Rasayely
International Journal of Equine Science
https://rasayely-journals.com/index.php/ijes
Vol 4(S1);38–48, 2025

Review Article Open Access

Gut Microbiota and Sleep Deprivation: Potential Interactions and Implications for Equine Research – Scoping Review

Anny R. C. Gomes[1,*], Tiago M. Oliveira[1,*], Guilherme G. Campos[1], and Raquel Y. A. Baccarin[1]

[1]*Department of Veterinary Clinical Sciences, School of Veterinary Medicine and Animal Science, University of São Paulo, São Paulo, 05508 270, Brazil.*

[*]Authors to whom any correspondence should be addressed; Emails: anny.gomes@usp.br (A.R.C.G.); tiagooliveira@usp.br (T.M.O.)

Received: 29 August 2025; Revised: 19 October 2025; Accepted: 26 November 2025; Published: 17 December 2025

Academic Editor: Ivana Schork, Hartpury University, United Kingdom

Abstract

Background: Sleep deprivation negatively affects metabolism and the immune system. Among other factors, this condition disrupts homeostasis by altering the gut microbiota, which plays a crucial role in maintaining physiological balance in humans and rodents, including sleep regulation. However, to date, no study has directly investigated this interaction in horses. **Aims:** This review aims to examine the interplay between sleep deprivation and gut microbiota from a translational perspective, emphasizing the gut–brain axis and the roles of sleep and microbiota in equine health. **Materials and Methods:** Original research articles, meta-analyses, and literature reviews relevant to the topic were included. Database searches were conducted using carefully selected keywords to ensure comprehensive coverage of pertinent literature. **Results:** Sixty-three studies were reviewed, including original studies, systematic and narrative reviews, observational studies, randomized clinical trials, and experimental investigations, published between 1975 and 2025. Sleep deprivation was found to alter the gut microbiota, increasing inflammatory markers and modifying metabolic pathways, predisposing the organism to disease. Conversely, the gut microbiota also influences sleep, indicating a bidirectional relationship. Although studies on horses are lacking, preliminary evidence suggests the presence of the gut–brain axis in this species. Sleep disruption may therefore negatively influence the gut microbiota of horses, as reported in other animals. **Conclusion:** Sleep deprivation negatively affects the body by altering the gut microbiota in humans and rodents. Although evidence links gut microbiota and sleep to equine health, important gaps remain regarding how sleep deprivation affects microbial balance, inflammation, metabolism, and disease risk.

Keywords

Microbiome; gut bacteria; microbial imbalance; metabolism; inflammation

1. Introduction

Sleep is a brain-regulated process that triggers a complex network of physiological cascades essential for maintaining the body's homeostatic balance and overall health [1]. Sleep deprivation occurs when rest is insufficient, whether due to reduced sleep duration or poor sleep quality, leading to decreased performance, reduced alertness, and adverse effects on health [2].

Sleep deprivation is a common issue in humans, affecting 37% to 58% of the population [3], and is considered a significant public health concern, as it is recognized as a risk factor for the development of various diseases [4]. This condition triggers systemic physiological alterations, particularly impacting metabolism and immune system function [5–11]. Sleep deprivation also affects horses, being associated with decreased well-being and behavioral changes [12–14].

One mechanism recently identified as central to the harmful effects of sleep deprivation is the alteration of the gut microbiota. This condition can disrupt the microbial community in the intestine, with studies demonstrating its impact on the diversity and abundance of bacteria inhabiting the gastrointestinal tract **[7,15–17]**. Changes in the microbiota resulting from inadequate sleep have been associated with increased inflammatory markers and alterations in energy metabolic pathways, establishing a direct link to metabolic and immune dysfunction in the host **[7,15–19]**.

The gut microbiota performs metabolic, neurological, immunological, and structural functions, and maintaining a balanced microbial community is essential for overall health **[20–22]**. In humans, the existence of the gut–brain axis has gained increasing attention, with various studies demonstrating that the gut microbiota directly interacts with sleep regulation, reinforcing the hypothesis of a bidirectional relationship between the two **[23–26]**.

In horses, no studies to date have directly addressed this topic. However, there is evidence that both sleep deprivation and gut microbiota influence the health and well-being of these animals, being associated with the development of metabolic disorders, cognitive deficits, and behavioral disturbances. These findings suggest the possible existence of a gut–brain axis in this species as well **[13,27–29]**.

The objective of this literature review was to investigate the interaction between sleep deprivation and the gut microbiota from a translational perspective, considering scientific evidence derived from studies conducted in humans and laboratory animals, and emphasizing the gut–brain axis and the influence of sleep and gut microbiota on equine health.

2. Materials and Methods

2.1. Target Questions and Search Strategy

The search strategy and the selection of keywords were guided by the research questions. These strategies were developed based on keywords and terms defined using the components of the PICO model, ensuring greater accuracy in the search process. In addition, Boolean operators ("AND" and "OR") were applied, as needed, to optimize the results obtained **[30,31]**. **Table 1** presents the guiding research questions, along with the detailed PICO framework used to define keywords and their synonyms.

Based on the keywords and search terms defined using the PICO model, search strategies were developed to address the research questions and subsequently applied to the selected databases. The following are the search strategies defined for Questions 1, 2, and 3, respectively:

2.1.1. Question 1

(humans OR women OR men OR children OR adolescents OR equines OR horses OR mares OR stallions OR foals) AND ("sleep deprivation" OR "chronic sleep deprivation" OR "acute sleep deprivation" OR sleep OR "sleep disorders") AND ("adequate sleep" OR "normal sleep" OR "healthy sleep" OR "physiological sleep" OR "sleep quality") AND ("physiological changes" OR "metabolic consequences" OR "inflammatory consequences" OR health OR homeostasis OR metabolism OR inflammation).

2.1.2. Question 2

(humans OR women OR men OR children OR adolescents OR equines OR horses OR mares OR stallions OR foals) AND ("gut microbiota" OR "microbial community" OR "dysbiosis" OR "microbiota imbalance") AND (normobiosis OR "balanced microbiota" OR "equilibrated microbiota") AND (metabolism OR "metabolic changes" OR "metabolic consequences" OR "energy metabolism" OR "immune function" OR "immune response" OR "immune system" OR inflammation OR cytokines OR "acute-phase proteins" OR "nervous system" OR "gut–brain axis" OR "neurological function" OR health).

2.1.3. Question 3

(humans OR women OR men OR children OR adolescents OR equines OR horses OR mares OR stallions OR foals OR rats OR mice OR laboratory animals) AND ("sleep deprivation" OR "sleep restriction" OR "chronic sleep deprivation" OR "acute sleep deprivation" OR "gut microbiota" OR "intestinal flora" OR dysbiosis OR microbiome OR "microbial community") AND ("adequate sleep" OR "normal sleep" OR "sufficient sleep" OR "sleep quality" OR "balanced microbiota" OR normobiosis OR "equilibrated microbiota") AND (metabolism OR "metabolic changes" OR "metabolic consequences" OR "energy metabolism" OR "immune function" OR "immune response" OR "immune system" OR inflammation OR cytokines OR "acute-phase proteins" OR health OR "health status" OR "disease risk" OR "chronic diseases" OR well-being).

