





Research Article

Molecular Detection of Virulence Factors in *Salmonella* serovars Isolated from Poultry and Human Samples

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Received 16 November 2022; Revised 30 January 2023; Accepted 2 February 2023; Published 2 March 2023

Academic Editor: Sumanta Nandi

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Salmonellosis is a common infectious disease in humans caused by *Salmonella* spp., which in recent years has shown an increase in its incidence, with products of avian origin being a common source of transmission. To present a successful infective cycle, there are molecular mechanisms such as virulence factors that provide characteristics that facilitate survival, colonization, and damage to the host. According to this, the study aims to characterize the virulence factors of *Salmonella* spp. strains isolated from broilers ($n = 39$) and humans ($n = 10$). The presence of 24 virulence genes was evaluated using end-point PCR. All the strains of *Salmonella* spp. isolated from broiler chickens revealed presence of 7/24 (29, 16%) virulence genes (*lpfA*, *csgA*, *sitC*, *sipB*, *sopB*, *sopE*, and *sivH*). Regarding the strains isolated from cases of gastroenteritis in humans, all strains contained (14/24, 58, 33%) virulence genes (*lpfA*, *csgA*, *pagC*, *msgA*, *spiA*, *sitC*, *iroN*, *sipB*, *orgA*, *hilA*, *sopB*, *sifA*, *avrA*, and *sivH*). In summary, the presence of virulence genes in different strains of *Salmonella* isolated from broilers and humans could be described as bacteria with potential pathogenicity due to the type and number of virulence genes detected. These findings are beneficial for the pathogenic monitoring of *Salmonella* in Colombia.

1. Introduction

Salmonellosis is a foodborne disease with the greatest impact worldwide on both humans and animals [1, 2]. This disease is caused by the *Salmonella* in which more than 2,700 serotypes have been reported so far [3]. In humans, the consumption of chicken meat and eggs that were contaminated is considered to develop the disease because they are considered the main reservoir and vehicle of *Salmonella* [1, 4]. Moreover, food contamination could occur in various stages of the food chain such as production, distribution, and sale [5]. The serotype, infective dose, virulence factors, and host immunity will influence the disease's clinical presentation [6].

Salmonellosis in humans is characterized by symptoms such as acute fever, abdominal pain, diarrhea, nausea, and vomiting; however, immunocompromised people and children under 5 years of age and older adults can present severe symptoms [7, 8]. In 2018, the EU member states reported 5146 foodborne outbreaks where 33% correspond to illnesses caused by *Salmonella* [7].

Host-pathogen interactions in bacteria can modulate the expression of some genes to adapt to the environment, influencing their ability to cause illness [9]. Therefore, the virulence genes facilitate the survival, colonization, and damage of the host [10]. Expression of virulence genes will initiate when *Salmonella* spp. faces the hostile environment

of the hosts' gastrointestinal tract compound of a wide variety of conditions such as osmolarity, oxygen tension, and pH which favor interaction with the target cell during pathogenesis [2]. The molecular mechanisms of pathogenicity used by *Salmonella* involve genes, grouped in regions called pathogenicity islands that provide new characteristics that allow it to undergo a successful infective cycle [11]. These genetic segments linked to virulence functions are known as *Salmonella* pathogenicity-island (SPI) and *Salmonella* has 24 identified [12]. In addition, SPI could be transmitted between bacteria by horizontal gene transfer and is related to virulence mechanisms such as host colonization, capsules, toxins, invasiveness, biofilm, fimbriae, flagella, serotype conversion, and secretion systems [12–14].

Overall, bacterial virulence factors are critical elements for systemic infections [11]. As a result, the pathogenicity of *Salmonella* has been associated with the number and type of virulence genes present in the chromosomal SPIs [15]. For example, genes such as *SopB/SigD* and *SopE2* allow a rapid internalization of the bacteria playing an important role in *Salmonella* virulence [16]. Moreover, genes involved in the intracellular survival of *Salmonella* play a significant role in systemic disease in humans [17]. Meanwhile, adherence factors like fimbrial operons mediate the attachment of *Salmonella* serovars to epithelial cell lines [18]. Besides, *Salmonella* virulence plasmid plays a crucial role in enhancing the ability of particular serovars to multiply in tissues outside the intestinal tract [19]. Other genes, such as *cdtB*, code for the CdtB subunit considered as a toxin with a possibly important role in the unusually lengthy, persistent, and development of systemic diseases [20, 21].

