bowel disease further underscore this association. In women, active gastrointestinal symptoms were significantly associated with sexual dysfunction, and severe depressive symptoms emerged as the strongest independent predictor. In men, anxiety symptoms were most strongly linked to ED. Interestingly, these psychological variables were more predictive than objective measures of mucosal inflammation, suggesting a complex interplay between intestinal immune activity, emotional well-being, and sexual health[122]. Taken together, these findings support the notion that inflammation and intestinal permeability can indirectly affect sexual functioning through neuroimmune and neuroendocrine pathways, particularly by altering mood, stress response, and hormone regulation.

The table below summarizes key mechanisms linking gut microbiota, inflammation, and sexual health (Table 2). Dysregulation of dopamine and serotonin transmission due to gut inflammation may thus impair pleasure perception, reducing sexual interest and satisfaction. The gut-brain connection is largely mediated by enteric nervous system and autonomic nervous system, particularly through vagal afferents that transmit information from the gut to the brain[123]. Approximately 80% of vagal fibers carry afferent signals, emphasizing the strong influence of gut microbiota-derived metabolites and immune factors on CNS function. This bidirectional communication is essential for emotional well-being and sexual function, as disruptions in vagus nerve signaling have been linked to mood disorders[123]. Stimulation of the vagus nerve has been shown to positively modulate neurotransmitter levels, particularly serotonin and dopamine, which are crucial for reward processing and sexual arousal. The cumulative evidence suggests that gut microbial activity shapes emotional and behavioral responses, including those related to sexual health, although the exact mechanisms remain to be fully elucidated (Figure 1).

PSYCHOLOGICAL ASPECTS OF THE INTERACTION BETWEEN GUT MICROBIOTA AND SEXUAL WELL-BEING

Through the gut-brain axis, the gut microbiota can regulate sex hormone production and metabolism, such as testosterone and estrogen, which are essential for healthy sexual function. Dysbiosis can disrupt the production of essential microbial metabolites, including SCFAs, that support hormonal regulation and vascular health[124]. These disruptions may lead to imbalances in sex hormone levels, increased inflammation, and endothelial dysfunction, all of which are critical factors for sexual function[125]. Furthermore, the gut microbiota's impact on the brain-gut axis can contribute to psychological factors, such as sexual performance anxiety (SPA) or low libido, reinforcing the interconnection between microbiota health and sexual well-being (Figure 2).

SPA is one of the most common sexual disorders. It manifests as a form of anxiety that occurs during or before sexual activity. People who suffer from it may have concerns about their performance, partner satisfaction, erection, or orgasm.

Table 2 Impact of gut microbiota on inflammation and sexual health

Microbiota factor	Effect on inflammation	Pathways and mechanisms	Impact on sexual health
SCFA production	Reduces pro-inflammatory cytokines (IL-6, TNF-α) and enhances anti-inflammatory responses (IL-10)	Activates GPCRs, inhibits NF-κB signaling, enhances Treg activity	Modulates neurotransmitter levels, improving libido and mood stability
Gut barrier integrity	Strengthens intestinal epithelium, reducing systemic endotoxin levels	Increases tight junction proteins (occludin, claudin), reduces LPS translocation	Prevents inflammatory-induced sexual dysfunction (*e.g.*, erectile dysfunction); Improved gut integrity supports neurotransmitter balance, positively affecting sexual desire and pleasure
Dysbiosis (microbial imbalance)	Promotes endotoxemia, activates TLR4 signaling, increases systemic inflammation	Activates TLR4, increases pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), disrupts HPA axis function	Neuroinflammation disrupts dopamine and serotonin homeostasis, potentially reducing libido and motivation; Contributes to decreased libido, erectile dysfunction, and hormonal imbalances
Neurotransmitter synthesis	Modulates 5-HT, DA, NE, Ach and GABA production, impacting brain function	Regulates tryptophan metabolism, modulates serotonin biosynthesis *via* gut-derived 5-HT; NO and PGs influence DA, 5-HT, NE, and ACh release in key brain regions	Reduced serotonin synthesis may lead to mood instability and decreased sexual interest; Altered neurotransmitter release may impair arousal, orgasm, and emotional connection; Influences arousal, mood, and stress-related sexual disorders

SCFA: Short-chain fatty acid; IL: Interleukine; TNF-α: Tumor necrosis factor α; GPCRs: G-protein coupled receptors; NF-κB: Nuclear factor kappa B; TLR4: Toll-like receptor 4; HPA: Hypothalamic-pituitary-adrenal; 5-HT: Serotonine; DA: Dopamine; NE: Norepinephrine; Ach: Acetylcholine; NO: Nitric oxide; PGs: Prostaglandins; LPS: Lipopolysaccharide; Treg: Regulatory T cell; GABA: Gamma-aminobutyric acid.

