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Article

Analysis of Gut Microbiota and Metabolic Indicators in Patients with Obesity and Type 2 Diabetes Mellitus

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Abstract: The aim of this article is to compare gut microbiota composition together with anthropometric and biochemical indicators between patients with obesity (prior to semaglutide administration) and patients with early T2DM, and to evaluate the discriminatory value of the studied markers. Materials and methods. A cross-sectional study included 51 adults: an obesity group (n = 44; 36 women, 8 men) examined before initiation of the GLP-1 receptor agonist semaglutide, and a T2DM group (n = 7 women) diagnosed within the preceding three years who had received oral hypoglycaemic agents previously but none during the last three months. Anthropometric data and a biochemical panel (glucose, hepatic enzymes, lipid profile, renal markers) were obtained. Results. Fasting glucose was, by design, markedly higher in the T2DM group (8.84 ± 0.49 vs 4.80 ± 0.53 mmol/L; $p < 0.001$), whereas anthropometric and lipid indicators did not differ significantly. The T2DM group showed higher abundance of Faecalibacterium prausnitzii (9.43 ± 1.90 vs 7.70 ± 1.36 lg Copy/ml; $p = 0.023$), Klebsiella oxytoca ($p = 0.015$) and Clostridium difficile ($p = 0.018$), with a trend toward higher Bifidobacterium spp. and total bacterial mass, while Escherichia coli ($p = 0.017$) and Enterococcus spp. ($p = 0.036$) were higher in the obesity group. Fasting glucose correlated positively with Clostridium perfringens ($\varrho = 0.36$), Clostridium difficile ($\varrho = 0.34$) and F. prausnitzii ($\varrho = 0.29$), and BMI with Candida spp. ($\varrho = 0.31$). In ROC analysis, F. prausnitzii (AUC = 0.77), urea (AUC = 0.73) and Bifidobacterium spp. (AUC = 0.71) showed moderate discrimination between the groups. Conclusion. Early, treatment-naïve T2DM and pre-treatment obesity are associated with distinct intestinal microbial signatures detectable by targeted PCR profiling. Selected taxa, in particular F. prausnitzii and Bifidobacterium spp., warrant further evaluation as adjunct biomarkers of metabolic phenotype.

Keywords: Gut microbiota; obesity; type 2 diabetes mellitus; GLP-1 receptor agonist; semaglutide; Faecalibacterium prausnitzii; dysbiosis; Kolonoflor-16; ROC analysis.

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Introduction

Obesity and type 2 diabetes mellitus (T2DM) constitute two of the most pressing non-communicable disease burdens of the twenty-first century, and their pathophysiology is now understood to extend well beyond caloric imbalance and insulin resistance. Over the past decade, the intestinal microbiota has emerged as an active metabolic organ that influences energy harvest, bile-acid metabolism, low-grade systemic inflammation and incretin secretion. Disturbances in microbial community structure, collectively termed

dysbiosis, have been repeatedly linked to the development and progression of metabolic disease [1, 2].

Mechanistically, the gut microbiota shapes host metabolism through the production of short-chain fatty acids (SCFAs), modulation of intestinal barrier integrity and regulation of the enteroendocrine axis, including secretion of glucagon-like peptide-1 (GLP-1) [3,4]. Butyrate-producing commensals such as Faecalibacterium prausnitzii [1], a member of the phylum Firmicutes that constitutes more than 5% of the faecal community in healthy adults, exert anti-inflammatory effects and have been shown to improve insulin sensitivity and lipid metabolism in experimental models of T2DM [5]. Likewise, the mucin-degrading species Akkermansia muciniphila is inversely associated with adiposity, glucose intolerance and metabolic endotoxaemia, and is regarded as a promising next-generation probiotic [6].

Pharmacological modulation of this axis has attracted particular attention. GLP-1 receptor agonists, of which semaglutide is the most widely prescribed long-acting representative, achieve substantial weight reduction and glycaemic control, and there is accumulating evidence that part of their benefit is mediated through favourable shifts in microbial composition, including enrichment of A. muciniphila and SCFA-producing taxa [7]. Characterising the baseline microbiota of patients who are candidates for such therapy is therefore of considerable clinical interest, both for understanding inter-individual variability in treatment response and for identifying microbial biomarkers of metabolic phenotype [8].

Most published work has relied on next-generation sequencing in heterogeneous, often medicated cohorts, which complicates the isolation of disease-specific signals from pharmacological confounders. Targeted quantitative real-time PCR panels, such as the Kolonoflor-16 assay (Alfalab, Russia), offer a standardised and reproducible alternative that quantifies a defined set of beneficial and opportunistic taxa in absolute terms, lending itself to routine clinical comparison [9].

