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ARTICLE

Molecular epidemiology of group B streptococci in Ireland reveals a diverse population with evidence of capsular switching

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Abstract The molecular epidemiology of group B Streptococcus (GBS) in Ireland was investigated. Invasive (n=132) and non-invasive (n=45) isolates, collected in 2007–2011, were analysed by multilocus locus sequence typing, capsular polysaccharide (CPS) serotyping, profiling of surface proteins, pilus islands (PI), and antimicrobial susceptibility. Isolates grouped into 45 sequence types and five main clonal complexes (CC). CC1, CC17 and CC23 represented 67.2 % of isolates and the most prevalent serotypes Ia, III and V. Serotype and surface protein genes were largely predictive of CC. Accordingly, CPS V/*alp3*, CPS Ib/CPS II/*bca*+*bac*, and CPS Ia/*eps* predominated in CC1, CC12 and CC23, respectively, and CPS III/rib in CC17 and CC19. Supporting their vaccine potential, all isolates harboured at least one PI, of which the PI-1+PI-2a combination was most prevalent. Macrolide resistance was found in 18.6 % of isolates. *erm*(B) and the globally disseminated CC1/CPS V were the most common resistance mechanism and CC/CPS type, respectively. CC17, significantly associated with neonatal disease, was also prevalent in pregnant adults, but was underrepresented among non-pregnant adults. Two of 46 CC17 isolates (typically CPS III) were CPS IV. Sequence analysis confirmed capsular switching and their relatedness to CC17/CPS IV strains recently characterized in France. CPS IV, detected only in invasive isolates (6.8 %), was most prevalent in adults (12 %) and showed an increase in prevalence to that reported (1.4 %) for invasive isolates in Ireland 1997–1999. Increases in serotype IV and evidence of capsular switching in CC17 highlights the importance of ongoing surveillance of GBS and may have implications for vaccine development strategies.

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Introduction

Streptococcus agalactiae (group B Streptococcus [GBS]), is an opportunistic pathogen that is carried asymptomatically in the gastrointestinal and outer genitourinary tract of healthy adults with vaginal carriage rates of 10–36 % in women [1]. GBS is a leading cause of invasive infections in neonates and an emerging pathogen of adults [1–3]. Neonatal GBS disease is classified as either early-onset disease (EOD, 1–6 days) or late-onset disease (LOD; 7–89 days). The principal risk factor for neonatal disease, particularly EOD, is maternal colonization. The pathogenesis of LOD is less well understood, and may additionally be hospital or community acquired [1]. The incidence of invasive GBS disease of neonates is about 0.5–3 per 1000 lives births worldwide. However, a decline in EOD has occurred in countries where antenatal risk- or screening-based guidelines and prophylaxis have been implemented [1].

GBS possess ten distinct capsular polysaccharide (CPS) types: Ia, Ib and II–IX. The principal disease-associated serotypes are 1a, Ib, II, III and V [1–10]. CPS serotyping has been the classical method used to distinguish strains. More recently, molecular methods including pulsed-field gel electrophoresis (PFGE), multilocus sequence typing (MLST) and whole genome comparisons have been utilized to distinguish lineages and study diversity [11, 12]. MLST has identified five major

clonal complexes (CC) termed CC1, CC12 (also termed CC9, CC8 and CC10), CC17, CC19, and CC23. The sequence type (ST)-17 lineage, also termed the hypervirulent lineage, has been associated with enhanced invasiveness of neonates [13].

Available evidence supports the concept of an open pan-genome for GBS consisting of a core, stable genome and, a flexible, dispensable genome much of which encodes putative virulence-associated surface and secreted proteins [12]. The distribution and polymorphisms of these latter genes have also been employed to examine GBS diversity [14–17].

The continued prevalence of GBS disease and recognized limitations of prophylactic antibiotic use has switched the focus of preventive measures to vaccine development that would additionally target LOD and adults [1, 18]. Multivalent CPS-based conjugate vaccines targeting Ia, Ib, II, III and V have had encouraging results, although there may be less coverage in some global locations [1, 18]. Recent genomic and proteomic approaches have identified several protective protein antigens with likely greater coverage [19, 20]. Epidemiological and genomic studies performed to date have highlighted temporal and geographical differences in GBS [5, 9, 10, 18, 21, 22]. However, on-going surveillance of GBS is warranted in this pre-vaccine era for the provision of pre-vaccine population baselines, to facilitate rational vaccine design and monitor the impact of any future vaccines.