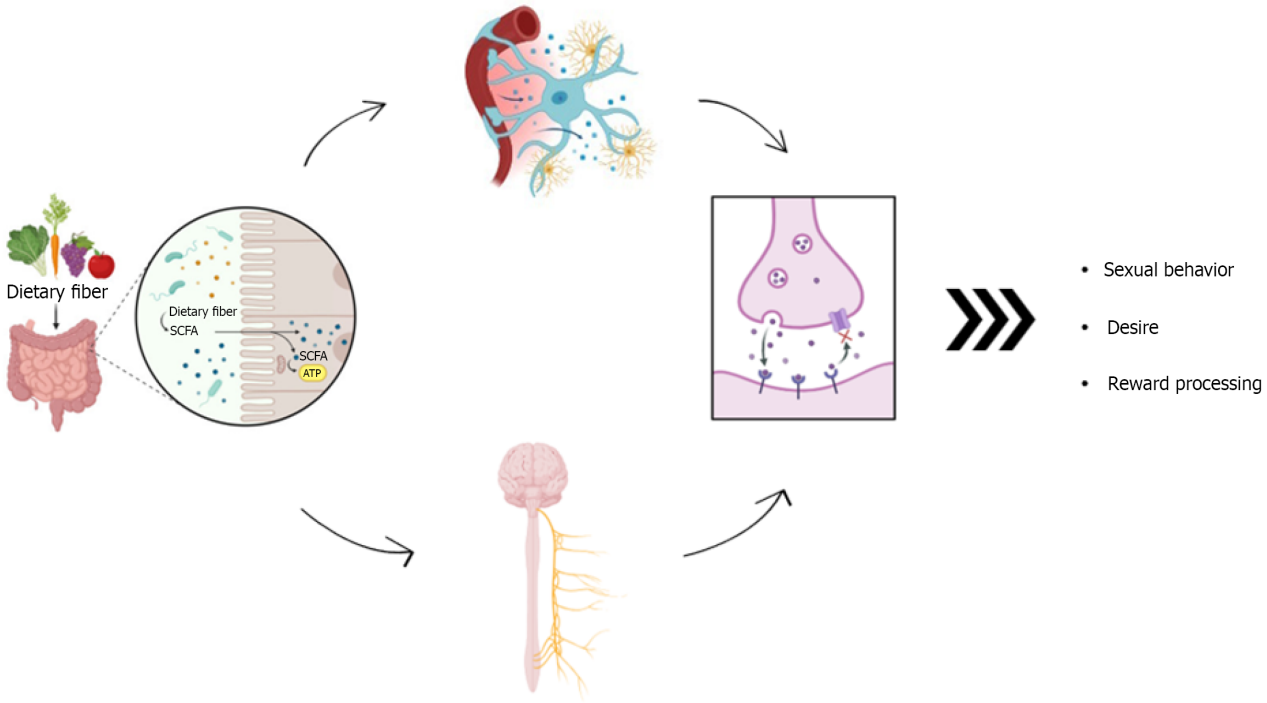

Figure 1 Short-chain fatty acids and the gut-brain-sex connection. The intricate interplay between short-chain fatty acid production, sexual hormones, immune signaling, and vagal communication underscores the importance of gut microbiota in regulating mood and sexual function. SCFA: Short-chain fatty acids.

This condition can affect both men and women, negatively impacting sexual and relational quality of life. The primary causes include high expectations, where the pressure to satisfy one's partner or meet idealized standards of sexual performance plays a significant role. Negative past experiences, including previous sexual difficulties, may trigger a cycle of fear and anxiety. Psychological and physical stress, along with fatigue, can exacerbate anxiety-related symptoms. Body image concerns, particularly low self-esteem and dissatisfaction with one's appearance, often heighten insecurities. Relationship difficulties, whether due to emotional conflicts or communication barriers, can further contribute to performance anxiety. Additionally, misinformation and unrealistic beliefs about sexuality and performance may intensify psychological distress[126,127].

In men, common manifestations include difficulty achieving or maintaining an erection (ED), premature ejaculation, or delayed ejaculation. In women, the condition often presents as a lack of sexual desire, difficulty with arousal, or pain during intercourse, like dyspareunia[128]. The physical reactions associated with SPA may include sweating, rapid

