

ORIGINAL RESEARCH ARTICLE

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INTESTINAL DYSBIOSIS AND ALTERATIONS OF CROSS-FEEDER BACTERIA IN THE DEVELOPMENT OF ANTIMICROBIAL RESISTANCE

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Abstract

Intestinal dysbiosis represents a significant disruption of the gut microbiota and is closely associated with the development of antibiotic resistance. This study aims to evaluate the role of alterations in cross-feeder bacteria in this process. A retrospective analysis was conducted on six dysbiosis tests (GA-map® Dysbiosis Test) performed over the past six months at the Intermedica Medical Center Laboratory. The study included six intestinal microbiota profiles obtained from patients routinely referred for microbiota evaluation at the Intermedica Medical Center Laboratory. The profiles were analyzed retrospectively, without additional study-specific inclusion criteria. Changes in microbiota composition and the presence of key metabolic bacteria were analyzed. A reduction in bacterial diversity and alterations in the commensal flora were observed in most cases. The most significant finding was a decrease in cross-feeder bacteria, which play a crucial role in the production of short-chain fatty acids (SCFAs). This reduction was associated with an increase in opportunistic microorganisms. The observed patterns suggest that antibiotic use creates selective pressure that promotes resistance through mechanisms such as horizontal gene transfer and biofilm formation. Intestinal dysbiosis, particularly the disruption of cross-feeder bacteria, plays a key role in the development of antibiotic resistance. Monitoring the gut microbiota and implementing targeted therapeutic interventions—including probiotics, prebiotics, and the rational use of antibiotics—are essential for maintaining intestinal homeostasis and preventing the spread of antimicrobial resistance.

Keywords: Intestinal dysbiosis, antibiotic resistance, microbiota, cross-feeder bacteria, SCFAs, Albania.

1. Introduction

The intestinal microbiota constitutes a highly complex microbial ecosystem composed of bacteria, viruses, fungi, and archaea that coexist in symbiosis with the human host. Under physiological conditions, the gut microbiota demonstrates stability, resilience, metabolic cooperation, and functional diversity. These characteristics are essential for maintaining intestinal homeostasis, regulating immune responses, supporting metabolic processes, and preventing colonization by pathogenic microorganisms [1, 2].

The composition of the microbiota is dynamic and influenced by multiple factors, including age, diet, medication exposure, antibiotic use, environmental conditions, and immune status. A healthy gut microbiota is generally characterized by high taxonomic diversity, functional redundancy, and stable metabolic interactions [3, 4].

Recent research has emphasized that the microbiota should not be viewed solely as a bacterial composition, but rather as a functional ecological network. Cross-feeder bacteria are one of the most important components of this network. These microorganisms participate in cooperative metabolic pathways where metabolites produced by one bacterial group become substrates for another. This interaction is essential for efficient fermentation and the production of short-chain fatty acids (SCFAs), particularly butyrate, acetate, and propionate [5–7].

SCFAs play a critical role in maintaining intestinal barrier integrity, regulating inflammation, modulating immune responses, and supplying energy to colonocytes. Disruption of these metabolic networks may lead to impaired barrier function, increased intestinal permeability, inflammation, and reduced colonization resistance [8, 9].

Antibiotic exposure is one of the most important factors capable of inducing dysbiosis. Ecological disruption of the intestinal microbiota may favor opportunistic microorganisms and contribute to the expansion of antimicrobial resistance (AMR). Although dysbiosis does not directly generate resistance genes, it may create a favorable environment for the persistence, dominance, and horizontal transfer of resistant microorganisms [9–11].

The primary aim of this study was to evaluate the role of intestinal dysbiosis in the development of antimicrobial resistance through analysis of functional microbiota alterations.

The specific objectives were (i) to analyze structural and functional changes of the intestinal microbiota under dysbiosis conditions; (ii) to evaluate alterations in cross-feeder bacterial networks; (iii) to assess the relationship between SCFA-associated bacteria and intestinal ecological stability; and (iv) to investigate the association between dysbiosis severity and AMR potential.

2. Materials and Methods

2.1. Study Design

A retrospective descriptive case-series study was conducted to evaluate functional alterations of the intestinal microbiota associated with dysbiosis and their potential relationship with antimicrobial resistance (AMR). The analysis included six GA-map® Dysbiosis Tests performed during the previous six months at Intermedica Medical Center Laboratory, Albania.

2.2. Study Population

The analyzed microbiota profiles represented a heterogeneous group of patients routinely referred for intestinal microbiota evaluation in daily clinical laboratory practice. The study population reflected real-world clinical conditions rather than a selected disease-specific cohort.

