

INVESTIGATION OF SULFONAMIDE AND TETRACYCLINE RESISTANCE GENES IN THE GUT MICROBIOTA OF COLORECTAL CANCER PATIENTS

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ABSTRACT

Antimicrobial resistance (AMR) remains a critical global health challenge, and Vietnam is no exception. The human gut harbors trillions of microorganisms and serves as a reservoir for AMR genes. Alterations in gut microbiota composition have been associated with various diseases, including colorectal cancer (CRC), highlighting the need to investigate whether these changes affect the reservoir of AMR genes. Our study aimed to examine the prevalence and distribution of sulfonamide and tetracycline resistance genes in the gut microbiota of CRC patients using real-time quantitative PCR. A total of 112 fecal samples were collected from the 108 Military Central Hospital, comprising 57 CRC patients and 55 healthy controls. DNA was extracted using the DNeasy PowerSoil Pro Kit and used for the quantification of AMR and 16S rRNA genes. The results revealed a high prevalence of *sul1*, *sul2*, *tetM*, *tetX*, and *tetQ* in the gut microbiota, with an average of more than 10^{11} copies per gram of feces among participants. Notably, gene copy numbers showed considerable inter-individual variation, especially for the *tetX* gene. A significantly higher level of *sul1* was detected in CRC patients compared to the healthy group. In addition to CRC status, several clinical and demographic factors, including gender, BMI, blood pressure, living region, and CRC stage, were found to influence the relative abundance of these genes. Correlation analysis indicated moderate associations between specific bacterial genera and the relative abundance of the studied AMR genes. Overall, our findings highlight the gut microbiota community as a potential source of AMR gene dissemination and emphasize the need for improved monitoring and regulation of antibiotic use in clinical settings, particularly for patients with CRC.

Keywords: Antimicrobial resistance genes, sulfonamides, tetracyclines, colorectal cancer, gut microbiota, qPCR.

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INTRODUCTION

Antimicrobial resistance poses a severe global health threat, with approximately 4.95 million deaths globally caused by bacterial infections in 2019 (Antimicrobial Resistance, 2022). In Vietnam, rampant overuse and improper use of antibiotics have resulted in a significant rise in AMR (Nguyen et al., 2022). The spread of AMR genes can be accelerated through horizontal gene transfer, a process by which bacteria carrying AMR genes pass them to other bacteria via mechanisms such as transformation, transduction, and conjugation. This allows the recipient bacteria to acquire resistance traits and proliferate (Liu et al., 2022). Our human body harbors a vast and diverse community of microorganisms that reside throughout different parts of the body. Among these communities, an estimated 100 trillion microorganisms colonize the gastrointestinal tract, known as gut microbiota, which is primarily composed of 300 to 500 different bacterial species with smaller proportions of fungi and viruses (Savage, 1977; Guarner & Malagelada, 2003). Bacteria within the gut microbiota serve as a critical reservoir for the mobilization of AMR genes from commensal to pathogenic bacteria. These bacteria can survive antimicrobial exposure either through pre-existing resistance mechanisms or by acquiring new mutations. Moreover, long-term consumption or misuse of antibiotics can alter the composition and resilience of the gut microbiota, which leads to the selection pressure. As a result, the composition of the bacterial population shifts, with resistant strains becoming more dominant and the non-pathogenic gut commensal bacteria are reduced (van Schaik, 2015).

Each individual possesses a distinct composition of gut microbiota, initially shaped by maternal microbes transferred from the skin, vaginal canal, and feces, and gradually developing into a mature community within the first two years of life (Claesson et al., 2011). Over a lifetime, various factors such as age, diet, geography, stress, medication use, and disease can trigger alterations in the gut microbial communities (Cresci & Bawden,