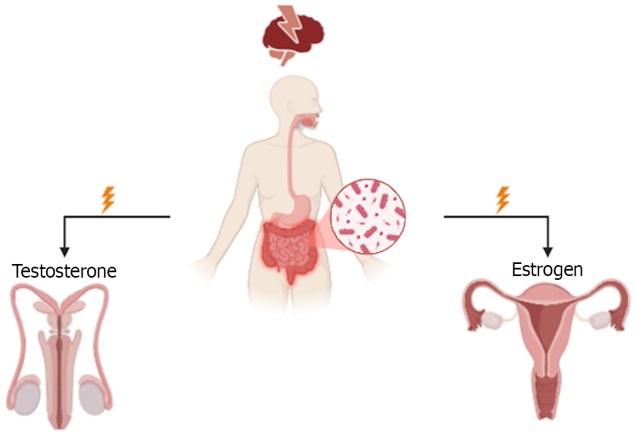

Figure 2 Psychological factors in the relationship between gut microbiota and sexual health. Sexual health is closely linked to the gut microbiota, which regulates hormonal balance and psychological well-being through the microbiota-gut-brain axis. This system influences sex hormone production and metabolism, such as testosterone and estrogen, which are essential for healthy sexual function. Dysbiosis can lead to hormonal imbalances, inflammation, and endothelial dysfunction, negatively affecting sexuality. Additionally, microbiota impacts psychological health, contributing to issues such as performance anxiety and decreased libido, reinforcing its key role in sexual well-being.

heartbeat, trembling, and muscle tension before or during sexual activity. Psychological reactions often include obsessive worry, loss of focus, feelings of shame, or guilt. SPA can have several negative consequences on sexual experiences, including a self-perpetuating cycle, where the fear of failure increases tension, which raises the risk of sexual difficulties, reinforcing the cycle of anxiety. It may lead to avoidance, as individuals start to avoid intimacy to escape anxiety, ultimately impacting their relationship. Additionally, it reduces pleasure, as anxiety distracts from the present moment, diminishing emotional connection and engagement.

These concerns can trigger the release of stress hormones, such as epinephrine and norepinephrine, which constrict blood vessels. This mechanism can compromise sexual health, creating a vicious cycle of anxiety and stress, which fuels and maintains sexual dysfunction[129]. Also, gut microbiota can act through the gut-brain axis, leading to the occurrence of psychological ED, but also organic ED, causing vascular endothelial dysfunction and disordered sex hormone metabolism, and interfering with lipid metabolism, immunity, and endocrine regulation[130].

Reduced diversity of the gut microbiota, as identified through gene sequencing studies in ED patients, correlates with modifications in sex hormone levels and overall sexual health[131]. Most research on SPA has focused on male experiences, particularly erectile functioning and premature ejaculation. However, little attention has been paid to women's experiences and the role of relationship dynamics in SPA-related problems[132]. From a female perspective, sexual well-being changes throughout different life stages, influenced by hormonal, physical, and emotional factors. During the menstrual cycle, hormonal fluctuations impact sexual desire[133]. Ovulation often increases libido due to higher estrogen levels, while the luteal and menstrual phases may lead to reduced desire and discomfort for some women. In pregnancy, the first trimester can bring a decline in sexual interest due to nausea, fatigue, and anxiety. The second trimester often sees an improvement in libido and sexual satisfaction due to increased blood flow and lubrication. In the third trimester, physical discomfort, body changes, and psychological concerns about childbirth may reduce intimacy[134]. After childbirth, a woman's body undergoes significant physical and emotional changes that can impact sexuality. Hormonal shifts, such as a drop in estrogen and oxytocin fluctuations, can reduce libido and cause vaginal dryness, affecting comfort during intercourse. Physical recovery from childbirth, including perineal trauma or cesarean delivery, may lead to pain or fear of intimacy. Body image concerns, influenced by weight changes, stretch marks, and self-perception, often affect confidence and sexual desire. Emotional factors like fatigue, stress from caring for a newborn, and postpartum depression can further influence sexual relationships[135]. Menopause presents unique challenges, with reduced estrogen causing vaginal dryness, thinning tissues, and discomfort during sex[136]. Lower testosterone levels can decrease libido, while physical and emotional changes, such as hot flashes, mood swings, and self-esteem issues, may also impact sexual health. However, treatments like hormone replacement therapy and open communication with a partner can help maintain a satisfying sex life. The prevalence of sexual problems increases with age (from 44.6% between 45 years and 64 years to 80.1% over 65 years)[137]. A recent study[138] demonstrated an inverse relationship between health anxiety and sexual desire, along with a direct relationship between health anxiety and sexual pain. Changes in gut microbiota diversity during menopause can interact with health anxiety, leading to reduced sexual desire[139]. Addressing these imbalances through microbiota-targeted interventions may alleviate anxiety and promote positive sexual health outcomes for women[140].