2.3. Microbiota Analysis

Intestinal microbiota evaluation was performed using the GA-map® Dysbiosis Test, a molecular diagnostic assay based on bacterial DNA detection and 16S rRNA profiling technology. The method combines bacterial DNA extraction, amplification of bacterial 16S rRNA regions, and hybridization-based detection to characterize intestinal microbial composition and functional ecological patterns. The test evaluates multiple microbiota domains, including bacterial diversity, broad commensal bacteria, anti-inflammatory bacterial groups, probiotic/lactic acid bacteria, cross-feeder bacterial networks, opportunistic microorganisms, and the Dysbiosis Index (DI). GA-map® was selected because it provides standardized functional microbiota profiling applicable in routine clinical laboratory practice.

The study focused primarily on functional ecological alterations rather than isolated taxonomic changes. The following microbiota parameters were evaluated: alpha diversity, broad commensal bacteria, cross-feeder bacterial networks, SCFA-associated bacteria, anti-inflammatory bacterial groups, probiotic/lactic acid bacteria, opportunistic microorganisms, and ecological AMR-associated potential. Attention was directed toward cross-feeder bacteria because of their central role in microbial metabolic cooperation and short-chain fatty acid (SCFA) production.

Cases were categorized according to the Dysbiosis Index (DI) generated by the GA-map® system. The DI ranges from 1 to 5, with DI1 indicating minimal or absent dysbiosis; DI2, mild dysbiosis; DI3, moderate dysbiosis; DI4, severe dysbiosis; and DI5, very severe dysbiosis. In the present study, no cases were classified as DI4.

Although the GA-map® Dysbiosis Test does not directly identify antimicrobial resistance genes, microbiota profiles were interpreted for ecological conditions potentially favoring AMR development. These included reduction of colonization resistance, expansion of opportunistic microorganisms, disruption of SCFA-producing bacterial networks, and ecological destabilization associated with resistant microorganism persistence and horizontal transfer potential [12–14].

2.4. Statistical and Functional Analysis

Given the limited sample size ($n = 6$), statistical evaluation remained descriptive. The analysis focused primarily on identifying biological trends and progressive ecological alterations associated with increasing dysbiosis severity. Functional interpretation was supported using descriptive comparative analysis, heatmap visualization models, radar profile analysis, and ecological progression models illustrating functional deterioration from DI1 to DI5.

3. Results

3.1. Dysbiosis Distribution

A total of six microbiota profiles were included in the present analysis. According to the GA-map® DI, one case corresponded to DI1, three cases to DI2, one case to DI3, and one case to DI5. The mean Dysbiosis Index of the cohort was 2.5, indicating a predominance of mild-to-moderate ecological alterations rather than complete microbiota collapse. The distribution of Dysbiosis Index categories is summarized in Table 1 and illustrated in Figure 1.

Table 1. Distribution of dysbiosis index.

Dysbiosis Index	Number of Cases	Interpretation
DI1	1	Normobiotic / minimal alterations
DI2	3	Mild dysbiosis / functional deviation
DI3	1	Moderate dysbiosis
DI4	0	Severe dysbiosis
DI5	1	Very severe dysbiosis

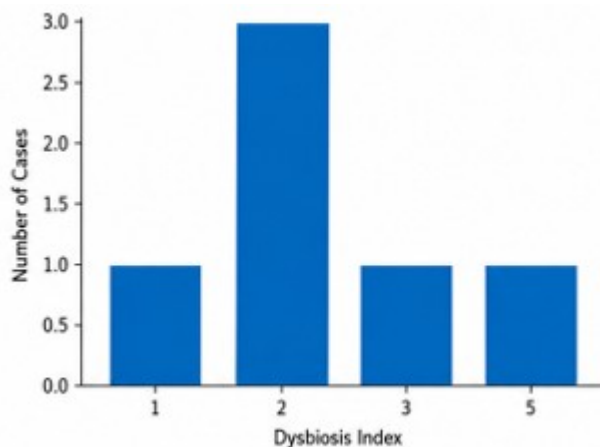


Figure 1. Distribution of dysbiosis severity across the cohort.

DI2 represented the most frequent microbiota profile. Very severe dysbiosis (DI5) was identified in one patient only. Most patients demonstrated progressive ecological dysfunction rather than complete microbial depletion.

3.2. Functional Microbiota Alterations

Progressive dysbiosis severity was associated with increasing ecological destabilization of the intestinal microbiota. The most consistent alteration observed throughout the analyzed profiles involved disruption of cross-feeder bacterial networks. The case-specific functional profiles are presented in Table 2. Increasing Dysbiosis Index severity was associated with progressive impairment of cross-feeding interactions, reduction of SCFA-producing bacteria, reduction of anti-inflammatory bacterial groups, expansion of opportunistic microorganisms, and increased ecological AMR-associated potential [5, 15].