2.2. Article Inclusion and Exclusion Criteria

The inclusion and exclusion criteria prioritized scientific literature providing the most robust and consistent evidence on the topic addressed in this review. Full-text articles from clinical trials and observational studies, as well as literature reviews (narrative or systematic) and meta-analyses published in scientific journals and written in English, were considered.

Excluded were book chapters, conference abstracts, case reports, articles not available in full text in English, studies irrelevant to the research objectives or unrelated to the topic, as well as duplicate studies identified across multiple databases.

2.3. Databases Used and Research Period

The literature searches were conducted in three databases of broad scientific relevance: PubMed, Scopus, and Web of Science. The research process was conducted between February and May 2025, during which previously established search questions were applied in a standardized manner to ensure comprehensiveness and reproducibility in identifying studies. Although not a systematic review, we followed structured search strategies to identify relevant studies across human, rodent, and equine research.

Table 1: Guiding research questions and breakdown of the PICO model for defining keywords and synonyms to be used in the search strategies.

Question 1: What is sleep deprivation, and what are the physiological (metabolic and inflammatory) and health consequences for humans and horses?		
PICO		Keywords/Synonyms/Search Terms
Patient or Population	Humans and horses	Humans, women, men, children, teenagers, equines, horses, mares, stallions, foals
Intervention	Sleep deprivation	Sleep deprivation, chronic sleep deprivation, acute sleep deprivation, sleep, sleep disorders
Comparison/Control	Adequate/healthy sleep	Adequate sleep, normal sleep, healthy sleep, physiological sleep, sleep quality
Outcome	Physiological changes (metabolic and inflammatory) and health consequences	Physiological changes, metabolic consequences, inflammatory consequences, health, homeostasis, metabolism, inflammation
Question 2: What is the influence of the intestinal microbiota on the metabolism, the immune system, the neurological system, and the health of humans and horses?		
PICO		Keywords/Synonyms/Search Terms
Patient or Population	Humans and equines	Humans, women, men, children, teenagers, equines, horses, mares, stallions, foals
Intervention	Changes in the intestinal microbiota	Gut microbiota, microbial community, dysbiosis, microbiota imbalance
Comparison/Control	Balanced microbiota	Normobiosis, balanced microbiota, balanced microbiota
Outcome	Effects on metabolism, immune system, neurological system, and general health	Metabolism, metabolic changes, metabolic consequences, energy metabolism, immune function, immune response, immune system, inflammation, cytokines, acute phase proteins, nervous system, gut-brain axis, neurological function, health
Question 3: How are sleep deprivation and gut microbiota related, and how does this interaction influence health, metabolism, and inflammation?		
PICO		Keywords/Synonyms/Search Terms
Patient or Population	Humans, horses, mice, and laboratory animals	Humans, women, men, children, adolescents, equines, horses, mares, stallions, foals, rats, mice, laboratory animals
Intervention	Sleep deprivation and changes in intestinal microbiota	Sleep deprivation, sleep restriction, chronic sleep deprivation, acute sleep deprivation, gut microbiota, gut flora, dysbiosis, microbiome, microbial community
Comparison/Control	Adequate sleep and balanced microbiota	Adequate sleep, normal sleep, sufficient sleep, quality of sleep, balanced microbiota, normobiosis, balanced microbiota
Outcome	General health, metabolism, immune system, and inflammatory processes	Metabolism, metabolic changes, metabolic consequences, energy metabolism, immune function, immune response, immune system, inflammation, cytokines, acute phase proteins, health, health status, disease risk, chronic diseases, well-being

3. Results and Discussion

3.1. Research Results

A total of 53,341 results were obtained from the established search strategies. Details on the number of studies retrieved by each strategy in each of the databases used are presented in **Table 2**.

From the total records obtained in the initial search, 63 articles were selected for this review after applying the predefined inclusion and exclusion criteria. These studies comprised narrative and systematic reviews, observational studies, randomized clinical trials, and experimental research. The included articles were published between 1975 and 2025, with the majority concentrated in the period from 2019 to 2024.

3.2. Sleep and Sleep Deprivation: A Human and Horse Perspective

Sleep is an essential physiological process generated by a complex cascade of events primarily involving the central nervous system (CNS) **[1]**. Sleep and wakefulness states are coordinated by neural networks and regulated by circadian

mechanisms [1]. Wakefulness is sustained by a network of subcortical structures in which neurochemicals such as norepinephrine, serotonin, histamine, dopamine, acetylcholine, and orexin act, all originating from different neural nuclei [32]. For sleep initiation and maintenance, it is necessary to suppress the activation of this ascending arousal system, a function performed by inhibitory neurons in the ventrolateral preoptic area, which remain active throughout sleep [1]. The trigger for the activation of this brain region is believed to be extracellular adenosine, which activates these neurons, triggering sleep [1,32].

In addition to endogenous mechanisms, external factors also influence the sleep–wake cycle. These include exposure to light and darkness, as well as social stimuli. In low-light conditions, the pineal gland secretes melatonin, a hormone that directly affects the mechanisms that regulate sleep [1]. In humans, environmental factors such as artificial light and noise are known to affect sleep quality; these effects are similarly observed in horses, with studies showing that nighttime lighting, ambient sounds, pain, and environmental changes interfere with sleep patterns [14,33,34].

The quantity and organization of sleep vary among animal species. For humans, the recommended sleep duration is 7 to 9 hours per night, in a monophasic pattern [1]. Rats and mice, which are widely used in sleep research as comparative models for humans, exhibit a polyphasic sleep pattern characterized by multiple short sleep episodes distributed throughout the day [35,36]. These species sleep an average of up to 18 and 12 hours per day, respectively [35,37], with a greater concentration of sleep during the light phase and higher activity during the dark phase [35,36]. On the other hand, horses sleep between 3 and 4 hours per day, in a polyphasic and fragmented pattern throughout the night and day, with a greater concentration of sleep periods during the night [14,38]. Despite these differences, sleep is equally important in these species for maintaining health and well-being.

Both humans and horses experience two main types of sleep: NREM (non–rapid eye movement) sleep (stages N1, N2, and N3) and REM (rapid eye movement), also known as paradoxical sleep. N1 sleep corresponds to light sleep, N2 to an intermediate stage of sleep, and N3 to deep slow-wave sleep. REM sleep occurs after stage N3 and is characterized by rapid eye movements and muscle atonia [1,14,32]. During these phases, critical events occur, such as memory consolidation, strengthening of the immune system, hormonal regulation, maintenance of cardiovascular health, and removal of metabolic waste from the CNS through the glymphatic system [1].

In horses, REM sleep occurs predominantly in the sternal or lateral recumbency position, accounting for approximately 30 to 60 minutes of total daily sleep time [38]. Therefore, persistent reductions in recumbency time may indicate partial sleep deprivation, compromising sleep quality [33].

