

Gut Microbiome Report — Human

Patient: Homo sapiens · M3 Age: 37 years Sex: M Sample date: 2026-05-03 Report date: 2026-07-05 Lab: OmicaLabs SL — Pipeline: GenePortal Human Microbiome v2.1 (shotgun metagenomics)

Notice: Clinical support report. It does not constitute a medical diagnosis. Clinical interpretation requires correlation with the complete clinical picture and complementary tests.

1. Main clinical picture (37y, M)

Pre-intervention assessment in a metabolic context: microbiome profile with severe dysbiosis; interpret together with metabolic parameters and a history of recent antibiotic therapy if applicable. No bacterial or eukaryotic pathogens of high clinical priority detected. Profile compatible with moderate-severe dysbiosis (severely dysbiotic band).

2. Patient clinical data

Field	Value
Species	human
Age	37
Sex	M
Weight (kg)	88.0
Diet	omnivorous (traditional Chinese pattern, rice/vegetables/meat)
Antibiotics in the last 30 d	False
Antibiotic (drug)	—
Probiotics	False
Sample date	2026-05-03

Field	Value
Storage	—
Geography (ISO)	CN

2.1 Clinical context

Reason for the test: Evaluation of gut dysbiosis in recently diagnosed type 2 diabetes with poor metabolic control despite metformin. Suspected contribution of the microbiome to insulin resistance.

Main symptoms: - Polyuria and polydipsia (3 months) - Persistent fatigue - Occasional blurred vision - No specific gastrointestinal symptoms

Relevant history: - Type 2 diabetes diagnosed 10 months ago (HbA1c 8.9% at diagnosis) - Current HbA1c 7.8% despite metformin 2000 mg/day + glibenclamide 5 mg/day - BMI 28.3 kg/m² (class II overweight for the Asian population) - HOMA-IR 4.8 (significant insulin resistance) - Mixed dyslipidemia: TG 220 mg/dL, HDL 34 mg/dL, LDL 145 mg/dL - Abdominal ultrasound: moderate hepatic steatosis (no fibrosis)

Current medication: - Metformin 1000 mg/12h - Glibenclamide 5 mg/day - Atorvastatin 20 mg/day

Comorbidities: - Mixed dyslipidemia - Non-alcoholic hepatic steatosis (NAFLD) - Overweight (BMI 28.3)

Family history: - Mother: type 2 diabetes (diagnosed at age 52) - Father: hypertension, MI at age 64

3. Sample quality (QC)

Metric	Value	Status
Total reads	44,177,224	—
Host reads	0.0%	OK
Post-filter reads	44,172,806	—
Classified reads	18,653,735	—
NTC	—	—

Metric	Value	Status
Mock community	—	—
Batch QC	Not evaluated	—

4. Taxonomic composition

4.1 Top 5 phyla

Phylum	Abundance %
Pseudomonadota (Proteobacteria)	95.21
Bacillota (Firmicutes)	3.21
Actinomycetota (Actinobacteria)	1.56
Bacteroidota (Bacteroidetes)	0.02

4.2 Top 10 genera

Genus	Abundance %
Escherichia	87.47
Klebsiella	8.52
Raoultella	0.01
Shigella (see enteroinvasive E. coli (EIEC))	1.42
Citrobacter	0.06
Salmonella	0.34
Enterobacter	0.03
Kluyvera	0.01
Leclercia	0.01
Kosakonia	0.00

4.4 Alpha diversity

Index	Value	Healthy range
Shannon	0.521	2.5 - 4.5 (healthy)
Simpson	0.227	0.8 - 0.98 (healthy)
Chao1	128.1	—
Observed ASVs	—	—
Pielou evenness	0.126	—

5. Dysbiosis Index (iHMP HMP2 proxy)

- Dysbiosis Index (z-score sum): 10.898 (severely dysbiotic)
- Bacillota / Bacteroidota ratio: 208.934 (firmicutes_dominant)
- β distance (Bray-Curtis vs healthy centroid): 0.9997
- β distance (Euclidean, log10): 1.0920
- Method: iHMP_HMP2_zscore_proxy_human_v2
- Reference: iHMP_HMP2_2019 + Gevers2014_CellHostMicrobe + Gupta2020_NatureComms + Toto2024_PedIBD

| Notice: not equivalent to the clinical DI by qPCR.

