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Original Research Article

POTENTIAL ROLE OF MICROBIOTA–GUT–BRAIN AXIS DYSFUNCTION IN PROCRASTINATION-LIKE BEHAVIOR IN DIABETES

Authors: Happy Isibor[1] and Celestine Oghenеruro Akpovwre[2]

[1]Department of Pharmacology, Faculty of Allied Health Sciences, Delta State University, Abraka.

[2]Department of Pharmacology and Therapeutics, Basic Clinical Sciences, Edo State University, Iyamho.

Corresponding Author: Happy Isibor
Email: happy-isibor@delsu.edu.ng,
https://orcid.org/0000-0002-0582-440X,
+2347036261290

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ABSTRACT
Procrastination, the voluntary delay of intended actions despite anticipated negative consequences, represents a significant behavioral barrier to effective diabetes self-management. While traditionally conceptualized as a psychological or personality-related trait, emerging evidence suggests that procrastination-like behaviors in diabetes may have neurobiological underpinnings rooted in the pathophysiology of the disease itself. This review synthesizes current evidence linking microbiota–gut–brain axis (MGBA) dysfunction to the triad of dopaminergic signaling deficits, neuroinflammation, and cognitive impairment that collectively may drive procrastination-like behaviors in diabetic patients. We proposed a mechanistic framework whereby diabetes-induced gut dysbiosis triggers systemic and neuroinflammation via microbial metabolite dysregulation and increased intestinal permeability, which in turn disrupts central dopaminergic reward circuitry and impairs executive functions, particularly cognitive flexibility, inhibition, and planning. These converging pathologies may contribute to a neurobehavioral state characterized by reduced motivational drive, impaired self-regulation, and diminished capacity for goal-directed behavior, manifesting clinically as health-related procrastination. Understanding this axis offers novel therapeutic opportunities targeting the gut microbiome, inflammatory pathways, and dopaminergic signaling to improve behavioral outcomes in diabetes care.

Keywords: Microbiota–gut–brain axis, diabetes, procrastination, dopamine, neuroinflammation, cognitive impairment, executive function, self-management

INTRODUCTION

Type 2 diabetes mellitus (T2DM) affects approximately 600 million people worldwide, with projections approaching 800 million by 2030. Beyond its well-established peripheral metabolic complications, diabetes is increasingly recognized as a condition that profoundly affects brain function (Erhunmwunse *et al*., 2025; Malih, 2026). Patients with diabetes face unique challenges in adopting and maintaining healthy behaviors, a reality that has profound implications for disease outcomes. Effective diabetes management relies heavily on executive functions: the cognitive capacities that enable planning, inhibition of impulses, cognitive flexibility, and goal-directed behavior (Shanley *et al*., 2026). Yet diabetes itself is associated with impairments in these very functions, creating what has been described as a "double whammy": patients have greater need for executive control while possessing fewer neural resources to exert it (Stajkovic & Stajkovic, 2025). Procrastination, the delay of intended actions despite awareness of negative consequences, represents a critical behavioral manifestation of this neurocognitive vulnerability (Amalia *et al*., 2026; Hu *et al*., 2026). Health-related procrastination in patients with T2DM has been identified as a multidimensional process that begins when patients perceive the hardships of self-care as their primary concern (Liu *et al*., 2025). This procrastination manifests across multiple health-related dimensions and is particularly evident in self-care and treatment adherence (Far *et al*., 2025). The consequences are clinically significant: oscillatory health-related procrastination leads to mental harm, improper disease control, and progressive "health deficit" (Merie *et al*., 2026). Traditional models of procrastination have emphasized psychological factors such as poor self-regulation, fear of failure, and task aversiveness (Merie *et al*., 2026). However, a growing body of evidence suggests that procrastination-like behaviors in diabetes may be driven, at least in part, by neurobiological alterations induced by the disease itself. This review advances a novel integrative framework: that microbiota–gut–brain axis dysfunction operating through disruptions in dopaminergic signaling, neuroinflammation, and cognitive impairment constitutes a neurobiological driver of procrastination-like behavior in diabetes.