2015). Among various diseases, alteration of the gut microbiota linked to CRC has been extensively studied (Ahn et al., 2013; Nhung et al., 2024). This disease remains a major contributor to cancer-related mortality worldwide, with rising incidence particularly in developing countries (Arnold et al., 2017). A study involving 1,605 individuals, including 748 CRC patients, demonstrated that CRC patients exhibit a significantly higher burden of AMR genes and pathogenic bacteria in their gut microbiota compared to healthy subjects. Interestingly, pathobionts carrying *sul2*, such as *Escherichia coli* and *Clostridium symbiosum* were significantly more abundant in CRC patients versus healthy controls (Liu et al., 2025). Furthermore, numerous studies have consistently revealed that infections with AMR bacteria significantly worsen clinical outcomes in cancer patients, resulting in higher morbidity, mortality, and longer hospital stays (Nanayakkara et al., 2021). Hence, a comprehensive understanding of the gut resistome in CRC patients may offer valuable insights into controlling the dissemination of antimicrobial resistance by the gut microbiota and enhancing cancer treatment efficacy, particularly in Vietnamese patients. In this study, we conducted research on the investigation of the prevalence and abundance of sulfonamide (*sul1*, *sul2*) and tetracycline resistance (*tetM*, *tetX*, *tetQ*) genes in the gut microbiota of CRC patients using real-time PCR. These specific genes were chosen based on their established high frequency of detection among AMR genes in clinical and environmental contexts (Bengtsson-Palme et al., 2015; Suzuki et al., 2015).

MATERIALS AND METHODS

Sample collection

Fifty-seven CRC patients and 55 healthy individuals were recruited from the 108 Military Central Hospital in Hanoi, Vietnam. As most CRC patients were over the age of 50, healthy controls were selected from the same age group. Stool samples were self-collected by participants at the hospital. Clinical data were obtained through interviews or extracted from

medical records. Comorbidities were recorded in all participants. All fecal samples were stored at -80 °C until further processing. Ethical approval for the recruitment of participants was granted by the Hanoi Medical University Institutional Review Board (No. 503/GCN-HĐĐĐNCYSH-ĐHYHN, date 10/05/2021).

DNA genomic extraction

DNA was isolated from 250 mg of fecal matter using the DNeasy PowerSoil Pro Kit (QIAGEN, Germany) following the manufacturer's guidelines. DNA quality and concentration were determined by the NanoDrop 2000 spectrophotometer (Thermo Fisher Scientific, USA).

Quantitative PCR analysis

The abundance of sulfonamide (*sul1*, *sul2*) and tetracycline (*tetM*, *tetX*, *tetQ*) resistance genes, and 16S rRNA genes was quantified using FastGene 2x IC Green qPCR Universal Mix on a QuantStudio 5 system. Primer sequences and qPCR conditions were applied as previously described (Tran et al., 2025) and are provided in Table 1. Calibration curves for each gene were generated from purified amplicons to ensure accurate quantification. Copy numbers were calculated for absolute abundance (copies/gram of feces) and normalized to 16S rRNA gene abundance for relative quantification.

Table 1. Primer sets utilized for quantitative PCR

No.	Target gene	Primer name	Primer sequence (5'-3')	Annealing temperature	Melting temperature	Product size (bp)
1	16S rRNA	16S-F	TGTGTAGCGGTGAAATGCG	62 °C	85 °C	140
		16S-R	CATCGTTTACGGCGTGGAC			
2	<i>sul1</i>	Sul1-F	CGCACCGGAAACATCGTGCAC	65 °C	87–88 °C	163
		Sul1-R	TGAAGTTCCGCCGCAAGGCTCG			
3	<i>sul2</i>	Sul2-F	TCCGGTGGAGGCCGGTATCTGG	57.5 °C	90 °C	191
		Sul2-R	CGGGAATGCCATCTGCCTTGAG			
4	<i>tetQ</i>	TetQ-F	AGAATCTGCTGTTTGCCAGTG	63 °C	86–88 °C	167
		TetQ-R	CGGAGTGTCAATGATATTGCA			
5	<i>tetX</i>	tetX-F	AGCCTTACCAATGGGTGTAAA	57 °C	91 °C	278
		tetX-R	TTCTTACCTTGGACATCCCG			
6	<i>tetM</i>	TetM-F	ACAGAAAGCTTATTATATAAC	55 °C	81 °C	171
		TetM-R	TGGCGTGTCTATGATGTTTAC			

Gut microbiota data

The 16S metagenomic data of the gut microbiota from all participants were obtained from previously published studies (Nhun et al., 2023; Nhun et al., 2024). The raw sequencing data were reprocessed using QIIME2 v2024.5 (Bolyen et al., 2019). Taxonomic classification at the genus level was assigned using the SILVA v138 reference database (Quast et al., 2013). Relative abundance was calculated as the proportion of reads assigned to each genus out of the total reads per sample.