5.1 Contribution per taxon

Taxon	Abundance %	z-score	Direction	Contribution
Faecalibacterium	0.003	-3.71	health	+3.71
Roseburia	0.001	-3.25	health	+3.25
Alistipes	0.002	-1.41	health	+1.41
Prevotella	0.00 (not detected)	0.00	health	0.00
Ruminococcus	0.002	-0.57	health	+0.57
Coprococcus	0.001	-0.36	health	+0.36

Taxon	Abundance %	z-score	Direction	Contribution
Subdoligranulum	0.00 (not detected)	0.00	health	0.00
Akkermansia	0.00 (not detected)	0.00	health	0.00
Escherichia coli	87.47	1.59	dysbiosis	+1.59
Clostridium (sensu stricto)	0.00 (not detected)	0.00	dysbiosis	0.00

- 4/10 panel taxa not detected — the DI should be interpreted with caution.
- Δ Centroid calibrated against a European population reference (European DMP (Gacesa 2022, n=8.208)); local Spanish cohort pending. The β distance and the derived DI have reduced accuracy for non-European populations; interpret as a guide.

6. Indicator species (SCFA producers)

Genus	Detected %	Healthy range	Status
Faecalibacterium	0.003	—	low
Roseburia	0.001	—	low
Alistipes	0.002	—	normal
Prevotella	0.00 (not detected)	—	normal
Ruminococcus	0.002	—	normal
Coprococcus	0.001	—	normal
Subdoligranulum	0.00 (not detected)	—	low
Akkermansia	0.00 (not detected)	—	normal
Escherichia coli	87.47	—	high
Clostridium (sensu	0.00 (not	—	low

Genus	Detected %	Healthy range	Status
stricto)	detected)		

SCFA producers total: 0.49 % (healthy > 12 %)

7. Species-specific biomarkers (clinical enrichment)

Human clinical biomarkers (SoTA 2020-2025 — Gupta 2020 + Frioux 2023 + Zheng 2024)

- Enterosignatures (NMF soft): ES-B=0%, ES-P=0%, ES-F=0%, ES-Bf=1%, ES-Ec=99%
- EDS (Enterotype Dysbiosis Score): 0.01 (defined enterotype)
- GMHI (Gut Microbiome Health Index): -2.49 (strongly not healthy)
- GMWI2 (Chang/Gupta 2024): -0.844 (strongly not healthy) [14S/15NS taxa detected]
- ICB = 0.0% (critical). Derived from the weighted sum of producer genera (Faecalibacterium, Agathobacter, Roseburia, Eubacterium, Anaerobutyricum, Anaerostipes, Coprococcus, Butyricicoccus, Subdoligranulum). It is NOT a fecal SCFA concentration; qualitative correlation with butyrogenic production.
- C. difficile risk: high (critical butyrogenic capacity (< 3%); Shannon < 2.0; Enterobacteriaceae bloom > 5%)
- Disease signatures:
 - IBD Panel (Zheng 2024) — NOT ASSESSABLE: 0 enriched spp, AUC=0.0 — 0 depleted detected. SCREENING ONLY — NOT a diagnosis.

CRC / IBD — Screening (NOT a diagnosis)

- IBD Panel Zheng 2024 — NOT ASSESSABLE
 - IBD Panel NOT assessable: dominance of Escherichia coli (87.3%) and extreme diversity (Shannon=0.52) — pattern compatible with severe dysbiosis / pharmacological confounding effect (metformin, PPI, recent antibiotic), not interpretable as an IBD signature. Repeat after microbiota normalization if clinical suspicion persists.
 - Ref: Zheng et al. 2024, Nature Medicine 30:3555-3567, PMID: 39367251

Quality Control

- Batch QC status: Not evaluated
- Beneficial genera: detected
 - Faecalibacterium altered (0.00%) — Major butyrate producer, anti-inflammatory

(*F. prausnitzii*)