Table 2. Functional alterations according to dysbiosis severity.

Case	DI	Diversity	Cross-Feeders	Anti-inflammatory Bacteria	Opportunists	AMR Potential
Case 1	2	Expected	Mild alteration	Mild alteration	Mild	Mild
Case 2	1	Expected	Minimal alteration	Preserved	Absent	Low
Case 3	2	Slightly lower	Moderate alteration	Mild alteration	Mild	Mild
Case 4	3	Expected	Moderate alteration	Moderate alteration	Moderate	Moderate
Case 5	2	Expected	Moderate alteration	Moderate alteration	Mild	Mild
Case 6	5	Lower than expected	Severe alteration	Severe alteration	Severe	High

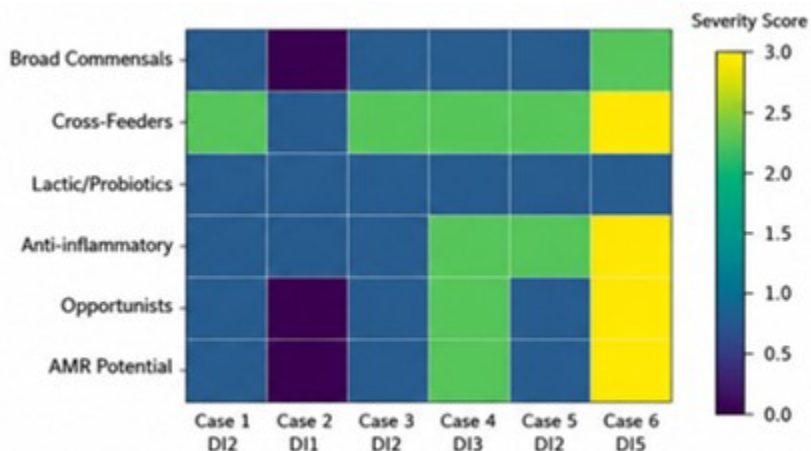


Figure 2. Functional ecological heatmap according to dysbiosis severity.

The degree of ecological dysfunction progressively increased with higher DI levels, particularly in relation to cross-feeder disruption and opportunistic microbial expansion.

The heatmap (Figure 2) demonstrated progressive ecological deterioration from DI1 toward DI5, early disruption of cross-feeder bacterial networks, increasing opportunistic microorganisms with higher DI, and progressive elevation of ecological AMR-associated potential. The visualization suggests that dysbiosis progression represents functional ecological destabilization rather than isolated bacterial alteration.

3.3. Cross-Feeder Bacterial Alterations

Cross-feeder bacteria represented the principal functional finding of the study. Alterations in these metabolic cooperation networks appeared early, even in mild dysbiosis (DI2), suggesting that ecological dysfunction may precede complete microbiota collapse [7, 16]. The main bacterial groups and their proposed metabolic roles are summarized in Table 3. The principal bacterial groups involved included *Ruminococcus bromii*, *Bacteroides/Parabacteroides*, *Eubacterium* spp., *Bifidobacterium*, and *Lactobacillus*.

Table 3. Main cross-feeder bacteria and functional significance.

Bacterial Group	Main Function	Clinical Significance
<i>Ruminococcus bromii</i>	Resistant starch degradation	Promotes fermentation and SCFA production
<i>Bacteroides/Parabacteroides</i>	Complex carbohydrate degradation	Provides substrates for cross-feeding
<i>Eubacterium</i> spp.	Fermentative metabolism	Supports butyrate network
<i>Bifidobacterium/Lactobacillus</i>	Lactate production and barrier support	Probiotic and anti-inflammatory role

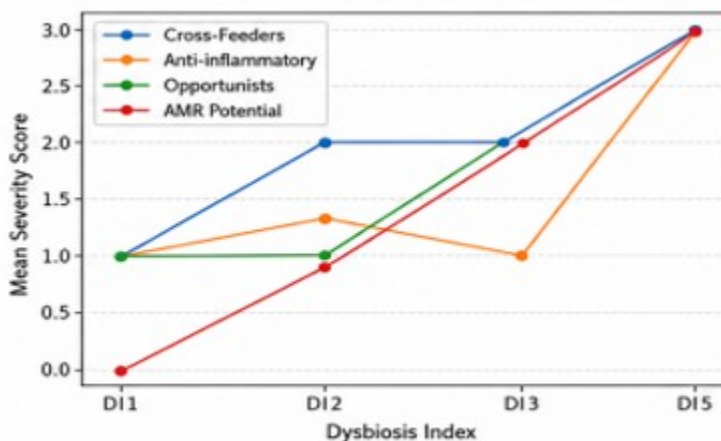


Figure 3. Functional progression of cross-feeder alterations from DI1 to DI5.