Despite being a public health concern, there are insufficient studies on virulence factors in *Salmonella* spp. isolates from broilers and humans in Colombia; also, without specific information, it is difficult to predict the success of *Salmonella* control schemes. Thus, it is important to know the genomic particularities in each of the serotypes belonging to this genus; this allows to clarify the bacterial dynamics in the different animal hosts and prevent outbreaks in humans and animals [11]. Thus, the aim of this study was to evaluate the potential virulence of *Salmonella* isolates from poultry and human by detecting the presence of 24 genes involved in virulence and pathogenicity using the polymerase chain reaction (PCR). Accordingly, the results of this study could lay the foundation for further research on public health security and food safety problems caused by *Salmonella* infections in Colombia.

2. Materials and Methods

2.1. *Salmonella* Strains. In this study, 49 *Salmonella enterica* strains from the Bacterial Strain Collection of the Laboratory of Immunology and Molecular Biology were included, and *Salmonella enteritidis* (ATCC® 13076™) were used as a positive control. The strains were previously serotyped using the Kauffmann–White scheme and correspond to the serotypes, namely, *S. enteritidis* ($n = 4$), *S. typhimurium* ($n = 2$), *S. braenderup* ($n = 1$), *S. newport* ($n = 1$), *S. grupensis* ($n = 1$), and *S. uganda* ($n = 1$) isolated from cases of

gastroenteritis in humans [22] and *S. paratyphi* B ($n = 24$) and *S. heidelberg* ($n = 15$) isolated from poultry farms located in the region of Tolima [23] and Santander [24].

2.2. DNA Extraction. Fresh bacterial colonies were used for Genomic DNA (gDNA) extraction using the Invisorb Spin Universal Kit (Stratec Molecular, Berlin, Germany) following the protocol suggested by the fabricant and were stored at -20°C until further use. Molecular confirmation of *Salmonella* isolates was done by amplification of a fragment of *invA* gene (accession number M90846.1) by endpoint PCR.

2.3. Virulence Genes. The molecular characterization of 24 genes involved in virulence and pathogenicity was conducted using the gDNA of *Salmonella* spp. (Table 1). A single PCR assay was used to detect each one of the 24 virulence genes. Primers and annealing temperature used for PCR are listed in Table 1. The reactions were carried out following the manufacturer's recommendation for the GoTaq® Flexi DNA Taq polymerase (Promega, Madison, WI, United States), $1\ \mu\text{L}$ of DNA, and $1\ \mu\text{L}$ of each primer ($10\ \text{pmol}/\mu\text{L}$). The ProFlex™ 3×32 -well PCR System (Applied Biosystems, Carlsbad, CA, United States) was used to perform the amplification using an initial denaturation for 3 minutes at 95°C , 35 cycles of denaturation for 30 seconds at 95°C , 30 seconds of annealing (Table 1), extension at 72°C , and final extension for 5 minutes at 72°C . The PCR products were detected by electrophoresis in agarose gel using HydraGreen (ACTGene, Piscataway, NJ, United States) as an intercalant agent, and the visualization of the gel was conducted in the gel documentation equipment ENDURO GDS (Labnet International, Edison, NJ, United States).

3. Results

3.1. Confirmation of *Salmonella*. All *Salmonella* strains amplified the expected DNA fragment of the *invA* gene that was used to confirm the *Salmonella* genus (Figure 1).