Diabetes-Associated Cognitive Impairment: The Executive Function Deficit

Executive Dysfunction in Diabetes

Large-scale epidemiological, longitudinal, and neuroimaging studies consistently demonstrate accelerated cognitive aging and increased dementia risk in individuals with T2DM (Lucia *et al*., 2025; Zeng *et al*., 2025). A comprehensive meta-analysis of 60 studies comparing 9,815 individuals with T2DM to 69,254 controls found that diabetes is associated with worse performance on cognitive tests measuring executive functions the abilities involved in the control of emotions, behaviors, and thought (Ahmed *et al*., 2025). Executive functions inhibit habitual thinking patterns, knee-jerk emotional reactions, and reflexive behaviors (Buck, 2025). Critically executive functions, particularly cognitive flexibility, are disproportionately affected in diabetes, independent of overt cerebrovascular disease (Mehta & Jayanna, 2025; Yu *et al*., 2025). Studies using translational touchscreen-based paradigms in db/db mice, a genetic model of T2DM, have demonstrated that diabetic animals display intact associative learning but marked deficits in executive cognitive flexibility, characterized by increased perseveration and failure to adapt to changed reward contingencies (Alasousi *et al*., 2026; Ghosh-Swaby, 2025). This selective impairment pattern sparing basic learning while compromising higher-order cognitive control has direct relevance to procrastination, as the ability to flexibly adjust behavior in response to changing circumstances is essential for sustained self-management (Trajković *et al*., 2026).

Neural Substrates of Diabetes-Related Cognitive Dysfunction

The neural underpinnings of diabetes-associated cognitive impairment involve disruptions in critical brain regions for cognitive processing, particularly the prefrontal cortex and parietal lobe. These regions are central to executive functions including planning, decision-making, and inhibitory control (Liu *et al*., 2025; Yu *et al*., 2025). Diabetes induces alterations in brain function and plasticity that can lead to cognitive impairment, with cognitive impairments potentially hindering positive changes to healthy behaviors in diabetes care (Chen *et al*., 2025; Yu *et al*., 2025).

Hippocampal pathology is also prominent, with T2DM and inflammatory cytokines such as interleukin-6 (IL-6) associated with hippocampus atrophy, amyloid pathology, tau accumulation, and neurofilament pathology at preclinical stages of diabetes-related cognitive impairment (Gupta *et al*., 2025; Hallab *et al*., 2025). These structural changes provide a neural substrate for the executive dysfunction that predisposes to procrastination-like behaviors.

The Microbiota–Gut–Brain Axis in Diabetes

Gut Dysbiosis in Diabetes

The microbiota–gut–brain axis (MGBA) is a bidirectional communication network that integrates neural, endocrine, and immune pathways between the gastrointestinal tract and the central nervous system (Salam, 2025; Zhao *et al*., 2025). In diabetes, gut microbiota composition is profoundly altered by a state termed dysbiosis. This dysbiosis has been linked to increased intestinal permeability and systemic inflammation, which can exacerbate insulin resistance and cognitive impairment (Nikolaidis *et al*., 2025; Shafik *et al*., 2025). Gut microbial dysbiosis contributes to metabolic dysfunction through multiple mechanisms: altered production of microbial metabolites including short-chain fatty acids (SCFAs) and bile acids, increased translocation of lipopolysaccharide (LPS), and induction of systemic low-grade inflammation (Sharma *et al*., 2026). These microbial changes are not merely epiphenomena but actively participate in disease pathogenesis by modulating host metabolism, immunity, and neuroendocrine signaling (Chen *et al*., 2026).

Microbiota-Derived Signals and Neuroinflammation

The gut microbiome influences brain function through multiple pathways. Microbial metabolites, including SCFAs, can cross the blood–brain barrier and influence the production of neurotransmitters including dopamine and serotonin (Anih *et al*., 2025). Conversely, dysbiosis promotes neuroinflammation through several mechanisms: LPS and other microbial products activate Toll-like receptors (particularly TLR4) on glial cells, triggering NF-κB-mediated inflammatory cascades; SCFA depletion reduces anti-inflammatory signaling; and increased intestinal permeability allows bacterial products to enter the circulation, promoting systemic inflammation that secondarily affects the brain (Katsigianni & Koutsouraki, 2026; Sarkar *et al*., 2026). In diabetic cognitive impairment, chronic inflammation is a crucial pathogenic factor. Pterostilbene, a natural compound with anti-inflammatory properties, has been shown to alleviate diabetic cognitive dysfunction by suppressing the TLR4/NF-κB pathway through the MGBA, reducing both intestinal inflammation and neuroinflammation while improving gut microbiota homeostasis and increasing SCFA levels (Zhang *et al*., 2026; Zhou *et al*., 2026). Similarly, probiotic interventions such as Lactobacillus casei Zhang have been shown to alleviate T2DM-induced cognitive impairment by modulating the MGBA, restoring SCFA production (particularly butyrate), attenuating hippocampal inflammation, and normalizing hippocampal metabolism (Lawal *et al*., 2025).