The bidirectional interaction between the gut, microbiota, and brain further underscores the importance of this system in sexual well-being. Psychological stress significantly impacts the gut microbiota, reducing microbial diversity and promoting dysbiosis, which directly affects sexual health[141,142]. Chronic stress tends to lower testosterone levels, a hormone essential for maintaining sexual desire in both men and women. The effect is amplified by increased cortisol, which inhibits sex hormone production, leading to a decrease in libido. In men, stress can cause difficulties with erection, premature ejaculation, or delayed ejaculation, often linked to reduced blood flow to the genitals and muscle tension induced by sympathetic nervous system activation[143]. In women, it may manifest as difficulties with arousal,

insufficient lubrication, and pain during intercourse, such as in dyspareunia[144,145]. Stress also interferes with the quality of sexual experience by reducing the ability to focus on the present moment, diminishing physical and emotional engagement. Concerns about performance and intrusive thoughts related to external factors divert attention from intimacy, compromising connection with one's partner. Additionally, prolonged stress can foster a sense of dissatisfaction or frustration in sexual life, negatively influencing emotional relationships and increasing the risk of conflicts or emotional distancing.

Dysbiosis triggers a cascade of physiological and psychological disruptions, including immune activation and inflammation that contribute to systemic dysfunction and impaired sexual health; disrupted neurotransmitter regulation, particularly serotonin, which influences mood, behavior, and sexual desire; enhanced stress reactivity, increasing vulnerability to SPA and other mental health challenges[146]. Through its interaction with sex hormones, the gut microbiota can regulate key aspects of sexual health. In fact, sex hormone regulation can disrupt the production and metabolism of testosterone, estrogen, and other essential hormones, affecting libido and sexual function in both men and women. The impact of dysbiosis on erectile function has been linked to endothelial and metabolic dysfunctions that contribute to ED, exacerbating preexisting conditions of stress or anxiety[147]. The gut microbiota serves as a critical bridge between mental health, physical well-being, and sexual health. Understanding and modulating the interactions between stress, the microbiota, and sex hormones offer new opportunities to enhance quality of life and sexual well-being for both men and women[148].

Sexual dysfunction, such as low sexual desire, premature ejaculation and ED are frequently associated with affective disorders[149]. Depression is intricately linked to sexual health, affecting not only physical aspects of sexual function but also emotional and relational dynamics. This relationship is particularly evident in cases of anhedonic depression, where pronounced loss of pleasure has been particularly linked to severe sexual problems[150]. Depression affects sexual health through multiple pathways, including hormonal imbalances, neurotransmitter disruptions, and the psychological impact of low mood, fatigue, and reduced self-esteem. One of the key mechanisms underlying the connection between depression and sexual dysfunction is the alteration of neurochemical pathways. Depression is associated with lower levels of serotonin, dopamine, and norepinephrine, neurotransmitters critical for mood regulation, arousal, and sexual desire. For example, reduced dopamine activity has been linked to diminished libido and difficulty experiencing pleasure, while imbalances in serotonin can contribute to delayed orgasm or anorgasmia. Depression often causes hormonal disruptions, such as dysregulation of the HPA axis[151]. Chronic activation of the HPA axis due to stress or depressive episodes can lead to elevated cortisol levels, which negatively impact sex hormone production. In men, this can result in reduced testosterone levels, contributing to ED and loss of sexual desire. In women, hormonal imbalances can interfere with ovulation, arousal, and lubrication, leading to diminished sexual satisfaction. Psychological factors further compound the issue. Feelings of worthlessness, guilt, and low self-esteem, which are common in depression, can undermine confidence in intimate relationships. Negative body image and fear of rejection exacerbate these challenges, creating barriers to sexual intimacy. Additionally, the fatigue and lack of motivation associated with depression can diminish interest in sexual activity, contributing to a cycle of avoidance and disconnection from one's partner. The relationship between depression and sexual dysfunction is bidirectional[152]. While depression can lead to sexual difficulties, unresolved sexual dysfunction often deepens depressive symptoms[153]. For instance, men experiencing ED may feel inadequate or anxious, which can worsen their mood. Similarly, women facing challenges like low libido or pain during intercourse may feel frustrated or disconnected, intensifying feelings of sadness or hopelessness[154]. Antidepressant medications, although essential for treating depression, can also exacerbate sexual dysfunction[155-157]. These side effects often discourage adherence to treatment, potentially prolonging depressive episodes and associated sexual health challenges.

Female sexual dysfunction (FSD) is a significant but often overlooked aspect of this interplay. In women, depression predominantly affects desire and arousal, leading to difficulties in achieving orgasm or experiencing pleasure. The psychological burden of female sexual dysfunction, combined with societal stigmas, often discourages women from seeking help, perpetuating cycles of distress and dissatisfaction in intimate relationships[158]. Male sexual dysfunction related to depression typically manifests as a combination of reduced libido, ED, and difficulties with ejaculation. For men, the stigma surrounding both mental health and sexual performance can be particularly challenging, leading to delays in seeking treatment and further exacerbating the issue[159].