Sleep deprivation, defined as inadequate rest due to reduced sleep quantity or quality, leads to decreased performance, reduced alertness, and significant health problems [2]. It can be classified as acute when there is an absence or restriction of sleep for one or two consecutive days, or chronic when characterized by excessive daytime sleepiness resulting from the habit of sleeping less than necessary for at least three months [39]. In horses, sleep deprivation can occur in situations such as changing environments, hospitalization, transport, and temporary housing during competitions [33,34,40].

The repercussions of sleep deprivation affect multiple systems, such as metabolism, leading to insulin resistance and type 2 diabetes [4,10,11], testosterone suppression with consequent loss of libido [41,42], and increased appetite due to ghrelin and leptin imbalances [1,37]. In the cardiovascular system, it promotes hypertension, endothelial dysfunction, an increased risk of arrhythmias, and a higher likelihood of cardiac events [1,4]. In the immune system, it reduces antibody production and increases inflammatory cytokines [1,4]. In the nervous system, it compromises memory consolidation and glymphatic system function [1,4]. Psychologically, it is associated with an increased risk of depression, mood swings, irritability, and reduced well-being [1,17].

In horses, sleep deprivation also has significant consequences, including reduced well-being, cognitive deficits, and decreased concentration and vigor during exercise [14,28,40]. However, there are still significant gaps to be addressed regarding the effects of this condition in this species, particularly concerning the influence of sleep deprivation on the gut microbiota, its repercussions on systemic inflammation and metabolism, and, as observed in humans, its potential role as a predisposing factor for the development of diseases.

The following topics will cover, in detail, the impacts of sleep deprivation on the gut microbiota, metabolism, and the immune system, based on evidence from humans, laboratory animals, and horses.

3.3. Influence of Sleep Deprivation on Metabolic and Immune Functions: Health Impacts

Sleep deprivation is common among humans and represents an important public health issue because it is associated with the development of diseases [3,4]. The impacts of sleep deprivation on the general health status of individuals are related to the metabolic and immunological changes caused by this disorder. This fact has been investigated in research carried out in humans and laboratory animals [5–11].

Two studies analyzed the effects of chronic REM sleep deprivation on depressive behavior and the metabolic and immunological profiles of Wistar rats [6,7]. To this end, the animals' urinary metabolic profile, levels of the pro-inflammatory cytokines IL-6 and TNF-α [6,7], and serum C-reactive protein concentrations [7] were evaluated. The results indicated that sleep deprivation caused significant metabolic changes. Among the observed changes were disturbances in the metabolic pathways of glyoxylate and dicarboxylate; glycine, threonine, and serine; nicotinamide and nicotinate; and arginine and proline [6,7]. Furthermore, alterations in the metabolism of beta-alanine and pyruvate were also identified [7]. These compounds participate mainly in energy metabolism pathways and contribute to the maintenance of cellular functions, while arginine and proline are essential for the biosynthesis of amino acids [6].

Table 2: Number of results obtained from the databases for each search strategy, according to the research question from which it was derived.

Search Strategy	PubMed	Scopus	Web of Science	Total
Question 1	4,551	24,701	7,673	36,925
Question 2	111	231	7,673	8.015
Question 3	579	3	7,819	8,401
Total	5,241	24,935	23,165	53,341

These findings are consistent with a previous study that used a sleep deprivation model in mice to evaluate the hepatic proteome and metabolome. In that study, the animals were subjected to sleep deprivation for 20 hours daily for ten days [9]. The authors also observed energy-metabolic changes after sleep deprivation, with the glutathione, pyruvate, and fructose and mannose metabolic pathways being the most affected, with a reduction in the activity of these pathways, and these changes were linked to the development of obesity [9].

Sleep deprivation has also been associated with metabolic disorders such as insulin resistance and diabetes [5]. In people suffering from chronic sleep deprivation, defined as a rest routine of less than 6 hours per day, extending sleep time resulted in a significant improvement in fasting insulin sensitivity and pancreatic β-cell function, even without changes in diet or body weight [8]. Both acute 24-hour sleep deprivation and sleep restriction to just 4 hours per night for four days led to increased plasma insulin levels compared to individuals who had regular sleep [11]. Between the two sleep deprivation models, the sleep-restricted group had higher insulin levels and a significant reduction in the Matsuda index, which assesses insulin sensitivity, suggesting insulin resistance [11].

These findings corroborate a systematic review, which, through analysis of several studies on the topic, found that reduced sleep time is significantly associated with insulin resistance, concluding that regular sleep of 7 hours per night may help prevent metabolic diseases such as type 2 diabetes [10].

Regarding the inflammatory profile of the animals in the studies discussed, there was an increase in pro-inflammatory cytokines [6,7] and C-reactive protein [7], suggesting that sleep deprivation induced a state of systemic inflammation. In addition to these parameters, the authors also measured hormones of the hypothalamic–pituitary–adrenal axis, which were increased after sleep deprivation, indicating increased stress in the animals [6,7].

Sleep-deprived rats exhibited depressive behavior, as assessed through behavioral tests [6,7]. The authors linked this behavioral change to metabolic, hormonal, and inflammatory changes resulting from the sleep disorder, suggesting that chronic sleep deprivation may negatively impact mental health [6,7]. However, another study suggested that acute 24-hour sleep deprivation in humans may have a beneficial effect on symptoms of depression [5]. This effect could be related to sleep-deprivation-induced metabolic changes, resulting in increased levels of serotonin and some of its precursors, such as tryptophan [5].

3.4. Interactions between Sleep and Gut Microbiota: A Bidirectional Relationship that Affects Health

Sleep and the gut microbiota have a bidirectional interaction [24]. The composition of the microbiota directly affects sleep through bacterial metabolites, such as short-chain fatty acids (SCFAs), and directly influences the serotonergic system. Conversely, sleep deprivation can alter the gut microbial composition, contributing to dysbiosis and inflammation [24].

Serotonin is a precursor to melatonin, the main hormone involved in regulating the sleep–wake cycle, and enteroendocrine cells are responsible for producing approximately 90% of this neurotransmitter in humans [43–45]. The gut microbiota can affect serotonin production by promoting the synthesis of its precursors, such as tryptophan [46]. A clinical study demonstrated that the administration of a probiotic based on *Bifidobacterium breve* increased fecal levels of tryptophan and its metabolites, favoring the regulation of systemic serotonin [46].

A systematic review identified 15 clinical trials that evaluated the effects of probiotic use on sleep-related parameters. The results showed that probiotic supplementation for a period of 4 to 6 weeks was associated with improved scores on the Pittsburgh Sleep Quality Index (PSQI), which assesses sleep quality [26]. Furthermore, other studies have observed similar benefits in human patients after probiotic use [23,25], reinforcing the hypothesis that the gut microbiota directly influences sleep regulation.