The MGBA–Neuroinflammation–Cognition Axis in Diabetes

The link between gut dysbiosis and cognitive impairment in diabetes is increasingly well-established. Gut microbiota dysbiosis has been linked to increased intestinal permeability and systemic inflammation, which can exacerbate insulin resistance and cognitive impairment (Abildinova *et al*., 2024; Zhang *et al*., 2022). Altered microbial composition influences neurodegenerative and cognitive disorders through effects on MGBA signaling, with changes in microbial metabolites modulating neuroinflammation, microglial activation, and metabolic signaling along the axis, contributing to cognitive decline (Lei *et al*., 2025).

Recent studies have demonstrated that interventions targeting the MGBA can ameliorate diabetes-associated cognitive decline. Semaglutide, a glucagon-like peptide-1 receptor agonist, significantly alleviates cognitive impairment in diabetic mice by inhibiting hippocampal neuron loss, improving synaptic ultrastructure, mitigating neuroinflammation, and inducing significant alterations in gut microbiota composition (Si *et al*., 2025; Qi *et al*., 2026). These findings suggest a correlation between the protective effects against diabetes-associated cognitive decline and MGBA modulation.

Dopaminergic Signaling Dysfunction in Diabetes

Insulin and Dopamine: An Interlinked System

Central insulin and dopamine action are fundamentally interlinked, impacting food intake, reward, and mood (Kleinridders & Pothos, 2019; Wallace & Fordahl, 2022). Insulin receptors are highly expressed in the dopaminergic midbrain, particularly in the ventral tegmental area (VTA), the primary dopamine source region and the striatum (Uzay *et al*., 2026). Evidence from rodent and human studies has accumulated indicating that insulin modulates dopaminergic signaling and reward behavior (Tongta *et al*., 2025). Brain insulin resistance, a hallmark of T2DM directly alters central dopamine pathways, which may result in motivational deficits and depressive symptoms.

Insulin resistance in the brain causes hyperphagia, anxiety, and depressive-like behavior while compromising the dopaminergic system (Kontogianni, *et al*., 2026). Such effects can induce reduced compliance to medical treatment.

Dopaminergic Deficits and Reward Processing in Diabetes

In T2DM and insulin resistance, the brain's reward system can become less sensitive, with reduced dopamine release or blunted dopaminergic responses. This manifests clinically as diminished motivation, reduces anticipation of reward, and decreases satisfaction from goal-directed activities (Fanelli, 2025); Kleinridders & Pothos, 2019). Everyday tasks feel effortful and unrewarding, while quick, high-reward activities become more appealing. Structural and functional brain changes in T2DM are distributed throughout the principally dopaminergic reward circuitry, suggesting a neurobiological basis for motivational and decisional aspects of anhedonia (Lisco *et al*., 2023). Anhedonic symptoms, including motivational and decisional symptoms, may arise from neuroendocrine effects on dopaminergic reward pathways. This shift in effort-related reward choices has consequential effects on energy expenditure, self-care, and eating behaviors that directly affect T2DM outcomes (Lisco *et al*., 2023).

Inflammation-Induced Dopaminergic Dysfunction

Neuroinflammation and metabolic dysfunction impair dopamine neurotransmission, serving as a critical mechanism underpinning motivational deficits. Low-grade inflammation signals the brain to induce a sustained reduction of striatal dopamine motivation signaling. This inflammatory suppression of dopaminergic function represents a key pathway through which diabetes-associated neuroinflammation contributes to reduced motivation and procrastination-like behaviors (Felger & Treadway, 2017).

