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Virulence Profiles and Antimicrobial Resistance of *Streptococcus agalactiae* Infective and Colonizing Strains from Argentina

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Abstract

Streptococcus agalactiae (GBS) is a colonizing agent in pregnant women, the main cause of invasive neonatal infections, and the reason of serious diseases in non-pregnant adults. Several virulence determinants are involved in the pathogenesis. These include capsular polysaccharide, surface-localized proteins, and toxins. Penicillin is considered the first choice antibiotic for the treatment and prophylaxis; erythromycin, clindamycin and fluoroquinolones are recommended alternatives for penicillin-allergic GBS carriers or patients. Our objective was to investigate the virulence genetic characteristics and the antimicrobial susceptibility of 162 GBS colonizing and infective isolates recovered in Argentina. Serotypes Ia and III were the most prevalent ones, followed by Ib, II, V, IV and non-typeable. In relation to the 13 virulence genes screened, *cpsA*, *cylE*, *hylB, lmb*, and *scpB* were the most prevalent and could be postulated as vaccine epitopes; *bca*, *rib*, *bac*, *hvgA*, *spb1*, PI, PI-2a, and PI-2b were detected in lesser frequencies. No significant association was found between serotypes or virulence genes and colonizing or infective isolates but, on the contrary, significant association was observed between some genes and the most prevalent serotypes, la and III. The cluster analysis showed 52 virulence profiles and, antimicrobial resistance tests, 16 profiles, some with up to 4 resistances. Tetracycline resistance was significantly associated with colonizing isolates. Genes *tetM* and *ermB* conferring resistance to tetracyclines and macrolides, respectively, were the most commonly identified. Our findings show that GBS colonizing and infective isolates circulating in Argentina share similar features in terms of serotype and virulence genes and show a high level of antimicrobial resistance.

Introduction

Streptococcus agalactiae, or Group B *Streptococcus* (GBS), is the leading cause of neonatal sepsis and meningitis and an important cause of infections in pregnant and nonpregnant adults, particularly among the elderly and those with underlying comorbidities [1, 2]. On the other hand, GBS is part of the normal gastrointestinal or genitourinary flora of healthy adults.

Asymptomatic colonization of pregnant women is the leading source of neonatal GBS infection, and has also been associated with an increased risk of prematurity and stillbirth. The vaginal colonization rate in pregnant women is not equal between different geographical areas. The estimated maternal GBS colonization worldwide average is 18%, with a range between 11 and 35% [3]. In Argentina, although information regarding national GBS colonization rate is scarce, regional data show that between 1.4 and 18% of pregnant women are colonized by GBS [4]. In order to prevent this route of transmission, universal screening for recto- vaginal GBS colonization is recommended for pregnant women between 35 and 37 weeks of gestation and carriers receive intrapartum antibiotic prophylaxis (IAP). In Argentina, the search for GBS in all pregnant women is mandatory since 2008, according to the National Law N° 26369. IAP has reduced the incidence of early onset neonatal disease without a notable impact on the incidence of late-onset neonatal disease [5].

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Beta-lactams remain appropriate for first line treatment and prophylaxis as GBS is still sensitive to penicillin but isolates with reduced susceptibility have been reported [6, 7]. Erythromycin, clindamycin and fluoroquinolones are recommended alternatives for penicillin-allergic GBS carriers or patients or when therapeutic failure is suspected. Increased frequency of resistance to these non-beta-lactam antibiotics has been observed, therefore continued monitoring of antimicrobial resistance (AMR) is essential [8].

GBS has a variety of virulence factors that facilitate its ability to cause disease. Several virulence determinants are involved in the adhesion and invasion of host cells, as well as in evasion from the immune system [9]. These include capsular polysaccharide (CPS), regulatory proteins, surface-localized proteins, and toxins. The CPS is an antigenic determinant and a major virulence factor as it interferes with complement mediated killing [10]. GBS can be classified into ten serotypes based on CPS types (Ia–Ib, II–IX) [11]. CPS is presented in combination with different surface proteins including α-C and ß-C, Rib, Lmb, C5a peptidase, HylB, and β-haemolysin, among others [9]. Vaccines targeting capsule polysaccharides and common proteins are under development. The frequency of the genes that encode them varies by origin of the strains, geographic location as well as serotypes [12].