- Bifidobacterium altered (0.46%) — SCFA/lactate, immune modulation; neonatal dominance
- Roseburia altered (0.00%) — Butyrate producer (*R. intestinalis*), depleted in IBD
- Agathobacter altered (0.00%) — Butyrate producer (*A. rectalis* = ex-*E. rectale*), fiber fermentation
- Ruminococcus altered (0.00%) — Resistant starch degrader (*R. bromii*), key for fiber
- Bacteroides altered (0.02%) — SCFA, bile acid metabolism — DUAL ROLE (commensal + opportunist)
- Alistipes altered (0.00%) — SCFA producer — DUAL ROLE (associations with depression, CRC)
- Pathogenic alerts (3):
 - [MODERATE] Salmonella elevated (0.34% > 0.1%) — Notifiable enteropathogen
 - [MODERATE] Escherichia coli elevated (87.47% > 3.0%) — Commensal overgrowth with no virulence genes detected (STEC/EPEC/EIEC pathotypes only if stx1/stx2/eae/ipaH present in VFDB)
 - [MODERATE] Klebsiella elevated (8.52% > 0.5%) — MDR risk, potential CRE (*K. pneumoniae*)
 - ⚠ Shigella detected (1.42%) is probably cross-mapping of enteroinvasive *E. coli* (EIEC, ipaH+). *E. coli* is 61.7x more abundant. Confirm with specific ipaH PCR + MLST if there is compatible clinical presentation.

HIGH ALERTS (6 alerts)

- ACTION: Dysbiosis index=10.9 — severely dysbiotic band (> 10.0)
- ACTION: Critically low alpha diversity (Shannon=0.52) — below the healthy range 2.5-4.5, predisposes to pathobiont overgrowth and loss of microbial resilience
- ACTION: Butyrogenic capacity 0.0% — CRITICAL depletion
- ACTION: HIGH *C. difficile* risk: critical butyrogenic capacity (< 3%); Shannon < 2.0; Enterobacteriaceae bloom > 5%
- ACTION: CRITICAL DOMINANCE: *Escherichia coli* represents 87.3% of the total microbiome — risk of bacterial translocation, endotoxemia and loss of barrier function
- ACTION: critical butyrate depletion

MODERATE ALERTS (8 alerts)

- Faecalibacterium 0.00% — major butyrate producer depleted

- [MODERATE] Salmonella elevated (0.34% > 0.1%) — Notifiable enteropathogen
- [MODERATE] Escherichia coli elevated (87.47% > 3.0%) — Commensal overgrowth with no virulence genes detected (STEC/EPEC/EIEC pathotypes only if stx1/stx2/eae/ipaH present in VFDB)
- [MODERATE] Klebsiella elevated (8.52% > 0.5%) — MDR risk, potential CRE (K. pneumoniae)
- Salmonella overgrowth
- main butyrate producer low
- butyrate producer low
- elevated MDR risk

INFORMATIVE (2 alerts)

- IBD Panel NOT assessable: dominance of Escherichia coli (87.3%) and extreme diversity (Shannon=0.52) — pattern compatible with severe dysbiosis / pharmacological confounding effect (metformin, PPI, recent antibiotic), not interpretable as an IBD signature. Repeat after microbiota normalization if clinical suspicion persists.
- Disease signature (IBD Panel Zheng 2024 — NOT ASSESSABLE, AUC=None): 0 enriched spp — NOT a diagnosis
- Clinical pattern: Dysbiotic pattern with Enterobacteriaceae overgrowth (Enterococcus/Escherichia/Klebsiella) and Faecalibacterium depletion — the differential includes recent antibiotic therapy, chronic PPI, ketogenic diet or chronic inflammation. No clinical metadata to characterize it; correlate with history.
- Estimated age: 37 years (declared) (based on patient data)

Clinical interpretation

ALERT: Community dominated by Escherichia (ES-Ec) with strong membership (0.99). This enterotype is associated with inflammatory bowel disease and ecological disruption (Frioux 2023, Keller 2024). Butyrogenic capacity is CRITICALLY reduced. Butyrate is the main fuel for colonocytes and its depletion is associated with intestinal inflammation and an ecological risk of C. difficile colitis. ALERT: Elevated ecological risk of C. difficile colitis. Confirm with toxin A/B PCR + GDH if there is diarrhea. IBD Panel NOT assessable: dominance of Escherichia coli (87.3%) and extreme diversity (Shannon=0.52) — pattern compatible with severe dysbiosis / pharmacological confounding effect (metformin, PPI, recent antibiotic), not interpretable as an IBD signature. Repeat after microbiota normalization if clinical suspicion persists.

