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Letter to the Editor

Vir typing for the analysis of group C and group G streptococcal genotypes

Group C and group G streptococci (GCS and GGS) cause a wide variety of suppurative and nonsuppurative infections.[1] The epidemiology of GCS and GGS is considered to be important, since infections caused by GCS and GGS have increased in recent years.[2,3] Studies in the Aboriginal population of Australia suggest that they may also have rheumatogenic potential.[4] Vir typing is considered to be a good epidemiological tool for the identification of group A Streptococcus (GAS) genotypes and involves strain discrimination based on the vir regulon, which consists of structurally related genes of the *emm* family.[5] The vir regulon of GCS and GGS can also be amplified using GAS-specific primers.[6] Hence the present study was aimed to determine the genetic variability among the GCS and GGS by vir typing.

A total of 117 GCS and GGS isolates were included in the study. The majority of the isolates ($n = 88$) were from throat cultures of asymptomatic children attending four different schools in Chennai City. The remaining isolates were from pharyngotonsillitis/pyoderma ($n = 26$), blood ($n = 2$), and aspirated fluid ($n = 1$). DNA was extracted from fresh subcultures of bacterial isolates by alkali lysis method.[6] PCR for vir regulon amplification and subsequent digestion with *Hae*III for analysis of restriction fragment length polymorphism (RFLP) was performed using previously described primers and conditions.[5]

The RFLP patterns were visually compared. VT8 was the most common (18.8%), followed by VT37 (4.3%), VT1 (3.4%), and VT28 (3.4%). A total of 61 vir type (VT) patterns were obtained from the 117 strains by RFLP analysis, accounting for 52.13% diversity. Thirty-three *emm* types and subtypes were identified among these 61 vir types. Since the majority of the strains had been *emm* sequence typed we attempted to compare the *emm* type with the vir type, but found virtually no concordance (Table 1). Previous studies on GAS have shown that *emm* types could be related to vir types in the majority of cases.[7] However no study has so far compared *emm* types and vir types of GGS and GCS. Vir typing also appeared to be less discriminatory than *emm* typing in this study, since some of the common types such as VT8 were represented by more than one *emm* type. However this could have been because of the fact that our RFLP patterns were based on digests using only one enzyme (*Hae*III). Use of the second enzyme *Hinf*I is known to

Table 1
Distribution of *vir* types among group C Streptococcus and group G Streptococcus strains

Vir type	No. of isolates	Source of isolation (throat/skin/invasive)	Biotype	*Emm* type
VT1	4	2/1/1	SDSD, SDSE, and SESZ	*stG6792.3*, *stG2574.0*, *stG866.0*
VT2	1	1/0/0	SESZ	*stG652.0*
VT3	3	3/0/0	SDSD, SDSE, and SESZ	*stG866.0*, *stG6792.3*, *stC36.0b*
VT4	2	2/0/0	SDSD and SESZ	*stG866.0*, *stG485.0*
VT5	2	0/2/0	SDS and SDSE	*stG652.1*, *emm80.0*
VT6	1	1/0/0	SESZ	*stC1400.0*
VT7	1	1/0/0	SDSE	*stG4974.0*
VT8	22	21/1/0	SDSD, SDSE, and SESZ	*stC2Sk.0*, *stC5345.1*, *stG166b*, *stG245.0*, *stG4974.0*, *stG2574.0*, *stG653.1*, *stG840.0*, *stGLp1.0*
VT9	2	2/0/0	SDSE	*stG6792.3*
VT10	1	1/0/0	SDSE	*stG485.0*
VT11	1	1/0/0	SDSD	*stG245.0*
VT12	1	1/0/0	SDSE	*stG7882.1*
VT13	1	1/0/0	SDSE	*stG480.0*
VT14	2	2/0/0	SDSE and SESZ	*stC36.4*
VT15	1	1/0/0	SDSE	*Emm* gene not amplified
VT16	1	1/0/0	SDSE	*stC345.1*
VT17	2	2/0/0	SDSE	*stG245.0*
VT18	1	1/0/0	SESZ	*stC1400.0*
VT19	3	3/0/0	SDSE and SESZ	*emm103.0*, *stG245.1*, *stC839.4*
VT20	1	0/1/0	SDSE	*stG1750.0*
VT21	1	1/0/0	SESZ	*stC5345.1*
VT22	1	1/0/0	SDSE	*stC5345.1*
VT23	2	2/0/0	SDSE	*stG643.0*, *stG653.0*
VT24	1	1/0/0	SDSE	*stG653.0*
VT25	1	1/0/0	SDSE	*stC1400.0*
VT26	2	2/0/0	SDSE and SESZ	*stG4974.0*, *stG485.0*
VT27	1	1/0/0	SDSE	*stG6792.3*
VT28	4	4/0/0	SDSE	*stG653.0*, *stG2574.0*, *stG4974.0*, *stG866.0*