The aim of this investigation was to perform molecular epidemiological analysis on a collection of GBS isolates collected across all age groups with a view to identifying major invasive lineages and to examine GBS population diversity in Ireland.

Materials and methods

Bacterial isolates

A total of 177 isolates of GBS recovered from clinical specimens of patients in maternity, paediatric and adult hospitals in Ireland were collected during the period October 2007 to June 2011, and submitted for typing to the Epidemiology and Molecular Biology Unit. Invasive isolates (n=132), defined as GBS cultured from blood or another sterile site, included those from EOD (n=38), LOD (n=32), two cases of delayed LOD (92 days and 112 days), pregnant women (n=29, age range 20–42 years) and non-pregnant adults (n=31, age range 34–81 years). Sixteen neonatal invasive cases were confirmed to be meningitis by culture and/or PCR detection of GBS in cerebrospinal fluid. The remaining 56 cases were blood culture positive only. Noninvasive isolates (n=45) recovered from non-sterile sites included vaginal swabs, skin swabs and urine collected from non-sepsis/meningitis patients or healthy pregnant women (age range, 5 days–71 years). Isolates were grown overnight at 37°C in 5 % CO_2 on Columbia horse blood agar plates or in Todd Hewitt broth containing 0.2 % yeast extract.

DNA Extraction from bacterial cultures

Genomic DNA was extracted from GBS cells, pre-treated with mutanolysin, using the Puregene genomic DNA purification kit (Qiagen).

CPS serotyping

CPS serotypes were determined by latex co-agglutination (Statens Serum Institute) and by two multiplex PCR assays to detect *cps* Ia, Ib and II to VIII and *dltR* as an GBS internal positive control [23, 24]. A singleplex assay was used to detect *cps* IX [25]. Capsular switching of CC17 isolates was confirmed by sequence analysis of the conserved *neuB-neuA* and *cpsE-cpsG* regions of *cps* loci previously shown to possess nucleotide polymorphisms indicative of capsular serotype [26, 27]. *cps* and *neu* oligonucleotide primers are detailed in online resource 1.

MLST

Gene fragments of the seven housekeeping genes employed in MLST were amplified by PCR and sequenced as previously described [11]. ST and allele numbers were assigned using the *S. agalactiae* MLST database (http://pubmlst.org/saglactiae). STs were grouped into defined CCs using the eBURST program (http://eburst.mlst.net) and a criterion of sharing identical alleles at six loci with at least one other member of the group.

Surface protein genes and pilus islands

Multiplex PCR reactions to detect *bca*, *rib*, *eps*, *alp2*, *alp3*, *alp4* and *bac* were described elsewhere [16, 17]. Pilus-islands 1(PI-1), PI-2a and PI-2b were detected using previously described primer pairs [19]. All primers are detailed in online resource 1. Absence of pilus genes was confirmed by using additional pilus-specific primers described by Margarit et al. [19].

Antimicrobial susceptibility testing and detection of macrolide resistant genes

All GBS isolates were tested for susceptibility to erythromycin, clindamycin, benzylpenicillin, ampicillin, cefotaxime and vancomycin using E-test® (bioMérieux, Marcy l'Etoile, France). Methods and interpretative criteria were according to Clinical and Laboratory Standards Institute (CLSI) guidelines [28]. *erm*(B), *erm*(A) [*erm*(TR) subclass], *mef*(A), and

mef(E) were detected as previously described [29–31] using primer pairs detailed in online resource 1.

Statistical analysis

The chi-squared, Fisher's exact tests as well as odds ratio (OR) and 95 % confidence intervals (CI_{95}) were used, as appropriate, to evaluate associations between groups or lineages associated with certain characteristics. *P* values below 0.05 were considered statistically significant. The adjusted Wallace coefficient [32], calculated to examine concordance between typing methods, was performed using tools available at www.comparingpartitions.info.

Accession numbers cpsE-cpsG and *neuB-neuA* sequences of GBS148, GBS150, GBS175, GBS173 and GBS85 were deposited in GenBank; accessions numbers for these sequences are KF984492–KF984501, respectively.