Limitations (12)

- There is NO ACMG equivalent for microbiome interpretation. These metrics are orientative and must be integrated with the complete clinical picture.

- Enterotypes: they are NOT discrete categories (Costea 2018, Bulygin 2023). They represent continuous trends on a non-linear manifold.
- GMHI requires species-level data. MetaPhlAn 4 uses GTDB taxonomy (SGBs) while GMHI uses NCBI. The SGB→NCBI mapping is approximate.
- DI V2 panel (10 genera): asymmetric 8:2, AUC=0.745, external validation: independent cohort ongoing.
- The F/B ratio is NOT a reliable standalone biomarker (Finucane 2014 PLOS ONE; Magne 2020 Nutrients). Interpret with DI, diversity and clinical context.
- Detection of ARGs from a metagenome does NOT equate to phenotypic resistance. EUCAST 2024: VME < 5% in only 44% of antibiotics.
- Disease signatures (IBD/CRC/T2D/obesity) are SCREENING TOOLS — NOT diagnostic.
- Reference ranges come mainly from the Dutch Microbiome Project (n=8,208, European population). Their applicability to other populations requires validation.
- Alpha diversity (Shannon) is a coarse ecological measure that does NOT predict clinical outcomes by itself.
- Q2PD (Zielinska et al. 2025, GigaScience 14:giaf015) is a functional index based on MetaCyc metabolic pathways. Currently NOT AVAILABLE.
- Drugs that alter intestinal transit (loperamide, opioids) or gastric pH (PPI) can modify the microbiome composition by changes in residence time and luminal acidity. Interpret with caution if the patient received them in the 2 weeks before sample collection.
- This report is for research use and clinical context. It does not constitute a medical diagnosis. ISO/TS 24420:2023 covers metagenomic data processing.
- Pharmacological confounders detected in the patient's medication: Metformin alters the gut microbiome composition (increases Akkermansia and Escherichia, reduces Bacteroides). A known confounder in microbiome studies in T2D.

References (25)

- Frioux et al. 2023, Cell Host & Microbe 31:1111-1125 (5 ES, 5,230 metagenomes)
- Keller et al. 2024, bioRxiv 2024.08.13.607711 (fuzzy enterotyping, EDS, 16,772 metagenomes)
- Bulygin et al. 2023, PeerJ 11:e15764 (no discrete clusters — manifold learning)
- Gupta et al. 2020, Nature Comms 11:4635 (GMHI, 50 species, AUC=0.73)
- Sun et al. 2024, npj Biofilms 10:96 (GMHI validation, 6,314 metagenomes, AUC=0.825)
- Gevers et al. 2014, Cell Host Microbe 15:382-392 (MD-index)
- Zheng et al. 2024, Nature Medicine 30:3555-3567 (IBD panels, 5,979 metagenomes)
- Piccinno/Segata/Huttenhower 2025, Nature Medicine (CRC pooled 3,741, AUC=0.85)

- Mei/Huttenhower et al. 2024, Nature Medicine 30:2265-2276 (T2D, 8,117 metagenomes)
- Chanda & De 2024, Gut Microbes 16:2304900 (obesity 25 spp, AUC=0.85)
- Gacesa et al. 2022, Nature 604:732-739 (DMP, n=8,208, 9 core species)
- Asnicar et al. 2021, Nature Medicine 27:321-332 (PREDICT 1, n=1,098)
- Andreu-Sanchez/Segata/Fu et al. 2025, Cell (32,152 metagenomes, 42 countries)
- Mancabelli et al. 2024, mSystems (6,653 samples, 5 life stages)
- Shao et al. 2024, Nature Microbiology (1,288 UK neonates, 3 community states)
- Vaiserman et al. 2020, BMC Microbiol 20:114 (F/B age-stratified, n=1,550)
- Finucane et al. 2014, PLOS ONE 9:e84689 (F/B NOT reliable standalone biomarker)
- Magne et al. 2020, Nutrients 12:1474 (F/B critical review)
- Vital et al. 2017, mBio 8:e01492-17 (butyrate producers)
- Dong et al. 2024, Nature Comms (VFDB 2.0 + MetaVF, 62,332 VFGs)
- Lessa et al. 2015, NEJM 372:825-834 (C. difficile epidemiology)
- EUCAST 2024: metagenomic ARG $\neq$ phenotypic AST
- Zielinska et al. 2025, GigaScience 14:giaf015 (Q2PD, PMID: 40117176, pathway-based dysbiosis, >8,000 samples, outperforms GMHI/hiPCA for IBD)
- ISO/TS 24420:2023 — shotgun metagenomic data processing
- STROBE-Metagenomics, Lancet Infect Dis, Oct 2025

