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Original article

Molecular epidemiological characteristics of monophasic *Salmonella enterica* serovar Typhimurium variants in Korea

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Abstract

Background: Monophasic *Salmonella enterica* serovar Typhimurium variants, particularly the serotype I 4,[5],12:i:-, have globally emerged as important foodborne pathogens associated with multidrug resistance. However, their molecular epidemiology and clonal distribution in South Korea remain unclear. This study investigated the distribution, clonal characteristics, and antimicrobial resistance profiles of *S*. Typhimurium variants circulating in South Korea.

Methods: In total, 173 *S*. Typhimurium variant strains were collected from 18 university hospitals between 2019 and 2020. Isolates were identified and serotyped according to the Kauffmann–White scheme. Gene patterns were determined based on seven phase 1 and 2 flagellar antigen-associated genes. Antimicrobial susceptibility was examined using broth microdilution.

Results: The monophasic variant I 4,[5],12:i:- (n = 131) was the most prevalent, followed by I 4,[5],12:i:1,2 (n = 38) and I 4,[5],12:-:- (n = 4). Gene patterns dominant among monophasic isolates included P5 (n = 72), P8 (n = 29), and P9 (n = 28). The P5 pattern corresponded to the typical Spanish clone. Monophasic variants showed higher resistance rates to ampicillin and third-generation cephalosporins than those of other variants, with higher cephalosporin resistance observed in P8 than in P5.

Conclusion: Among the isolates included in this study, the monophasic variant I 4,[5],12:i:- was more frequently identified than typical *S*. Typhimurium. Most I 4,[5],12:i:- isolates showed gene patterns previously associated with the Spanish clone and high rates of antimicrobial resistance. Overall, these findings highlight the need for continued surveillance of I 4,[5],12:i:- and its antimicrobial resistance patterns.

Keywords: Drug Resistance, Bacterial; Molecular epidemiology; *Salmonella* Typhimurium

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Introduction

Background

Salmonella enterica is one of the leading causes of foodborne bacterial illness worldwide [1], with > 2,500 serotypes identified using the Kauffmann–White scheme [2]. The epidemiology and clinical severity of

salmonellosis vary with serotype, and certain serotypes are associated with specific reservoirs and severe disease in humans [3,4].

Salmonella enterica serovar Typhimurium (antigenic formula: I 4,[5],12:i:1,2) is one of the most prevalent serotypes worldwide. However, human infections caused by its variants, including monophasic variant (I 4,[5],12:i:-), have increased [5-7]. Accurately identifying these variants is important as they may differ in antimicrobial resistance and transmission patterns.

To improve the identification of *S.* Typhimurium variants, the European Food Safety Authority (EFSA) has proposed the molecular approaches targeting the phase 1 and phase 2 flagellar genes by Tennant et al. [8]. Further, Bugarel et al. [9] have proposed a refined polymerase chain reaction (PCR)-based classification system that defined 11 gene patterns (P1–P11).

S. Typhimurium variants, including I 4,[5],12:i:-, are increasing in Asia including Japan and South Korea [5-7]; however, the data on their molecular epidemiology and clonal distribution are limited.

Objectives

This study aimed to investigate the distribution and molecular epidemiological characteristics of *S. enterica* serovar Typhimurium variants in South Korea.

Methods

Study design

This retrospective surveillance study was based on laboratory investigations. The study was undertaken according to the Microbiology Investigation Criteria for Reporting Objectively (MICRO) framework for the reporting and interpretation of clinical microbiology data [10].

Laboratory studies

Bacterial isolates

In total, this study included 173 *S.* Typhimurium variant strains collected from 18 hospitals across South Korea between 2019 and 2020. The participating hospitals were located in Seoul, Incheon, Gyeonggi, Gangwon, Chungcheong, Daegu, Ulsan, Busan, Gwangju, Jeolla, and Jeju. All isolates were recovered from clinical specimens submitted for diagnostic testing and were not obtained through non-diagnostic surveillance or screening. The specimen sources included stool (n = 126), blood (n = 36), and other specimens (n = 11).