Results

Serotyping

There was agreement between latex agglutination and PCR determination of CPS serotype for all isolates with the exception of three (ST-504/CPS III, ST-196/CPS IV and ST-19/CPS V) that failed to agglutinate specific antiserum. Serotypes III (35 %), Ia (21.5 %), V (16.9 %) and II (12.4 %) were the most common overall (Table 1). Invasive isolates in neonates, pregnant women and non-pregnant adults exhibited a significant difference in serotype distribution ($P=0.0013$) that was likely due to the predominance of type III (51.4 %) in neonates (36.8 % EOD; 67.6 % LOD), types Ia and III (34.5 % and 31 %, respectively) in pregnant women and, the under representation of type III ($n=3$) in non-pregnant adults where types II and V predominated (each accounting for 25.8 %). The distribution of CPS types in invasive isolates was not significantly different from the non-invasive population ($P=0.36$). Notably, however, CPS IV which accounted for 6.8 % of invasive isolates, was absent from non-invasive isolates.

MLST analysis

The 45 STs clustered into the five recognized clonal complexes (CC1, CC12, CC17, CC19 and CC23), a minor complex (CC22) and six singletons (Table 1 and Table 2). Also, 46 % ($n=21$) of STs were novel (see Table 2) with the greatest number in CC17. CPS distribution in the CCs was largely clonal ($P<0.0001$) and the population possessed a high $AW_{ST\rightarrow CPS}=0.834$ (CI_{95} 0.743–0.925) indicating that 83 % of any pair of isolates of the same ST shared the same CPS type. A lower concordance between CC and CPS type ($AW_{CC\rightarrow CPS}=0.636$, CI_{95} 0.548–0.724) was observed due to the presence of multiple CPS types in some CCs (Table 2).

CPS IV grouped in CC1 ($n=6$), a singleton (ST-452) and two CC17 isolates. The detection of two CPS IV isolates in CC17, usually associated with CPS III, was suggestive of capsular switching and consisted of an ST-291 (a single locus variant [SLV] of ST-17) and an ST-607 (SLV of ST-291) isolate recovered from unrelated invasive cases in a pregnant woman and a fatal case of EOD, respectively. The CPS type of the two CC17/CPS IV isolates (GBS150 and GBS175, respectively) was confirmed by sequence analysis of the conserved *cpsE-cpsG* and *neuB-neuA* regions of the *cps* operon which revealed that the sequences were 100 % identical to an ST1/CPS IV isolate (GBS148) in this study and CC209361, a ST-17/CPS IV strain recently characterized by Bellais et al. [33] but differed from two CC17/CPS III isolates (ST-17 [GBS173] and ST291 [GBS85]) by 7 and 17 SNPs for the *cpsE-cpsG* and *neuB-neuA* regions, respectively (data not shown).

CC17 was the most prevalent cause of invasive neonatal disease (45.8 %), followed by CC1 (15.3 %) and CC23 (12.5 %). The ST-17 lineage was significantly associated with neonatal disease (LOD, $P<0.0001$; EOD, $P=0.0029$) when compared to its prevalence in adult invasive disease. Moreover the lineage accounted for 53.8 % of confirmed LOD meningitis cases ($n=7/13$) and a higher prevalence in LOD (55.9 %) than EOD (36.8 %), although it did not reach significance ($P=0.16$). CC23 (41.4 %) and CC17 (24 %) were most prevalent in pregnant women. In contrast to the rest of the invasive population, only one CC17 isolate was detected in non-pregnant adults ($P<0.0001$). In this latter group, CC1 and CC12 together accounted for 64.5 % of isolates although only CC1 exhibited a significant association compared to its prevalence in neonates ($P=0.018$) and pregnant women ($P=0.017$). CC23 and CC19 lineages were more prevalent in non-invasive isolates (28.9 % and 20 %, respectively) than in invasive isolates (19.7 % and 9 %, respectively).

Distribution of surface protein genes and pilus islands

alp genes were detected in all but two isolates (ST-8 and ST-23), *alp4* was not detected and *bac* was prevalent only in CC12 (Table 2). *rib*, mainly confined to CC17 and CC19 ($P<0.0001$), was the most prevalent (39.5 %) and *alp2* (present mostly in CC23) was least prevalent (3.4 %). Due to the prevalence of some alp protein genes in more than one CC and CPS type, CC and CPS type was more predictive of alp protein gene ($AW_{cc\rightarrow alp}=0.699$, CI_{95}: 0.615–0.783; $AW_{cps\rightarrow alp}=0.698$, CI_{95}: 0.595–0.801) than vice versa ($AW_{alp\rightarrow cc}=0.425$, CI_{95}: 0.337–0.0.512; $AW_{alp\rightarrow cps}=0.523$, CI_{95}: 0.406–0.0.639) confirming the usefulness of *alp* genes in population