The progression model (Figure 3) illustrated early impairment of cross-feeding pathways beginning in DI2, gradual loss of metabolic cooperation, severe ecological dysfunction in DI5, and parallel increase of AMR-associated ecological instability.

3.4. SCFA-Associated Dysfunction and Barrier Integrity

Reduction of SCFA-producing and anti-inflammatory bacterial groups became progressively more pronounced with increasing dysbiosis severity. SCFA-associated bacteria and their physiological functions are presented in Table 4. Patients with advanced dysbiosis demonstrated impaired butyrate-associated metabolism, increased intestinal permeability, chronic inflammatory activation, reduced mucosal barrier integrity, and increased susceptibility to opportunistic colonization [8, 9, 15].

Table 4. SCFA-producing bacteria and functional impact.

Bacteria/Metabolite	Main Role	Effect of Reduction
<i>Faecalibacterium prausnitzii</i>	Butyrate production; anti-inflammatory activity	Increased inflammation and barrier dysfunction
<i>Roseburia</i> spp.	Butyrate production	Reduced energy supply to colonocytes
<i>Akkermansia muciniphila</i>	Mucin turnover and barrier support	Increased intestinal permeability
Butyrate	Main energy source for colonocytes	Leaky gut and chronic inflammation
Propionate/Acetate	Hepatic and systemic metabolism	Reduced metabolic regulation

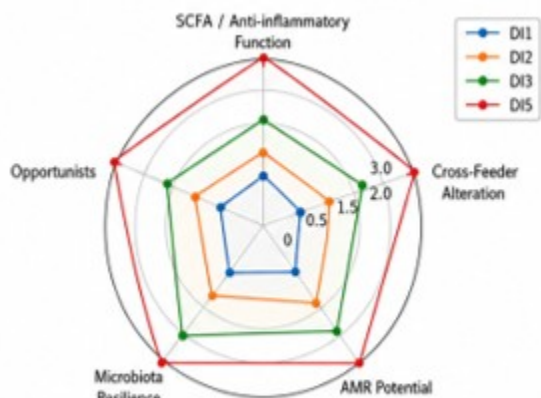


Figure 4. Radar model of functional ecological alterations.

The most severe SCFA-associated functional reduction was observed in the DI5 profile.

The radar chart (Figure 4) demonstrated preserved ecological resilience in DI1, early disruption of cross-feeding and SCFA pathways in DI2, metabolic destabilization in DI3, and severe multi-functional ecological collapse in DI5.

3.5. Opportunistic Microorganisms and Ecological AMR Potential

A progressive increase in opportunistic microorganisms was observed with increasing Dysbiosis Index severity. Higher DI profiles were associated with increased ecological instability, reduced colonization resistance, expansion of opportunistic bacterial groups, and ecological conditions favorable for antimicrobial resistance persistence and dissemination [12, 13]. The opportunistic microbial groups and their potential ecological relevance to AMR are summarized in Table 5.

Table 5. Opportunistic microorganisms and ecological AMR potential.

Opportunistic Group	Ecological Interpretation	Association with AMR
<i>Enterobacter/Citrobacter/Salmonella</i>	Facultative anaerobes favored in dysbiosis	Frequently associated with MDR and ESBL markers
<i>Escherichia/Shigella</i> group	Marker of inflammation and dysbiosis	Potential reservoir of resistance genes
Proteobacteria	Ecological destabilization marker	Increased resistome potential
<i>Bacteroides fragilis</i>	Opportunistic anaerobic species	Possible coexistence with resistance elements

Note: MDR- Multidrug-Resistant; ESBL- extended-spectrum β -lactamase.

Although GA-map® does not directly identify antimicrobial resistance genes, the observed ecological profiles suggest microbiota conditions potentially favorable for resistome expansion and persistence of resistant microorganisms.