The present study was designed to compare gut microbiota composition, together with anthropometric and biochemical indicators, between two clinically distinct but metabolically related groups: patients with obesity examined immediately before initiation of semaglutide, and patients with recently diagnosed T2DM who were free of glucose-lowering medication at the time of sampling. We further sought to quantify the associations between individual taxa and metabolic parameters and to assess, by means of receiver operating characteristic (ROC) analysis, the extent to which microbial and biochemical markers discriminate between the two phenotypes [10].

Materials and Methods

Study design and participants

This study enrolled 51 adult participants allocated to two groups. The obesity group comprised 44 patients (36 women and 8 men) with established obesity who were assessed at baseline, prior to administration of the GLP-1 receptor agonist semaglutide. The T2DM group comprised 7 women diagnosed with type 2 diabetes mellitus within the preceding three years; all had previously received oral hypoglycaemic agents but had taken none during the three months immediately before sampling, thereby minimising the confounding influence of current pharmacotherapy on the intestinal microbiota.

In the obesity group the age distribution was as follows: one participant aged 18–19 years, ten aged 20–29, eighteen aged 30–39, eight aged 40–49, five aged 50–59, one aged 60–69 and one aged 70–75 years. In the T2DM group, four participants were aged 40–59 years and three were aged 60–75 years; the T2DM group was thus, on average, older and exclusively female.

Anthropometric and biochemical measurements. For each participant, body weight, waist circumference and body mass index (BMI) were recorded. A fasting venous

blood sample was analysed for the following indicators: plasma glucose; the hepatic transaminases alanine aminotransferase (ALT) and aspartate aminotransferase (AST); total bilirubin; the renal markers urea and creatinine; and a complete lipid profile comprising total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol. All assays were performed using standard automated clinical-chemistry methods.

Gut microbiota analysis

Gut microbiota composition was determined from faecal samples using the Kolonoflor-16 quantitative real-time PCR assay (Alfalab, Russia). The panel quantifies the total bacterial mass together with beneficial and opportunistic taxa, namely Lactobacillus spp., Bifidobacterium spp., Escherichia coli, Bacteroides spp., Faecalibacterium prausnitzii, Bacteroides thetaiotaomicron, Akkermansia muciniphila, Enterococcus spp., enteropathogenic Escherichia coli, Klebsiella pneumoniae, Klebsiella oxytoca, Candida spp., Staphylococcus aureus, Clostridium difficile, Clostridium perfringens, Proteus vulgaris/mirabilis, Citrobacter spp. and Enterobacter spp. Results are expressed as decimal logarithms of genome equivalents per gram of faeces (lg copy/ml); taxa below the detection threshold were assigned a value of zero.

Statistical analysis

Continuous variables are presented as mean ± standard deviation and, where appropriate, as median with interquartile range. Given the small and unequal group sizes and the non-normal distribution of microbial counts, between-group comparisons were performed using the non-parametric Mann–Whitney U test. Associations between metabolic parameters and microbial abundances across the whole cohort were quantified with the Spearman rank correlation coefficient (ϱ). The discriminatory performance of individual markers in separating T2DM from obesity was assessed by ROC analysis, with the area under the curve (AUC) reported alongside the sensitivity and specificity at the optimal Youden cut-off. A two-sided p-value < 0.05 was considered statistically significant. Analyses were conducted in Python 3 (pandas, SciPy and scikit-learn libraries).

Results

Demographic characteristics

The two groups differed in their demographic structure. The obesity group was predominantly composed of younger adults, with the largest stratum (18 of 44 participants) aged 30–39 years, and included both sexes (82% women). The T2DM group was smaller, exclusively female and concentrated in the older age strata, with all seven participants aged 40 years or above. The distribution of age and sex across the two groups is summarised in Table 1 and depicted in Figure 1 [11].

Table 1. Demographic characteristics of the study groups.

Characteristic	Obesity (n = 44)	T2DM (n = 7)
Women, n	36	7
Men, n	8	0
Age 18–29 years, n	11	0
Age 30–39 years, n	18	0
Age 40–59 years, n	13	4
Age 60–75 years, n	2	3