Table 1 Distribution of invasive and non-invasive GBS isolates according to capsular polysaccharide serotype (CPS) and clonal complex (CC)

Isolate	Invasive				Noninvasive	
	EOD	LOD	Pregnant adults	Non-pregnant adults		Total
Ia	5 (CC23)	4 (CC23) 1 (SS)	10 (CC23)	4 (CC23)	13 (CC23), 1 (SS)	38
Ib	2 (CC12), 2 (SS)	1 (CC12)	1 (CC12)	3 (CC12)	3 (CC12)	12
II	1 (CC1), 2 (CC12), 1 (CC19), 2 (CC22)	1 (CC22), 1 (CC12)	1 (CC12)	1 (CC1), 5 (CC12), 2 (CC19)	3 (CC12), 1 (CC1), 1 (CC22)	22
III	13 (CC17), 1 (CC19)	19 (CC17), 2 (CC19), 2 (SS)	6 (CC17), 2 (CC19), 1 (CC23)	1 (CC17) 1 (CC19) 1 (CC23)	8 (CC19), 4 (CC17), 1 (SS)	62
IV	1 (CC1), 1 (CC17)		3 (CC1), 1 (CC17)	1 (SS) 2 (CC1)		9
V	5 (CC1), 1 (CC19)	3 (CC1)	1 (CC12), 2 (CC19), 1 (CC23)	8 (CC1)	7 (CC1), 1 (SS), 1 (CC19)	30
VI–IX	1 (CC1)			1 (CC1), 1 (SS)	1 (CC1)	4
Total	38	34	29	31	45	177

SS singletons

Clonal complexes are indicated in parentheses

subtyping. *alp3* was confined to one CC viz. CC1 and accounted for 100 % of CC1/CPS V (n=23). The remaining isolates in CC1 largely presented with *eps* associated with 100 % of serotype IV isolates and also CC23/CPS Ia isolates (31/36, P<0.0001) (Table 2).

All isolates possessed one or more pilus islands with frequencies of 75.7 %, 71.2 % and 28.2 % for PI-1, PI-2a and PI-2b, respectively. The most prevalent combination was PI-1+PI-2a (48.6 %) which was detected in several CCs (CC1, CC12, CC19) (Table 2). The PI-1+PI-2b combination was unique to CC17. Whereas, CC23 and CC22 were notable for the virtual absence of PI-1.

Antimicrobial resistance

All isolates were susceptible to penicillin, ampicillin, cefotaxime and vancomycin. Erythromycin resistance (18.6 %; n=33) was distributed in all clonal complexes and all but two serotypes (VII and IX) (Table 3). Constitutive MLS_B ($cMLS_B$) accounted for 54.5 % (n=18) of resistant isolates. Sixteen $cMLS_B$ isolates harboured *erm*(B) and two isolates harboured *erm*(TR). Whereas isolates displaying inducible MLS_B ($iMLS_B$) resistance (27.3 %; n=9) and the M phenotype (18.2 %; n=6) presented with *erm*(TR) and *mef*(E), respectively. *mef*(E) was only detected in invasive isolates whereas *erm*(B) and *erm*(TR) together accounted for 14 % and 16 % of invasive and non-invasive isolates, respectively. Of all ST and serotype combinations, only ST1/CPS V was significantly associated with erythromycin resistance (P=0.0002; OR:6.7, 95 % CI: 2.545–17.64). Moreover, 42.8 % (n=15) of CC1 was erythromycin resistant and accounted for 67 % and 50 % of iMLSB and $cMLS_B$, respectively. CC23/CPS Ia predominated (n=4/6) in isolates displaying the M phenotype (P=0.0017).

Discussion

GBS is a leading cause of invasive disease of neonates and an emerging pathogen of adults [1, 18]. This report presents, for the first time, a comprehensive molecular analysis of GBS isolates circulating in Ireland. The 177 isolates in this study belonged to 45 STs which clustered into the five main CCs that have been detected worldwide [5, 11, 14, 15, 34]. CC23, CC17 and CC1 together accounted for 67.2 % of isolates and the most prevalent capsular serotypes Ia, III and V detected in Europe and the United States [3, 6, 9, 10, 35].