Gut health significantly influences not only physical aspects, but also self-perception, impacting confidence and sexual relationships. This connection becomes especially pronounced in the context of eating disorders, where disruptions in the gut microbiota, psychological distress, and sexual dysfunction create a complex and multifaceted challenge. Anorexia nervosa (AN), bulimia nervosa (BN), and obesity exemplify how these interactions manifest through different mechanisms[160]. Hormonal imbalances, such as reduced estrogen or testosterone levels, are common in AN and contribute to a decrease in libido, intimacy difficulties, and sexual dysfunction. Women with AN often experience amenorrhea due to endocrine disruption, further diminishing sexual health, while men may suffer from reduced testosterone levels, leading to low sexual desire and ED[161]. The intense body image distortion and low self-esteem associated with AN amplify intimacy challenges, creating barriers to healthy relationships and reinforcing isolation[162]. In AN, severe caloric restriction and selective eating habits lead to lower gut microbiota diversity, which is associated with poorer health outcomes[163]. This dysbiosis exacerbates symptoms of anxiety, depression, and body image distortion, while impairing sexual health[164]. The bidirectional communication between the gut and brain underscores the profound role of gut health in both mental and sexual well-being.

BN, characterized by episodes of binge eating followed by compensatory behaviors such as self-induced vomiting or laxative use, causes profound gastrointestinal and hormonal imbalances[165]. These mechanisms alter the dopaminergic system, affecting impulse control, pleasure, and sexual gratification[166,167]. The guilt and shame associated with bulimic behaviors can lead to low self-esteem and difficulty experiencing sexual desire in a healthy way, creating a conflicted relationship with one's body. The weight fluctuations typical of BN further aggravate negative body

perception, increasing discomfort in sexual interactions and fear of judgment from a partner[168]. Physiologically, electrolyte depletion and hormonal alterations associated with bulimia can reduce vaginal lubrication and cause menstrual irregularities, further compromising female sexual function[169]. In men, hormonal fluctuations can lower libido and increase vulnerability to ED and ejaculation difficulties[170]. Obesity is often associated with psychological and metabolic dynamics similar to those seen in BN, significantly impacting sexual health. Excess adipose tissue leads to increased estrogen levels in men and decreased testosterone levels, negatively affecting libido and erectile function. In women, hormonal imbalances can result in irregular menstrual cycles, difficulty with arousal, and reduced genital sensitivity[171,172]. Insulin resistance and chronic inflammation, both common in obesity, impair blood circulation, further compromising sexual response in both men and women[173,174]. Psychologically, body image dissatisfaction, fear of judgment, and SPA can lead to decreased sexual activity, dissatisfaction, and difficulties in intimate relationships [175].

The role of gut microbiota in regulating sexual health is increasingly recognized as a key factor in these conditions. Alterations in gut microbiota composition, commonly found in individuals with eating disorders, influence neurotransmitter production, such as serotonin and dopamine, which are crucial for mood regulation, sexual desire, and pleasure perception[176,177]. Dysbiosis also affects sex hormones and metabolism, contributing to the persistence of both sexual and psychological dysfunctions[178-180]. Understanding the link between eating disorders, obesity, and sexual health is essential for developing targeted intervention strategies[181]. Multidisciplinary therapeutic approaches that combine psychological, nutritional, and endocrinological support can help restore a healthy relationship with the body and sexuality. Restoring gut health may alleviate depressive symptoms while simultaneously improving sexual desire, arousal, and overall satisfaction, creating a holistic pathway for enhancing mental and sexual well-being[182]. Addressing body image concerns and strengthening self-esteem are fundamental steps in promoting a fulfilling and unconditioned sexuality.

GUT MICROBIOTA, DIET, AND MENTAL HEALTH: IMPLICATIONS FOR SYSTEMIC AND SEXUAL WELL-BEING

Maintaining homeostasis in the human gut microbiota is vital for overall health[183]. Disruptions to this equilibrium can compromise intestinal barrier integrity and promote chronic inflammation[184], potentially contributing to the development of various gut microbiota-associated diseases, including obesity, metabolic syndrome, liver cirrhosis, neurodegeneration, cardiovascular diseases, inflammatory bowel disease, celiac disease, irritable bowel syndrome, diabetes, and several autoimmune conditions[185]. Several factors positively influence gut microbiota composition, thereby promoting systemic health benefits. Among these, dietary interventions play a crucial role in modulating microbiota dynamics. These strategies encompass a broad range of approaches, including the consumption of fermented foods, adherence to fiber-rich diets, and the incorporation of probiotics, prebiotics, and synbiotics[186]. Such interventions are versatile and accessible in various forms, ranging from everyday foods to specialized medical products and pharmaceuticals, making them adaptable to individual health needs and preferences.