8. Functional profiling (HUMANn 3 + dbCAN 3)

Most represented metabolic pathways (top 10)

Metabolic pathway	Abundance (RPKM)	Clinical interpretation
PWY0-1586: peptidoglycan maturation (meso-diaminopimelate containing)	103,845	Cell wall synthesis — Peptidoglycan maturation.
PWY-7238: sucrose biosynthesis II	58,924	Carbohydrate metabolism — Sucrose biosynthesis. Indicates active bacterial glycolytic activity.
PWY-7111: pyruvate fermentation to isobutanol (engineered)	58,014	Fermentation — Pyruvate fermentation to isobutanol. Bacterial fermentative activity.
NONOXIPENT-PWY: pentose phosphate pathway (non-oxidative	57,411	Pentose phosphate — Pentose phosphate pathway (non-oxidative branch). Bacterial

Metabolic pathway	Abundance (RPKM)	Clinical interpretation
branch) I		NADPH generation.
VALSYN-PWY: L-valine biosynthesis	55,839	Amino acid synthesis — Valine biosynthesis. Basal metabolic activity.
PWY-5941: glycogen degradation II	55,024	Glycogen metabolism — Bacterial glycogen degradation. Energy storage.
ILEUSYN-PWY: L-isoleucine biosynthesis I (from threonine)	51,150	Amino acid synthesis — Isoleucine biosynthesis. Basal metabolic activity.
PWY0-1477: ethanolamine utilization	48,139	Bacterial metabolic activity — Bacterial metabolic pathway — part of the basal metabolism of the gut microbiome.
P461-PWY: hexitol fermentation to lactate, formate, ethanol and acetate	46,847	Bacterial metabolic activity — Bacterial metabolic pathway — part of the basal metabolism of the gut microbiome.
CALVIN-PWY: Calvin-Benson-Bassham cycle	23,334	Energy metabolism — Calvin-Benson-Bassham cycle. Bacterial CO ₂ fixation.

777 pathways detected in total by HUMAnN 3. The 10 with the highest abundance (RPKM) are shown, excluding unclassified reads.

Note: The most represented pathways correspond mainly to basal (housekeeping) metabolic activity of the commensal microbiota — glycolysis, amino acid synthesis, nucleotides and cell wall. Their clinical relevance is global (it indicates metabolic activity of the microbiome), not individual. Alterations in the relative abundance of specific pathways (butyrate, bile acids, LPS) are more informative than the presence of housekeeping pathways.

8.1 Overall functional assessment

Of the 10 most abundant pathways, 6 are housekeeping pathways (basal metabolism: glycolysis, amino acids, nucleotides, cell wall) and 4 are specialized-metabolism pathways (fermentation, energy storage, isoprenoids).

Interpretation: The diversity of active metabolic pathways indicates a metabolically competent microbiota. The presence of multiple housekeeping pathways at high abundance is expected in a metabolically active microbiome — it means the bacteria are alive, reproducing and processing nutrients.

Clinical relevance of the functional pathways: the presence of housekeeping pathways is necessary but not sufficient for a healthy microbiome. What is clinically relevant is the depletion of butyrate-specific production pathways (butyryl-CoA acetate CoA-transferase pathway) and the elevation of pro-inflammatory pathways (LPS synthesis, flagella). These functional alterations are already reflected in the biomarkers of section 7 (butyrogenic capacity, *C. difficile* risk) and are more clinically actionable than the individual abundance of each housekeeping pathway.