At each participating hospital, isolates recovered from clinical specimens were stored in 10% skim milk at −70°C and transported frozen to Inje University Busan Paik Hospital, where all subsequent experiments were performed.

The analysis included only the first isolate of each *Salmonella enterica* serovar Typhimurium variant obtained from each patient during the study period. Duplicate isolates from the same patient were excluded.

Identification and serotyping

All isolates were confirmed as *S. enterica* using the VITEK MS system with database version 3.0 (bioMérieux SA). Serotyping was performed using slide agglutination with somatic (O) and flagellar (H) antisera (BD Difco™, Becton, Dickinson and Company), and antigenic formulas were determined according to the Kauffmann–White scheme [11]. Phase inversion was performed to assess phase 2 H-antigen expression and to distinguish the monophasic variants from biphasic *S*. Typhimurium.

Gene pattern analysis

All isolates were screened for seven molecular markers associated with phase 1 and 2 flagellar antigens, including the *fljAB* operon, and gene patterns were assigned accordingly (Table 1 and 2). P5 and P8 were considered Spanish clone-associated patterns, whereas P7 and P9 were considered U.S. clone-associated patterns [9].

Genomic DNA was extracted using InstaGene Matrix (Bio-Rad Laboratories). PCR amplification was performed using AccuPower® PCR PreMix (BIONEER) according to the manufacturer's instructions. Primers targeting the *fliA–fliB* intergenic region were used at 20 pmol per reaction, whereas all other primers were used at 10 pmol per reaction.

The PCR conditions comprised an initial denaturation step at 95°C for 5 min, and a final extension at 72°C for 7 min. The detailed cycling conditions and primer sequences are provided in Table 1 [8,9,12].

Table 1. Primers and polymerase chain reaction conditions used in this study

Targets	Name	Primer sequence (5′–3′)	Size (bp)	Cycling profile
Intergenic region of *fliA–fliB*	FFLIB	CTG GCG ACG ATC TGT CGA TG	964	(30 cycles)
	RFLIA	GCG GTA TAC AGT GAA TTC AC		94°C, 30 s/54°C, 30 s/72°C, 90 s
fljB	Sense-59	CAA CAA CAA CCT GCA GCG TGT GCG	1389	(30 cycles)
	Antisense-83	GCC ATA TTT CAG CCT CTC GCC CG		94°C, 30 s/62°C, 30 s/72°C, 30 s
fliC	Sense-60	ACT CAG GCT TCC CGT AAC GC	550	(30 cycles)
	Antisense-i	ATA GCC ATC TTT ACC AGT TCC		94°C, 40 s/58°C, 20 s/72°C, 20 s
STM2757	STM2757-F	AAC CGT ACA GGG TTT ATA CGC C	91	(35 cycles)
	STM2757-R	TTA TCG TGC CGC CGA ATT ATG G		95°C, 15 s/60°C, 1 min/72°C, 30 s
mdh	*mdh*-F	TGC CAA CGG AAG TTG AAG TG	260	(30 cycles)
	mdh-R	CGC ATT CCA CCA CGC CCT TC		94°C, 30 s/55°C, 30 s/72°C, 30 s
fljA	*fljA*-F	TCC GAA GCC AGA ATC AAA TTT TCC	105	(35 cycles)
	fljA-R	TAC GTT TTA ATG ATA TCC CTG TTC G		95°C, 15 s/60°C, 1 min/72°C, 30 s
hin	*hin*-F	CGC CCC GGC CTG AAA CGA	334	(35 cycles)
	hin-R	CGA CTA ATC TGT TCC TGT TCA TGT T		95°C, 15 s/60°C, 1 min/72°C, 30 s

Table 2. Antigenic formula and gene pattern of *Salmonella* Typhimurium variants

Gene pattern[a)]	Antigenic formula (n, %)			Total
	I 4,[5],12:-:-	I 4,[5],12:i:-	I 4,[5],12:i:1,2 (Typhimurium)	
P1 (+/+/+/+/+/+/+)	4 (100.0)	2 (1.5)	32 (84.2)	38 (22.0)
P2 (+/+/−/+/+/+/+)	−	−	6 (15.8)	6 (3.5)
P5 (+/+/+/+/−/−/−)	−	72 (55.0)	−	72 (41.6)
P8 (−/+/+/+/−/−/−)	−	29 (22.1)	−	29 (16.8)
P9 (−/+/+/+/−/−/+)	−	28 (21.4)	−	28 (16.2)
Total	4 (100.0)	131 (100.0)	38 (100.0)	173 (100.0)

[a)](STM2757/*mdh*/*fliA*-*fliB*/*fliC*/*fljB*/*fljA*/*hin*); +, positive; −, negative.