Probiotics, prebiotics, and synbiotics have been clearly defined in scientific literature. Probiotics are "live microorganisms that, when administered in adequate amounts, confer a health benefit on the host"[187]. Prebiotics are defined as substrates selectively utilized by host microorganisms to confer a health benefit. Synbiotics, initially considered a mere combination of probiotics and prebiotics, are now defined as a mixture comprising live microorganisms and substrate(s) selectively utilized by host microorganisms that confers a health benefit on the host[188]. Postbiotics refer to health-promoting bioactive compounds derived from microorganisms and represents a significant innovation in microbiota modulation. Unlike probiotics, which must be administered in a viable form, postbiotics consist of inactivated microorganisms and their components that still exert beneficial effects on the host[189].

Recent studies indicate that supplementation with specific probiotic strains, particularly *Lactobacillus* and *Bifidobacterium*, enhances levels of key neurotransmitters such as serotonin and dopamine, which are essential for mood regulation and cognitive functions[190-192]. These effects positively impact mood and indirectly impact sexual function. The primary mechanism underlying these benefits is the modulation of the gut-brain axis[9]. In a physiologically healthy state, microbial communities and host cells interact in a balanced manner, maintaining homeostasis within the gut-brain axis. This equilibrium is fundamental to the proper function of interconnected biological networks and plays a critical role in overall health and well-being. Emerging evidence suggests that the gut microbiota has coevolved with the gut-brain axis, influencing brain development, function, and mood-related processes. Dysregulation of the gut-brain-microbiota axis has been implicated in neuropsychiatric disorders such as major depressive disorder[193], anxiety[194], and bipolar disorder[195], reinforcing its significance in mental health. Additionally, probiotics contribute to reducing oxidative stress and systemic inflammation, factors closely associated with mood disorders and sexual dysfunctions. By modulating signaling pathways involving GABA, oxytocin, and brain-derived neurotrophic factor, probiotics influence host behaviors such as anxiety, appetite, and mood[196,197]. These effects are mediated *via* vagal nerve activation, further highlighting the gut-brain axis's role in neurobehavioral regulation.

In addition to a balanced diet, probiotic supplementation is crucial for maintaining gut microbiota health and represents a promising, non-invasive therapeutic strategy for managing mood disorders and sexual dysfunctions. Probiotics hold significant potential as interventions for these conditions by modulating the gut-brain axis, reducing oxidative stress, and alleviating systemic inflammation. However, further research is needed to identify the most effective probiotic strains and establish optimal dosages for achieving clinically meaningful outcomes. Complementing probiotic use, a balanced diet plays a crucial role in maintaining a healthy gut microbiota, a well-documented relationship[198,199]. As

highlighted by Wilson *et al*[200], geographical dietary patterns significantly shape gut microbiota composition, reflecting the interaction between local food practices and microbial ecology. For instance, in rural African populations, traditional high-fiber diets are associated with a predominance of butyrate-producing bacteria, such as *Faecalibacterium*, which confer protective effects against non-communicable diseases. In contrast, urbanization and the adoption of Westernized diets, characterized by high-fat and low-fiber intake, lead to reduced microbial diversity and an increased prevalence of dysbiosis-related conditions, including obesity and colorectal cancer. A similar trend is observed in Asia, where the transition from low-fat, high-carbohydrate diets to high-fat Westernized diets has been linked to decreased SCFA-producing bacteria and an increase in inflammatory disorders. These findings underscore the critical influence of regional dietary patterns on gut microbiota composition and their broader implications for health.

High fiber diets, including whole grains, fruits, vegetables, and legumes, support the growth of beneficial gut bacteria, particularly SCFA-producing species such as *Bifidobacterium* and *Faecalibacterium*. According to Tan *et al*[201], dietary fibers and the SCFAs derived from their fermentation by gut microbiota play a crucial role in regulating mucosal immunity and preventing chronic diseases. SCFAs interact with specific receptors [G-protein-coupled receptor (GPR) 41, GPR43] on intestinal epithelium and immune cells, enhancing barrier integrity, reducing inflammation, and improved vascular function[202]. This is particularly relevant for sexual health, as enhanced vascular performance and reduced inflammation contribute to optimal sexual function. Additionally, fermented foods such as yogurt, kefir, sauerkraut, and miso serve as natural sources of probiotics, further promoting gut microbiota stability and resilience. Preliminary research suggests that incorporating fermented foods into the diet can improve cardiovascular parameters, boost energy levels, and enhance sexual desire, likely due to their influence on gut-brain axis signaling and metabolic processes[203].