Our hypothesis is that GBS is differentiated into subpopulations according to whether they are infective or colonizing strains. This study aimed to analyse the virulence profiles and antimicrobial resistance of *Streptococcus agalactiae* isolates from the Argentina Pampa region.

Materials and Methods

Isolates Collection

Between 2010 and 2020, we received 189 *S. agalactiae* isolates obtained and identified using standard biochemical criteria and sent from health centers (three hospitals and three biochemical laboratories) of the Argentinean Pampa region of Argentina. The isolates identified by routine diagnostics as GBS were classified as infective or colonizing strains. Sources of the infective isolates were symptomatic non-pregnant people. This group included mostly adults of different ages (with the exception of three infants), both females and males, with symptomatic disease. We received infective isolates recovered from blood, soft tissue, urinary and vaginal infections. Sources of the colonizing isolates were pregnant women tested between 35 and 37 weeks of pregnancy. The isolates were recovered from vaginal swabs (according to the Argentinean regulation 26369/2008). Of 189 isolates received, 162 could be confirmed by amplifying a region of the monocopy regulatory gene *dltR*, specific to *S. agalactiae* [13].

Serotyping and Detection of Virulence-Associated Genes

The capsular type identification, Ia, Ib, II–IX, was determined by PCR according to Imperi et al. [14]. A total of thirteen virulence genes associated with adhesion and colonization, invasion, tissue damage and/or immune evasion were amplificated by PCR. The genes encoding virulence factors analysed were: *bca* and *bac* (alpha and beta subunits of protein C), *lmb* (laminin-binding protein), *rib* and *spb1* (surface proteins), *cpsA* (capsule component, survival of the pathogen in the host), *scpB* (peptidase C5a), *cylE* (pore-forming toxin β-haemolysin), *hylB* (enzyme, degradation hyaluronic acid) [15–18] and PI1, PI2a and PI2b (pilus structures, colonization and invasion of host tissues and formation of biofilms) [19]. Also, a 210 bp genetic region encoding the S10 domain of the HvgA surface protein, from the gbs2018 allele, described as specific for ST-17 was amplified [13]. The primers used to amplify DNA regions specific to virulence genes in GBS isolates are listed in Suppl. Mat. Table 1. The DNA template for PCR assays was obtained by boiling frozen bacteria suspended in sterile water for 10 min. The PCR products were visualized in 2% agarose gel stained by ethidium bromide.

Antimicrobial Susceptibility

Antimicrobial susceptibility to clindamycin (CLI), erythromycin (ERY), levofloxacin (LEV), penicillin (PEN) and tetracycline (TET), was tested in 93 isolates by disc diffusion method according to Clinical and Laboratory Standards Institute [20]. The interpretation of results for NOR was performed following the recommendations of the Clinical and Laboratory Standards Institute [21] (Suppl. Mat. Table 2). A bacterial suspension in sterile saline solution from an overnight pure culture, adjusted to a turbidity of 0.5 on the McFarland scale, was inoculated on a Muller-Hinton agar (Britania) plate, supplemented with 5% ovine blood. Antibiotic discs (Britania) were placed on the agar surface and plates were incubated overnight (16–18 h) at 37 °C in an atmosphere with 5% CO_2. Antimicrobial resistance (AMR) was defined as non-susceptibility to a given antimicrobial by combining intermediate (or susceptible with increased exposure) and resistant categories into a single category. On the other hand, AMR genes were investigated. The macrolide resistance gene *ermB*, was amplified by PCR according to Zhou et al. [22], tetracycline resistance genes *tetM and tetO,* according to Lopardo et al. [23], lincosamide resistance