Integrating the Pathways: A Mechanistic Model of Procrastination-Like Behavior

From Gut Dysbiosis to Behavioral Dysregulation

Synthesizing the evidence presented above, we propose an integrated mechanistic model whereby microbiota–gut–brain axis (MGBA) dysfunction drives procrastination-like behavior in diabetes through a sequence of converging pathophysiological events. The process is initiated by the diabetic state itself, which induces a pathological shift in the gut microbiome characterized by reduced microbial diversity, depletion of short-chain fatty acid (SCFA)-producing bacteria, and increased abundance of pathobionts (Di Vincenzo *et al*., 2024). This diabetes-associated dysbiosis, in turn, compromises the integrity of the intestinal barrier, leading to increased permeability ("leaky gut") and facilitating the translocation of bacterial products such as lipopolysaccharides into the systemic circulation. These microbial components trigger a state of low-grade systemic inflammation, which, via both humoral and vagal neural pathways, culminates in neuroinflammation within the central nervous system—marked by microglial activation, astrocytosis, and elevated pro-inflammatory cytokines in brain regions critical for executive function and reward processing (Guo & Russo, 2026). The ensuing neuroinflammation directly disrupts dopaminergic signaling in the mesolimbic and neocortical pathways; compounded by central insulin resistance, this disruption reduces dopamine availability and blunts reward sensitivity (Kleinridders & Pothos, 2019). The combined effect of dopaminergic hypofunction and neuroinflammation is a measurable impairment of core executive functions particularly cognitive flexibility, inhibitory control, and planning which are precisely the cognitive capacities required for sustained diabetes self-management (Yu *et al*., 2025). Ultimately, this neurocognitive deficit state manifests behaviorally as procrastination: reduced motivation to initiate health-related actions, impaired ability to resist immediate gratifications in favor of long-term health goals, and diminished capacity to adapt self-management strategies to changing circumstances.