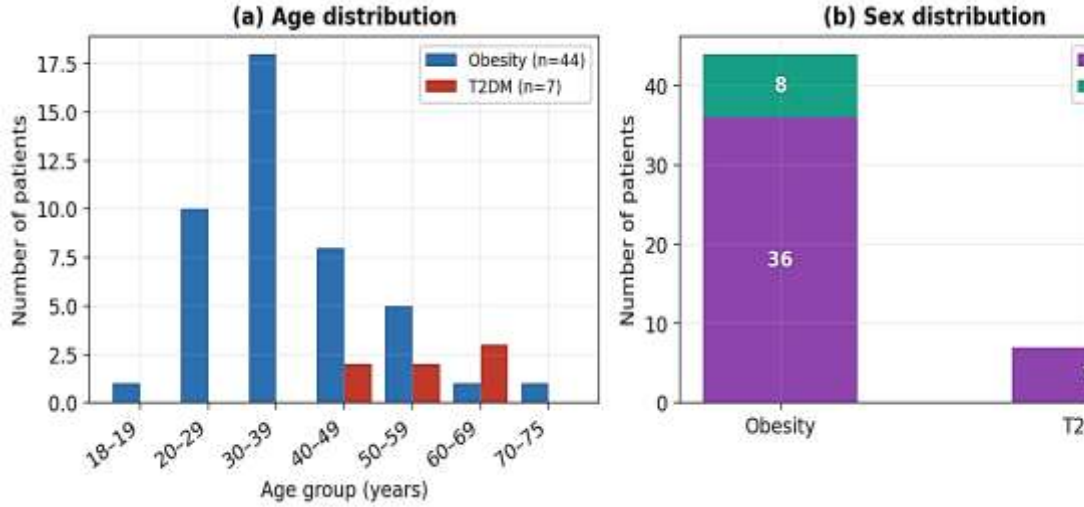

Figure 1. Distribution of participants by age group (a) and by sex (b) in the obesity and T2DM groups.

Anthropometric and biochemical indicators

Anthropometric indicators were comparable between the groups: mean body weight, waist circumference and BMI did not differ significantly (all $p > 0.16$), and both groups were, on average, in the class II obesity range (BMI ≈ 36 kg/m²). As expected from the case definition, fasting glucose was the single most discriminating biochemical variable, being markedly elevated in the T2DM group (8.84±0.49 vs 4.80±0.53 mmol/L; $p<0.001$). Creatinine was numerically higher in the T2DM group, consistent with its older age structure, although the difference did not reach significance (72.54 ± 15.17 vs 64.54 ± 7.38 μmol/L; $p = 0.129$). The lipid profile, hepatic transaminases and total bilirubin were similar between the groups. The full comparison is presented in Table 2 and the key indicators are illustrated in Figure 2 [12].

Table 2. Comparison of anthropometric and biochemical indicators between groups.

Indicator	Obesity (n = 44)	T2DM (n = 7)	p
Weight, kg	100.80 ± 10.62	95.14 ± 4.38	0.162
Waist circumference, cm	108.82 ± 10.13	108.14 ± 8.13	0.978
BMI, kg/m²	36.84 ± 4.75	36.15 ± 1.86	0.816
Glucose, mmol/L	4.80 ± 0.53	8.84 ± 0.49	<0.001
ALT, U/L	25.05 ± 19.86	26.00 ± 5.51	0.100
AST, U/L	25.34 ± 10.55	25.71 ± 5.22	0.475
Total bilirubin, μmol/L	12.13 ± 2.89	10.74 ± 0.96	0.261
Urea, mmol/L	5.04 ± 1.16	4.83 ± 2.68	0.057
Creatinine, μmol/L	64.54 ± 7.38	72.54 ± 15.17	0.129
Total cholesterol, mmol/L	5.52 ± 0.42	5.54 ± 0.31	0.956
Triglycerides, mmol/L	2.10 ± 0.38	2.02 ± 0.27	0.680
LDL cholesterol, mmol/L	3.37 ± 0.38	3.45 ± 0.23	0.763
HDL cholesterol, mmol/L	1.21 ± 0.14	1.18 ± 0.12	0.600