9. Pathogens detected (bacterial + eukaryotic + fungi)

9.1 Bacterial pathogens

[MODERATE] *Salmonella enterica* — MODERATE - Abundance: 0.00% | Reads: 6,315 - Clinical significance: SPI-1/SPI-2 virulence genes NOT detected by metagenomics. Possible commensal non-typhoidal *Salmonella* or limited shotgun sensitivity for these genes (low genomic coverage of the pathogen at low relative abundance). Interpret in clinical context: if the patient is asymptomatic, probable carrier. If there is acute diarrhea, confirm with stool culture + serotyping.

[MODERATE] *Escherichia coli* (pathotypes) — MODERATE - Abundance: 0.00% | Reads: 2,954,029 - Clinical significance: Enteropathogenic *E. coli* virulence genes (stx1/stx2/eae/ipaH/elt/est/aggR/afa) NOT detected by metagenomics. *E. coli* is a ubiquitous commensal of the human gut. Pathogenicity depends on the pathotype determined by specific virulence genes: STEC/STEC (stx1/stx2+eae), EPEC (eae), ETEC (elt/est), EIEC (ipaH), EAEC (aggR/afa). Without these determinants, the detected strain probably represents non-pathogenic commensal colonization. Interpret in clinical context: if there is acute diarrhea or HUS, confirm with multiplex pathotype PCR + Shiga toxin in stool. Mandatory notification (RD 1054/2002) applies only to laboratory-confirmed STEC.

9.1.x Taxonomic ambiguities (probable cross-mapping)

△ Note: *Shigella* detected (1.42%) is probably cross-mapping of enteroinvasive *E. coli* (EIEC, ipaH+). *E. coli* is 61.7x more abundant. Confirm with specific ipaH PCR + MLST if there is compatible clinical presentation.

9.3 Fungi (mycobiome)

Note on fungal detection by shotgun metagenomics: Fungal biomass in stool is typically <0.1% of the total metagenome (Nash et al. 2017, Microbiome). The sensitivity of shotgun for fungi is lower than for bacteria due to low abundance, a cell wall resistant to lysis (chitin/glucans), and large genomes with lower coverage per sequencing depth. A negative result does NOT rule out a clinically relevant fungal infection. Always interpret in the clinical and dietary context of the patient.

Classification methodology: Fungi are classified according to the BugID/BugSeq matrix (2026): clinical classification of the species (according to WHO/ECDC priority lists) × universal signal threshold. The threshold has been calibrated against the natural distribution of fungal abundance in the human fecal metagenome: the breakpoint at 10 RPM separates the normal mycobiotic background from elevated signal, consistent with the MetaMIC minimum reporting. An absolute floor of 30 reads filters cross-mapping noise. IMPORTANT: There are no species-validated thresholds in the literature for fungi in stool by shotgun — these thresholds are for screening, not diagnostic. Any detection at trace level requires orthogonal confirmation (culture + ITS PCR) before making clinical decisions.

Mycobiome — traces without clinical significance

- Traces of environmental/dietary fungal DNA were detected (8 species: *Aspergillus flavus*, *Candida albicans*, *Candida auris*, *Candida glabrata*, *Candida parapsilosis*...; 506 total reads) in an immunocompetent outpatient. No clinical significance. Confirm with culture + ITS PCR only if there are risk factors (immunosuppression, hospitalization, TPN, prior antifungals).

Confirmatory gold standard for fungi: Culture on CHROMagar/Sabouraud medium + MALDI-TOF + ITS1/ITS2 PCR. Taxonomic identification by shotgun has screening value; species confirmation and the antifungal susceptibility profile require isolation in culture.

△ Very low abundance (<0.01%). Detection of fungi at trace level in shotgun metagenomics may reflect: (1) real subclinical colonization, (2) environmental contamination (ubiquitous spores), or (3) dietary origin (bread, beer, cheese, probiotics). The fungal cell wall (chitin/glucans) is resistant to standard DNA extraction lysis, which underestimates the real abundance. Correlate with clinical findings and consider targeted culture if there are risk factors (immunosuppression, prolonged hospitalization, parenteral nutrition, broad-spectrum antibiotic therapy).