Few studies address the relationship between the gut microbiota and sleep duration, and there is no consensus on the subject [26]. However, an experimental study in mice associated administration of the *Lactiplantibacillus plantarum P72* strain with reduced sleep latency time and increased sleep duration [47]. To assess these effects, anesthetics were used as sleep inducers, defining latency as the interval between anesthetic administration and sleep onset, and duration as the time between sleep onset and the animals' awakening [47]. The authors found that in cultured neuronal cells with serotonin secretion suppressed by corticosteroids, administration of the strains led to increased serotonin secretion, suggesting a possible mechanism involving modulation of serotonergic neurotransmission [47].

Tests in mice and rats have shown that butyrate administration can increase the duration of NREM sleep [48]. In one experimental group, the animals received a gavage solution containing tributyrin, a butyrate prodrug, and experienced an approximately 50% increase in NREM sleep duration. In another group, sodium butyrate was administered intraportally at a dose of 1 g/kg, resulting in a 70% increase [48].

Administration of a butyrate supplement to patients with ulcerative colitis improved indices assessing the participants' sleep quality [49]. In this study, butyrate also reduced inflammatory markers such as calprotectin and C-reactive protein, compared to the group of patients receiving placebo [49]. These findings demonstrate that bacterial metabolites also act as sleep modulators.

As shown in sections 3.2 and 3.3, sleep and the gut microbiota influence metabolism and the immune system, and their disturbances may represent a risk factor for the development of diseases. In recent years, evidence from clinical and preclinical studies has demonstrated that interactions between sleep and the gut microbiota may represent a fundamental link in understanding the inflammatory and metabolic changes triggered by sleep deprivation **[7,15–19]**.

Sleep deprivation has been associated with changes in the composition of the gut microbiota, favoring an increased abundance of bacteria potentially harmful to the host, while reducing the presence of beneficial bacteria **[15,17]**. Mice subjected to 72 hours of paradoxical sleep deprivation showed a significant reduction in the alpha and beta diversity of the gut microbiota **[17]**. Furthermore, an increased abundance of certain bacteria, such as *Enterobacter* and *Candidatus arthromitus*, was observed, which demonstrated a positive correlation with plasma TNF-α concentrations, also elevated in the sleep-deprived group, and a negative correlation with the expression of genes related to intestinal barrier integrity (OCLN and ZO-1), which were significantly reduced compared to the control group **[17]**.

On the other hand, genera considered beneficial, such as *Lactobacillus*, *Muribaculum*, *Parasutterella*, *Ruminococcus*, *Monoglobus*, and *Akkermansia*, among others, were less abundant in the sleep-deprived group and demonstrated a negative correlation with TNF-α levels, as well as a positive correlation with the expression of genes associated with maintaining intestinal barrier integrity **[17]**. The increase in plasma lipopolysaccharide (LPS) after sleep deprivation suggested increased intestinal permeability **[17]**. Some of these bacterial genera, which were reduced in sleep-deprived mice, are also recognized for their beneficial effects in studies conducted with humans and horses. Among them, the *Lactobacillus* genus stands out, being widely described as beneficial in both species **[3,25,50,51]** and commonly used in probiotic formulations with positive results **[3,25,50,51]**. The Ruminococcaceae family, which includes the *Ruminococcus* genus, also appears to exert beneficial effects in both horses and humans, particularly through species within this genus that possess the ability to degrade dietary fiber **[27,50,52,53]**.

Complete sleep deprivation for 48 hours in mice reduced alpha diversity and altered beta diversity of the gut microbiota compared to the control group ($p < 0.05$) **[16]**. The composition of the gut microbiota was also impacted, with significant changes in several bacterial genera **[16]**. After 48 hours of sleep deprivation, there was a reduction in bacteria from the genera *Butyricicoccus*, *Butyricimonas*, *Alistipes*, *Intestinimonas*, and *Lactobacillus* **[16]**. Conversely, there was a significant increase in the genus *Streptococcus*, whose presence has been previously associated with deleterious effects and disease development **[16]**.

The changes in the gut microbiota were accompanied by a reduction in fecal butyrate, which was positively correlated with the abundance of bacteria from the genus *Butyricimonas*, which were reduced in the sleep-deprived group **[16]**.

Significant differences were observed in microbial metabolism after sleep deprivation in mice. Pathways related to lipid metabolism, short-chain fatty acids, and amino acid and tryptophan metabolism were the most affected **[16]**. However, there was an increase in the activity of metabolic pathways associated with the biosynthesis of LPS and their structural proteins **[16]**. These findings reinforce that sleep deprivation not only affects microbial composition but also compromises essential metabolic functions **[16]**. The microbial changes observed after 48 hours of sleep deprivation were normalized or close to normal after a week of recovery sleep **[16]**. However, functional changes in the microbiota persisted even after the recovery period **[16]**.

Changes in urinary metabolites of sleep-deprived mice were measured, and alterations, primarily in energy metabolism pathways, were observed **[7]**. Bacteria from the genera *Oscillospira*, *Parabacteroides*, *Ruminococcus*, *Phascolarctobacterium*, and *Aggregatibacter* were associated with the metabolic changes identified **[7]**.

In humans, the results observed were similar, as both acute and chronic sleep deprivation caused alterations in the fecal microbiome as well as in inflammatory and metabolic parameters **[15,18]**. Forty hours of sleep deprivation in young individuals resulted in reduced alpha and beta diversity of the gut microbiota and a decrease in the abundance of butyrate-producing bacteria **[15]**. Another study, conducted with university students, showed that acute sleep deprivation for 24 hours led to changes in alpha and beta diversities of the gut microbiota **[18]**. Microbial composition was also affected, with a reduction in the abundance of bacteria from the phyla Bacteroidetes and Actinobacteria, while members of the phyla Firmicutes and Proteobacteria increased **[18]**.

Pro-inflammatory cytokines IL-6, IL-1β, and TNF-α were elevated after 40 hours of sleep deprivation, while IL-10 decreased **[15]**. To confirm the involvement of the gut microbiota in the inflammation triggered by sleep deprivation, an experimental phase was conducted with Specific Pathogen-Free (SPF) and Germ-Free (GF) mice **[15]**. It was observed that 24 hours of sleep deprivation in SPF mice led to increased IL-6 and TNF-α levels, along with reduced IL-10 **[15]**. These effects were not observed in GF animals after sleep deprivation, indicating that the gut microbiota plays a fundamental role in the deleterious effects of sleep deprivation **[15]**.

Serum endotoxins were elevated in SPF mice after sleep deprivation, whereas no such increase was observed in GF mice **[15]**. When a solution containing fecal microbiota from sleep-deprived human participants was administered to GF mice, the animals exhibited increased concentrations of endotoxins, IL-1β, IL-6, and TNF-α, along with a reduction in IL-10, compared with GF mice inoculated with baseline microbiota from participants (before deprivation) **[15]**.