Statistical analysis

Data visualization and statistical analyses were performed using R v.4.2.1. Mann-Whitney

U tests were used to compare the relative abundance of AMR genes between two groups, while comparisons of more than two groups were done using the Kruskal-Wallis test. Spearman correlation analysis between gut bacterial genera and the relative abundance of sulfonamide and tetracycline resistance genes was carried out using the *cor.test* function. Spearman's rank correlation coefficients (ρ) were visualized with the *pheatmap* v1.0.13 package.

RESULTS

Participants' characteristics

A total of 112 fecal samples were collected, comprising 57 CRC patients aged 47–76 years

and 55 healthy controls aged 48–79 years. Age distribution was categorized into three groups (≤ 59 , 60–70, and 70–80 years), with most participants falling into the ≤ 59 and 60–70 age groups, accounting for 70.1% of CRC patients and 89.1% of healthy controls. Most participants had a normal BMI, including 45 CRC patients (78.9%) and 46 healthy individuals (83.6%). The gender distribution

was approximately equal in both groups. Participants were recruited from diverse urban and peri-urban communities across northern Vietnam, with nearly 20% residing in Hanoi. Type 2 diabetes (T2D) was more prevalent in the CRC group, with 10 patients affected, while hypertension was observed in both CRC patients and healthy controls, consistent with its common occurrence in the elderly (Table 2).

Table 2. Characteristics of patients with CRC and healthy controls

Characteristic	CRC patients (n = 57)	Healthy controls (n = 55)
<i>Age at diagnosis (years old)</i>		
≤ 59	21 (36.8%)	23 (41.8%)
60–70	19 (33.3%)	26 (47.3%)
70–80	17 (29.8%)	6 (10.9%)
<i>BMI categories</i>		
Normal weight	45 (78.9%)	46 (83.6%)
Overweight	6 (10.5%)	8 (14.5%)
Underweight	6 (10.5%)	1 (1.8%)
<i>Gender</i>		
Female	25 (43.9%)	29 (52.7%)
Male	32 (56.1%)	26 (47.3%)
<i>Living region</i>		
Hanoi	10 (17.5%)	13 (23.6%)
Other cities	47 (82.5%)	42 (76.4%)
<i>Comorbidity</i>		
T2D	10 (17.5%)	1 (1.8%)
Hypertension	16 (28.1%)	9 (16.4%)
<i>Tumor size (cm)</i>		
2–5 cm	35 (61.4%)	
5–10 cm	22 (38.6%)	
<i>Tumor, node and metastasis (TNM) stage</i>		
Stage I	7 (12.3%)	
Stage II	20 (35.1%)	
Stage III	25 (43.9%)	
Stage IV	5 (8.8%)	
<i>Tumor location</i>		
Left colon	29 (50.9%)	
Right colon	13 (22.8%)	
Transverse colon	5 (8.8%)	
Rectum	10 (17.5%)	

Note: Data are presented as number (percentage).

Among CRC patients, tumor sizes ranged from 2 to 10 cm, with a median size of 4.3 cm, and 61.4% of patients had tumors

measuring between 2 and 5 cm. All cancer stages were represented among the CRC patients, with stages III and II being the most

common, accounting for 43.9% and 35.1% of patients, respectively. Tumor locations included the left, right, and transverse colon, as well as the rectum, with the left and right colon being the most frequently affected sites, in 29 (50.9%) and 13 (22.8%) patients, respectively (Table 1)

Relationship between clinical characteristics and sulfonamide and tetracycline resistance genes