Our findings support others showing that the ST-17 lineage represents a successful clone with enhanced invasiveness for neonates and a high prevalence in LOD [22, 36]. CC23/CPS Ia and CC1 (represented by serotype V [n=8] and others [n=3]) were the second most prevalent causes of neonatal invasive disease. The incidence of neonatal CPS V (12.5 %) was somewhat higher than some other European countries (6.3–8 %) but lower than in Scandinavia (17–22 %) [10, 21, 33, 37, 38]. Whereas, the prevalence of CPS Ia (13.8 %) was similar to prevalence in Germany, Norway and Sweden (10–15 %) [21, 37, 38], but lower than recently reported in England, Wales and Spain (22–28 %) [3, 10]. The under representation of CC17 and dominance of CC1 and CC12 in older non-pregnant adults is similar to reports elsewhere [5, 39] and that the increase in invasive disease in non-pregnant adults coincided in part with the emergence of CPS V in the 1990s [2]. In pregnant women, the predominance of CC23 and CC17 in bacteraemia cases is in keeping with vaginal colonisation by these clonal complexes [40]. Whereas, CC19 was more prevalent in non-invasive isolates in agreement with other studies [21].

In this report, CPS IV was detected only in the invasive population and, though at an overall low prevalence (6.8 %),

Table 2 Distribution of sequence types (ST), capsular serotypes (CPS) and surface protein genes among GBS clonal complexes

CC	ST	CPS	*alp* and *bac*	Pilus Islands
1 (35)	1 (26), 2 (2), 136 (3), 196 (2), 507 (1), 514 (1)	II (3), IV (6), V (23), VI (2), VII (1)	*alp3* (25), *eps* (8), *rib* (1) *bca* (1)	PI-1 (33), PI-2a (34),PI-2b (1)
12 (23)	8 (7), 10 (3), 12 (9), 43 (1), 509 (1), 513 (1), 527 (1)	Ib (10), II (12), V (1)	*bca* (22), *bac* (22),	PI-1 (23), PI-2a (23)
17 (45)	17 (37), 291 (2), 512 (1), 515 (1), 557 (1),564 (2), 607(1)	III (43), (IV (2)	*rib* (45)	PI-1 (44), PI-2b (45)
19 (21)	19 (10), 27 (1), 28 (3), 110 (3), 182 (1), 504 (1), 510 (1), 566 (1)	III (14), II (3), V (4)	*rib* (20), *eps* (1)	PI-1 (20), PI-2a (21)
23 (39)	23 (32), 88 (4), 144 (1), 163 (1), 506 (1)	Ia (36), III (2), V(1)	*eps* (31), *alp2* (5), *rib* (2)	PI-1 (6), PI-2a (39)
22 (4)	22 (3), 525 (1)	II (4)	*bca* (4)	PI-2a (4)
SS (10)	4 (1), 7 (1), 452 (1), 505 (1), 508 (1), 511 (1), 516 (1), 526 (1), 529 (1), 565 (1)	Ia (2), Ib (2), III (3), IV (1), V(1), IX (1)	*bca* (4), *eps* (2), *alp2* (1), *bac* (4), *rib* (2)	PI-1 (9), PI-2a (5), PI-2b (4)

SS singletons

Number of isolates are indicated in parentheses. Novel STs are underlined

showed an increase to that previously reported (1.38 % in 1997–1999) for invasive GBS in Ireland [41]. CPS IV (12 %) in adult invasive disease was higher than in the United States (5.7 %), Australia (5 %), and other European countries (0.9–7 %) [2, 3, 5, 8, 42, 43]. An increased prevalence of CPS IV was also recently reported among colonizing isolates in Ireland (1–2 % in 1997–1998, 7.5 % in 2004 and 15 % in 2006) [44, 45]. Increases in CPS IV have been reported in the United States among colonizing (0.6 % in 1993–2002 to 8.4 % in 2004–2006) and invasive isolates (0.2 % in 1998/1999 to 5.7 % in 2005/2006) [2, 43]. High prevalences (18–26 %) were also reported in other geographical locations [7, 46, 47]. The lower incidence of serotype IV in neonates (2.7 %) was similar to the rest of Europe (1 %–3 %) [3, 10, 33, 35]. Significantly, two of nine CPS IV invasive cases were due to CC17. CC17/CPS IV was first reported as a novel ST (ST-291) in Portugal 2004–2006 [35] and subsequently reported at low frequencies elsewhere [33, 43, 48]. Genome analysis confirmed capsular switching in three ST-291/CPS IV strains recently isolated in France [33]. The two invasive CC17/CPS IV isolates detected here possessed *pheS-25, gdhA-7* and *pgm-6* alleles (data not shown) and *cps* IV sequences identical to the ST-291/CPS IV French isolates indicating that CC17/CPS IV may have evolved from a common ancestor that successfully disseminated. The detection of capsular switching in CC17 highlights the need for improved surveillance of CPS IV and the importance of sub-typing to identify these strains.