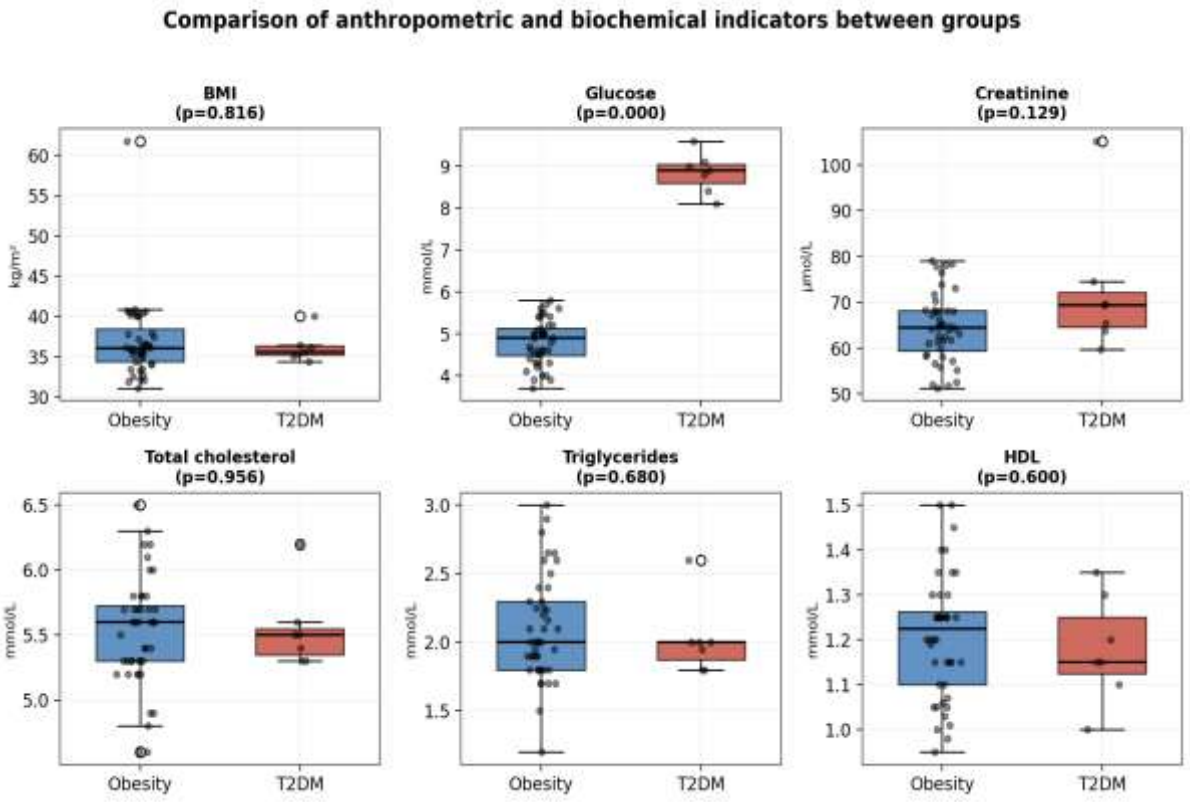

Figure 2. Box-and-whisker plots of selected anthropometric and biochemical indicators by group.

Gut microbiota composition

Quantitative profiling with Kolonoflor-16 revealed a distinct microbial pattern in each group (Table 3, Figure 3). Total bacterial mass tended to be higher in the T2DM group (12.29 ± 1.11 vs 11.39 ± 1.10 lg copy/ml; p = 0.052). Among beneficial taxa, the T2DM group displayed significantly higher abundance of Faecalibacterium prausnitzii (9.43 ± 1.90 vs 7.70 ± 1.36 lg copy/ml; p = 0.023) and a trend toward higher Bifidobacterium spp. (p = 0.065) and Akkermansia muciniphila (p = 0.155). In contrast, Escherichia coli was significantly higher in the obesity group (8.20 ± 1.32 vs 6.86 ± 1.21 lg copy/ml; p = 0.017), as was Enterococcus spp. (7.84 ± 1.58 vs 6.14 ± 1.95; p = 0.036) [13].

Among opportunistic taxa, Klebsiella oxytoca (4.43 ± 1.40 vs 2.93 ± 1.32; p = 0.015) and Clostridium difficile (1.57 ± 1.13 vs 0.73 ± 0.54; p = 0.018) were significantly more abundant in the T2DM group, with Clostridium perfringens showing a parallel borderline trend (p=0.052). Conversely, Proteus vulgaris/mirabilis and Enterobacter spp. were numerically higher in the obesity group. Taken together, the early-T2DM phenotype was characterised by a higher absolute bacterial load and enrichment of both butyrate-producing commensals and selected opportunistic organisms, whereas the pre-treatment obesity phenotype showed relative enrichment of E. coli and Enterococcus spp [14].

Table 3. Comparison of gut microbiota abundance.

Taxon	Obesity (n = 44)	T2DM (n = 7)	p
Total bacterial mass	11.39 ± 1.10	12.29 ± 1.11	0.052
Lactobacillus spp.	6.41 ± 1.23	7.00 ± 1.15	0.207
Bifidobacterium spp.	8.25 ± 1.26	9.29 ± 1.38	0.065
Escherichia coli	8.20 ± 1.32	6.86 ± 1.21	0.017

Taxon	Obesity (n = 44)	T2DM (n = 7)	p
Bacteroides spp.	9.02 ± 1.34	9.57 ± 1.13	0.190
Faecalibacterium prausnitzii	7.70 ± 1.36	9.43 ± 1.90	0.023
Bacteroides thetaiotaomicron	7.23 ± 1.87	8.43 ± 2.94	0.197
Akkermansia muciniphila	5.73 ± 2.53	7.57 ± 3.41	0.155
Enterococcus spp.	7.84 ± 1.58	6.14 ± 1.95	0.036
E. coli enteropathogenic	3.77 ± 1.20	3.57 ± 1.40	0.734
Klebsiella pneumoniae	4.59 ± 1.11	4.57 ± 1.13	0.929
Klebsiella oxytoca	2.93 ± 1.32	4.43 ± 1.40	0.015
Candida spp.	4.75 ± 1.51	4.71 ± 1.25	0.989
Staphylococcus aureus	4.11 ± 1.13	4.14 ± 1.21	0.831
Clostridium difficile	0.73 ± 0.54	1.57 ± 1.13	0.018
Clostridium perfringens	0.84 ± 0.75	1.57 ± 1.13	0.052
Proteus vulgaris/mirabilis	5.07 ± 1.48	3.86 ± 1.86	0.091
Citrobacter spp.	3.68 ± 1.23	3.43 ± 1.51	0.774
Enterobacter spp.	4.91 ± 1.55	4.00 ± 1.63	0.173