WARNING — Dietary false positives: Detection of *Saccharomyces cerevisiae* DNA (baker's/brewer's yeast) is common in fecal shotgun and almost always reflects recent dietary intake (bread, beer, wine, cheese, *S. boulardii* probiotics), NOT infection. ALWAYS interpret in dietary context before attributing clinical significance.

9.4 Viruses / phages

- Phage: *Klebsiella* phage LASTA: 0.01%
- Phage: *Klebsiella* phage SopranoGao: 0.0095%
- Phage: *Klebsiella* phage vB_KpnP_P184: 0.0040%
- Phage: *Klebsiella* phage vB_KpP_FBKp27: 0.0030%

Clinical note: The viruses detected are commensal bacteriophages, normal components of

the gut microbiome with no known clinical implication. No pathogenic human DNA viruses were detected above reporting thresholds.

9.x Taxonomic ambiguities (probable cross-mapping)

The following findings are flagged as probable cross-mapping with a more abundant taxon. They must not be interpreted as confirmed pathogens; they require specific PCR/MLST before clinical decisions.

- ⚠ Shigella detected (1.42%) is probably cross-mapping of enteroinvasive E. coli (EIEC, ipaH+). E. coli is 61.7x more abundant. Confirm with specific ipaH PCR + MLST if there is compatible clinical presentation.

10. Antibiotic resistance (AMR genes)

[OK] No acquired resistance genes were detected in the assembled contigs (AMRFinderPlus + VFDB).

Note: The absence of ARGs in a metagenome does NOT guarantee phenotypic susceptibility. EUCAST 2024: VME < 5% in only 44% of antibiotics. Requires culture + antibiogram.

11. Prioritized recommendations

Priority	Action	Justification	Gold standard	Timeframe
PRIORITY	Rule out C. difficile	High risk due to critical butyrogenic capacity + low Shannon	PCR toxin A/B + GDH (if diarrhea)	24-48 h
FOLLOW-UP	Urgent clinical evaluation + confirmatory PCR of detected pathogens. Re-test microbiome in 4-8 weeks after resolution of the acute episode.	Decided by severity — Critical	Urgent evaluation + confirmatory PCR	4-8 weeks after resolution

12. Clinical conclusions

- Dysbiosis index: 10.898 (severely dysbiotic band).
- Enterotype Dysbiosis Score: 0.01 (defined enterotype).
- Reactive commensal expansion of Salmonella enterica, Escherichia coli without virulence genes (VFDB negative) — does not suggest acute infection. No specific clinical action required.

- No acquired antibiotic resistance genes were detected.
- ALERT: Elevated *C. difficile* risk. Rule out with PCR toxin A/B + GDH if diarrhea.
- Notice: report of. It does not constitute a medical diagnosis. Interpretation requires complete clinical correlation.

13. Methodology and limitations — regulatory compliance

- STORMS guidelines, Mirzayi et al. Nat Med 2021
- MlxS-Human v6.3.0 (Genomic Standards Consortium)
-
- EUCAST 2025 + IDSA/SHEA antimicrobial stewardship
- Pipeline: GenePortal Human Microbiome v2.1 (shotgun metagenomics)
- Generated: 2026-07-05 22:57

Limitations:

- Shotgun DNA does not detect RNA viruses (coronavirus, rotavirus, calicivirus, astrovirus); it requires RNA-seq or specific PCR.
- Relative abundance does not equate to viability or an absolute CFU count; the official numerical DI requires quantitative qPCR.
- Shotgun provides AMR genotype but not a phenotypic profile (MIC); it requires targeted culture + antibiogram.
- Critical eukaryotic pathogens (e.g. *Giardia*, *Cryptosporidium*) require specific PCR as the gold standard — shotgun sensitivity at ~3-6M reads is limited.
- Clinical interpretation requires correlation with the clinical picture, diet, previous treatments and other complementary tests.

14. Validation

Role	Name	License number
Analysis lead (bioinformatics)	_____	OmicaLabs SL — Pipeline GenePortal Human Microbiome v2.1
Clinical validation lead	Juan Carlos Barba Carretero	Nº Col. 002395189P

OmicaLabs SL — Clinical decision support report. Processing, analysis and interpretation performed in accordance with STORMS (Mirzayi 2021), MlxS-Human v6.3.0 and ISO/TS 24420:2023 for shotgun metagenomic data processing.