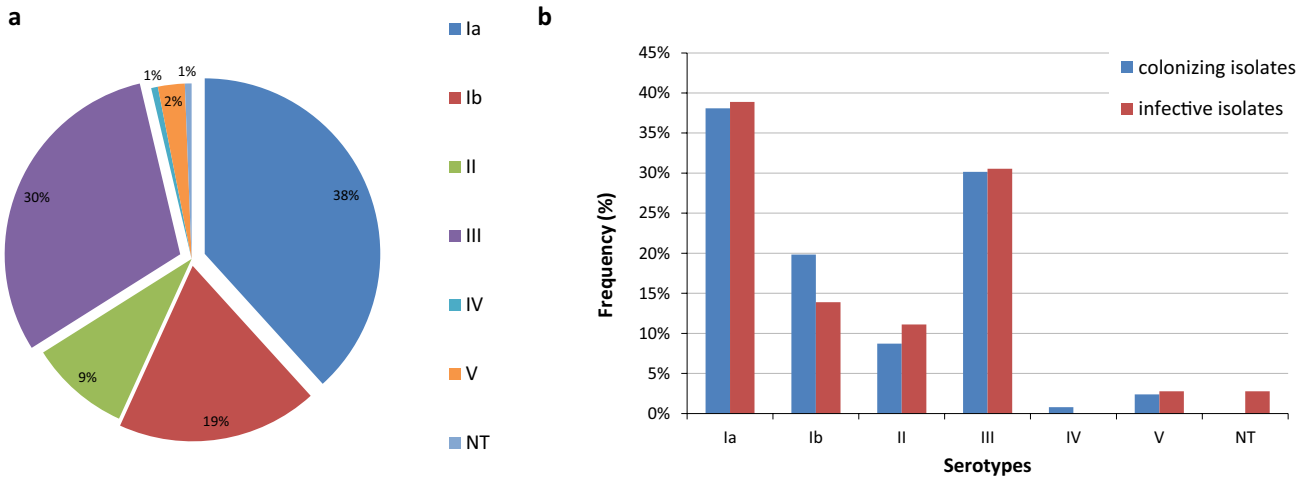

Fig. 1 Distribution of serotypes among GBS Argentinean isolates, general (**a**) and by source (**b**). No significant association was found between serotypes and colonizing or infective isolates ($p > 0.05$). *NT* non-typeable

gene *linB*, according to Bozdogan et al. [24] (Suppl. Mat. Table 1).

Data Analysis

The statistical associations between serotypes, virulence genes, antimicrobial resistance and isolates source, and between serotypes and virulence genes were analysed by 2×2 contingency tables, chi-square test (χ^2), and Fisher exact test, with a confidence level of 95%, using the software Epi Info™ 7.1.5.2.

Taking into account the combinations of the genes detected in the present study, virulence profiles were defined. A cluster analysis was carried out using the UPGMA clustering method. The dendrogram was generated using the BioNumerics v.6.6 software.

Results

Molecular Identification, Virulence and Serotypes of *S. agalactiae*

Among the 162 isolates, the percentages of colonizing and infective isolates were 78% and 22%, respectively. Six serotypes and non-typeable isolates were detected. Serotypes Ia and III were the most prevalent ones (38% and 30%, respectively) followed by Ib, II, and V (Fig. 1a). Also, among colonizing isolates a serotype IV isolate was detected, meanwhile among infective isolates, an isolate was non-typeable. No significant association was found between the serotypes and colonizing or infective GBS isolates ($p > 0.05$) (Fig. 1b).

In relation with virulence-associated genes screened, *cpsA, cylE, hylB* (100%), *lmb* (98%), and *scpB* (91%) were the most prevalent, meanwhile, the other ones were detected in lesser frequencies, PI-2a (73%), *bca* (66%), PI-1 (50%), *rib* (32%), *bac* (23%), PI-2b (14%) and *spb1* (12%). The genes *bac*, *rib* and PI-2a predominated in colonizing isolates over infective ones, and on the contrary, *spb*1, PI-1 and PI-2b predominated in infective isolates. However, no significant association was found between virulence genes and colonizing or infective strains ($p > 0.05$) (Table 1).

The genes *rib* and PI-2b were detected only in serotypes III, Ia, and II, meanwhile *spb1*, in III and Ia isolates (Fig. 2). The *hvgA* gene (reported as specific for the ST-17) was detected in 33 isolates (20%). The majority (82%) of the *hvgA*-positive strains were from colonizing samples. Surprisingly, in addition to amplifying in serotype III isolates (73%), it was detected in some Ia and Ib colonizing isolates. On the other hand, *hvgA*-positive infective isolates belonged only to serotype III.