The prevalence of sulfonamide and tetracycline resistance genes was investigated in gut microbiota communities from CRC patients and healthy individuals. Specifically, we focused on the detection of five genes *sul1*, *sul2*, *tetQ*, *tetX* and *tetM*, which are frequently observed in clinical and environmental settings. The *sul2* gene was detected in 111 out of 112 samples (99.1%),

absent in only one healthy individual. *Sul1* was found in 106 out of 112 samples (94.6%), and was undetectable in six healthy samples (5.4%). Similarly, *tetQ*, *tetM*, and *tetX* were detected in 108 (96.4%), 108 (96.4%), and 90 (80.4%) samples, respectively. These *tet* genes were absent in several CRC and healthy control samples. Among the five genes, *tetQ* exhibited the highest abundance, with an average of 4.2×10^{11} copies/g. The average gene copy numbers for *sul2*, *tetX*, *tetM*, and *sul1* were 7.6×10^{10} , 3.2×10^{10} , 1.5×10^{10} , and 2.2×10^8 copies/g, respectively. The 16S rRNA gene, used as a reference for total bacterial content, was detected in all samples, with an average of 1.38×10^{12} copies/g. The distribution of these genes varied considerably among subjects, especially for the *tetX* gene, as indicated by its high standard deviation (Fig. 1).

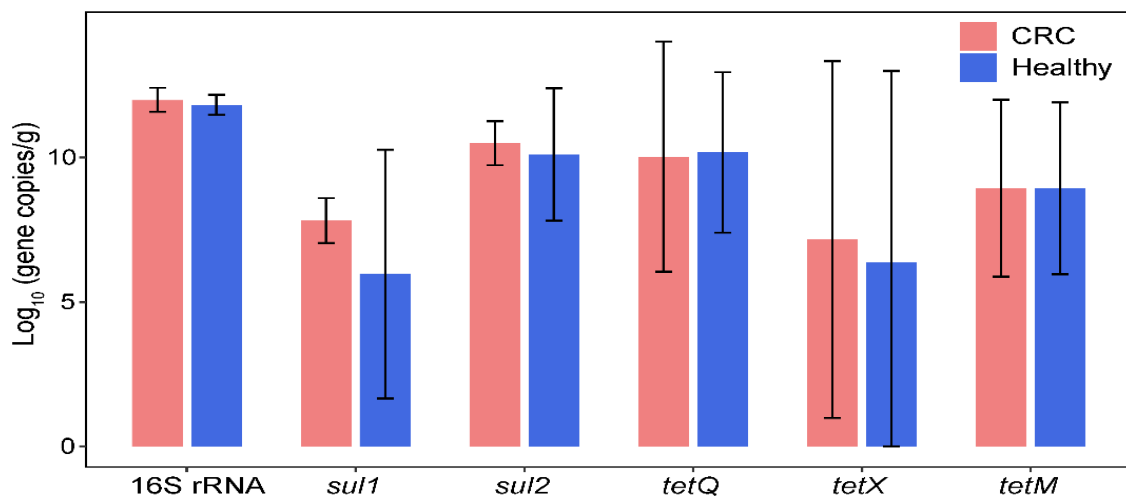


Figure 1. Bar plot showing the mean and standard deviation of the absolute abundance (copies per gram of feces) of 16S rRNA gene, sulfonamide resistance genes (*sul1*, *sul2*), and tetracycline resistance genes (*tetQ*, *tetX*, *tetM*) in colorectal cancer (CRC) patients (pink) and healthy individuals (blue). Abundance values were displayed on a log₁₀ scale

Next, all ARGs were normalized to 16S rRNA gene abundance to account for variations in microbial biomass across samples. The relative abundance of *sul1* was significantly higher in CRC samples compared to healthy individuals (Mann–Whitney U test, $p = 0.043$; Table 3 & Fig. 2A). Additionally, the relative abundance of *tetQ* was significantly elevated in

females compared to males (Mann–Whitney U test, $p = 0.043$; Table 3 & Fig. 2B). Compared to participants from other cities, individuals from Hanoi showed a lower trend in *sul1* abundance (Mann–Whitney U test, $p = 0.078$), while the reduction in *tetQ* abundance was statistically significant (Mann–Whitney U test, $p = 0.009$; Table 2 & Fig. 2C). Furthermore,

tetM abundance was significantly higher in participants with hypertension compared to those with normal blood pressure (Mann–Whitney U test, $p = 0.047$; Table 2 & Fig. 2D). We further investigated the association between ARG abundances and clinical characteristics within the CRC and healthy groups. In the CRC group, the relative abundances of *sul2*, *tetQ*, *tetX* and *tetM* were associated with clinical factors such as hypertension status, and tumor, node and metastasis (TNM) stage. A higher relative

abundance of *tetQ* was observed in CRC stage I (1.9 ± 2.9) compared to later stages, with mean relative abundances of 0.5 ± 1.6 in stage II, 0.5 ± 1.4 in stage III, and 0.3 ± 0.6 in stage IV. A similar pattern was observed for *tetX*, with a higher abundance in stage I (0.2 ± 0.4) compared to stage II (0.02 ± 0.07) and stage IV (0.005 ± 0.01). Additionally, demographic factors including age, BMI, gender, and living region were found to be linked with the abundance of *sul2*, *tetQ*, and *tetM* (Table 3).