Antimicrobial susceptibility testing

Sensititre KRCDC2F MIC plates (Thermo Fisher Scientific) were used for antimicrobial susceptibility testing. The following antimicrobials were tested: amikacin, ampicillin, azithromycin, cefotaxime, cefoxitin, ceftazidime, ceftriaxone, chloramphenicol, ciprofloxacin, colistin, gentamicin, imipenem, nalidixic acid, streptomycin, tetracycline, and trimethoprim–sulfamethoxazole (SXT).

Minimum inhibitory concentrations were determined according to the Clinical and Laboratory Standards Institute (CLSI) M100 ED36:2026 guidelines [13]. CLSI breakpoints were not available for amikacin, cefoxitin, ceftazidime, gentamicin, nalidixic acid, and streptomycin; therefore, susceptibility to these antimicrobials was interpreted based on the National Antimicrobial Resistance Monitoring System criteria [14]. Colistin susceptibility was interpreted according to the European Committee on Antimicrobial Susceptibility Testing (version 16.0) [15].

ACT/S resistance was defined as resistance to ampicillin, chloramphenicol, and SXT. The ACSSuT resistance profile indicated resistance to ampicillin, chloramphenicol, streptomycin, sulfonamides, and tetracycline [16].

Statistical analysis

Statistical analyses were performed to evaluate differences in antimicrobial resistance rates according to the antigenic formula and gene patterns. Categorical variables were expressed as numbers and percentages.

For the antimicrobial resistance analysis presented in Table 3, resistance rates between P5 and P8 isolates were compared using the chi-square test or Fisher's exact test, as appropriate. Statistical analyses were performed using SPSS version 29.0 (IBM Corp.). Statistical significance was defined as a two-tailed P-value < 0.05.

Table 3. Antimicrobial resistance and gene patterns of *Salmonella* Typhimurium variants

Antimicrobial agent	I 4,[5],12:i:1,2 (Typhimurium)			I 4,[5],12:-:-	I 4,[5],12:i:-					
	P1 (n = 32)	P2 (n = 6)	Total (n = 38)	P1 (n = 4)	P1 (n = 2)	P5 (n = 72)	P8 (n = 29)	P9 (n = 28)	Total (n = 131)	*P*-value (P5 vs. P8)
Amikacin[a)]	0	0	0	0	0	0	0	0	0	–
Ampicillin	46.9	50.0	47.4	25.0	0	91.7	79.3	3.6	68.7	0.098
Azithromycin	0	0	0	0	0	2.8	0	0	1.5	1.000
Cefotaxime	3.1	0	2.6	0	0	13.9	41.4	3.6	17.6	0.006
Cefoxitin[a)]	3.1	0	2.6	0	0	4.2	0	0	2.3	0.555
Ceftazidime[a)]	0	0	0	0	0	4.2	37.9	0	10.7	< 0.001
Ceftriaxone	3.1	0	2.6	0	0	11.1	41.4	3.6	16.0	0.002
Chloramphenicol	25.0	50.0	28.9	25.0	0	31.9	41.4	0	26.7	0.489
Ciprofloxacin (Intermediate rates, %)	6.3 (40.6)	16.7 (50.0)	7.9 (42.1)	0 (100)	0 (0)	0 (27.8)	6.9 (31.0)	0 (0)	1.5 (22.1)	0.080
Colistin[b)]	0	0	0	0	0	0	0	7.1	1.5	–
Gentamicin[a)]	28.1	0	23.7	25.0	0	8.3	0	0	4.6	0.178
Imipenem	0	0	0	0	0	0	0	0	0	–
Nalidixic acid[a)]	43.8	0	36.8	100	0	0	3.4	0	0.8	0.287
Streptomycin[a)]	21.9	33.3	23.7	25.0	0	86.1	79.3	3.6	65.6	0.385
Tetracycline	34.4	33.3	34.2	0	0	84.7	75.9	0	63.4	0.389
Trimethoprim–sulfamethoxazole	12.5	50.0	18.4	0	0	27.8	0	0	15.3	< 0.001

Values are presented as percentages of resistant isolates unless otherwise indicated.