Significant association was observed between the presence of virulence genes and the most prevalent serotypes, Ia and III. The *bca* and PI-2a genes were significantly associated with serotype Ia (OR 2.54, $p < 0.05$; OR 3.61, $p < 0.05$, respectively) compared to isolates of serotype III; and, *rib*, *spb1*, PI-1 and PI-2b were significantly associated with serotype III, compared to serotype Ia (OR 56, $p < 0.05$; OR 15.93, $p < 0.05$; OR 3.75, $p < 0.05$; OR 19, $p < 0.05$, respectively). The *bac* gene was not significantly associated with either serotype.

The cluster analysis taking into account the virulence genes showed 52 profiles, being 31 of them unique and the remaining 21 shared by 2 to 24 isolates. Each profile comprised 4 to 12 genes. The principal profile was *bca-lmB-hylB-cylE-scpB-PI-2a-cpsA* ($n = 24$, 15%), followed

Table 1 Distribution of virulence-associated genes in GBS and association analysis between virulence genes and source

Virulence genes	No. (%) of isolates			Association between virulence gene presence and source*	
	Total ($N=162$)	Colonizing isolates ($N=126$)	Infective isolates ($N=36$)	OR (95% CI)	p
bac	37 (23)	31 (25)	6 (17)	1.63	>0.05
bca	107 (66)	83 (66)	24 (67)	0.96	>0.05
cpsA	162 (100)	126 (100)	36 (100)	Undefined	–
cylE	162 (100)	126 (100)	36 (100)	Undefined	–
hvgA	33 (20)	27 (21)	6 (17)	1.36	>0.05
hylB	162 (100)	126 (100)	36 (100)	Undefined	–
lmb	159 (98)	125 (99)	34 (94)	7.35	>0.05
rib	52 (32)	42 (33)	10 (28)	1.3	>0.05
scpB	148 (91)	116 (92)	32 (89)	1.45	>0.05
spb1	19 (12)	13 (10)	6 (17)	0.57	>0.05
PI-1	81 (50)	60 (48)	21 (58)	0.64	>0.05
PI-2a	119 (73)	95 (75)	24 (67)	0.25	>0.05
PI-2b	23 (14)	16 (13)	7 (19)	0.6	>0.05

*No significant association was found between virulence genes and colonizing or infective strains ($p>0.05$)

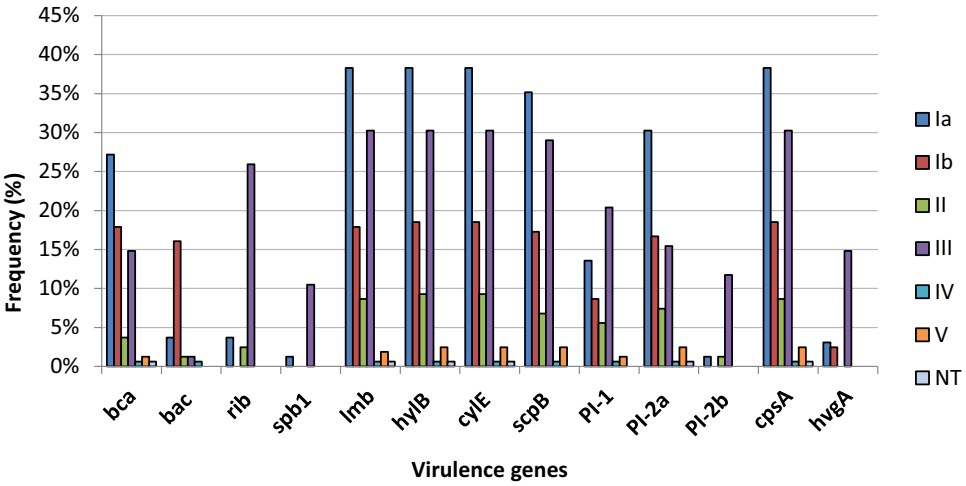

Fig. 2 Distribution of virulence-associated genes in Argentinean GBS by serotype. The genes *rib* and PI-2b were detected only in serotypes III, Ia, and II, meanwhile *spb1*, in III and Ia isolates

by *bca-bac-lmb-hylB-cylE-scpB-PI2a-cpsA/bca-bac-lmb-hylB-cylE-scpB-PI-1-PI-2a-cpsA* ($n=13$, 8% each one), *lmb-hylB-cylE-scpB-PI2a-cpsA* ($n=12$, 7%), and *bca-lmb-hylB-cylE-scpB-PI-1-PI-2a-cpsA* ($n=11$, 7%). No one of these mentioned profiles could be associated with a particular serotype (Fig. 3).

