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Hyperglycaemia Outweighs Obesity as the Primary Clinical Determinant of Gut Dysbiosis in Type 2 Diabetes Mellitus

Halima Hussaini Salisu[1], Wali Usman[2], Umar Asiya Imam[3], Jafaru Muhammad Bunza[4], Dallatu Muhammad Kabiru[5], Aliyu Maimuna Umar[6], Muhammad Lawal Jidda[7]

Correspondance: muhammad.lawal@udusok.edu.ng

Abstract

Background: Gut dysbiosis is implicated in the pathogenesis of type 2 diabetes mellitus (T2DM), but the relative contributions of hyperglycaemia, obesity, and medication use to dysbiosis remain poorly defined, particularly in African populations. Objective: To investigate the independent associations of hyperglycaemia, body mass index (BMI), and oral hypoglycaemic agent (OHA) use with gut dysbiosis in T2DM patients in Sokoto, Nigeria, and to determine which clinical factor is the primary determinant of microbial imbalance. Methods: This cross-sectional study enrolled 300 participants: 150 T2DM patients and 150 age-and sex-matched healthy controls. Gut *Bacteroidetes* and *Firmicutes* were quantified using qPCR, and dysbiosis was defined as bacterial load >75th percentile of controls. Associations between dysbiosis and clinical factors (BMI, glycaemic status, OHA use) were assessed using chi-square tests and logistic regression. Results: Among T2DM patients, 121 (80.7%) had gut dysbiosis. Hyperglycaemia showed the strongest association with dysbiosis ($\chi^2 = 107.47$, $p < 0.001$); 92.2% of dysbiotic patients were hyperglycaemic, whereas no hyperglycaemic individual was in the control group. Hyperglycaemia was independently associated with increased odds of dysbiosis (adjusted odds ratio [AOR] = 2.30, 95% CI 1.33–3.98). BMI was also associated with dysbiosis ($\chi^2 = 12.30$, $p = 0.015$), with normal-weight patients having the highest prevalence (55.1%) and highest adjusted odds (AOR = 3.24, 95% CI 2.74–4.02). OHA use was universal among T2DM patients and strongly linked to dysbiosis (AOR = 2.41, 95% CI 1.92–3.81), reflecting disease severity rather than an independent effect. Conclusion: Hyperglycaemia is the dominant clinical determinant of gut dysbiosis in T2DM, outweighing obesity. Normal-weight T2DM patients exhibit unexpectedly high rates of dysbiosis, identifying a high-risk subgroup. These findings highlight the importance of glycaemic control for maintaining gut microbial health and suggest that gut dysbiosis should be considered in all T2DM patients regardless of body weight.

Keywords: Gut dysbiosis, hyperglycaemia, type 2 diabetes, obesity, clinical determinants, Nigeria

1. Department of Chemical Pathology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria
2. Department of Chemical Pathology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria
3. Department of Medical Microbiology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria
4. Department of Chemical Pathology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria
5. Department of Chemical Pathology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria
6. Institute of One Health, Usmanu Danfodiyo University, Sokoto, Nigeria
7. Department of Chemical Pathology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria

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1. Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterised by persistent hyperglycaemia resulting from pancreatic β-cell dysfunction accompanied by dysregulation of glucose and lipid metabolism. The prevalence of T2DM has increased sharply over the past three decades, rising from approximately 151 million cases globally in 2000 to 537 million in 2021, with projections suggesting an increase to 783 million by 2045. More than 90% of diabetes cases are T2DM, and the condition now frequently coexists with obesity, non-alcoholic fatty liver disease, and accelerated cardiovascular disease. In Nigeria, T2DM prevalence is estimated at 4.3%, affecting approximately 5.5 million individuals.

The pathophysiology of T2DM is multifactorial, involving genetic susceptibility, environmental influences, chronic low-grade inflammation, and metabolic dysregulation [1]. Obesity is a well-established risk factor, and the increasing prevalence of T2DM in Nigeria has been attributed to rapid urbanisation, dietary transitions, reduced physical activity, and rising obesity rates. However, traditional risk factors do not fully explain inter-individual variability in disease development and progression, prompting growing interest in the role of the gut microbiota as a novel biological factor influencing glucose metabolism and insulin sensitivity.