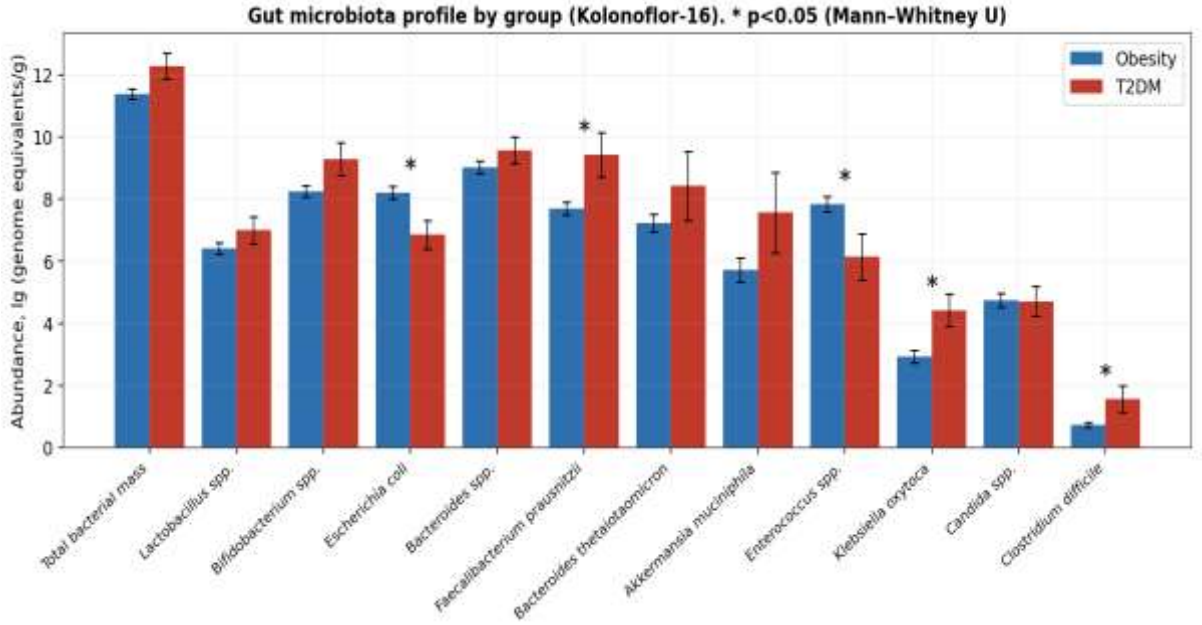

Figure 3. Mean gut microbiota profile of the two groups measured by Kolonoflor-16.

Correlation analysis

Spearman correlation analysis across the whole cohort (n = 51) identified several significant associations between microbial abundances and metabolic parameters (Table 4, Figure 4). Fasting glucose correlated positively with Clostridium perfringens (ϱ=0.36; p=0.010), Clostridium difficile (ϱ = 0.34; p = 0.016), Lactobacillus spp. (ϱ = 0.31; p = 0.027), Klebsiella oxytoca (ϱ = 0.30; p = 0.036) and Faecalibacterium prausnitzii (ϱ=0.29; p=0.042), and negatively with Proteus vulgaris/mirabilis (ϱ =−0.30; p=0.035). Body mass index correlated positively with Candida spp. (ϱ = 0.31; p = 0.026), Klebsiella pneumoniae (ϱ =

0.30; p = 0.033) and Enterococcus spp. (ϱ = 0.29; p = 0.038). The relationship between glucose and F. prausnitzii is shown in Figure 6a, and the overall balance between beneficial and opportunistic taxa is illustrated in Figure 6b.

Table 4. Statistically significant Spearman correlations between metabolic parameters and microbial taxa (whole cohort, n = 51).