Alterations in the gut microbiota were accompanied by reduced production of short-chain fatty acids, with a decrease in fecal butyrate levels **[15,18]**. This was further associated with a reduction in the abundance of butyrate-producing bacteria **[15]** and with increased serum endotoxins in mice subjected to 72 hours of sleep deprivation **[18]**. Another parameter indicating impairment of the intestinal barrier was the reduction in the levels of tight junction proteins, such as ZO-1, claudin-1, and occludin **[18]**.

In addition to acute deprivation models, studies in humans and mice have also investigated chronic sleep deprivation and sleep restriction, and the results were similar, showing that chronic sleep deprivation likewise affects metabolic and immune functions [18,19,54]. A group of participants was subjected to sleep restriction, being limited to only 5 hours of sleep per day for a period of one week [18]. In this group, no significant alterations in the gut microbiota were observed; however, there was a reduction in the bacterial genera *Faecalibacterium* and *Bifidobacterium*, which include important producers of short-chain fatty acids [18].

Another study showed different results; however, participants were restricted to 4.25 hours of sleep per day for two days [54]. After this period of deprivation, significant changes in the gut microbiota were observed, including an increase in the Firmicutes-to-Bacteroidetes ratio, a shift associated with obesity and insulin resistance [54]. The families Coriobacteriaceae and Erysipelotrichaceae were also increased, both of which have been linked to metabolic disorders [54]. In addition to these changes, there was a reduction in the abundance of the phylum Tenericutes, a group associated with metabolic regulation [54]. After the period of sleep deprivation, a 40% increase in insulin resistance was also observed, measured by the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index, along with a 22% reduction in postprandial insulin sensitivity, measured by the Matsuda index [54]. These results demonstrate that the alterations caused by sleep deprivation interfere with energy metabolism, predisposing patients to metabolic disorders [54].

In mice, partial sleep deprivation for 6 hours per day over a period of 8 weeks caused alterations in the gut microbiota, with significant increases in the families/taxa Tannerellaceae, Rhodospirillales, *Alistipes*, and *Parabacteroides* [19]. These changes were accompanied by increases of 69.28%, 61.18%, and 50.76% in the cytokines IL-1β, IL-6, and TNF-α, respectively, showing a significant difference ($p < 0.001$) compared with animals with regular sleep [19].

In the experiment, a positive correlation was observed between IL-1β and the abundance of Muribaculaceae ($r = 0.497$, $p < 0.05$), and a negative correlation with the abundance of Lachnospiraceae ($r = -0.583$, $p < 0.05$) [19]. TNF-α, in turn, showed positive correlations with the abundance of Erysipelotrichaceae, Burkholderiaceae, and Tannerellaceae ($r = 0.492$; $r = 0.646$; $r = 0.726$; all $p < 0.05$) [19]. The cognitive status of the animals after sleep deprivation was also evaluated using the Morris water maze test, which assesses spatial learning and memory in mice [19]. Sleep-deprived animals performed worse in the test, indicating cognitive deficits [19].

Another interesting finding from studies evaluating the relationship between sleep disorders and the gut microbiota is that the use of probiotics may serve as a tool to improve sleep quality and to reverse deleterious effects, such as cognitive deficits and inflammation caused by sleep deprivation [3,26,46,47]. Currently, the main strains used in humans for sleep deprivation interventions include *Levilactobacillus brevis*, *Bifidobacterium*, *Lacticaseibacillus paracasei*, and *Lactiplantibacillus plantarum*, among others [3].

3.5. Influence of Gut Microbiota and Sleep on the Health and Welfare of Horses

Companion animals are following the increasing trend of obesity observed in humans [55]. Obesity and overweight affect between 25% and 50% of cats, dogs, and horses in developed countries, conditions that are directly related to the development of metabolic and inflammatory diseases, as well as to the impairment of animal welfare [55].

In horses, bacteria of the genus *Butyrivibrio* and of the family Prevotellaceae have been associated with obesity [56]. In addition, obese animals showed a higher abundance of bacteria from the phylum Firmicutes and a lower abundance of the phylum Bacteroidetes compared with lean or normal-weight horses [56]. This imbalance, characterized by an increased Firmicutes-to-Bacteroidetes ratio, is also observed in obese humans [56].

Insulin resistance is an important factor linked to obesity and the development of metabolic syndrome [55]. Although obese horses did not present insulin levels significantly different from those found in lean or normal-weight animals, a positive correlation between plasma insulin and glucose concentrations and obesity was observed, indicating a state of insulin resistance [56]. Obese horses also showed higher levels of glucose, triglycerides, and leptin, reinforcing an altered metabolic profile associated with obesity [56].

Equine metabolic syndrome (EMS) is characterized by insulin resistance and obesity and resembles human metabolic syndrome [27]. It includes predisposing components related to physical inactivity and diets high in sugars and starch, which are determining factors for the development of obesity [57]. The gut microbiota of horses with EMS was compared with that of healthy animals maintained under the same dietary conditions [27]. Horses with EMS showed lower representation of the genus *Fibrobacter* and reduced microbial diversity, an alteration associated with obesity and metabolic changes [58]. In contrast, healthy animals displayed greater abundance of the genus *Fibrobacter* and the family Ruminococcaceae, microorganisms linked to the digestion of dietary fibers and the production of SCFAs, such as butyrate, which is beneficial for intestinal health [27].

The gut microbiota has been implicated in the onset of important gastrointestinal disorders such as colic syndrome, colitis, gastric ulcers, and diarrhea [59]. Colic syndrome is one of the most common conditions in horses [60] and represents an important cause of mortality in the species. Nevertheless, its etiopathogenesis remains not fully understood [59,60], and the role of the gut microbiota in this process has become the focus of increasing investigation [60].

Studies have shown that the gut microbiota of horses with colic syndrome differs significantly from that of healthy animals [59–61]. One such study reported a reduction in microbial diversity, a decrease in *Fibrobacter* populations, and an increase in *Streptococcus*, the latter being associated with adverse health effects in other species [16,60].

Alterations in the equine gut microbiota have been observed both during episodes of abdominal pain and throughout recovery, and they appear to vary according to the primary cause of colic [59,61]. A key question that

remains unresolved is whether these microbial changes are a cause or a consequence of the disease, a topic that warrants further investigation [61].

The gut microbiota has also been associated with the development of stereotypies in horses, strengthening the hypothesis of the existence of the gut–brain axis in this species [13,29]. In horses with crib-biting behavior, a reduction in bacteria of the Bacteroidales family and an increase in the phyla Bacillota and Clostridia were observed compared with animals without behavioral disorders [29]. In addition to alterations in microbial composition, metabolic changes in the intestinal community were also reported, with the crib-biting group showing increased activity of pathways related to the biosynthesis of amino acids such as L-methionine and L-lysine [29].

Another study reported similar results, demonstrating that oral and locomotion-related stereotypies were associated with gut microbiota composition, with approximately 24% of oral stereotypies and 16.2% of locomotion-related stereotypies explained by the microbial differences observed in the study animals [13]. The study also revealed that 13% of aggressive behaviors and 9% of hypervigilance episodes were linked to variations in the gut microbiota [13]. These findings reinforce the influence of the gut microbiota on behavioral aspects and welfare indicators in horses.