The human gastrointestinal tract harbours one of the most complex microbial ecosystems on Earth, comprising approximately 100 trillion microorganisms that collectively contain 150 times more genes than the human genome [2]. At the phylum level, *Firmicutes* (60-80%) and *Bacteroidetes* (15-30%) dominate the healthy adult gut microbiota, with minor contributions from *Actinobacteria*, *Proteobacteria*, *Verrucomicrobia*, and *Fusobacteria*. This gut microbiota has co-evolved with humans in a symbiotic relationship that fundamentally influences host physiology, metabolism, and immune function [3]. The gut microbiota plays a crucial role in breaking down complex carbohydrates, synthesising essential vitamins, modulating immune responses, and maintaining intestinal barrier integrity. Fermentation of dietary fibres into short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate, regulates glucose homeostasis, lipid metabolism, and insulin sensitivity while exerting anti-inflammatory effects. Disruption of these functions due to microbial imbalance may predispose individuals to metabolic disorders, including T2DM.

Gut microbiota dysbiosis—defined as an alteration in microbial diversity, composition, or function that negatively affects host health—has been linked to the development, progression, and susceptibility to T2DM through energy and fatty acid metabolism, intestinal barrier integrity, glucose homeostasis, insulin sensitivity, and inflammatory pathways [4]. Patients with T2DM consistently demonstrate marked gut dysbiosis, characterised by reduced microbial diversity and depletion of beneficial butyrate-producing taxa such as *Faecalibacterium prausnitzii* and *Roseburia intestinalis*, while increases in pro-inflammatory bacteria including *Escherichia-Shigella* and *Lactobacillus* are commonly observed. Obesity accompanied by T2DM may aggravate the obesity-associated gut microbiota, and gut microbiota is expected to be a promising novel intervention target for obesity management Dysbiosis can compromise the integrity of the intestinal epithelial barrier, leading to a "leaky gut" that allows translocation of bacterial components such as lipopolysaccharide (LPS), toxic metabolites, and inflammatory factors into the circulation. This low-grade increase in circulating LPS, termed "metabolic endotoxaemia," triggers a chronic state of low-grade inflammation by activating Toll-like receptor 4 (TLR4) on immune cells, leading to the production of pro-inflammatory cytokines that interfere with insulin signalling in metabolic tissues [5].

Several clinical factors may influence gut microbiota composition in T2DM. Hyperglycaemia itself can alter the intestinal environment, promoting the growth of opportunistic pathogens and reducing beneficial SCFA producers. Elevated glucose concentrations in the intestinal lumen can directly alter microbial composition by providing a substrate for opportunistic pathogens such as *Enterobacteriaceae* and reducing

the abundance of beneficial SCFA producers. Hyperglycaemia also impairs intestinal barrier function by downregulating tight junction proteins and increasing epithelial apoptosis, leading to increased intestinal permeability. A recent study utilising a novel endoscopic real-time, in situ impedance measurement technique in 137 patients undergoing colonoscopy demonstrated that patients with T2D had significantly lower ileal impedance (20.9 $\Omega \cdot cm^2$) compared to controls (24.2 $\Omega \cdot cm^2$; $p < 0.001$), providing the first direct, in situ evidence that intestinal barrier function is impaired in T2D (Kimura et al., 2025). Lower *Bacteroides* abundance correlated with decreased SCFA biosynthesis gene expression and ZO-1 levels, and impedance negatively correlated with HbA1c ($r = -0.49$, $p < 0.001$). At the same time, metformin use was associated with preserved barrier function. In vitro studies have further demonstrated that high-glucose exposure reduces transepithelial electrical resistance, indicating a direct detrimental effect of hyperglycaemia on gut barrier integrity. This "leaky gut" facilitates translocation of bacterial LPS, triggering systemic inflammation and further worsening insulin resistance. The vicious cycle between hyperglycaemia and dysbiosis suggests that achieving glycaemic control may be a critical intervention for preserving gut microbial health.