Parameter	Taxon	ϱ	p
Glucose	Clostridium perfringens	0.36	0.010
Glucose	Clostridium difficile	0.34	0.016
Glucose	Lactobacillus spp.	0.31	0.027
Glucose	Klebsiella oxytoca	0.30	0.036
Glucose	Faecalibacterium prausnitzii	0.29	0.042
Glucose	Proteus vulgaris/mirabilis	−0.30	0.035
BMI	Candida spp.	0.31	0.026
BMI	Klebsiella pneumoniae	0.30	0.033
BMI	Enterococcus spp.	0.29	0.038

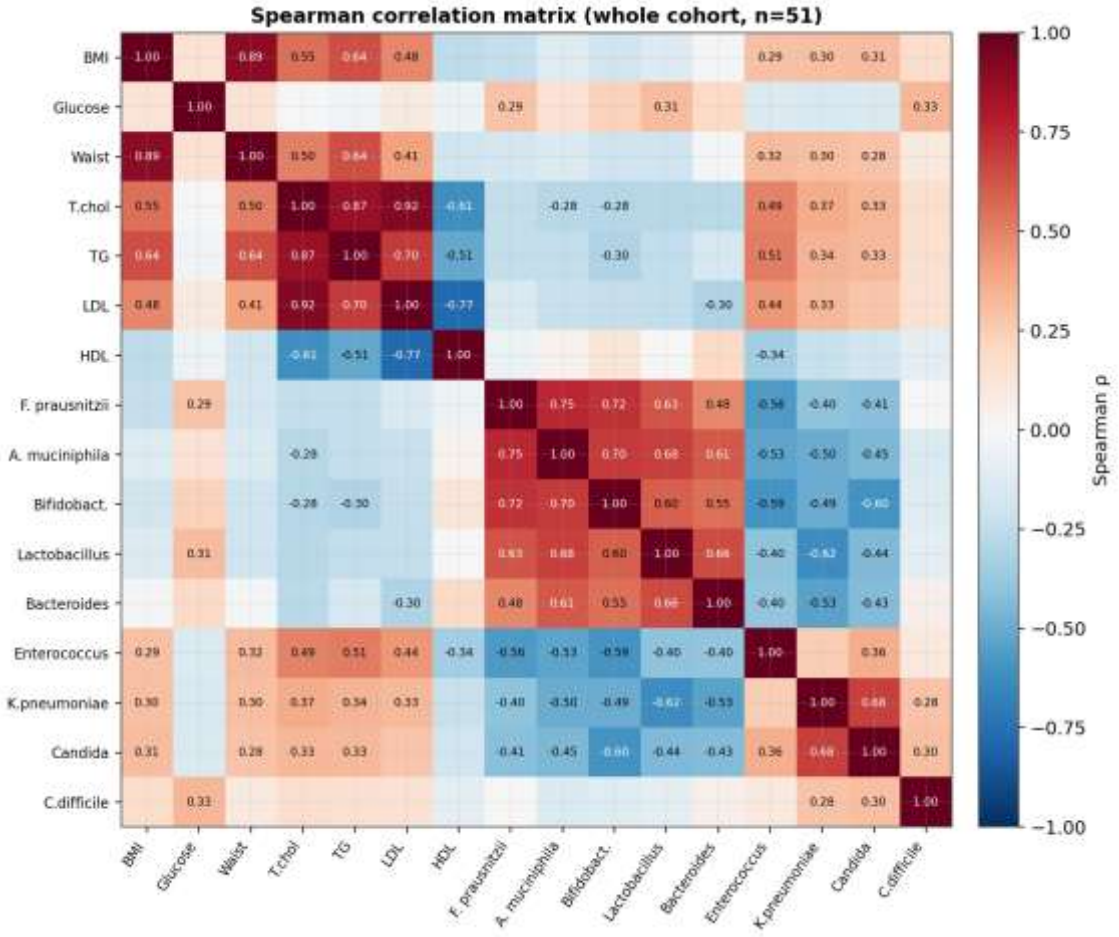

Figure 4. Spearman correlation matrix of selected metabolic indicators and microbial taxa across the whole cohort.

ROC analysis

The discriminatory ability of individual markers to separate the T2DM phenotype from obesity was assessed by ROC analysis (Table 5, Figure 5). Fasting glucose achieved perfect separation (AUC = 1.00), which is an expected, essentially definitional, consequence of the diagnostic criteria for diabetes rather than an independent finding. More informative were the microbial and renal markers: among these, Faecalibacterium prausnitzii showed the strongest discrimination (AUC = 0.77; sensitivity 0.43, specificity 0.96), followed by urea (AUC = 0.73), Bifidobacterium spp. (AUC = 0.71), creatinine (AUC = 0.68), Akkermansia muciniphila (AUC = 0.67) and Bacteroides thetaiotaomicron (AUC = 0.65). These values indicate moderate, hypothesis-generating discriminatory capacity for the microbial signature beyond the trivial separation provided by glycaemia [15].

Table 5. ROC analysis for discrimination of T2DM from obesity.