Table 2. Comparison of sulfonamide and tetracycline resistance genes across clinical characteristics

Characteristics	<i>sul1</i>	<i>sul2</i>	<i>tetQ</i>	<i>tetX</i>	<i>tetM</i>
All samples (CRC and HC samples)					
CRC condition: CRC vs. Healthy	0.043	0.926	0.498	0.517	0.496
Age category: ≤ 59 , 60–70 vs. 70–80	0.504	0.374	0.819	0.402	0.859
BMI category: Normal weight vs. Overweight	0.582	0.384	0.608	0.972	0.424
Gender: Male vs. Female	0.610	0.681	0.026	0.967	0.425
Living region: Hanoi vs. Other cities	0.078	0.218	0.009	0.817	0.798
T2D condition: T2D vs. Non-T2D	0.969	0.907	0.992	0.291	0.977
Blood pressure: Hypertension vs. Normal	0.101	0.240	0.106	0.787	0.047
CRC samples					
Age category: ≤ 59 , 60–70 vs. 70–80	0.435	0.399	0.916	0.757	0.840
BMI category: Normal weight vs. Overweight	0.205	0.195	0.428	0.803	0.204
Gender: Male vs. Female	0.477	0.325	0.942	0.949	0.962
Living region: Hanoi vs. Other cities	0.569	0.114	0.104	0.563	0.698
T2D condition: T2D vs. Non-T2D	0.844	0.926	0.875	0.412	0.941
Blood pressure: Hypertension vs. Normal	0.465	0.043	0.010	0.160	0.035
Tumor size: 2–5 cm vs. 5–10 cm	0.776	0.183	0.166	0.915	0.755
Tumor, node and metastasis (TNM) stage: 4 stages	0.398	0.121	0.029	0.011	0.258
Tumor location: 4 locations	0.918	0.427	0.354	0.095	0.904
HC samples					
Age category: 50–60, 60–70 vs. 70–80	0.935	0.014	0.195	0.157	0.535
BMI category: Normal weight vs. Overweight	0.712	0.759	0.902	0.703	0.026
Gender: Male vs. Female	0.413	0.477	0.007	0.899	0.136
Living region: Hanoi vs. Other cities	0.098	0.945	0.039	0.819	0.968
Blood pressure: Hypertension vs. Normal	0.275	0.255	0.614	0.166	0.657

Note: Statistical significance was assessed using the Mann-Whitney U test (two groups) and the Kruskal-Wallis test (> 2 groups). Bold text indicated statistical significance ($p < 0.05$).

Correlation analysis between gut bacterial genera and sulfonamide and tetracycline resistance genes

Spearman's correlation analysis was conducted between gut microbiota at the genus level and the relative abundance of

sulfonamide and tetracycline resistance genes. A total of 38 classified genera were found to be moderately correlated with at least one of the studied ARGs, with absolute Spearman's rank correlation coefficients ($|\rho|$) ranging from 0.3 to 0.5. Among the resistance genes, *tetQ* showed the highest number of correlations,

followed by *sul2* and *tetX*. Genera associated with these three genes clustered into two distinct groups. Cluster A, comprising 18 genera, was mainly positively correlated with *sul2*, *tetQ*, and *tetX* (cluster A, Fig. 3). Notably, *Odoribacter*, *Alistipes*, *Anaerotignum*, *Dysosmobacter*, *Phascolarctobacterium*, *Butyrivimonas*, *Phocaea*, *Pseudoflavonifractor*, *Bacteroides*, *Parabacteroides*, and *Flintibacter* were associated with all three genes. In contrast,

Cluster B, consisting of 20 genera, of which 19 genera showed negative correlations with *tetQ*, while 4 and 8 genera were also negatively correlated with *sul2* and *tetX*, respectively (cluster B, Fig. 3). In comparison, *sul1* and *tetM* exhibited fewer moderate correlations. *Sul1* was positively correlated with five genera, including *Diplocloster*, *Anaerotruncus*, *Pseudomonas*, *Streptomyces*, and *Paenibacillus*. *TetM* was negatively correlated with *Odoribacter* and *Alistipes* (Fig. 2).