These effects also have important implications for sexual health. SCFAs produced by dietary fiber fermentation improve endothelial function and lower systemic inflammation, two factors that are essential for healthy sexual function [7]. Enhanced endothelial performance supports genital blood flow, which is fundamental to arousal and erectile function. At the same time, reduced inflammatory states can help preserve hormonal balance and libido. SCFAs exert these effects through multiple pathways, including the activation of G-protein-coupled receptors (GPR41 and GPR43) and increased nitric oxide bioavailability, which collectively contribute to improved vascular tone and anti-inflammatory signaling[204]. Recent studies suggest that SCFAs may also influence mucosal integrity and immune responses in the reproductive tract, thereby contributing to a favorable environment for sexual well-being[205]. Conversely, dysbiosis and decreased SCFA-producing bacteria have been associated with sexual dysfunctions, possibly due to the chronic low-grade inflammation and vascular impairment that accompany microbial imbalances. Therefore, beyond their known roles in metabolic and intestinal health, fiber-derived SCFAs may also serve as key mediators of diet and sexual health[206]. Emerging evidence supports the potential of nutritional interventions aimed at restoring SCFA levels, particularly through high-fiber diets, as a non-invasive approach to improving sexual health outcomes.

Moreover, fiber-rich diets sustain SCFA production, reinforcing their wide-ranging benefits for gut, systemic, and sexual health. Conversely, low-fiber diets characteristic of Westernized lifestyles are strongly associated with decreased SCFA levels[207]. In parallel, diets high in refined sugars and saturated fats exacerbate dysbiosis, leading to reduced microbial diversity and an overgrowth of pathogenic species, thereby increasing the risk of inflammatory, metabolic, and neurodegenerative diseases[208]. This dysbiotic state is closely linked to chronic low-grade inflammation, a known contributor to metabolic and sexual dysfunctions. To support gut and sexual health, dietary strategies should focus on fostering microbial diversity and reducing inflammation. Incorporating omega-3 fatty acids, abundantly found in fatty fish, flaxseeds, and walnuts, offers significant benefits due to their anti-inflammatory properties and their role in maintaining vascular health, which is vital for optimal sexual function[209]. Equally important is minimizing the intake of harmful foods high in refined sugars and saturated fats to preserve microbiota homeostasis and mitigate systemic inflammation. These dietary interventions promote gut health and exert cascading benefits on systemic and sexual health, underscoring the profound impact of nutrition on overall well-being. Further research is essential to refine and tailor dietary recommendations to effectively optimize individual health outcomes.

INTEGRATING PSYCHOLOGICAL THERAPIES AND PSYCHOBIOTICS: A NOVEL APPROACH TO GUT-BRAIN AND SEXUAL HEALTH

The integration of psychobiotics represents an innovative and multidisciplinary approach to addressing sexual dysfunctions by modulating stress, anxiety, and overall mental well-being, all of which are closely linked to sexual function. Psychobiotics are a specialized class of probiotics defined as bacteria that confer mental health benefits by influencing the gut-brain axis[210]. This axis facilitates bidirectional communication between the gastrointestinal tract and the CNS through neural pathways such as the vagus nerve, hormonal pathways involving cortisol and serotonin, and immune pathways mediated by cytokines. Psychobiotics exert broad effects on mental and physical health by targeting key pathways in the gut-brain axis. Their mechanisms of action encompass psychological effects on cognitive function and emotional regulation; systemic influences on the HPA axis and the body's stress response; and modulation of inflammation, which is often accompanied by elevated inflammatory markers[211]. These benefits are mediated by the production of neuroactive compounds such as serotonin, GABA and SCFAs, all of which play critical roles in brain function and emotional stability[212]. Additionally, psychobiotics enhance intestinal barrier integrity, regulate HPA axis activity, and reduce systemic inflammation, collectively promoting mental and physical well-being. They also show potential in alleviating anxiety, depression, and other psychosomatic disorders, conditions frequently associated with sexual dysfunction. Preclinical and clinical studies highlight their role in managing neuropsychiatric disorders, including depression, anxiety, attention-deficit/hyperactivity disorder, autism spectrum disorder, schizophrenia, Parkinson's

disease and Alzheimer's diseases[213-215].

Notable strains, such as *Lactobacillus rhamnosus* and *Bifidobacterium longum*, have demonstrated the ability to reduce anxiety and depressive symptoms through the GABA and serotonin production, key neurotransmitters for mood regulation and psychological well-being[216-218]. Although these strains have not yet been directly associated with sexual function in clinical studies, their influence on psychological states (particularly stress reduction, mood stabilization, and decreased HPA axis hyperactivation) suggests a plausible indirect benefit for sexual health. Improved mental well-being is closely linked to enhanced sexual desire, arousal, and satisfaction, especially in individuals with psychogenic sexual dysfunctions. Emerging evidence further suggests that strains may exert modulatory effects on endocrine pathways and vascular function, both of which are relevant to sexual physiology. For instance decreased systemic inflammation and cortisol levels, along with improved vascular tone and neurotransmitter availability, may help create a neuroendocrine environment conducive to sexual responsiveness.