4. Discussion

The findings of this pilot case series are consistent with the current concept that intestinal dysbiosis is not only a compositional alteration of gut bacteria, but a functional ecological disorder affecting metabolic

cooperation, barrier integrity, immune regulation, and colonization resistance. In our series, the most consistent alteration was observed in cross-feeder bacteria. This is in line with studies describing cross-feeding as a central mechanism in intestinal microbial metabolism, where primary degraders of complex carbohydrates provide substrates for other bacterial groups involved in SCFA production. Ríos-Covián et al. [15] emphasized that SCFA production depends strongly on cooperative microbial metabolism, particularly acetate-to-butyrate conversion and other interspecies metabolic interactions. Our results support this concept, as cross-feeder alteration appeared early, even in mild dysbiosis, suggesting that metabolic dysfunction may precede complete loss of microbiota diversity [5, 15, 16].

The reduction of SCFA-associated and anti-inflammatory bacteria observed in cases with higher Dysbiosis Index is also comparable with published evidence showing that SCFAs, especially butyrate, play a major role in colonocyte energy metabolism, intestinal barrier maintenance, and regulation of inflammation. Koh et al. [8] have shown that SCFAs act as key mediators between dietary fiber, microbial metabolism, and host physiology. In our data, the progressive alteration of SCFA-related bacterial groups suggests a biological pathway linking dysbiosis with impaired mucosal protection and inflammatory activation. Another important finding was the progressive increase in opportunistic microorganisms in higher DI profiles. This agrees with the concept of microbiota-mediated colonization resistance described by Buffie and Pamer [12] and later studies, where a stable microbiota prevents pathogen expansion through nutrient competition, antimicrobial metabolite production, immune stimulation, and maintenance of an unfavorable environment for pathogens. In our series, higher DI was associated with reduced ecological stability and increased opportunistic markers, supporting the hypothesis that dysbiosis weakens colonization resistance [8, 9, 12].

The relationship between dysbiosis and antimicrobial resistance observed in this study is also supported by literature showing that antibiotic-induced microbiota disruption may favor colonization by multidrug-resistant organisms. Studies on antibiotic effects on the human gut microbiome have demonstrated that broad-spectrum antibiotics reduce microbial diversity, alter SCFA-producing bacteria, and may promote the persistence or expansion of resistant microorganisms. Our findings are consistent with this model: patients with higher DI showed increased opportunists and higher ecological AMR potential. The GA-map® Dysbiosis Test does not directly detect antimicrobial resistance genes. Therefore, the AMR potential described in this study should be interpreted as an ecological risk profile rather than direct genetic confirmation of resistance. This distinction is essential. However, several studies describe the gut microbiota as an important reservoir of resistance genes and mobile genetic elements, which may be transferred between commensals and pathogens under selective pressure. In this context, our results suggest that dysbiosis may create favorable ecological conditions for resistome expansion rather than directly proving the presence of resistance genes [11, 14, 17].

Our findings are also consistent with studies describing antibiotics as major disruptors of gut microbiota resilience. After antibiotic exposure, recovery of the microbiota may be incomplete or delayed, especially when key functional groups such as butyrate producers and cross-feeders are reduced. In our series, DI5 showed the most severe profile, characterized by reduced diversity, altered cross-feeders, reduced anti-inflammatory bacteria, increased opportunists, and higher AMR potential. This pattern reflects loss of microbiota resilience and transition toward a less stable, more inflammatory intestinal ecosystem. Compared with larger studies and reviews, our results show the same biological direction, although on a smaller scale. The main contribution of this case series is the functional interpretation of dysbiosis: rather than focusing only on individual bacterial changes, the analysis highlights the progressive loss of ecological

function through the sequence: cross-feeding disruption → reduced SCFA production → impaired barrier function → increased opportunists → higher AMR ecological potential [10, 18, 19].

Overall, the results support the hypothesis that intestinal dysbiosis may contribute to antimicrobial resistance indirectly by altering the ecological conditions of the gut microbiota. This reinforces the need to consider microbiota status as a complementary element in the interpretation of antibiotic exposure, recurrent infections, and AMR risk [11, 17].

Limitations: The principal limitation of this study is the small sample size. Statistical inference remains limited, and the findings should primarily be interpreted as biologically relevant trends within a pilot observational case series.

5. Conclusions

Intestinal dysbiosis demonstrates a progressive ecological spectrum ranging from minimal functional alteration to severe microbiota destabilization. Cross-feeder bacteria appear to represent one of the most important functional components of intestinal microbial ecology. Disruption of cross-feeding networks contributes to reduced SCFA production, weakening of intestinal barrier integrity, increased inflammation, reduced colonization resistance, and ecological conditions favorable for AMR expansion. Functional microbiota analysis may complement conventional microbiological methods in the interpretation of antimicrobial resistance. Further studies involving larger patient populations are required to better clarify the relationship between dysbiosis, microbial ecological disruption, and antimicrobial resistance [5, 8, 15].

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Conflict of Interest: All authors declare no conflicts of interest.

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