3.2. Distribution of *Salmonella* Virulence Genes. All the isolates carried the *lpfA*, *csqA*, *invA*, *sivH*, *sopB*, and *sitC* genes ($n = 49/49$). Regarding poultry isolates, the detection rate of the *sopE* gene was 100% ($n = 39/39$), while fimbria-associated genes such as *sefA*, *lpfC*, and *lpfA* were present in 51.3% ($n = 20/39$), 87.2% ($n = 34/39$), and 97.2% ($n = 34/39$). Genes associated with type III secretion systems (TTSS) virulence that function as *orgA*, *prgH*, and *spaN* were found in 71.8% ($n = 37/39$), 74.4% ($n = 36/39$), and 79.5% ($n = 31/39$), respectively, of the poultry isolates that were analyzed (Table 2). The detection rate of an effector protein gene like *avrA* was 97.4% ($n = 38/39$). Gen with regulatory protein function as *hilA* was found in 94.9% ($n = 37/39$). Genes related to survival inside cells functions such as *pagC*, *spiA*, *msgA*, and *tolC* were present in 97.4% ($n = 38/39$), 97.4% ($n = 38/39$), 97.4% ($n = 38/39$), and 92.3% ($n = 36/39$) of the poultry strains that were analyzed. The detection rate of

TABLE 1: Primer sequences for virulence genes in *Salmonella* spp.

Virulence factor	Gene	Primer sequences	Annealing temperature (°C)	Amplicon size (bp)	References
Fimbriae	<i>lpfC</i>	F-GCCCCGCTGAAGCCTGTGTTGC R-AGGTCCGCGCTGTTTGAGGTTGGATA	58	641	
	<i>pefA</i>	F-GCGCCGCTCAGCCGAACCAG R-GCAGCAGAAGCCCAGGAAACAGTG	59	157	
	<i>lpfA</i>	F-CTTTCGCTGCTGAATCTGGT R-CAGTGTTAACAGAAACCAGT	46	250	
	<i>csgA</i>	F-TCCACAATGGGGCGGCGGCG R-CCTGACGCACCATTACGCTG	54	350	
	<i>sefA</i>	F-GATACTGCTGAACGTAGAAGG R-GCGTAAATCAGCATCTGCAGTAGC	54	488	
Plasmid	<i>spvB</i>	F-CTATCAGCCCCGCACGGAGAGCAGTTTTTA R-GGAGGAGGCGGTGGCGGTGGCATCATA	58	717	
	<i>tolC</i>	F-TACCCAGGCGCAAAAAGAGGCTATC R-CCGCGTTATCCAGGTTGTTGC	55	161	
Survival inside cells	<i>pagC</i>	F-CGCCTTTTCCGTGGGGTATGC R-GAAGCCGTTTATTTTTGTAGAGGAGATGTT	55	454	
	<i>msgA</i>	F-GCCAGGCGCACGCGAAATCATCC R-GCGACCAGCCACATATCAGCCTCTTCAAAC	57	189	
	<i>spiA</i>	F-CCAGGGTTCGTTAGTGTATTGCGTGAGATG R-CGCGTAACAAAGAACCCTAGTGATGGATT	56	550	
Toxins	<i>cdtB</i>	F-ACAACGTGTCGCATCTCGCCCCGTCATT R-CAATTTGCGTGGGTTCTGTAGGTGCGAGT	57	268	[25]
	<i>sitC</i>	F-CAGTATATGCTCAACGCGATGTGGGTCTCC R-CGGGGCGAAAATAAAGGCTGTGATGAAC	58	768	
Iron metabolism	<i>iroN</i>	F-ACTGGCACGGCTCGCTGTGCTCTAT R-CGCTTTACCGCCGTTCTGCCACTGC	58	1205	
	<i>prgH</i>	F-GCCCGAGCAGCCTGAGAAGTTAGAAA R-TGAAATGAGCGCCCCTTGAGCCAGTC	57	756	
	<i>spaN</i>	F-AAAAGCCGTGGAATCCGTTAGTGAAGT R-CAGCGCTGGGGATTACCGTTTTG	55	504	
Structure, the invasion-associated type III secretion system	<i>sipB</i>	F-GGACGCCGCCGGGAAAAACTCTC R-ACACTCCCCTCGCCGCCTTCACAA	58	875	
	<i>invA</i>	F-GTGAATTATCGCCACGTTCCGGGCAA R-TCATCGCACCGTCAAAGGAACC	55	284	
	<i>orgA</i>	F-TTTTTGGCAATGCATCAGGGAACA R-GGCGAAAAGCGGGACGGTATT	55	255	
Regulatory protein, the invasion-associated type III secretion system	<i>hilA</i>	F-CTGCCGCAGTGTTAAGGATA R-CTGTCGCCTTAATCGCATGT	50	497	
Effector protein, the invasion-associated type III secretion system	<i>sopB</i>	F-CGGACCGGCCAGCAACAAAACAAGAAGAAG R-TAGTGATGCCCGTTATGCGTGAGTGTATT	57	220	
	<i>sifA</i>	F-TTTGCCGAACCGCCCCACACAG R-GTTGCCTTTTCTTGCGCTTCCACCCATCT	58	449	
	<i>avrA</i>	F-AGCCTGGCGCTCGCCAAAAA R-GCGGTCTGCTTTATCGGACGGG	57	123	
	<i>sopE</i>	F-GAGGGCCGGGCAGTGTGAC R-CTTACGGGTCTGGCTGGCG	55	121	[26]
	<i>sivH</i>	F-AGCGCGCTGAATGCGGTGAT R-TCTTGTGCGCCACAGCAGG	55	121	