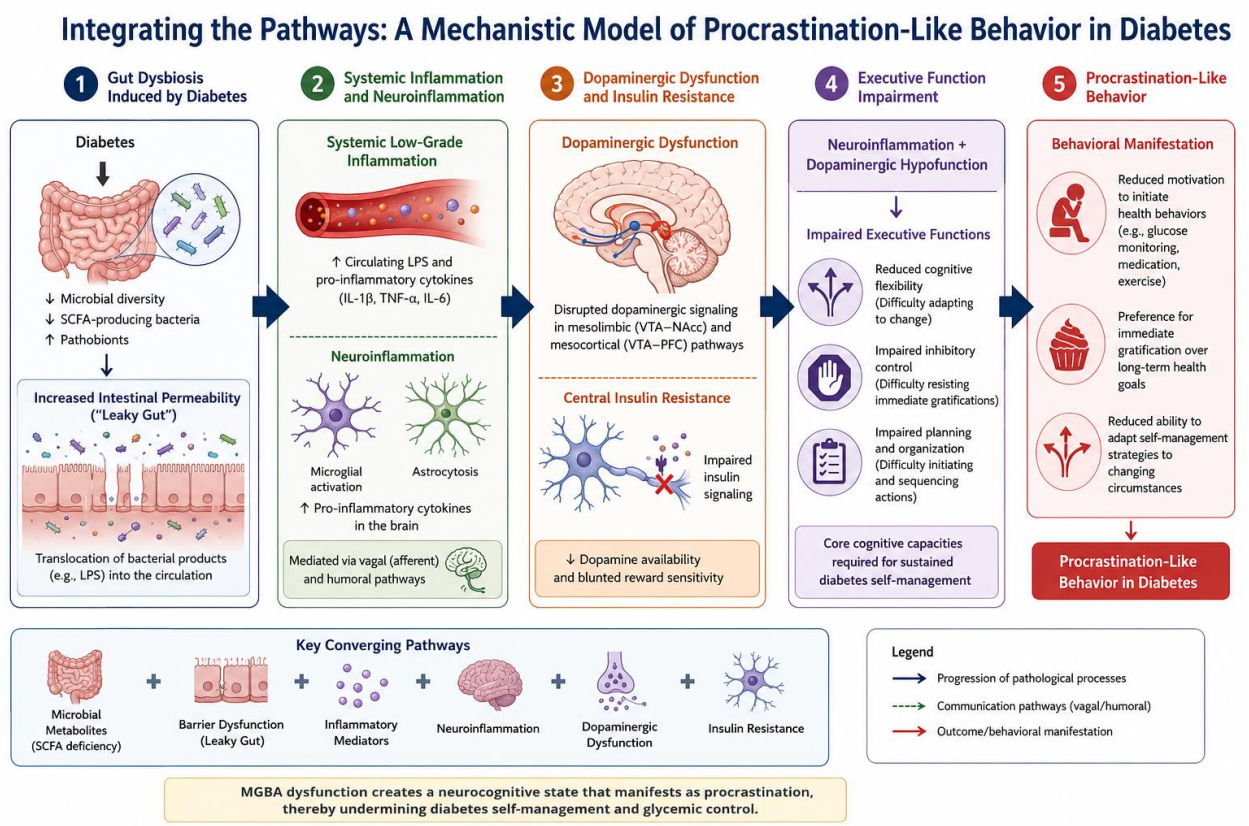

Figure 1: Proposed mechanistic model illustrating how diabetes-induced gut dysbiosis promotes procrastination-like behavior through the microbiota–gut–brain axis. Gut dysbiosis increases intestinal permeability and systemic inflammation, which trigger neuroinflammation and impair dopaminergic signaling and central insulin sensitivity. These changes disrupt executive functions, including cognitive flexibility, inhibitory control, and planning, ultimately reducing motivation for health-promoting behaviors, favoring immediate gratification, and impairing diabetes self-management.

The Procrastination Phenotype in Diabetes

Health-related procrastination in type 2 diabetes is not a simple character flaw but rather a multidimensional process shaped by multiple interacting factors. Its formation begins when patients perceive the hardships of self-care as their primary concern, with contributing elements including "the ominous shadow of the disease," family-related pressures, patient-specific factors, health system shortfalls, and sociocultural background (Shareinia *et al*., 2024). In response, patients often attempt to abandon diabetes self-care through strategies such as escaping the reality of the disease, self-medication, and procrastination itself (Mahetlane, 2025). Our sequential model suggests that these psychological and social factors do not operate in isolation but interact with underlying neurobiological vulnerabilities. The patient who experiences self-care as burdensome does so not only for psychological reasons; their neurocognitive apparatus compromised by MGBA dysfunction, neuroinflammation, and dopaminergic deficits is demonstrably less capable of sustaining the effortful, future-oriented behaviors that rigorous diabetes management requires. Thus, the pathway from diabetes to behavioral dysregulation is a coherent cascade, with each step logically giving rise to the next, and the final procrastination phenotype represents the behavioral endpoint of this neuro-immune-metabolic axis.

Therapeutic Implications and Future Directions

Targeting the Microbiota–Gut–Brain Axis

The MGBA offers multiple therapeutic entry points for addressing procrastination-like behaviors in diabetes. Probiotic interventions have shown promise: Lactobacillus casei Zhang has been demonstrated to alleviate T2DM-induced cognitive impairment by modulating the MGBA, restoring SCFA production, attenuating hippocampal inflammation, and normalizing hippocampal metabolites (Lawal *et al*., 2025). Dietary modifications, prebiotics, and fecal microbiota transplantation have also shown encouraging results in improving insulin sensitivity and cognitive function in both animal models and human studies (Abildinova *et al*., 2024). Pharmacological agents targeting the MGBA are emerging. Semaglutide, a GLP-1 receptor agonist, alleviates diabetes-associated cognitive decline through MGBA-mediated mechanisms, including modulation of gut microbiota composition, alteration of fecal and serum metabolites, and transcriptomic changes in brain tissue. Such agents may have dual benefits: improving metabolic control while simultaneously addressing the neurocognitive barriers to self-management (Si *et al*., 2025; Qi *et al*., 2026).