Marker	AUC	Sens.	Spec.	Direction*
Glucose	1.00	1.00	1.00	higher
Faecalibacterium prausnitzii	0.77	0.43	0.96	higher
Urea	0.73	0.57	0.91	lower
Bifidobacterium spp.	0.71	0.57	0.84	higher
Creatinine	0.68	0.57	0.80	higher
Akkermansia muciniphila	0.67	0.71	0.73	higher
Bacteroides thetaiotaomicron	0.65	0.43	0.98	higher
HDL cholesterol	0.56	0.71	0.50	lower

** Direction indicates whether higher or lower values of the marker are associated with the T2DM phenotype.*

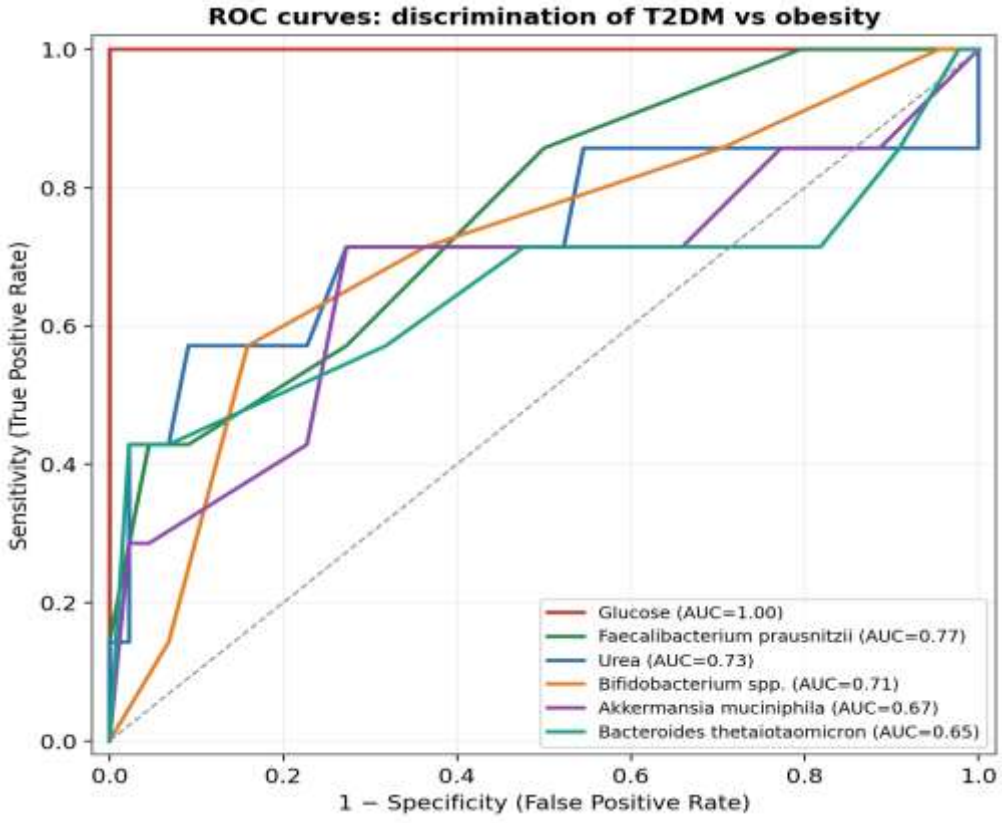

Figure 5. ROC curves for the discrimination of T2DM from obesity by individual markers. The diagonal represents chance performance.