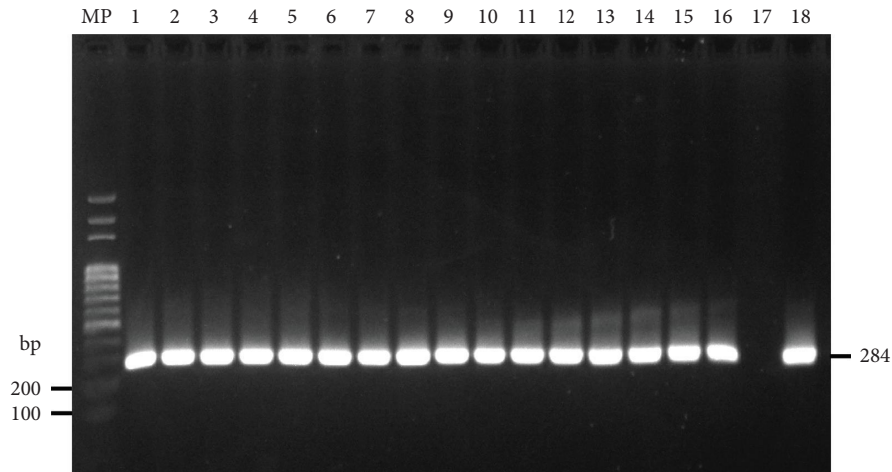


FIGURE 1: Gel image of the PCR amplification of a DNA fragment from the *invA* gene (284 bp) of representative *Salmonella* strains isolated from broiler farms. MP-100 bp DNA ladder (Solis BioDyne, Estonia); 1–4: *S. heidelberg*; 5–9: *S. paratyphi* B; 10: *S. newport*; 11–12: *S. enteritidis*; 13: *S. braenderup*; 14: *S. uganda*; 15: *S. grupensis*; 16: *S. typhimurium*; 17: negative control *E. coli* ATCC 25922; 18: positive control *S. enteritidis* ATCC 13076.

pSLT-mediated virulence genes such as *spvB* was 71.8% ($n = 37/39$). The *iroN* gene was present in 97.4% ($n = 38/39$) of the poultry isolates that were analyzed.

On the other hand, all the isolates from cases of gastroenteritis in humans carried *spiA*, *pagC*, *hilA*, *avrA*, *msgA*, *orgA*, and *iroN* genes (Table 2). The detection rates of *sefA*, *lpfC*, and *lpfA* were 90% ($n = 9/10$), 80% ($n = 8/10$), and 60% ($n = 6/10$), respectively, whilst those for *prgH* and *spaN* genes were 90% ($n = 9/10$) and 80% ($n = 8/10$), respectively. The *sopE* gene was present in 70% ($n = 7/10$) of the human isolates, respectively. The detection rates of *tolC* and *spvB* genes were 60% ($n = 6/10$) and 60% ($n = 6/10$), respectively.