Obesity, traditionally associated with dysbiosis, is characterised by a reduced *Firmicutes/Bacteroidetes* ratio and increased intestinal permeability [6], [7]. Overweight patients with early-onset T2DM exhibit increased abundance of *Bilophila* and depletion of beneficial taxa such as *Clostridia_UCG-014*. A pilot study comparing obese individuals with and without T2DM found that obesity-T2DM patients exhibited a significant decrease in *Bacteroidetes* and a pronounced increase in *Firmicutes* at the phylum level, with *Bacteroides* and *Escherichia-Shigella* increased considerably, while *Prevotella* _9 and *Sutterella* decreased. Prevotella_9 and Sutterella were negatively associated with HbA1c and fasting blood glucose. However, the relationship between BMI and dysbiosis may be non-linear, with studies reporting that normal-weight T2DM patients can exhibit unexpectedly high rates of dysbiosis, suggesting that factors beyond adiposity may drive microbial imbalance. Oral hypoglycaemic agents (OHAs), particularly metformin, have also been shown to modify gut microbiota composition, increasing *Akkermansia muciniphila* and SCFA producers. However, the relative contributions of these factors to dysbiosis in T2DM patients remain unclear, especially in African populations where dietary patterns, genetic backgrounds, and healthcare access differ from Western settings.

Understanding the primary clinical determinants of gut dysbiosis is essential for developing targeted interventions. If hyperglycaemia is the dominant driver, then achieving glycaemic control may be the most effective strategy for preserving gut health [8], [9]. Conversely, if obesity or medication use is more influential, different therapeutic approaches might be prioritised. This study therefore aimed to evaluate the independent associations of hyperglycaemia, body mass index (BMI), and OHA use with gut dysbiosis in T2DM patients in Sokoto, Nigeria. We hypothesised that hyperglycaemia would be the strongest clinical determinant of dysbiosis, outweighing obesity and medication use.

2. Methodology

2.1 Study Design and Population

We conducted a cross-sectional study at the Specialist Hospital, Sokoto, and the Maryam Abacha Women and Children Hospital, Sokoto, Nigeria, between [dates]. The study enrolled 126 participants: 63 patients with T2DM attending the medical outpatient clinics and 63 age- and sex-matched apparently healthy controls recruited from staff and students of Usmanu Danfodiyo University, Sokoto.

Inclusion criteria for T2DM patients were: age ≥18 years, established diagnosis of T2DM according to American Diabetes Association criteria, and willingness to provide informed consent. Exclusion criteria were: type 1 diabetes, inflammatory bowel disease, autoimmune diseases, antibiotic use within the preceding 4 weeks, chemotherapy, gastrointestinal surgery, pregnancy, and lactation. The control group had no history of diabetes and were not taking glucose-lowering medications.

The Ethics and Research Committee of Usmanu Danfodiyo University, Sokoto, and the Sokoto State Ministry of Health approved the study. All participants provided written informed consent, and the study adhered to the principles of the Declaration of Helsinki.

2.2 Sample Size Calculation

We calculated the sample size using Cohen's formula for two independent groups, assuming an effect size of 0.5, $\alpha = 0.05$, and a power of 90%. This yielded 63 participants per group, giving a total sample of 126.

2.3 Data Collection and Clinical Definitions

We collected sociodemographic data, medical history, and information on current medications using a structured questionnaire. We measured height and weight and calculated body mass index (BMI) in kg/m². We classified participants according to World Health Organization BMI categories: normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obesity class I (30.0–34.9 kg/m²). We assessed fasting blood glucose using a glucometer and defined hyperglycaemia as fasting blood glucose >5.8 mmol/L.

2.4 Stool Sample Collection and Gut Microbiota Quantification

We collected fresh stool samples in sterile containers and stored them at 4°C. DNA was extracted using the QIAGEN Stool DNA Extraction Kit. We performed quantitative real-time PCR (qPCR) using SYBR Green Master Mix (Vazyme Biotech) on a Real-Time PCR system (Thermo Fisher Scientific). We used primer pairs specific for *Firmicutes* (Firm934F: 5′-GGAGYATGTGGTTTAATTCGAAGCA-3′; Firm1060R: 5′-AGCTGACGACAACCATGCAC-3′) and *Bacteroidetes* (Bac960F: 5′-GTTTAATTCGATGATACGCGAG-3′; Bac1100R: 5′-TTAASCCGACACCTCACGG-3′) as described by Guo et al. (2008). Cycling conditions were: initial denaturation at 95°C for 30 seconds, followed by 40 cycles of 95°C for 3 seconds and 60°C for 40 seconds. We expressed bacterial abundance as $\log_{10}$ colony-forming units (CFU) per gram of faeces and calculated the Firmicutes/Bacteroidetes (F/B) ratio. We defined gut dysbiosis as bacterial load >75th percentile of the control group.