Antimicrobial Susceptibility

Regarding antimicrobial resistance (AMR), 93 isolates could be tested using a disc diffusion method. All analysed isolates were susceptible to penicillin. The resistance rates measured for clindamycin, erythromycin, norfloxacin, levofloxacin, and tetracycline were 14, 22, 41, 13, and 76% of the isolates, respectively (Table 2). No significant association was found between penicillin, clindamycin, erythromycin, norfloxacin, and levofloxacin resistances and colonizing or infective strains. On the contrary, tetracycline resistance was significantly associated with colonizing isolates (OR 4.5, $p<0.05$). Norfloxacin resistance was present in all the serotypes, except in NT. Serotype Ia isolates presented mostly resistance to TET (30/36) and NOR (13/36); serotype III isolates, to TET (26/32), ERY (9/32) and CLI (7/32), and serotype Ib, to LEV (8/19), NOR (12/19), TET (11/19), ERY (3/19) and CLI (1/19). All isolates resistant to levofloxacin were resistant, also, to norfloxacin ($N=12$).

Results showed 16 AMR profiles, some of them with resistance to up to 4 antibiotics and only four isolates were susceptible to 100% of the tested antibiotics. Among colonizing isolates, the profile TET-resistance (44%), followed by the profile NOR-TET-resistance (18%) predominated. Among the infective isolates, TET-resistance and LEV-NOR-resistance profiles (19% each one) predominated, followed by NOR-TET-resistance, CLY-ERY-TET- resistance, and NOR-resistance (13% each one) (Suppl. Mat. Table 3).

Detection of Antimicrobial Resistance Genes

In order to investigate genetic resistance mechanisms, all isolates ($N=162$) were screened by PCR for some genes accounting for resistance to several antibiotics. In relation to erythromycin resistance, the *ermB* gene was detected in 27% of the total isolates (43/162) and in 80 and 77%, of ERY and CLI-resistant isolates (according to phenotypic analysis), respectively. The gene *linB* was not detected in clindamycin-resistant isolates. In relation to tetracycline resistance, the ribosomal protection genes *tetM* and *tetO* were detected in 51% (83/162) and in 9% (15/162) of the total isolates, respectively, and in the 96% and 13% among TET-resistant isolates (according to phenotypic analysis), respectively.

Discussion

GBS colonizes the lower genital tract of approximately 18% of women globally as an asymptomatic member, but established in other host niches, however, GBS is highly pathogenic [3]. Also, this pathogen is able to colonize mammary glands and cause bovine mastitis [25]. The present study characterizes circulating GBS isolates that predominate among colonized pregnant mothers as well as infective cases in a region of Argentina.

GBS is encased by a capsular polysaccharide, based on which ten serotypes are distinguished. Among the Argentinean GBS isolates studied, six serotypes (Ia, III, Ib, II, IV, and V) were detected. Serotypes Ia and III together comprised almost 70% of the total number of isolates. Only one colonizing isolate showed serotype IV. Our results are somewhat similar to a recent study carried out on strains from pregnant women of Misiones province but not in terms of the frequencies of each one of them. The authors detected