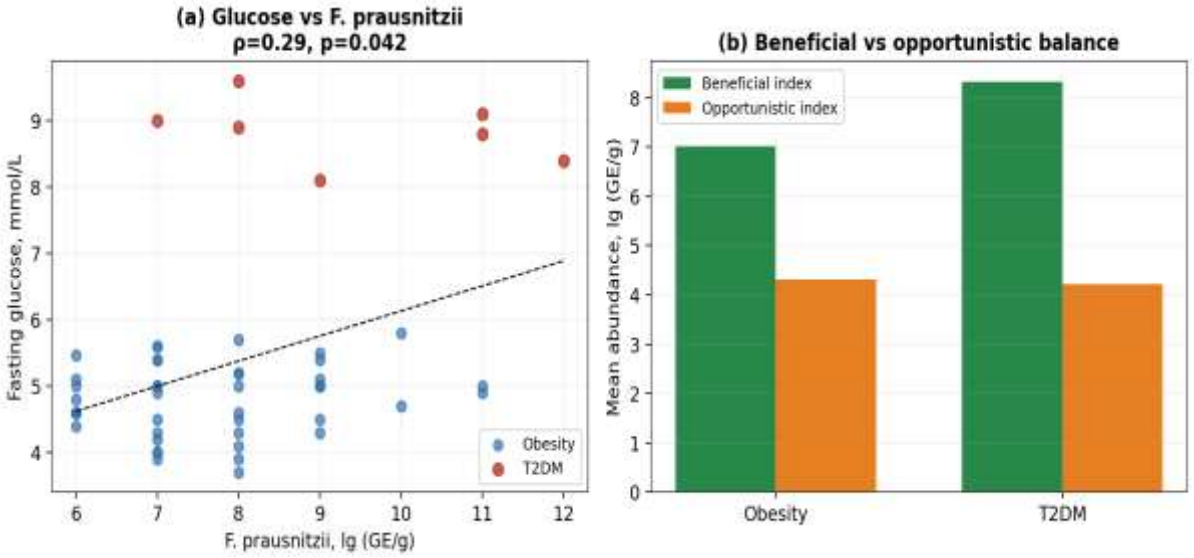

Figure 6. (a) Relationship between fasting glucose and Faecalibacterium prausnitzii abundance across the cohort, with linear trend line.

Discussion

This study compared the intestinal microbiota and metabolic profile of patients with obesity awaiting GLP-1 receptor agonist therapy and patients with recently diagnosed, currently untreated T2DM, using a standardised quantitative PCR panel. The principal observation is that the two metabolically related phenotypes carry distinguishable microbial signatures even when anthropometric and lipid indicators are largely similar, supporting the view that intestinal dysbiosis tracks with glycaemic dysregulation independently of body mass.

The strong positive correlations of fasting glucose with Clostridium perfringens, Clostridium difficile and Klebsiella oxytoca, together with the higher abundance of these opportunistic taxa in the T2DM group, are consistent with the broader literature describing an expansion of pro-inflammatory and facultatively anaerobic organisms in diabetic dysbiosis. An increased load of opportunistic Enterobacteriaceae and clostridia is thought to promote metabolic endotoxaemia and low-grade inflammation, which in turn aggravate insulin resistance.

A more nuanced finding concerns the butyrate-producing commensal Faecalibacterium prausnitzii, which in our cohort was higher in the T2DM group and showed the greatest discriminatory AUC among the microbial markers. This appears to contrast with the prevailing report that F. prausnitzii is depleted in established T2DM and exerts protective, insulin-sensitising effects. Several features of our design may reconcile this apparent discrepancy. First, the T2DM participants were recently diagnosed and, critically, had been free of glucose-lowering medication for three months, whereas the obesity comparison group was examined before any GLP-1 intervention; the relative enrichment of F. prausnitzii, Bifidobacterium spp. and A. muciniphila in the diabetic group may therefore reflect a compensatory or early-disease pattern rather than the depletion typically seen in long-standing, treated disease. Notably, prior work has reported that F. prausnitzii and A. muciniphila can increase in type 2 diabetic patients during weight-reducing and GLP-1-based interventions, underscoring that the abundance of these taxa is highly context- and treatment-dependent. Second, the small size of the T2DM group limits the precision of these estimates and may inflate the influence of individual outliers.

The relative enrichment of Escherichia coli and Enterococcus spp. in the obesity group, and the positive association of BMI with Candida spp., Klebsiella pneumoniae and Enterococcus spp., are in line with reports linking adiposity to an increased proportion of

facultative anaerobes and a reduced capacity for beneficial SCFA production. The mucin-degrading species A. muciniphila, widely regarded as inversely related to adiposity and metabolic syndrome and as a target of GLP-1 receptor agonist therapy, was numerically lower in the obesity group, in keeping with its proposed protective role, although the difference did not reach significance in this sample.

From a translational perspective, the moderate discriminatory performance of F. prausnitzii, Bifidobacterium spp. and A. muciniphila suggests that targeted microbial profiling could complement conventional biochemistry in metabolic phenotyping and, potentially, in predicting response to incretin-based therapy. Because the obesity group in this study represents a pre-treatment baseline, the present data also provide a reference point against which microbiota changes following semaglutide administration can be evaluated in future longitudinal work.

Conclusion

In this study, patients with pre-treatment obesity and patients with early, treatment-naïve type 2 diabetes mellitus exhibited distinct intestinal microbial signatures despite comparable anthropometric and lipid profiles. The diabetic phenotype was characterised by a higher total bacterial load and enrichment of Faecalibacterium prausnitzii, Bifidobacterium spp., Klebsiella oxytoca and Clostridium difficile, whereas the obesity phenotype showed relative enrichment of Escherichia coli and Enterococcus spp. Fasting glucose correlated with several opportunistic and commensal taxa, and ROC analysis identified F. prausnitzii, Bifidobacterium spp. and Akkermansia muciniphila as markers with moderate discriminatory value. These findings, although limited by the small and demographically unbalanced diabetic group, indicate that targeted quantitative microbiota profiling captures clinically meaningful differences between metabolic phenotypes and provide a baseline for future studies of GLP-1 receptor agonist–induced microbial remodelling.

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