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Table 1 (*Continued*)

Vir type	No. of isolates	Source of isolation (throat/skin/invasive)	Biotype	*Emm* type
VT29	4	4/0/0	SESZ and SDSE	*stG1750.0*, *stG866.0*, *stG10.0*, *stG653.0*
VT30	1	1/0/0	SDSE	*stC6792.3*
VT31	1	1/0/0	SDSE	*stC1400.0*
VT32	3	3/0/0	SDSE	*stG652.0*, *stC1400.0*
VT33	1	1/0/0	SDSE	*stGM220.0*
VT34	1	1/0/0	SDSE	*stC36.4*
VT35	2	1/0/1	SDSE	*stC6979.0*, *stG6792.3*
VT36	1	0/0/1	SDSE	*stG6792.3*
VT37	5	5/0/0	SDSE and SESZ	*stC5345.0*, *stGLp1.0*, *stG245.0*, *stG4974.0*, *stC1400.0*
VT38	1	1/0/0	SDSE	*stC1400.0*
VT39	2	2/0/0	SDSE	*stGLp1.0*
VT40	1	1/0/0	SDSD	*stG643.0*
VT41	2	2/0/0	SDSE	*stG6792.3*
VT42	2	1/1/0	SDSD	*stC1400.0*
VT43	1	1/0/0	SDSE	*stC1400.0*
VT44	1	1/0/0	SDSE	*stC5345.1*
VT45	1	1/0/0	SESZ	*stG6792.3*
VT46	3	3/0/0	SDSE	*stC36.4*
VT47	2	2/0/0	SDSE	*stG653.2*
VT48	1	1/0/0	SDSE	*stC36.0b*
VT49	1	1/0/0	SDSE	*stG4974.0*
VT50	1	1/0/0	SDSE	*stC36.4*
VT51	1	1/0/0	SDSE	*stGLp1.0*
VT52	1	1/0/0	SDSE	*stC5345.1*
VT53	1	1/0/0	SDSE	*Emm* gene not amplified
VT54	1	1/0/0	SDSE	*emm113.0*
VT55	2	2/0/0	SDSD	*stG6792.3*
VT56	1	0/1/0	SDSE	*stG1750.0*
VT57	1	1/0/0	SDSE	*Emm* gene not amplified
VT58	1	2/0/0	SDSE and SESZ	*stG6792.3*, *stG653.0*
VT59	1	1/0/0	SDSE	*stG480.0*
VT60	1	1/0/0	SDSE	*stC1400.0*
VT61	1	1/0/0	SDSE	*stG1750.0*

SDSD, *Streptococcus dysgalactiae* subsp. *dysgalactiae*; SDSE, *Streptococcus dysgalactiae* subsp. *equisimilis*; SESZ, *Streptococcus equi* subsp. *zooepidemicus*.

increase the discriminatory power of vir typing and yield information on subtypes.[7]

In conclusion, vir typing could be an alternative genotyping method for GCS and GGS isolates.

Conflict of interest: No conflict of interest to declare.

Ethics considerations: This work was approved by the institutional ethics committee and subjects gave informed consent to the work.

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