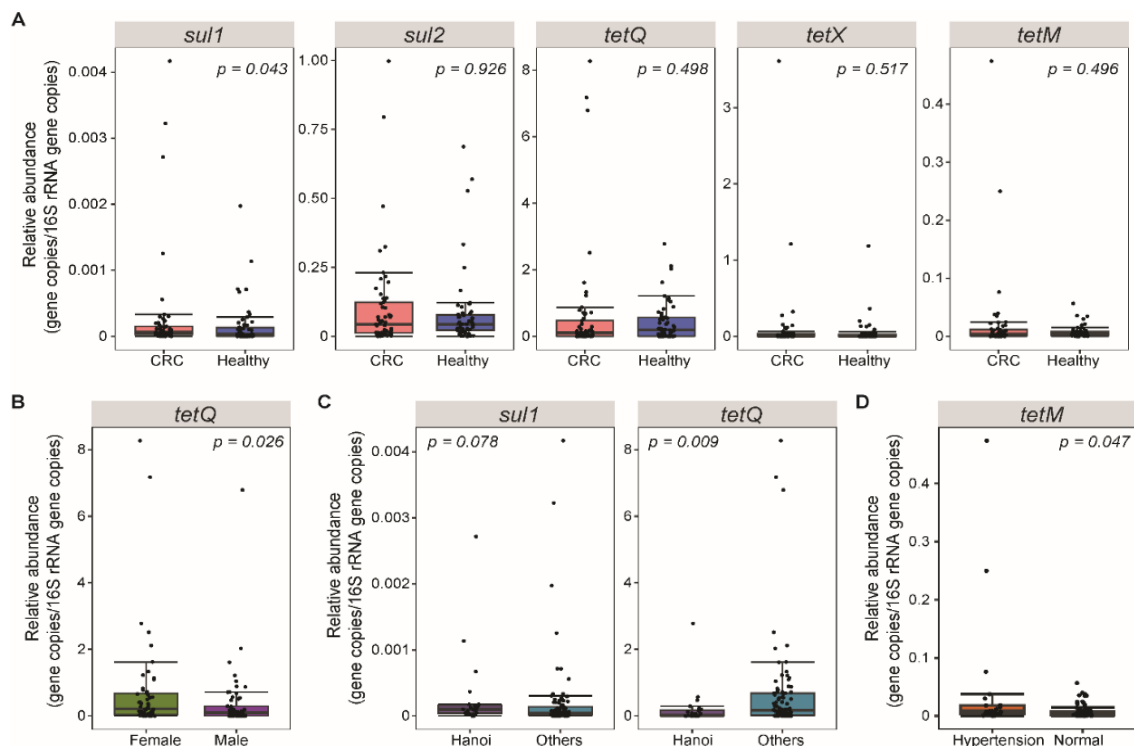


Figure 2. Boxplots illustrate the distribution of the relative abundance of sulfonamide and tetracycline resistance genes, grouped by (A) CRC condition, (B) gender, (C) living region, and (D) blood pressure. In panels (B) - (D), only genes with *p*-values less than 0.1 are displayed. Statistical significance between the two groups was assessed using the Mann–Whitney U test.