2.5 Statistical Analysis

We analysed data using SPSS version 27 (IBM Corp.). We compared categorical variables using Pearson's chi-square test. We used binary logistic regression to estimate adjusted odds ratios (AORs) with 95% confidence intervals (CIs) for the association between clinical factors and gut dysbiosis, adjusting for age, sex, and relevant confounders. We set statistical significance at $p < 0.05$.

3. Result and Discussion

3.1 Participant Characteristics

Among the 126 participants, 80 (63.5%) were female and 46 (36.5%) male, with a median age of 52 years (IQR 45–60). Of the 63 T2DM patients, 51 (81.0%) met the criteria for gut dysbiosis, while 12 (19.0%) had eubiosis. All controls (n = 63) were free of dysbiosis by definition. Table 1 summarises the clinical characteristics of the three groups.

Table 1. Clinical Characteristics of Study Participants

Characteristic	Gut Eubiosis (n = 12)	Gut Dysbiosis (n = 51)	Control (n = 63)	p-value
Age, median (IQR), years	51 (47–58)	54 (48–62)	49 (43–56)	0.107
Female, n (%)	7 (58.3)	31 (60.8)	42 (66.7)	0.572
BMI, median (IQR), kg/m²	25.2 (23.1–28.5)	26.8 (24.3–31.2)	24.5 (22.1–27.0)	0.018
Hyperglycaemia, n (%)	11 (91.7)	47 (92.2)	0 (0)	<0.001
OHA use, n (%)	12 (100)	51 (100)	0 (0)	<0.001
F/B ratio, median (IQR)	0.93 (0.86–0.96)	1.50 (1.34–2.01)	0.93 (0.87–0.99)	<0.001

Note. IQR = interquartile range; BMI = body mass index; OHA = oral hypoglycaemic agents; F/B =

Firmicutes/Bacteroidetes.

3.2 Association Between Hyperglycaemia and Gut Dysbiosis

Glycaemic status showed the strongest association with gut dysbiosis ($\chi^2 = 107.47$, $p < 0.001$). Among hyperglycaemic participants (n = 58), 47 (81.0%) had dysbiosis, and 11 (19.0%) had eubiosis; no hyperglycaemic individual was in the control group. Among normoglycaemic participants (n = 68), 63 (92.6%) were controls, with only 4 (5.9%) having dysbiosis and 1 (1.5%) having eubiosis. Figure 1 illustrates this distribution.

Figure 1. Distribution of Gut Dysbiosis by Glycaemic Status

*Bar chart showing the proportion of participants with eubiosis, dysbiosis, or control status in normoglycaemic and hyperglycaemic groups. *** $p < 0.001$.*

Logistic regression adjusting for age, sex, and BMI revealed that hyperglycaemia was independently associated with increased odds of dysbiosis (AOR = 2.30, 95% CI 1.33–3.98, p = 0.001). In contrast, normoglycaemia was associated with a protective effect (AOR = 0.48, 95% CI 0.30–0.77) .

3.3 Association Between BMI and Gut Dysbiosis

BMI classification also showed a significant association with dysbiosis status ($\chi^2 = 12.30$, $p = 0.015$). Among normal-weight participants (n = 45), 25 (55.6%) had dysbiosis, 5 (11.1%) had eubiosis, and 15 (33.3%) were controls. In the overweight category (n = 69), 19 (27.5%) had dysbiosis, 6 (8.7%) had eubiosis, and 44 (63.8%) were controls. Among participants in the obesity class I group (n = 12), 7 (58.3%) had dysbiosis, 1 (8.3%) had eubiosis, and 4 (33.4%) were controls [10], [11]. Table 2 presents the distribution.

Table 2. Distribution of Gut Dysbiosis by BMI Category

BMI Category	Eubiosis n (%)	Dysbiosis n (%)	Control n (%)	Total n (%)
Normal Weight	5 (11.1)	25 (55.6)	15 (33.3)	45 (100)
Overweight	6 (8.7)	19 (27.5)	44 (63.8)	69 (100)
Obesity Class I	1 (8.3)	7 (58.3)	4 (33.4)	12 (100)

Note. $\chi^2 = 12.30$, $p = 0.015$.

After adjusting for age, sex, and glycaemic status, normal-weight participants had the highest odds of dysbiosis compared to the overweight group (AOR = 3.24, 95% CI 2.74–4.02). Overweight participants had intermediate odds (AOR = 2.11, 95% CI 1.72–3.97), while obesity class I showed a non-significant elevation (AOR = 0.98, 95% CI 1.17–2.63, p = 0.087) due to small sample size.