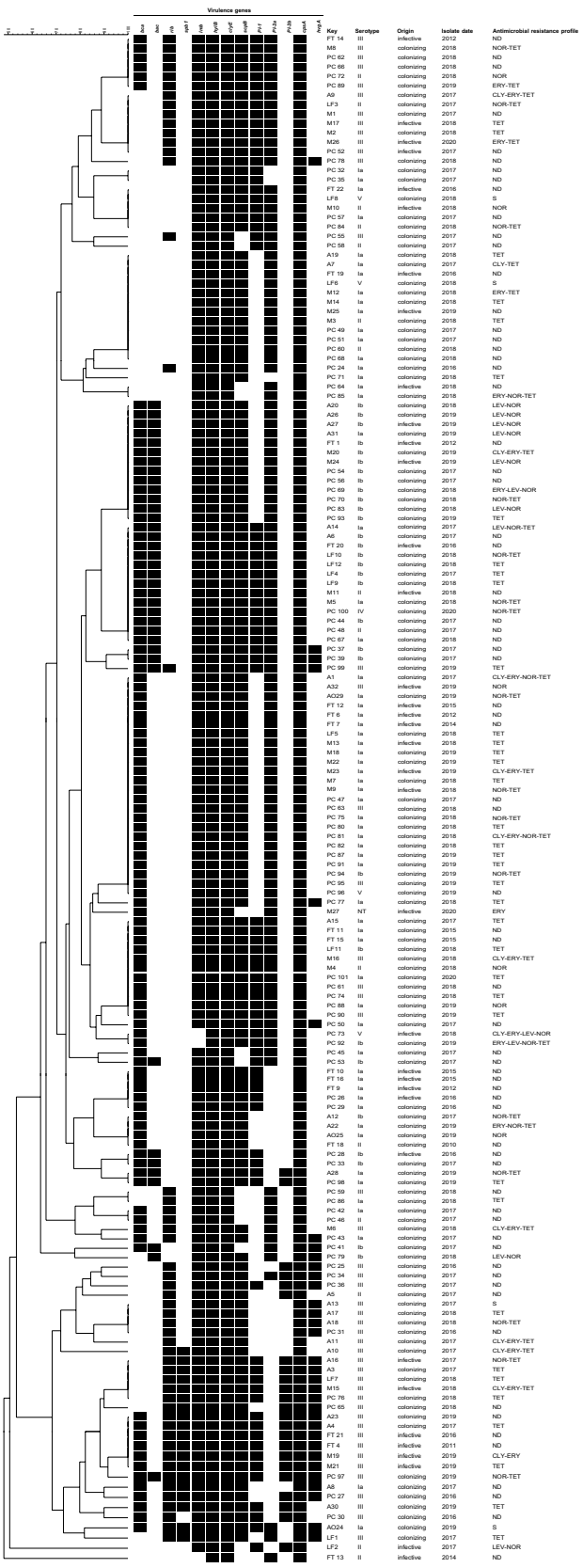

Fig. 3 Cluster analysis of GBS isolates from Argentina based on virulence-associated genes profiles. The presence (black) or absence (white) of genes, the isolate name, serotype, source, and isolation date of the isolates are shown. The antimicrobial resistance profiles are indicated on the right. The dendrogram was carried out by the UPGMA clustering method, and was generated using the BioNumerics v.6.6 software. *NT* non-typeable, *CLI* clindamycin, *ERY* erythromycin, *LEV* levofloxacin, *ND* no data, *PEN* penicillin, *S* susceptible to all tested antimicrobial agents, *TET* tetracycline

Table 2 Distribution of antimicrobial resistance in GBS and association analysis between antimicrobial resistance and source

Antimicrobial agent	No. (%) of isolates			Association between antimicrobial resistance and source*	
	Resistant isolates ($N=93$)	Colonizing isolates ($N=77$)	Infective isolates ($N=16$)	OR (95% CI)	p
Beta-lactams—Penicillin	0 (0)	0 (0)	0 (0)	–	–
Lincosamides—Clindamycin	13 (14)	9 (12)	4 (25)	0.39	> 0.05
Macrolides—Erythromycin	20 (22)	14 (18)	6 (38)	0.37	> 0.05
Quinolones—Norfloxacin	38 (41)	30 (39)	8 (50)	0.63	> 0.05
Quinolones—Levofloxacin	12 (13)	8 (10)	4 (25)	0.34	> 0.05
Tetracyclines—Tetracycline	71 (76)	63 (82)	8 (50)	4.5	< 0.05

*No significant association was found between penicillin, clindamycin, erythromycin, norfloxacin, and levofloxacin resistance and colonizing or infective strains ($p > 0.05$). Tetracycline resistance was significantly associated with colonizing isolates (OR 4.5, $p < 0.05$)

serotype Ia (33%) as the most frequent one followed by III (19%), Ib (15%), II (14%), V (7%), and IX [4]. A previous study, also in strains from Misiones pregnant women, showed a serotype distribution of Ia (40%), III, V, II, Ib, and IX [26]. On the other hand, in Argentinean multi-center studies, serotypes Ia, III and Ib (85% of the total) were the most frequently recorded serotypes among GBS strains from urinary infections [27], meanwhile Ia and III followed by serotypes II and IV were recorded among strains from invasive diseases [28]. A recently published study [29], with emphasis on Argentinean invasive strains collected during 1 year (2014–2015), reported results concordant with ours in relation in that serotypes Ia and III were the most frequent ones in colonizing and invasive isolates recovered from neonates; in invasive isolates from adults, on the other hand, serotypes Ib and Ia prevailed.