[a)]Clinical and Laboratory Standards Institute (CLSI) breakpoints have not been established for these antimicrobial agents; the interpretive standards used are the National Antimicrobial Resistance Monitoring System (NARMS)-established breakpoints for resistance monitoring, which should not be used to predict clinical efficacy.

[b)]European Committee on Antimicrobial Susceptibility Testing, 2026.

Results

Distribution of antigenic formulas and gene patterns

Among 173 *S*. Typhimurium variant isolates, the monophasic variant I 4,[5],12:i:- (n = 131, 75.7%) was the most prevalent, followed by typical *S*. Typhimurium I 4,[5],12:i:1,2 (n = 38, 22.0%), and the non-motile variant I 4,[5],12:-:- (n = 4, 2.3%). The monophasic variant I 4,[5],12:-:1,2 was not detected.

Gene pattern analysis revealed five patterns (P1, P2, P5, P8, and P9) based on the presence or absence of seven target genes (STM2757, *mdh*, intergenic regions of *fliA*–*fliB*, *fliC*, *fljB*, *fljA*, and *hin*) (Table 2). P5 was the most prevalent (n = 72), followed by P1 (n = 38), P8 (n = 29), P9 (n = 28), and P2 (n = 6).

P1 included 32 typical *S*. Typhimurium isolates, four I 4,[5],12:-:- isolates, and two I 4,[5],12:i:- isolates. P2 exclusively consisted of six typical *S*. Typhimurium isolates. P5, P8, and P9 were only detected in I 4,[5],12:i:- isolates. Both P5 and P8 lacked *fljA*, *fljB*, and *hin*, whereas P8 additionally lacked STM2757. P9 was positive for *hin*, but lacked *fljA*, *fljB*, and STM2757.

Among the I 4,[5],12:i:- isolates, 77.1% were classified as Spanish clones (P5, 55.0%; P8, 22.1%), whereas 21.4% corresponded to a U.S. clone (P9).

Antimicrobial resistance profiles

Resistance to ampicillin, streptomycin, and tetracycline was numerically more frequent in P5 and P8 than in the other gene patterns. The resistance rates at P5 and P8 were as follows: ampicillin (91.7% and 79.3%); streptomycin (86.1% and 79.3%); and tetracycline (84.7% and 75.9%), respectively (Table 3).

P8 showed significantly higher resistance to third-generation cephalosporins compared with that of P5 (cefotaxime: 41.4% vs. 13.9%; ceftazidime: 37.9% vs. 4.2%; ceftriaxone: 41.4% vs. 11.1%; $P < 0.05$). Among 72 P5 isolates, 23 (31.9%) were concurrently resistant to ampicillin, chloramphenicol, streptomycin, and tetracycline, corresponding to four of the five components of the characteristic ACSSuT resistance profile of the Spanish clone. Because sulfamethoxazole alone was not tested, the complete ACSSuT phenotype could not be determined. Twenty-three P5 isolates were resistant to four ACSSuT components (ampicillin, chloramphenicol, streptomycin, and tetracycline) and 18 of these were additionally resistant to SXT.

Typical *S*. Typhimurium (P1 and P2) and I 4,[5],12:-:- (P1) strains demonstrated high rates of intermediate resistance to ciprofloxacin (42.1% and 100%, respectively). Further, high rates of nalidixic acid resistance were observed in P1 isolates of typical *S*. Typhimurium (43.8%) and I 4,[5],12:-:- (100%).