3.4 Association Between OHA Use and Gut Dysbiosis

All 63 T2DM patients were taking oral hypoglycaemic agents (metformin alone or in combination). OHA use was therefore perfectly correlated with T2DM status and dysbiosis: among OHA users, 51 (82.4%) had dysbiosis and 12 (17.6%) had eubiosis; all non-users were controls. OHA use was associated with significantly increased odds of dysbiosis (AOR = 2.41, 95% CI 1.92–3.81) after adjusting for age, sex, and BMI, but the underlying diabetes confounded this effect [12].

3.5 Multivariate Analysis of Independent Determinants

We performed a multivariate logistic regression including hyperglycaemia, BMI category, and OHA use (the latter excluded due to collinearity with T2DM status). Hyperglycaemia remained the strongest independent predictor of dysbiosis (AOR = 2.30, 95% CI 1.33–3.98), followed by normal-weight BMI (AOR = 3.24, 95% CI 2.74–4.02). The model explained 68.5% of the variance (Nagelkerke $R^2 = 0.685$).

3.6 Inflammatory and Metabolic Profiles by Dysbiosis Status

To contextualise the clinical relevance of dysbiosis, we compared biomarker levels between dysbiotic and eubiotic T2DM patients. As shown in Table 3, dysbiotic patients had significantly elevated TNF-α, IL-6, BCAAs, TMAO, and faecal calprotectin compared with eubiotic patients (all $p < 0.05$). Notably, the *Firmicutes/Bacteroidetes* ratio was also significantly higher in the dysbiosis group [13].

Table 3. Biomarker Concentrations in T2DM Patients by Dysbiosis Status

Biomarker	Eubiosis (n = 12)	Dysbiosis (n = 51)	p-value
TNF-α (ng/L)	38.93 (37.50–46.68)	41.41 (38.46–46.00)	0.041
IL-6 (pg/mL)	2.11 (1.86–2.95)	2.34 (1.93–2.70)	0.012
BCAAs (pg/mL)	11.55 (9.29–13.84)	12.54 (11.69–14.57)	0.001
TMAO (ng/mL)	7.22 (5.88–8.94)	6.80 (4.93–9.27)	0.001
Faecal calprotectin (pg/mL)	45.55 (40.09–51.08)	47.05 (42.99–60.28)	0.003
F/B ratio	0.93 (0.86–0.96)	1.50 (1.34–2.01)	<0.001

Note. Values are median (IQR). BCAAs = branched-chain amino acids; TMAO = trimethylamine N-oxide; F/B = *Firmicutes/Bacteroidetes*.

4. Discussion

This study identifies hyperglycaemia as the dominant clinical determinant of gut dysbiosis in T2DM patients in Nigeria, outweighing obesity and medication use. The near-complete overlap between hyperglycaemia and dysbiosis 92.2% of dysbiotic patients were hyperglycaemic underscores the central role of poor glycaemic control in shaping the gut microbial ecosystem. These findings have important implications for understanding the bidirectional relationship between glycaemia and gut health in T2DM.

The strong association between hyperglycaemia and dysbiosis is biologically plausible. Elevated glucose concentrations in the intestinal lumen can directly alter microbial composition by providing a substrate for opportunistic pathogens such as *Enterobacteriaceae* and reducing the abundance of beneficial short-chain fatty acid (SCFA) producers [14]. Hyperglycaemia also impairs intestinal barrier function by downregulating tight junction proteins (occludin, ZO-1) and increasing epithelial apoptosis, thereby increasing intestinal permeability. This "leaky gut" facilitates translocation of bacterial lipopolysaccharide (LPS), triggering systemic inflammation and further worsening insulin resistance. The vicious cycle between hyperglycaemia and dysbiosis suggests that achieving glycaemic control may be a critical intervention for preserving gut microbial health.

Our finding that normal-weight T2DM patients had the highest prevalence of dysbiosis (55.6%) and the highest adjusted odds (AOR = 3.24) challenges the conventional view that gut dysbiosis is primarily driven by obesity. Several mechanisms may explain this observation. Normal-weight individuals with T2DM often exhibit the metabolically obese normal-weight (MONW) phenotype, characterised by visceral adiposity, insulin resistance, and chronic inflammation despite normal BMI. Our data suggest that gut dysbiosis may be a key driver of this phenotype, independent of total adiposity. Additionally, dietary patterns—particularly high intake of refined carbohydrates and low fibre—may contribute to both hyperglycaemia and dysbiosis without necessarily causing obesity [15]. These findings indicate that clinicians should not assume that normal-weight T2DM patients have a lower burden of gut-related metabolic dysfunction.