The replacement of hay with oats in the diet of ponies resulted in a significant increase in recumbency time and total sleep, with a 20% increase in the duration of recumbency and sleep [62]. The proposed explanation for this effect is that an oat-based diet provides less cerebral stimulation originating from the gastrointestinal tract, thereby reducing the activity of the reticular substance and favoring the induction and maintenance of sleep [62]. This study suggested the existence of a communication pathway between the gastrointestinal tract and the brain in horses; however, the possible involvement of the microbiota in this process was not addressed.

Sleep is closely linked to welfare in horses and may serve as an indicator of well-being. Sleep deprivation can reduce welfare, which in turn may directly interfere with sleep [14]. Horses with reduced REM sleep exhibited less time spent in recumbency and lower locomotor activity, suggesting impaired welfare [63]. REM sleep deprivation also appears to impair the cognitive function of horses. In one study, animals subjected to 72 hours of REM sleep deprivation showed a trend ($p = 0.08$) toward requiring more time to complete spatial memory tasks, commonly used to assess cognition, suggesting a decline in cognitive performance [28]. In the same study, the authors further reported that sleep deprivation may reduce horses' concentration during exercise and contribute to fatigue [37].

Although no studies have yet directly investigated the interaction between sleep disorders and the gut microbiota in horses, evidence from research in humans and rodents indicates that this interaction may play a relevant role in modulating inflammation and metabolism, as well as serve as a risk factor for the development of metabolic and inflammatory diseases. Furthermore, studies conducted in horses suggest that both sleep and gut microbiota composition are associated with the occurrence of metabolic, behavioral, and cognitive disorders, as well as with the maintenance of health and welfare in these animals.

4. Conclusion

Sleep and gut microbiota are fundamental for maintaining physiological homeostasis, playing crucial roles in metabolism and physiological balance. Evidence supports a bidirectional relationship, in which sleep deprivation disrupts gut microbial balance, triggering metabolic changes and low-grade systemic inflammation associated with disease development, while the microbiota also contributes to sleep regulation. Although no specific studies have yet been conducted in horses, indications suggest that, as in humans, sleep and gut microbiota influence health, welfare, and important physiological functions in these animals, supporting the presence of the gut–brain axis in this species. This review highlights the need for such research, given the potential implications for equine health, welfare, and athletic performance.

Authors' Contributions

Conceptualization: A.R.C.G.; Methodology: A.R.C.G., T.M.O., and R.Y.A.B.; Selection and analysis of articles: A.R.C.G.; Writing—original draft: A.R.C.G.; Writing—review & editing, and translation: A.R.C.G., T.M.O., and G.G.C.; Review: A.R.C.G., T.M.O., R.Y.A.B., and G.G.C. All authors have read and agreed to the published version of the manuscript.

Data Availability

Not applicable to the present study, as it is a literature review that did not involve the generation of new data.

Conflicts of interest

The authors declare no conflicts of interest.

Funding

This research received no external funding.

Ethical Approval

Not applicable, as the study consists of a literature review of previously published works.

Declaration of Generative AI and AI-Assisted Technologies

During the preparation of this work, the authors used ChatGPT version 5.1 for the purpose of translating the text from Portuguese into American English. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

References

[1] Baranwal N, Yu PK, Siegel NS. Sleep physiology, pathophysiology, and sleep hygiene. Progress in Cardiovascular Diseases 2023;77:59–69. https://doi.org/10.1016/j.pcad.2023.02.005.

[2] Abrams RM. Sleep deprivation. Obstetrics and Gynecology Clinics of North America 2015;42:493–506. https://doi.org/10.1016/j.ogc.2015.05.013.

[3] Chen H, Wang C, Bai J, Song J, Bu L, Liang M, *et al*. Targeting microbiota to alleviate the harm caused by sleep deprivation. Microbiological Research 2023;275:127467. https://doi.org/10.1016/j.micres.2023.127467.

[4] Liew SC, Aung T. Sleep deprivation and its association with diseases- a review. Sleep Medicine 2021;77:192–204. https://doi.org/10.1016/j.sleep.2020.07.048.

[5] Davies SK, Ang JE, Revell VL, Holmes B, Mann A, Robertson FP, *et al*. Effect of sleep deprivation on the human metabolome. Proceedings of the National Academy of Sciences 2014;111:10761–6. https://doi.org/10.1073/pnas.1402663111.

[6] Ma W, Zhou M, Gou X, Zhao L, Cen F, Xu Y, *et al*. Urinary metabolomic study of chlorogenic acid in a rat model of chronic sleep deprivation using gas chromatography-mass spectrometry. International Journal of Genomics 2018;2018:1–11. https://doi.org/10.1155/2018/1361402.

[7] Ma W, Song J, Wang H, Shi F, Zhou N, Jiang J, *et al*. Chronic paradoxical sleep deprivation-induced depressionlike behavior, energy metabolism and microbial changes in rats. Life Sciences 2019;225:88–97. https://doi.org/10.1016/j.lfs.2019.04.006.

[8] So-ngern A, Chirakalwasan N, Saetung S, Chanprasertyothin S, Thakkinstian A, Reutrakul S. Effects of two-week sleep extension on glucose metabolism in chronically sleep-deprived individuals. Journal of Clinical Sleep Medicine 2019;15:711–8. https://doi.org/10.5664/jcsm.7758.

[9] Hu S, Li P, Zhang R, Liu X, Wei S. Integrated metabolomics and proteomics analysis reveals energy metabolism disorders in the livers of sleep-deprived mice. Journal of Proteomics 2021;245:104290. https://doi.org/10.1016/j.jprot.2021.104290.

[10] Singh T, Ahmed TH, Mohamed N, Elhaj MS, Mohammed Z, Paulsingh CN, *et al*. Does insufficient sleep increase the risk of developing insulin resistance: a systematic review. Cureus 2022;14:e23501. https://doi.org/10.7759/cureus.23501.

[11] Souza JFT, Monico-Neto M, Tufik S, Antunes HKM. Sleep debt and insulin resistance: what's worse, sleep deprivation or sleep restriction? Sleep Science 2024;17:e272–80. https://doi.org/10.1055/s-0044-1782173.

[12] Aleman M, Williams DC, Holliday T. Sleep and sleep disorders in horses. AAEP Proceedings 2008;54:180–5.

[13] Mach N, Ruet A, Clark A, Bars-Cortina D, Ramayo-Caldas Y, Crisci E, *et al*. Priming for welfare: gut microbiota is associated with equitation conditions and behavior in horse athletes. Scientific Reports 2020;10:8311. https://doi.org/10.1038/s41598-020-65444-9.

[14] Greening L, McBride S. A review of equine sleep: implications for equine welfare. Frontiers in Veterinary Science 2022;9:916737. https://doi.org/10.3389/fvets.2022.916737.