These effects may positively impact sexual function by improving desire, arousal, and satisfaction, primarily through stress reduction and increased mental energy. In neurodegenerative conditions such as Alzheimer's and Parkinson's, psychobiotics contribute to cognitive preservation, oxidative stress reduction, and neuroprotection by modulating gut microbiota composition and inflammation[219]. By restoring microbial diversity and promoting the growth of beneficial bacteria, psychobiotics help re-establish gut-brain homeostasis. However, while their potential as a cost-effective and non-invasive therapy is promising, further studies are needed to determine the most effective strains, optimal dosages, and long-term effects for clinical integration.

Psychological therapies targeting the gut-brain axis, including gastrointestinal-specific cognitive-behavioral therapy (CBT), gut-focused hypnosis, and mindfulness-based approaches, have proven effective in managing disorders associated with gut-brain dysregulation[220]. CBT has demonstrated efficacy in treating gastrointestinal conditions such as irritable bowel syndrome, where stress and microbiota imbalances exacerbate symptoms. By reducing anxiety, promoting health-supporting behaviors and encouraging lifestyle changes that restore microbiota balance, CBT improves symptoms, stool consistency, quality of life, and emotional well-being[221]. Building on this foundation, integrating psychological therapies with psychobiotics represents a novel multidisciplinary approach. Psychobiotics modulate the gut-brain axis by producing neuroactive compounds, reducing systemic inflammation and reinforcing intestinal barrier integrity. These mechanisms directly influence emotional resilience and physiological processes, particularly the HPA axis, which plays a key role in stress regulation. Mindfulness, which enhances self-awareness and reduces stress[222], may act synergistically with psychobiotics to further stabilize mood, improve emotional regulation, and promote overall psychological and physical well-being.

This integrated approach holds promise in various clinical contexts, particularly in treating psychogenic sexual dysfunction, such as performance anxiety and low sexual desire driven by psychological stress. By addressing both the psychological and microbiotic contributors to these conditions, the combined use of psychobiotics with therapies like CBT and mindfulness may enhance sexual responsiveness and overall satisfaction. This convergence of psychological interventions and psychobiotics represents a promising frontier in clinical research, integrating insights from neuroscience, psychiatry, and microbiology to develop more effective treatments for complex disorders.

In the future, comprehensive therapeutic strategies that combine probiotics, dietary modifications, and psychological interventions may become a standard approach to managing sexual dysfunction associated with gut dysbiosis. Advances in microbiota research have enabled mapping of this intricate microbial community, revealing that each individual has a unique "microbial fingerprint" shaped by genetics and environmental interactions. These discoveries have paved the way for innovative therapeutic strategies, including tailored probiotics and prebiotics to support specific beneficial bacterial strains, personalized dietary interventions aimed at optimizing microbiota composition and reducing inflammation, microbiota-targeted pharmaceuticals designed to enhance therapeutic efficacy while minimizing side effects, and fecal microbiota transplantation, an emerging and effective treatment for dysbiosis-related diseases[223]. Personalized therapies based on individual microbiota profiles hold great potential for enhancing treatment efficacy and minimizing adverse effects. Long-term clinical trials are essential to validate these approaches and further elucidate their underlying mechanisms. Concurrently, increasing public awareness of microbiota's role in health may encourage the adoption of healthier lifestyles, thereby enhancing the preventive impact of these integrated strategies.

CONCLUSION

Gut microbiota plays a central role in the complex interplay between sexual health and mental well-being. Acting through the gut-brain axis, it influences key neurobiological processes such as neurotransmitter production, stress regulation, hormonal balance, and emotional resilience - all of which are critical for healthy sexual functioning. Dysbiosis has been linked to physiological conditions like ED and hormonal imbalances, but also to psychiatric disorders such as anxiety, depression, and SPA. Psychobiotics and microbiota-targeted therapies, especially when integrated with psychological interventions, offer promising avenues for addressing both the mental and physical dimensions of sexual dysfunction. A better understanding of the microbiota's influence on neuropsychiatric and sexual health may lead to more personalized, effective, and holistic therapeutic strategies. Future research should continue to explore these connections, with the aim of advancing integrated care models that bridge psychiatry, sexual medicine, and microbiome science.

FOOTNOTES

Author contributions: Marano G, Gaetani E, and Mazza M contributed to the conceptualization, supervision and management of the manuscript; Marano G and Mazza M performed the methodology; Anesini MB, Militenda M, Acanfora M, d'Abate C, Lisci FM, Pirona I, Traversi G, and Pola R provided resources and performed data curation; Marano G and Mazza M wrote the original manuscript, reviewed it and edited it; all authors reviewed and discussed the results and contributed to the final manuscript.

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