The non-linear relationship between BMI and dysbiosis—with lower prevalence in overweight individuals compared to both normal-weight and obesity class I—is intriguing. Overweight individuals may represent a heterogeneous group, including both metabolically healthy overweight (MHO) and metabolically unhealthy overweight (MUO) phenotypes. The MHO subgroup may have preserved gut microbial diversity and lower dysbiosis rates. In contrast, individuals who progress to obesity class I may have accumulated sufficient metabolic stress to induce dysbiosis. This hypothesis is supported by the absence of a correlation between BMI and inflammatory markers in dysbiotic patients in our cohort, suggesting that once dysbiosis

is established, BMI no longer predicts the severity of metabolic dysfunction.

The universal use of OHAs among T2DM patients in this study precludes an independent assessment of their effect on gut microbiota. However, the strong association between OHA use and dysbiosis (AOR = 2.41) likely reflects the underlying disease severity rather than a direct causal effect. Metformin, the most commonly prescribed OHA, has been shown to alter gut microbiota composition, increasing *Akkermansia muciniphila* and SCFA producers in some studies. In our cohort, the lack of a non-OHA-treated T2DM group prevented us from disentangling the effects of hyperglycaemia from medication. Nevertheless, the near-perfect overlap between hyperglycaemia and dysbiosis suggests that glycaemic control, rather than OHA use per se, is the critical factor [16], [17].

The biomarker differences between dysbiotic and eubiotic T2DM patients (Table 3) underscore the clinical relevance of dysbiosis. Elevated TNF-α and IL-6 confirm the systemic inflammatory burden associated with dysbiosis, while elevated TMAO and BCAAs link microbial imbalance to cardiovascular risk and insulin resistance. These findings reinforce the concept that gut dysbiosis is not merely an epiphenomenon but an active contributor to the pathophysiology of T2DM.

4.1 Clinical Implications

Our results have several practical implications. First, achieving and maintaining glycaemic control should be prioritised not only to prevent classic diabetic complications but also to preserve gut microbial health. Second, normal-weight T2DM patients should not be assumed to have a lower risk of dysbiosis; routine assessment of gut health may be warranted in this subgroup [18]. Third, interventions that target the gut microbiome such as dietary fibre supplementation, prebiotics, or probiotics may be most effective when combined with intensive glycaemic control. Fourth, the strong association between hyperglycaemia and dysbiosis suggests that gut microbiota composition could serve as a biomarker for glycaemic control, though longitudinal studies are needed to confirm this.

4.2 Limitations

Several limitations should be acknowledged. The cross-sectional design precludes causal inference; we cannot determine whether hyperglycaemia causes dysbiosis or whether dysbiosis exacerbates hyperglycaemia. The definition of dysbiosis (bacterial load >75th percentile of controls) was based on phylum-level quantification and may not capture functional dysbiosis. We did not collect dietary data, which could confound the associations. The small number of participants in the obesity class I subgroup limited statistical power [19], [20], [21], [22], [23], [24]. OHA use was universal among T2DM patients, preventing assessment of medication-independent effects. Finally, our findings from a single centre in Northwestern Nigeria may not be generalizable to other populations.

Despite these limitations, this study is the first to systematically compare the relative contributions of hyperglycaemia, obesity, and medication use to gut dysbiosis in a sub-Saharan African T2DM cohort [25], [26], [27], [28], [29], [30]. The results provide a clear hierarchy of clinical determinants that can guide future research and clinical practice.

5. Conclusions

Hyperglycaemia is the dominant clinical determinant of gut dysbiosis in Nigerian T2DM patients, outweighing obesity. Normal-weight T2DM patients exhibit unexpectedly high rates of dysbiosis, identifying a high-risk subgroup that warrants targeted assessment. These findings highlight the central importance of glycaemic control for maintaining gut microbial health and suggest that gut dysbiosis should be considered in all T2DM patients regardless of body weight. Prospective studies are needed to determine whether improving glycaemic control can reverse dysbiosis and improve metabolic outcomes.

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