3.3. Virulence Gene Patterns in *Salmonella* Isolates. Detection of the twenty-four virulence genes by PCR classified 49 selected *Salmonella* isolates into 21 patterns (Table 2). The virulence gene patterns of cases of gastroenteritis in humans were Enteritidis (III), Braenderup (XXII), Newport (XX), Grupensis (XXV), Uganda (XXIII), and Typhimurium (XXIV).

The pattern III was the most predominant that was detected in 10 isolates (Heidelberg 3, Paratyphi B 4, Enteritidis 2). In poultry farm isolates, pattern II with 21 virulence genes was detected in 8 isolates (Heidelberg 4, and Paratyphi B 4). The pattern I was detected in 6 isolates (Heidelberg 3, Paratyphi B 3). Patterns X, XI, XII, XIII, XIV, XV, XVII, XVIII, and XIX were detected only in serotypes of Paratyphi B. Also, patterns II, IV, VII, and IX were only observed in Heidelberg isolates. Patterns I, III, V, and VIII were observed in poultry isolates.

4. Discussion

Salmonella species are ubiquitous pathogens that are considered the major agents of foodborne disease worldwide [27, 28]. In *Salmonella* spp., physiological and environmental stimuli drive the expression of virulence genes, which

are responsible for the main pathogenic mechanisms in this microorganism [10, 29]. Virulence factors can maximize the fitness of pathogens via host exploitation [30]. Virulence factors are encoded by a number of genes and may be located on *Salmonella* pathogenicity islands (SPI), virulence plasmids (pSLT), bacteriophages, or at another location on the chromosome [25, 27, 31].

A few virulence factors are related with the cellular structure of the bacteria, such as fimbriae [32]. Fimbrial virulence genes represent a major player in pathogenesis by allowing bacteria to interact with host cells [33, 34]. In the present study, all the isolates carried the *lpfA* and *csgA* genes. Similarly, previous studies have found high detection of the *csgA* gene among *Salmonella* serotypes [25, 35]. The *csgA* gene is related to biofilm production and the maintenance of the bacteria in the environment, including inert surfaces [36]. Likewise, the presence of the *csgA* gene is relevant to public health because the *csg* genes in *Salmonella* are related to the ability to produce biofilms, leading to increased drug resistance [37]. In this way, the presence of the *csgA* gene in all the strains could suggest that the *Salmonella* strains could be kept on inert surfaces such as those used in food production, which is relevant to public health. In poultry isolates, the detection rate of fimbria-associated genes such as *sefA* and *pefA* were 51.3% and 74.4%, respectively, lower than the other virulence genes evaluated. Additionally, the detection rate of *pefA* was 60% in isolates from cases of gastroenteritis in humans. The presence of the *sefA* gene in *Salmonella* isolates is relevant because this gene is a promoter of the *sef* operon, and this operon is a mechanism by which *Salmonella* serotypes can adapt to an increasing number of hosts [25, 38]. Previous studies of virulence gene detection in *Salmonella* Heidelberg isolated from chicken carcasses did not report isolates with the *sefA* gene [25]. For this reason, it is possible that the presence of the *sefA* gene in *Salmonella* Heidelberg isolates could indicate a major virulence of the strains. Furthermore, *sefA* gene has been associated with the serotypes Enteritidis, Moscow, but the

TABLE 2: Patterns of virulence genes of *Salmonella* isolates obtained from poultry farms and cases of gastroenteritis in humans.