In contrast, P9 isolates showed low rates of resistance to most antimicrobials, with resistance to colistin observed in 7.1% of isolates and resistance to other antimicrobials occurring at rates below 4%.

ACT/S-resistant isolates

Overall, 25 isolates (14.5%) were classified as ACT/S-resistant isolates. These included seven typical *S*. Typhimurium isolates (P1, n = 4; P2, n = 3) and 18 I 4,[5],12:i:- isolates (all P5). No ACT/S-resistant isolates were identified among the P9 strains.

Discussion

Among the isolates collected in South Korea during the study period, I 4,[5],12:i:- was more frequently identified compared with typical *S*. Typhimurium. P5, P8, and P9 were the predominant gene patterns among I 4,[5],12:i:- isolates, with P5 and P8 previously associated with the Spanish clone and P9 with the U.S. clone. These gene patterns showed substantially different antimicrobial resistance profiles, with P8 isolates showing particularly high resistance to third-generation cephalosporins. These findings reveal the molecular epidemiological diversity of I 4,[5],12:i:- and the association between gene patterns and antimicrobial resistance profiles.

Salmonella serotypes are determined using a combination of somatic (O) and flagellar (H) antigens. Phase variation in *S*. Typhimurium is regulated by the coordinated expression of two flagellar proteins, FliC (phase 1 antigen) and FljB (phase 2 antigen). Expression switching occurs through inversion of the promoter region, which is mediated by the DNA invertase Hin. This results in *fliC* suppression by *fljA* and the subsequent expression of *fljB* [17, 18]. Gene disruption or deletion within the *fljAB* operon or associated regulatory regions can result in monophasic variants.

Salmonella Typhimurium (I 4,[5],12:i:1,2) expresses both phase 1 and phase 2 H antigens. However, based on differences in H antigen expression, three variants have been described: monophasic variant I 4,[5],12:i:-, the uncommon variant I 4,[5],12:-:1,2, and the non-motile variant I 4,[5],12:-:- [19-21].

The Centers for Disease Control and Prevention and the EFSA report that I 4,[5],12:i:- ranks among the top five most-detected serotypes in human clinical isolates. The I 4,[5],12:i:- variant is increasingly reported in North America, Europe, Asia, Oceania, and South America and is frequently associated with pork, poultry, and cattle products [6, 22-28].

A previous nationwide human surveillance in South Korea (2016–2017) reported that I 4,[5],12:i:- accounted for 16.7% of the isolates, surpassing the typical Typhimurium isolates (9.9%) [5]. In the present study, the ratio of I 4,[5],12:i:- to typical Typhimurium increased from 1.7:1 to 3.4:1, indicating that I 4,[5],12:i:- was more frequently identified than typical Typhimurium during the study period. However, this comparison was limited to these two serotypes and does not indicate predominance among all *Salmonella* serotypes. A recent nationwide surveillance from 2022 to 2024 identified *S*. Enteritidis (29.7%) as the most common serotype, whereas I 4,[5],12:i:- accounted for 10.5% of the isolates [29].

To improve *S.* Typhimurium variant identification, the EFSA has proposed molecular approaches targeting the phase 1 and phase 2 flagellar genes. Tennant et al. [8] analyzed genetic variations in *fljB*, the *fliB–fliA* intergenic region, and the phase 1 “i” antigen gene using PCR-based methods. However, inconsistent findings, including the unexpected absence or presence of certain flagella-associated genes, were occasionally observed. Subsequently, Bugarel et al. [9] proposed a refined PCR-based classification system defining 11 gene patterns (P1–P11) using additional markers, such as *fljA*, *fliC*, *hin*, STM2757, and *mdh*. This approach enabled improved molecular epidemiologic investigations and facilitated the differentiation of epidemic clones, including the Spanish (P5 and P8) and U.S. (P7 and P9) clones of I 4,[5],12:i:-.

In terms of clonal lineages, I 4,[5],12:i:- has been categorized into several epidemic lineages, including Spanish, U.S., and European clones [19, 27, 28, 30]. In the present study, most isolates showed P5 and P8

patterns, which have been associated with the Spanish clone, whereas P9 has been associated with the U.S. clone. However, as the P1–P11 classification is based on a limited set of PCR markers, these patterns do not allow definitive assignment of isolates to specific clonal lineages. In particular, presence or absence of the European clone, characterized by features such as ST34 and the ASSuT resistance phenotype, could not be determined using the methods employed in this study.