[15] Wang Z, Chen W-H, Li S-X, He Z-M, Zhu W-L, Ji Y-B, *et al*. Gut microbiota modulates the inflammatory response and cognitive impairment induced by sleep deprivation. Molecular Psychiatry 2021;26:6277–92. https://doi.org/10.1038/s41380-021-01113-1.

[16] Wang Q, Chen B, Sheng D, Yang J, Fu S, Wang J, *et al*. Multiomics analysis reveals aberrant metabolism and immunity linked gut microbiota with insomnia. Microbiology Spectrum 2022;10:e00998-22. https://doi.org/10.1128/spectrum.00998-22.

[17] Yang D-F, Huang W-C, Wu CW, Huang C-Y, Yang Y-CSH, Tung Y-T. Acute sleep deprivation exacerbates systemic inflammation and psychiatry disorders through gut microbiota dysbiosis and disruption of circadian rhythms. Microbiological Research 2023;268:127292. https://doi.org/10.1016/j.micres.2022.127292.

[18] Gao T, Wang Z, Dong Y, Cao J, Chen Y. Butyrate ameliorates insufficient sleep-induced intestinal mucosal damage in humans and mice. Microbiology Spectrum 2023;11:e02000-22. https://doi.org/10.1128/spectrum.02000-22.

[19] Zhang M, Zhang M, Kou G, Li Y. The relationship between gut microbiota and inflammatory response, learning and memory in mice by sleep deprivation. Frontiers in Cellular and Infection Microbiology 2023;13:1159771. https://doi.org/10.3389/fcimb.2023.1159771.

[20] Lozupone CA, Stombaugh JI, Gordon JI, Jansson JK, Knight R. Diversity, stability and resilience of the human gut microbiota. Nature 2012;489:220–30. https://doi.org/10.1038/nature11550.

[21] Adak A, Khan MR. An insight into gut microbiota and its functionalities. Cellular and Molecular Life Sciences 2019;76:473–93. https://doi.org/10.1007/s00018-018-2943-4.

[22] Chen Y, Zhou J, Wang L. Role and mechanism of gut microbiota in human disease. Frontiers in Cellular and Infection Microbiology 2021;11:625913. https://doi.org/10.3389/fcimb.2021.625913.

[23] Patterson E, Griffin SM, Ibarra A, Ellsiepen E, Hellhammer J. Lacticaseibacillus paracasei Lpc-37® improves psychological and physiological markers of stress and anxiety in healthy adults: a randomized, double-blind, placebo-controlled and parallel clinical trial (the Sisu study). Neurobiology of Stress 2020;13:100277. https://doi.org/10.1016/j.ynstr.2020.100277.

[24] Sen P, Molinero-Perez A, O'Riordan KJ, McCafferty CP, O'Halloran KD, Cryan JF. Microbiota and sleep: awakening the gut feeling. Trends in Molecular Medicine 2021;27:935–45. https://doi.org/10.1016/j.molmed.2021.07.004.

[25] Badrfam R, Zandifar A, Hajialigol A, Rashidian M, Schmidt NB, Morabito D, *et al*. Efficacy of probiotic supplements in improving the symptoms of psychosis, anxiety, insomnia, and anorexia due to amphetamine and methamphetamine use: a randomized clinical trial. Psychopharmacology 2024;241:1463–76. https://doi.org/10.1007/s00213-024-06577-x.

[26] Ito H, Tomura Y, Kitagawa Y, Nakashima T, Kobanawa S, Uki K, *et al*. Effects of probiotics on sleep parameters: a systematic review and meta-analysis. Clinical Nutrition ESPEN 2024;63:623–30. https://doi.org/10.1016/j.clnesp.2024.07.006.

[27] Elzinga SE, Weese JS, Adams AA. Comparison of the fecal microbiota in horses with equine metabolic syndrome and metabolically normal controls fed a similar all-forage diet. Journal of Equine Veterinary Science 2016;44:9–16. https://doi.org/10.1016/j.jevs.2016.05.010.

[28] Barbosa ÂP, Oliveira TM, Trindade PHE, Seidel SRT, Tokawa PKA, Jaramilo FM, *et al*. Sleep pattern interference in the cognitive performance of Lusitano horses. Animals 2024;14:334. https://doi.org/10.3390/ani14020334.

[29] Martínez-Aranzales JR, Córdoba-Agudelo M, Pérez-Jaramillo JE. Fecal microbiome and functional prediction profiles of horses with and without crib-biting behavior: A comparative study. Journal of Equine Veterinary Science 2024;142:105198. https://doi.org/10.1016/j.jevs.2024.105198.

[30] Roever L, Resende ES, Diniz ALD, Penha-Silva N, O'Connell JL, Gomes PFS, *et al.* Metabolic syndrome and risk of stroke. Medicine 2018;97:e9862. https://doi.org/10.1097/md.0000000000009862.

[31] Brockmeier AJ, Ju M, Przybyła P, Ananiadou S. Improving reference prioritisation with PICO recognition. BMC Medical Informatics and Decision Making 2019;19:256. https://doi.org/10.1186/s12911-019-0992-8.

[32] Carley DW, Farabi SS. Physiology of sleep. Diabetes Spectrum 2016;29:5–9. https://doi.org/10.2337/diaspect.29.1.5.

[33] Oliveira T, Santos A, Silva J, Trindade P, Yamada A, Jaramillo F, *et al.* Hospitalisation and disease severity alter the resting pattern of horses. Journal of Equine Veterinary Science 2022;110:103832. https://doi.org/10.1016/j.jevs.2021.103832.

[34] Oliveira TM, Zanotto GM, Trindade PHE, Baccarin RYA. Environment change causes a transient adverse impact on horse behavioral resting patterns. Applied Animal Behaviour Science 2025;282:106482. https://doi.org/10.1016/j.applanim.2024.106482.

[35] Soltani S, Chauvette S, Bukhtiyarova O, Lina J-M, Dubé J, Seigneur J, *et al.* Sleep–wake cycle in young and older mice. Frontiers in Systems Neuroscience 2019;13:51. https://doi.org/10.3389/fnsys.2019.00051.

[36] Aulehner K, Bray J, Koska I, Pace C, Palme R, Kreuzer M, *et al.* The impact of tethered recording techniques on activity and sleep patterns in rats. Scientific Reports 2022;12:3179. https://doi.org/10.1038/s41598-022-06307-3.

[37] Sharma S, Kavuru M. Sleep and metabolism: An overview. International Journal of Endocrinology 2010;2010:1–12. https://doi.org/10.1155/2010/270832.

[38] Williams DC, Aleman M, Holliday TA, Fletcher DJ, Tharp B, Kass PH, *et al.* Qualitative and quantitative characteristics of the electroencephalogram in normal horses during spontaneous drowsiness and sleep. Journal of Veterinary Internal Medicine 2008;22:630–8. https://doi.org/10.1111/j.1939-1676.2008.0096.x.