A characteristic of GBS strains is that most of the genes associated with the virulence encode proteins necessary for bacteria-host-cell interaction in the process of pathogenicity [30]. In relation to the virulence genes screened in this study, all of them were detected in some percentage. All isolates possessed *cpsA*, *hylB,* and *cylE*, at least one variant or a combination of the two pili island and nearly all isolates possessed *lmb*, *scpB*. These would suggest that these factors are crucial for colonization in humans.

The GBS hyaluronate lyase (HylB) degrades hyaluronic acid, the main component of human connective tissue, facilitating bacterial spread and immunity evasion [31]. This gene and *cylE* were detected in 100% of the strains, both infective and colonizing ones. Haemolytic activity in GBS is produced by the gene products of the *cyl* operon, and *cylE,* which encodes an N-acyl transferase, is necessary for pigment production [32]. This β-haemolysin is, also, a pore-forming toxin, involved in tissue damage, balance the pro- and anti-inflammatory responses of the infected host and the systemic spread of bacteria [33, 34].

The major strategy that GBS employs to colonize the lower genital tract is adherence to epithelial cells via surface-associated adhesins. A common ability conferred by these adhesins is GBS binding to components of the extracellular matrix. The laminin-binding protein (Lmb) mediates the union to laminin, a major component of the basement membrane in human tissues [35]. The peptidase C5a, encoded by the gene *scpB,* interferes with the recruitment of leukocytes at infection sites and binds to fibronectin to promote bacterial invasion of epithelial cells [36].

GBS encodes pili encoded in islands-1 and -2, (PI-1 and PI-2, respectively), with PI-2 comprising 2 variants [37]. The most common pilus here detected was PI-2a, agreeing with results from other groups [38].

The hypervirulent GBS adhesin (HvgA) is a critical virulence trait of neonatal GBS-associated-disease and serotype III has been found to exhibit specific neurotropism through expression of it [39] Gene *hvgA* was mostly present but not restricted to serotype III. It was also detected in Ia and Ib serotypes meanwhile McGee et al. [40] detected it in serotype IV isolates.

No significant association was found between virulence genes and colonizing or infective strains but, on the other hand, significant association was observed between some virulence genes and the most prevalent serotypes, la and III. The *bca* and PI-2a genes were significantly associated with serotype Ia (compared to isolates of serotype III) meanwhile *rib*, *spb1*, PI-1, and PI-2b were significantly associated with serotype III. The gene *bca* encodes the α-C surface protein,

which plays an important role in virulence when the bacterium joins the eukaryotic cell [41].

In order to have a global view of antimicrobial susceptibility occurrence, some isolates were tested against six antimicrobial agents. In Argentina, according to our results and previous information [27, 29, 42], GBS continues to be susceptible to penicillin. However, an important level of RAM for other antibiotics, in both colonizing and infective strains, was detected. Macrolide and lincosamide antibiotics are often used as an alternative in penicillin-allergic patients, and the emergence of resistance among GBS is an increasing problem in many parts of the world [8]. Recent reports on ERY and CLI susceptibility among isolates from infections in Argentina informed resistance rates between 17 and 24% and between 16 and 18%, respectively [29, 42]. In relation to resistance detected in colonizing isolates from Misiones, a recent publication revealed ERY and CLI-resistance rates of 6% and 5%, respectively [43]. Our results taking into account the total number of strains show similar average resistance rates (22 and 14%, respectively); considering only colonizing ones, our data are similar (18 and 12%, respectively) to those obtained from other geographical areas in South America [17, 44].