The Spanish monophasic clone, first reported in Spain in 1997, was predominantly associated with phage type U302 and a multidrug resistance profile including resistance to ampicillin, chloramphenicol, sulfonamides, gentamicin, streptomycin, tetracycline, and SXT [9, 31]. This clone is distinct from the classical multidrug-resistant *S*. Typhimurium DT104 lineage that emerged in the mid-1980s and is characterized by the ACSSuT resistance profile [32]. In the current study, 23 of 72 P5 isolates (31.9%) were resistant to four of the five ACSSuT components (ampicillin, chloramphenicol, streptomycin, and tetracycline) and 18 of these isolates were additionally resistant to SXT. However, because sulfamethoxazole alone was not tested, the complete ACSSuT phenotype could not be determined. These findings should therefore be interpreted as overlapping resistance characteristics rather than evidence of the classical ACSSuT phenotype. Interestingly, P8 isolates, although genetically related to the Spanish clone, showed markedly higher rates of resistance to third-generation cephalosporins than those of P5 isolates. This may limit the treatment options for invasive infections and pose critical clinical and public health challenges. This finding suggests the possible acquisition of additional resistance determinants, underscoring the need for continuous monitoring.

In contrast, the U.S. clone (P9) exhibits low levels of antimicrobial resistance [7]. Consistent with previous reports, the P9 isolates in this study showed low resistance rates, and no ACT/S-resistant isolates were identified. However, detection of colistin resistance in a subset of P9 isolates warrants further investigation into the genetic mechanisms underlying this phenotype.

Overall, three major gene patterns (P5, P8, and P9), previously associated with the Spanish and U.S. clones, were identified among the I 4,[5],12:i:- isolates in South Korea. Predominance of the Spanish clone suggests potential transmission through livestock reservoirs, particularly pork and poultry, as observed in Europe and North America. This finding raises concerns regarding foodborne transmission and highlights the need for integrated surveillance across the food production chain. Furthermore, significant differences in antimicrobial resistance profiles according to gene patterns highlight the epidemiological importance of molecular subtyping.

Considering the increasing prevalence of I 4,[5],12:i:- and its association with the ACT/S resistance phenotype, continued molecular surveillance is essential. Gene pattern-based subtyping may serve as a practical tool for epidemiological investigations and the development of antimicrobial treatment strategies. Overall, these findings highlight the importance of continuous national surveillance under the One Health framework and support the implementation of genomic surveillance and antimicrobial stewardship programs for mitigating the spread of high-risk clones.

Limitations

This study has a few limitations. First, isolates were collected only from university hospitals, which may

not fully represent the nationwide epidemiology. Second, whole-genome sequencing was not performed to confirm the clonal relationships and resistance determinants.

Conclusion

Monophasic *S.* Typhimurium I 4,[5],12:i:- was more frequently identified than typical *S*. Typhimurium among the isolates included in this study. Most I 4,[5],12:i:- isolates presented gene patterns previously associated with the Spanish clone, whereas a small proportion showed patterns associated with the U.S. clone. Distinct gene patterns were associated with pronounced differences in antimicrobial resistance profiles; particularly, the P8 isolates demonstrated increased resistance to third-generation cephalosporins. These findings highlight the epidemiological and public health relevance of I 4,[5],12:i:- and the importance of continued surveillance. Moreover, they indicate the practicality of gene pattern-based molecular subtyping for epidemiological surveillance and antimicrobial resistance monitoring in resource-limited settings. Overall, this study provides important evidence for public health policies and infection control strategies.

Ethics statement

This study was approved by the Institutional Review Board of Inje University Busan Paik Hospital, with a waiver of informed consent (Approval No. BPIRB NON2024-001).

Conflicts of interest

No potential conflicts of interest relevant to this article were reported.

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Data availability

Data are available upon reasonable request.

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