The serotype and ST distribution of surface protein genes and pilus islands was in keeping with other studies. Despite population diversity, there was good overall correlation between serotype, surface protein gene and STs [1, 5, 9, 14, 15, 34]. Some geographical differences in strain distribution were observed. ST-24/*bca* and ST-2/CPS V/*eps* isolates reported as sub-lineages in Mediterranean countries were absent in this study [5, 9]. Moreover, CC12 lacked *eps*- or CPS Ia-positive isolates documented in some studies [14, 15].

Current trialed pentavalent (Ia, Ib, II, III and V) and trivalent (Ia, Ib, III) conjugate vaccines would have reasonably good population coverage (93 % and 63 %, respectively) ([1]; http://clinicaltrials.gov/show/NCT01193920). However, the detection of CC17/CPS IV in different global locations raises concerns of possible serotype III replacement within this hypervirulent lineage. The presence of one or more pilus islands in all GBS isolates concurs with existing evidence supporting the use of pilin subunit proteins as vaccine candidates with likely greater global coverage [19, 34].

Despite recent reports of decreased susceptibility [49], all isolates were uniformly susceptible to penicillin, the first-line antibiotic. Resistance to macrolides and lincosamides, the alternative antimicrobials of choice, has however been increasing worldwide. The erythromycin resistance rate (18.6 %) reported here was higher than reported (11.4 %) in Ireland during 2004–2006 for colonizing GBS [50]. This latter

Table 3 Phenotypes and genotypes of macrolide resistant GBS isolates

CPS	ST	genotype	phenotype
Ia (6)	ST-23 (6)	*mefE* (5)	M (5)
		ermTR (1)	iMLSB (1)
Ib (1)	ST-12 (1)	*ermB* (1)	cMLSB (1)
II (3)	ST-12 (1)	*ermB*(1),	cMLSB (1)
	ST-12 (1), ST-22 (1)	*ermTR* (2)	iMLSB (2)
III (8)	ST-17 (5)	*ermB* (4)	cMLSB (4)
		mefE (1)	M (1)
	ST-19 (1)	*ermTR* (1)	cMLSB (1)
	ST-566 (1)	*ermB* (1)	cMLSB (1)
	ST-529 (1)	*ermB* (1)	cMLSB (1)
IV (1)	ST-514 (1)	*ermTR* (1)	iMLSB (1)
V (13)	ST-1 (13)	*ermB* (7)	cMLSB (8)
		ermTR (6)[a]	iMLSB (5)
VI (1)	ST-1 (1)	*ermB* (1)	cMLSB (1)

Number of isolates is indicated in parentheses. [a] iMLSB and cMLSB phenotypes were detected in five and one isolate, respectively

study [50] also reported a high prevalence of iMLSB/*erm*(TR) resistance (51 %/54 %). The high frequency of *erm*(B) in this study is similar to that reported in many studies [9, 42, 51]. However, high prevalence of *erm*(TR) was also reported in Canada and Portugal [10, 52] and may reflect temporal and geographical variations in circulating GBS resistant clones. The overall rate of erythromycin resistance detected in this study is within the ranges reported in Europe, South America and Canada (12–18.8 %) [3, 5, 9, 10, 52, 53] but less than higher rates recently reported in France (30 %) [42], the United States and Taiwan (46–54 %) [51, 54]. The prevalence of ST1/CPS V among resistant GBS in this study is consistent with successful clonal expansion of this strain [9, 10]. However, the detection of resistance in diverse ST and serotypes suggests many independent acquisitions of resistance determinants among the other four main CCs.

In conclusion, GBS in Ireland consists of a limited number of dominant lineages as detected elsewhere. Despite the clonality of the population, there was considerable population diversity favoring a multivalent vaccine approach for maximum coverage and prevention of genotype replacement. The emergence and expansion of virulent GBS lineages over the past five decades, increases in erythromycin and inducible clindamycin resistance rates, geographical strain variation and, the possibility for emergence of new virulent clones highlights the need for ongoing monitoring of GBS populations to facilitate vaccine and preventive strategies.

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Conflict of interest The authors declare no conflicts of interest.

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