Modulating Neuroinflammation and Dopaminergic Function

Anti-inflammatory strategies may mitigate the neuroinflammation that drives dopaminergic dysfunction and cognitive impairment. Interventions that suppress TLR4/NF-κB signaling through the MGBA, such as pterostilbene, have shown efficacy in alleviating diabetic cognitive dysfunction (Zhang *et al*., 2023). However, as demonstrated in studies using the anti-inflammatory peptide ARA 290, improvement in peripheral metabolic function and altered immune cell proportions may be insufficient to restore executive cognition unless brain-specific immunometabolic mechanisms are targeted (Alasousi *et al*., 2026). Dopamine-targeted interventions including dopamine receptor agonists and strategies to enhance central insulin sensitivity may improve motivational drive and reward processing in diabetic patients. The highly adaptable nature of the reward system offers hope: as metabolic health improves and the brain's energy supply stabilizes, dopamine signaling begins to rebalance. Behavioral interventions that create "micro-rewards" through small, achievable actions can gradually strengthen new neural pathways and rebuild motivation (Fanelli, 2025).

Cognitive and Behavioral Interventions

Understanding the neurobiological basis of procrastination in diabetes does not diminish the importance of behavioral interventions rather, it informs their design. Interventions should be tailored to accommodate executive function deficits: simplifying self-care routines, providing external structure and reminders, breaking tasks into smaller steps, and reducing the cognitive load of diabetes management. Cognitive training and compensatory strategies may help address diabetes-related cognitive changes. Aerobic exercise and cognitively challenging activities learning new things, solving difficult puzzles help optimize the brain structures that support executive function. Importantly, these interventions may have dual benefits for both cognitive function and metabolic control.

Future Research Directions

Several critical questions remain. First, longitudinal studies are needed to establish causality in the proposed pathway from gut dysbiosis to procrastination-like behavior. Second, the specific microbial taxa and metabolites most influential in driving neurocognitive dysfunction in diabetes require further elucidation. Third, the relative contributions of different inflammatory pathways and their interactions with dopaminergic signaling need to be mapped at the mechanistic level. Fourth, clinical trials are needed to test whether MGBA-targeted interventions can reduce procrastination and improve diabetes self-management outcomes.

Limitations and Methodological Considerations

Several limitations of this narrative review warrant acknowledgment. First, the proposed pathway from MGBA dysfunction to procrastination-like behavior remains largely speculative, as direct experimental or clinical studies integrating all components of this cascade are lacking. The evidence base is predominantly correlational, with causal relationships yet to be established.

Second, heterogeneity across studies include differences in models (animal versus human), designs (cross-sectional versus longitudinal), outcome measures, and participant characteristics limits generalizability. Mechanistic insights derive primarily from rodent studies, and translation to human behavioral phenotypes requires caution. Third, causality is uncertain. Bidirectional interactions along the MGBA and confounding variables such as medication use, diet, physical activity, and comorbid psychiatric conditions may influence both gut microbiome composition and procrastination-related behaviors. The relative contribution of neurobiological versus psychosocial factors remains unquantified. Fourth, therapeutic implications discussed are based on preliminary evidence and have not been specifically evaluated for effects on procrastination in diabetic populations. Clinical translation requires rigorous trials with behaviorally relevant endpoints. Finally, as a narrative review, this work is inherently selective. The synthesis reflects the authors' interpretation without a formal search protocol or standardized quality assessment, making the conclusions qualitative and hypothesis-generating rather than definitive.

CONCLUSION

In this review, we have proposed a plausible integrative framework whereby diabetes-induced MGBA dysfunction through disruptions in dopaminergic signaling, neuroinflammation, and cognitive impairment may contribute to procrastination-like behaviors that undermine diabetes self-care. While biologically plausible and supported by converging evidence, this model should be regarded as a working hypothesis rather than an established pathway. Direct evidence linking MGBA dysfunction to procrastination in diabetic patients is limited, and heterogeneity across studies, confounding factors, and the selective nature of narrative review synthesis necessitate cautious interpretation. Nevertheless, the model offers heuristic value for clinical practice and research, suggesting that addressing procrastination may require multidimensional approaches targeting both neurobiological substrates and behavioral factors. Interventions aimed at the MGBA, alongside cognitive and behavioral strategies tailored to executive function deficits, may offer adjunctive benefits. However, rigorous longitudinal, mechanistic, and interventional studies are needed before clinical translation. Until such evidence emerges, this framework serves primarily to generate testable hypotheses and guide future investigation. The path from mechanistic plausibility to clinical application remains long, warranting cautious optimism tempered by scientific rigor.

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