The relative abundance of AMR genes was calculated as the ratio of gene copies to 16S rRNA gene copies

DISCUSSION

Our study found a high copy number of sulfonamide and tetracycline resistance genes in gut microbiota in both CRC and healthy populations, revealing that the antibiotic resistant issue is not only restricted to those with

compromised health, but also widely occupied within the community. The abundance of these genes in healthy individuals suggests they may have become a component of the normal microbiota. The copy numbers of these genes predominate with frequent detection, aligning with previous reports on the antibiotic landscape

both globally and in Vietnam (Pavelquesi et al., 2021; Liu et al., 2025). According to a study conducted across 720 fish and shrimp farms in seven provinces of Vietnam, tetracycline and sulfonamide were among the top three most commonly used antibiotics in the country's livestock and aquaculture industries (Luu et al., 2021). In addition to being acquired by humans through direct contact or the food chain,

wastewater generated from these farming activities, containing high levels of antibiotic residues, often flows into nearby water bodies or seeps into the soil. In this way, antibiotics are rapidly introduced into the ecosystem, facilitating the spread of resistant bacteria as well as accelerating the dissemination of resistance genes in human (Uluseker et al., 2021).

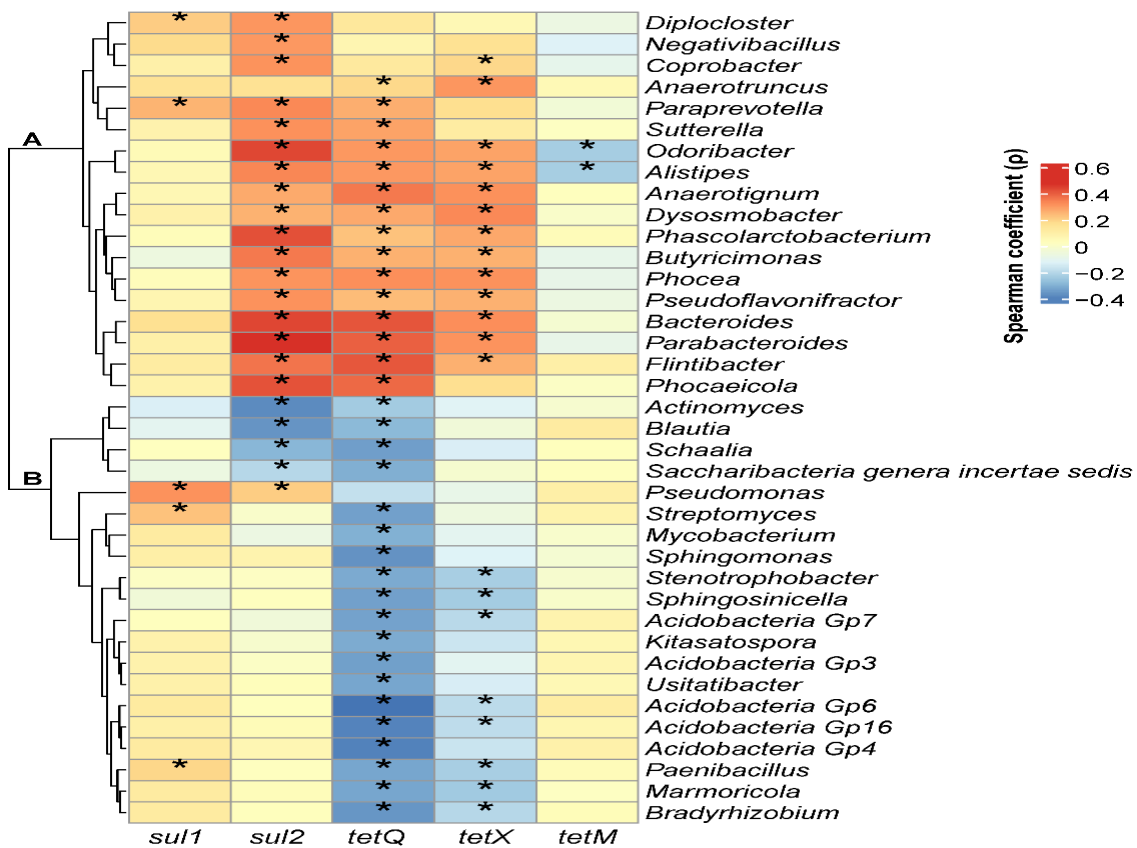


Figure 3. The heatmap shows Spearman correlation analysis between gut bacterial genera and the relative abundance of sulfonamide and tetracycline resistance genes. Hierarchical clustering using Euclidean distance grouped the genera into two distinct clusters. Colors in the heatmap represent Spearman's rank correlation coefficients (ρ), and asterisks indicate correlations with p -values less than 0.05. Only bacterial genera with a $|\rho|$ greater than 0.3 in correlation with any of the studied ARGs are presented