[39] Garbarino S, Lanteri P, Bragazzi NL, Magnavita N, Scoditti E. Role of sleep deprivation in immune-related disease risk and outcomes. Communications Biology 2021;4:1304. https://doi.org/10.1038/s42003-021-02825-4.

[40] Barbosa ÂP, Oliveira TM, Seidel SRT, Tokawa PKA, Jaramillo FM, Roncati NV, *et al.* Could restricting horses' REM sleep improve dressage performance? Applied Animal Behaviour Science 2025;291:106740. https://doi.org/10.1016/j.applanim.2025.106740.

[41] Mun JK, Choi SJ, Kang M-R, Hong SB, Joo EY. Sleep and libido in men with obstructive sleep apnea syndrome. Sleep Medicine 2018;52:158–62. https://doi.org/10.1016/j.sleep.2018.07.016.

[42] Su L, Zhang S, Zhu J, Wu J, Jiao Y. Effect of partial and total sleep deprivation on serum testosterone in healthy males: a systematic review and meta-analysis. Sleep Medicine 2021;88:267–73. https://doi.org/10.1016/j.sleep.2021.10.031.

[43] Shah PA, Park CJ, Shaughnessy MP, Cowles RA. Serotonin as a mitogen in the gastrointestinal tract: revisiting a familiar molecule in a new role. Cellular and Molecular Gastroenterology and Hepatology 2021;12:1093–104. https://doi.org/10.1016/j.jcmgh.2021.05.008.

[44] Poza JJ, Pujol M, Ortega-Albás JJ, Romero O. Melatonin in sleep disorders. Neurología (English Edition) 2022;37:575–85. https://doi.org/10.1016/j.nrleng.2018.08.004.

[45] Hou Y, Li J, Ying S. Tryptophan metabolism and gut microbiota: a novel regulatory axis integrating the microbiome, immunity, and cancer. Metabolites 2023;13:1166. https://doi.org/10.3390/metabo13111166.

[46] Tian P, Chen Y, Zhu H, Wang L, Qian X, Zou R, *et al.* Bifidobacterium breve CCFM1025 attenuates major depression disorder via regulating gut microbiome and tryptophan metabolism: A randomized clinical trial. Brain, Behavior, and Immunity 2022;100:233–41. https://doi.org/10.1016/j.bbi.2021.11.023.

[47] Lee D-Y, Baek J-S, Shin Y-J, Kim D-H. Alleviation of immobilization stress or fecal microbiota-induced insomnia and depression-like behaviors in mice by Lactobacillus plantarum and its supplement. Nutrients 2024;16:3711. https://doi.org/10.3390/nu16213711.

[48] Szentirmai É, Millican NS, Massie AR, Kapás L. Butyrate, a metabolite of intestinal bacteria, enhances sleep. Scientific Reports 2019;9:7035. https://doi.org/10.1038/s41598-019-43502-1.

[49] Firoozi D, Masoumi SJ, Mohammad-Kazem Hosseini Asl S, Labbe A, Razeghian-Jahromi I, Fararouei M, *et al.* Effects of short-chain fatty acid-butyrate supplementation on expression of circadian-clock genes, sleep quality, and inflammation in patients with active ulcerative colitis: a double-blind randomized controlled trial. Lipids in Health and Disease 2024;23:216. https://doi.org/10.1186/s12944-024-02203-z.

[50] Schoster A. Probiotic use in equine gastrointestinal disease. Veterinary Clinics of North America: Equine Practice 2018;34:13–24. https://doi.org/10.1016/j.cveq.2017.11.004.

[51] Moyseos M, Michael J, Ferreira N, Sophocleous A. The effect of probiotics on the management of pain and inflammation in osteoarthritis: a systematic review and meta-analysis of clinical studies. Nutrients 2024;16:2243. https://doi.org/10.3390/nu16142243.

[52] Costa MC, Weese JS. Understanding the intestinal microbiome in health and disease. Veterinary Clinics of North America: Equine Practice 2018;34:1–12. https://doi.org/10.1016/j.cveq.2017.11.005.

[53] Valentino V, De Filippis F, Marotta R, Pasolli E, Ercolini D. Genomic features and prevalence of Ruminococcus species in humans are associated with age, lifestyle, and disease. Cell Reports 2024;43:115018. https://doi.org/10.1016/j.celrep.2024.115018.

[54] Benedict C, Vogel H, Jonas W, Woting A, Blaut M, Schürmann A, *et al.* Gut microbiota and glucometabolic alterations in response to recurrent partial sleep deprivation in normal-weight young individuals. Molecular Metabolism 2016;5:1175–86. https://doi.org/10.1016/j.molmet.2016.10.003.

[55] Ragno VM, Zello GA, Klein CD, Montgomery JB. From table to stable: a comparative review of selected aspects of human and equine metabolic syndrome. Journal of Equine Veterinary Science 2019;79:131–8. https://doi.org/10.1016/j.jevs.2019.06.003.

[56] Biddle AS, Tomb J-F, Fan Z. Microbiome and blood analyte differences point to community and metabolic signatures in lean and obese horses. Frontiers in Veterinary Science 2018;5:225. https://doi.org/10.3389/fvets.2018.00225.

[57] Johnson PJ, Wiedmeyer CE, LaCarrubba A, Ganjam VKS, Messer NT. Diabetes, Insulin Resistance, and Metabolic Syndrome in Horses. J Diabetes Sci Technol 2012;6:534–40. https://doi.org/10.1177/193229681200600307.

[58] Turnbaugh PJ, Hamady M, Yatsunenko T, Cantarel BL, Duncan A, Ley RE, *et al*. A core gut microbiome in obese and lean twins. Nature 2009;457:480–4. https://doi.org/10.1038/nature07540.

[59] Chaucheyras-Durand F, Sacy A, Karges K, Apper E. Gastro-intestinal microbiota in equines and its role in health and disease: the black box opens. Microorganisms 2022;10:2517. https://doi.org/10.3390/microorganisms10122517.

[60] Thomson P, Garrido D, Santibáñez R, Lara F. Preliminary functional analysis of the gut microbiome in colic horses. Animals 2024;14:3222. https://doi.org/10.3390/ani14223222.

[61] Lara F, Castro R, Thomson P. Changes in the gut microbiome and colic in horses, are they cause or consequence? Open Veterinary Journal 2022;12:242. https://doi.org/10.5455/ovj.2022.v12.i2.12.

[62] Dallaire A, Rucklebusch Y. Sleep and wakefulness in the housed pony under different dietary conditions. Canadian Journal of Comparative Medicine 1974;38:65–71.

[63] Kelemen Z, Grimm H, Long M, Auer U, Jenner F. Recumbency as an equine welfare indicator in geriatric horses and horses with chronic orthopaedic disease. Animals 2021;11:3189. https://doi.org/10.3390/ani11113189.

How to Cite
Gomes ARC, Oliveira TM, Campos GG, Baccarin RYA. Gut Microbiota and Sleep Deprivation: Potential Interactions and Implications for Equine Research – Scoping Review. Int J Equine Sci 2025;4(S1):38–48.