Serotype	Presence of virulence gene																				Pattern				
	Fimbriae ^a					Type Three Secretion System gene function ^b							Survival inside cells ^c				d		e	f					
						Structure ^{b1}				Effector protein ^{b2}			b3												
	<i>sefA</i>	<i>lpfC</i>	<i>lpfA</i>	<i>pefA</i>	<i>csgA</i>	<i>sipB</i>	<i>orgA</i>	<i>prgH</i>	<i>spaN</i>	<i>avrA</i>	<i>sopB</i>	<i>sopE</i>	<i>sivH</i>	<i>hilA</i>	<i>pagC</i>	<i>spiA</i>	<i>msgA</i>	<i>toIC</i>	<i>sifA</i>	<i>spvB</i>		<i>iroN</i>	<i>sitC</i>	<i>cdtB</i>	
Heidelberg																								I	
Heidelberg																									I
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Paratyphi B																									XVIII
Paratyphi B																									XIII
Paratyphi B																									XIII
Paratyphi B																									III
Paratyphi B																									XIX
Newport																									XX
Enteritidis																									XXI
Enteritidis																									III
Enteritidis																									III
Braenderup																									XXII
Uganda																									XXIII
Enteritidis																									III
Typhimurium																									XXIV
Grupensis																									XXV
Typhimurium																									XXIV

*For PCR-based patterns, black area represents a positive result and white area represents negative result for the presence of a virulence gene. Virulence-related function is a Fimbriae gene function; b is type three secretion system gene function, b1 structure, b2 is effector protein, b3 is regulatory protein; c is survival inside cells gene function; d is plasmid gene function, e is iron metabolism gene function, and f is toxins gene function.

horizontal transfer methods allowed other serotypes to obtain different genes than that in the case of this study serotypes such as Paratyphi B and Braenderup, and Typhimurium carried the gene [39]. The absence of the *pefA* gene in some *Salmonella* serotypes is related to the location of the gene, which is plasmidial [40].

The type III secretion system (TTSS) encoded by *Salmonella* mediates, in a contact-dependent manner, the translocation of effector proteins from the bacterial cytoplasm into the host cell [41]. Some genes of TTSS are related to structure, effector protein, or regulatory protein of these systems [42]. The *sipB*, *invA*, *orgA*, *prgH*, and *spaN* genes are

associated to the structure of TTSS, which allows *Salmonella* to invade phagocytic and nonphagocytic cells [40, 43]. The *sipB* and *invA* genes were found in 100% of the isolates that were assessed ($n = 49/49$). The *sipB* gene may play a vital role in *Salmonella* pathogenesis [44]. In the case where detection rates of the *invA* gene were expected, this gene is recognized as a rapid detection agent for the genus *Salmonella*, and this gene also indicates that all the strains are able to produce gastroenteritis and invade the cells [45, 46]. A high prevalence of *orgA*, *prgH*, and *spaN* genes in poultry and human isolates were observed in this study (71–100%). In the same way, previous research has detected *sipB*, *orgA*, *prgH*, and *spaN* genes in *Salmonella* isolates from poultry-related sources [47].

Furthermore, TTSS is employed by *Salmonella* to inject different “effector proteins” into host cells [48]. Each effector protein activates or blocks a specific host cell signaling pathway to establish symbioses or infectious diseases [49]. Some genes that encode the effector proteins are *avrA*, *sopE*, *sopB*, and *sivH* [50]. All the isolates carried the *sivH*, *sopB*, and *sopE* genes whilst the human isolates analyzed were 70%. The *SopB* gene can regulate changes in phosphatidylinositol signaling that could generate chloride secretion by epithelial cells [51]. Thus, the significance of the presence of the *sopB* gene is because of the fact that strains with this gene can cause diarrhea, and this disease leads to the elimination of large numbers of bacteria in the host’s environment [52]. Consequently, the possession of this gene could increase the spread of *Salmonella*. High frequency of *sivH* and *sopE* may be explained by the fact that these genes are associated with an island which is unique to *Salmonella* infecting warm-blooded vertebrates [53, 54]. The detection rate of an effector protein gene like *avrA* gene was 97.4% in poultry and 100% in human isolates. *AvrA* protein plays a critical role in inhibiting inflammation, regulating epithelial apoptosis, and enhancing proliferation during bacterial infections [55–58]. On the other hand, gene with regulatory protein functioning as *hilA* was found in 94.9% in poultry isolates. All the isolates from cases of gastroenteritis in humans carried the *hilA* gene.