An elevated relative abundance of a sulfonamide resistance gene *sul1* was observed in CRC patients, suggesting selective pressure and persistence of AMR gene-harboring microbes in the CRC-associated gut environment. Although *sul2* was not found to be

significantly enriched in our study, it has been previously reported as enriched in the gut microbiota of CRC patients in a Hong Kong cohort (Liu et al., 2025). A case-control study conducted in the United Kingdom between 1989 and 2012, involving 28,980 CRC patients and

137,077 healthy individuals, reported an association between oral antibiotic use and CRC risk. Penicillins, including ampicillin and amoxicillin, were positively associated with colon cancer, while tetracyclines showed a negative association with rectal cancer (Zhang et al., 2019). In our study, no significant differences were observed in the abundance of tetracycline resistance genes based on CRC status or tumor location. However, a significant elevation of *tetQ* and *tetX* was detected in TNM stage I compared to the later stages. This could be due to alterations in gut microbiota composition across different TNM stages, which may influence the enrichment of tetracycline resistance genes. *TetQ* was also found at higher levels in individuals living in provinces outside of Hanoi. This may be explained by the fact that most agricultural farms are located outside the capital. As a result, environmental exposure to tetracyclines may be consistently higher in other cities compared to Ha Noi (Luu et al., 2021). Mechanistically, AMR gene enrichment may be driven by selective pressures from prior antibiotic use, gut inflammation, and altered microbial metabolism. The significant correlation between AMR burden and CRC highlights the need for further research into microbial-host interactions that may exacerbate tumorigenesis (Saus et al., 2019; Voskarides, 2022).

We observed a moderate correlation between a variety of bacterial genera and the AMR genes examined in our study, providing evidence that alterations in gut microbial composition of CRC patients are linked to differences in their AMR profiles. Specifically, *Parabacteroides* and *Bacteroides* showed a positive correlation with *tetQ* and *tetX*. *Parabacteroides* genus and three *Bacteroides* species (*Bacteroides salyersiae*, *Bacteroides cellulosilyticus*, and *Bacteroides caccae*) were previously reported to be enriched in CRC patients compared to healthy controls (Nhung et al., 2024). *Bacteroides* is known to be a primary reservoir of *tetQ*, one of the most abundant AMR genes in the human gut microbiota (Wu et al., 2021). Similarly, a large-scale metagenomic analysis encompassing 202,265 assembled

genomes from 12,829 human microbiome samples demonstrated that *tetX* genes are widely distributed among Bacteroidaceae colonized in the human gut (Zhang et al., 2021). Unlike *sul1*, *sul2* was positively associated with multiple commensal gut genera such as *Odoribacter*, *Phascolarctobacterium*, *Parabacteroides*, and *Bacteroides*. Given that *sul2* is frequently located on small plasmids (Skold, 2000), this genetic context may facilitate its dissemination across different bacterial genera. In addition, *Pseudomonas* was found to be positively correlated with both *sul1* and *sul2*, and has been identified as a potential carrier of *sul* genes through horizontal gene transfer (Byrne-Bailey et al., 2009). On the other hand, the beneficial genus *Blautia* exhibited a negative association with *sul2* and *tetQ*. *Blautia* species, known for its potential probiotic properties, have also been reported to be sensitive to a broad range of antibiotics, including tetracycline (Liu et al., 2021). Negative associations were also observed between several Acidobacteria genera and tetracycline resistance genes. This finding is in line with a previous report indicating that these genera, particularly Acidobacteria Gp16, are highly susceptible to tetracycline (Huang et al., 2014).

CONCLUSION

Our findings not only provide valuable insights into the prevalence of sulfonamide and tetracycline resistance genes across CRC and healthy individuals, but also underscore the correlation between specific gut genera and the relative abundance of *sul* and *tet* genes. The high prevalence of AMR genes in the gut microbiota, regardless of disease status, raises serious concerns about the potential for widespread dissemination of AMR genes within the ecosystem. Moreover, profiling AMR genes in the gut microbiota of CRC patients holds significant potential for informing more effective treatment strategies and to minimize the spread of resistance during therapy.

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