Resistance against macrolides and lincosamides among GBS may be occurring through two mechanisms, target site modification encoded by *erm* gene and through an active efflux pump [45]. In this study the *ermB* gene was detected in 80% of ERY-resistant isolates and, 77% of CLY-resistant isolates, explaining, at least partially, the resistance by a ribosome methylase which alter the binding of the antibiotic target site. The detection of *linB* (among other resistance determinants) in clindamycin-resistant isolates would explain the resistance observed to lincosamides, not explained by macrolides, however the *linB* gene was not detected in this study.

In relation to fluoroquinolones, an important therapeutic alternative, Arias et al. [29, 46] detected a LEV-resistance rate of approximately 15% in invasive GBS isolates. The authors discussed that the resistance percentage detected was higher than the latest reports from Argentina and the rest of the world, except Korea, China and Japan. In this study, the general percentage of resistance found was similar (13%).

No significant association was found between penicillin, clindamycin, erythromycin, norfloxacin, and levofloxacin resistances and colonizing or infective strains. On the contrary, tetracycline resistance was significantly associated with colonizing isolates. Resistance to tetracycline could be attributed, at least partially, to *tetM* and, *tetO*, genes encoded ribosome protection. High rates of tetracycline have been found previously in Argentinean infective GBS isolates [23], and rates even higher than those found by us have been detected for other geographical areas [47]. It is known that tetracycline resistance in GBS is ubiquitously high, and has been proposed that the acquisition of resistance genes *tetO* and *tetM* by a subset of GBS clones has led to their selection and expansion [48]. According to our data, those tetracycline resistant clones would have spread mainly among the colonizing strains.

GBS leads a double life as both an asymptomatic colonizer and a potent pathogen. Although most people who are colonized with GBS do not experience invasive disease, invasion of GBS into host niches outside of the gastrointestinal and/or vaginal mucosa causes severe damage to the host, resulting in severe outcomes, especially in new-borns [49]. Our findings show that both colonizing and infective isolates share similar features in terms of capsular serotype and virulence genes, suggesting that there are no a GBS subpopulation with a particular propensity to cause disease in adults. This research contributes to epidemiological surveillance in public health and provides data of interest about GBS strains circulating in Argentina. Data on serotypes and virulence genes, particularly those encoding surface proteins, can be useful to evaluate the effectiveness in the region of a GBS vaccine; data on antimicrobial resistance are necessary to evaluate which would be the best prophylactic/therapeutic option.

Conclusions

This is the first time that the virulence profiles of GBS colonizing and infective Argentinean strains have been investigated and compared. Genes *cpsA, cylE, hylB, lmb*, and *scpB* were the most prevalent and could be postulated as vaccine epitopes. Also, this work highlights the genetic diversity of GBS isolates circulating in the Pampean region of Argentina.

Penicillin-resistant GBS was not found, but resistance levels to tetracycline, fluoroquinolones, macrolides, and lincosamides were high. Our and worldwide reports on emerging multi-drug resistant GBS isolates reinforce the need for continued surveillance.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00284-022-03050-w.

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Author Contributions LH: Formal analysis, Investigation, Methodology, Writing—Original Draft. JC: Formal analysis, Visualization. FT, SA: Resources, Investigation. AB: Conceptualization, Methodology, Investigation. MS: Conceptualization, Writing—Review & Editing, Supervision, Project administration, Management.

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Data Availability All data generated or analysed during this study are included in this published article (and its supplementary information file).

Code Availability Not applicable.

Declarations

Conflicts of interest The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Ethics Approval The research was adjusted to the principles of Good Clinical Research Practices and adheres to the precepts established by the Declaration of Helsinki. The study followed the regulations stated in the Guide for Research in Human Health (Resolution 1480/11, Health Ministry of Presidency of the Argentine Nation, and Buenos Aires Province law No. 11,044 with its Regulatory Decree 3385/08 related to research on human beings). The data obtained were analysed anonymously, ensuring the patients' confidentiality who were assigned an alphanumeric code. The confidentiality rules were respected according to Law 25326 (Protection of Personal Data) and articles 51 and 52 of the Civil and Commercial Code of the Argentine Nation.

Informed Consent Not applicable.

Consent for Publication Not applicable.

Something about Declaration of Deposition in Repositories Not applicable.

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