Some virulence genes may contribute to survival within the macrophage or intracellular survival, for example, *pagC*, *spiA*, *msgA*, and *tolC* genes [59]. The *pagC* gene is ubiquitously distributed among *Salmonella* serotypes [60]. As a result, prevalence found in *pagC* gene was 97.4% in poultry and 100% in human isolates. The detection rate of the *spiA*, *msgA*, and *tolC* genes was higher than 92.3% in all the poultry isolates that were analyzed. On the other hand, all the isolates from cases of gastroenteritis in humans carried *pagC*, *spiA*, and *msgA* genes. The high frequency of the *spiA* gene in poultry and human samples is considered critical due to the function of the gene that is related to the ability of the *Salmonella* serotypes to produce biofilms [25]. Biofilm is an important public health problem; it enhances resistance to physical forces, the host immune system, and antimicrobials [61, 62]. In this way, *Salmonella* strains with the *spiA* gene would survive longer in poultry farms and could contaminate meat and eggs, where contaminated food is a vehicle in the transmission of *Salmonella* to humans. The detection rate of *tolC* in human isolates was 60%. The *tolC*

gene plays a crucial role in the excretion of a wide range of molecules, including antibiotics [63].

The detection rate of pSLT-mediated virulence genes such as *spvB* was 71.8% in poultry and 60% in human isolates, and the frequencies may be explained by the *spvB* gene that is located on virulence plasmids [64]. However, the *spvB* gene that is present in these isolates is relevant because *spv* genes are highly associated with strains that cause nontyphoid bacteremia and disseminated infection in humans [17, 65]. In addition, genes related with iron metabolism such as *iroN* gene that are related to iron acquisition were 97.4% in poultry and 100% in human isolates that were analyzed [66]. Also, the *sitC* gene is another gene that is related to iron metabolism, and this gene encodes an important transporter of iron [67], and all the isolates carried the gene. Previously, the presence of the *spaN* gene was reported, but the *iroN* gene was not associated to bacteria isolated from poultry sources [68]. In the case of *S. Heidelberg*, the fifteen strains carried the two genes. The significance of *iroN* gene cluster that is present on the *Salmonella* isolates is because of the fact that the iron gene that is present represents an adaptation to life at inflamed mucosal surfaces [69]. On the other hand, Webber et al. reported that 88.9% (*iroN*; 112/126) and 79.4% (*sitC*; 100/126) of the *Salmonella Heidelberg* carried the gene [25]. Nevertheless, the presence of the virulence genes does not indicate that the bacteria is pathogenic, it necessarily combined the expression of multiple genes [70]. Finally, we suggest performing other methodologies to confirm the expression of genes or proteins related to virulence factors for a better characterization of each *Salmonella* strains.

5. Conclusions

An analysis of the virulence genes of *Salmonella enterica* was conducted to assess its pathogenic potential. In summary, this study provided a better insight into the epidemiology and pathogenicity of *Salmonella* serovars circulating in two Colombia regions. Also, the presence of virulence genes in different strains of *Salmonella* isolated from broilers and humans could describe it as bacteria with potential pathogenicity due to the type and number of virulence genes detected. In this way, we recommend active surveillance to have updated information on the pathogenicity of *Salmonella enterica* strains circulating and preventing outbreaks of *Salmonella* infection.

Data Availability

The data were obtained from the study. Also, all the datasets generated or analyzed during this study are included in this manuscript.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors’ Contributions

M.P.H.-S. and I.S.R.-B. conceptualized the study; methodology was given by M.P.H.-S., S.C.-M., M.A B.-M., and

K.J.L.-V.; formal analysis was done by M.P.H.-S. and K.J.L.-V.; M.P.H.-S. and K.J.L.-V. wrote the original draft; M.P.H.-S., K.J.L.-V., and I.S.R.-B. reviewed and edited the manuscript; M.P.H.-S., K.J.L.-V., and I.S.R.-B. supervised the study; funding acquisition was done by M.P.H.-S. and I.S.R.-B. All authors have read and agreed to the published version of the manuscript.

Acknowledgments

The authors specially thank the Tolima poultry producers, who have allowed sampling in their farms and identified problems to improve the processes within the companies and productions to provide a better poultry product. This research was funded by the Research Office of the University of Tolima (project number 60130521) and Colciencias (grant number 907-2021).

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