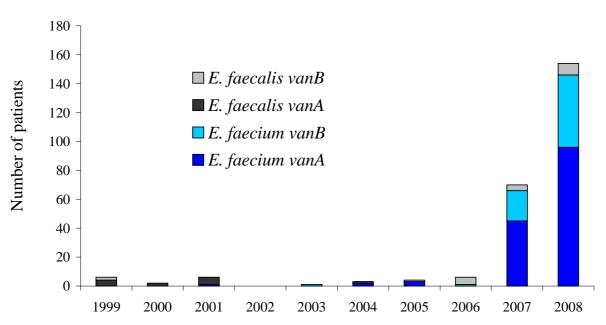
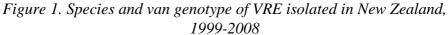
Vancomycin-resistant enterococci (VRE) confirmed in 2008

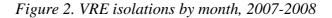
VRE from 153 patients were referred to ESR in 2008. The species and van genotype distribution of the VRE from these 153 patients is shown in Figure 1. 95 patients had vanA *E. faecium*, 50 had vanB *E. faecium*, and 8 had vanB *E. faecalis*. There were no isolates of vanA *E. faecalis*.

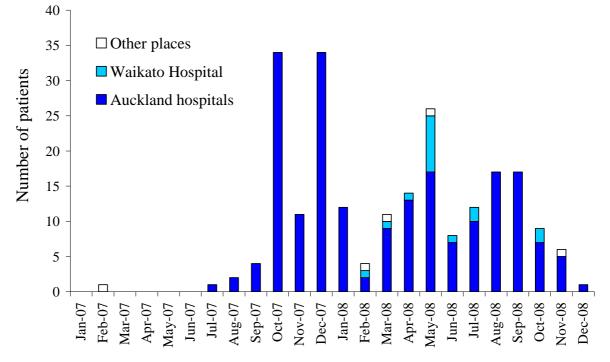




The number of VRE referred to ESR in 2008 was over twice the number referred in 2007 (Figure 1). Before 2007, VRE were isolated from no more than six patients in any one year. The increases in VRE isolates since 2007 are due to an outbreak of vancomycin-resistant *E. faecium* in Auckland City Hospital in 2007, which continued into 2008, along with additional vancomycin-resistant *E. faecium* outbreaks in other Auckland hospitals and Waikato Hospital in 2008. In 2008, 87.0% percent of patients with VRE were from Auckland hospitals (57.4% Auckland City Hospital, 17.3% Middlemore Hospital and 12.3% North Shore Hospital) and 9.8% from Waikato Hospital. A more detailed breakdown of the sources of the VRE referred in 2008 is shown in Table 1.

Figure 2 shows the epidemic curve for the VRE outbreaks in Auckland hospitals over the 2007-2008 period and Waikato Hospital in 2008. The outbreaks in Auckland hospitals were contained by the end of 2008. The outbreak in Waikato Hospital patients was confined to the February-July 2008 period, as the two isolates in October did not belong to the outbreak strain.





Assignment of isolations to a month was based on specimen date, not the date the isolate was referred to ESR.

In 2008, the majority (138, 89.6%) of the VRE were isolated from rectal swabs or faecal specimens as the result of screening for the organism. The remaining VRE were isolated from blood (3, 1.9%), urine (7, 4.5%) and other miscellaneous diagnostic specimens (6, 3.9%).

Table 1 shows the various VRE strains identified by pulsed-field gel electrophoresis (PFGE) typing in 2008. Two vanA *E. faecium* strains (EfN and EfAF) were isolated from patients in Auckland City, Middlemore and North Shore Hospitals, and another two vanA *E. faecium* strains (EfAG and EfAH) were isolated from Auckland City and Middlemore Hospital patients. Similarly, three of the vanB *E. faecium* strains (EfO EfQ and EfAB) were common to patients in Auckland City and Middlemore Hospitals. With the exception of one isolate of strain EfAD from an Auckland City Hospital patient who had been in Waikato Hospital, the two vanB *E. faecium* strains (EfAD and EfAE) isolated from patients in Waikato Hospital were distinct from the VRE strains isolated in Auckland hospitals.

Seven of the eight vanB *E. faecalis* isolates belonged to strain EfJ. This strain has been predominant among vanB *E. faecalis* isolates since 2005, and all isolates of this strain have come from the Northland or Auckland area. Most cases appear to be sporadic.

Species	<i>van</i> gene	Referred from	PFGE profile/'strain' ¹	Number of patients ²
E. faecium	А	Auckland City Hospital	EfN	32
			EfAF	25
			EfAG	3
			EfAH	1
			distinct ³	3
		Middlemore Hospital	EfN	2
			EfAF	1
			EfAG	1
			EfAH	8
			distinct	2
		North Shore Hospital	EfN	14
			EfAF	6
		Waikato Hospital	distinct	2
	В	Auckland City Hospital	EfO	9
			EfQ	5
			EfAB	8
			$EfAD^4$	1
			distinct	2
		Middlemore Hospital	EfO	1
			EfQ	3
			EfAB	9
			distinct	1
		Waikato Hospital	EfAD	9
		-	EfAE	3
			distinct	2
		Wellington Hospital	distinct	1
E. faecalis	В	Auckland City Hospital	EfJ	4
		Auckland community	EfJ	1
		Whangarei Hospital	EfJ	2
		Wellington Hospital	distinct	1

Table 1. VRE referred to ESR, 2008

1 In-house pulsed-field gel electrophoresis (PFGE) profile designations. The PFGE profiles of isolates designated as the same strain share ≥90% similarity. PFGE profile designations in boldface are profiles of strains that were also identified in 2007.

3 Distinct isolates that do not share \geq 90% PFGE profile similarity with any other VRE isolate.

4 Patient transferred from Waikato Hospital.

² Two vanA *E. faecium* strains (EfN and EfAF) were isolated from one patient. This patient is included in the counts for both strains. Another eight patients were identified with the same strain of VRE in different hospitals and are included in the counts for each hospital.

The antimicrobial susceptibility among the 2008 VRE isolates is shown in Table 2. All VRE tested were susceptible to linezolid. Almost all VRE were multiresistant to \geq 3 antibiotic classes in addition to glycopeptides. The vanA genotype typically confers resistance to both vancomycin and teicoplanin. However, 15 vanA *E. faecium* isolates were teicoplanin susceptible (MICs 1-4 mg/L). These isolates all belonged to the same strain (EfAF).

	Percent resistance			
Antimicrobial agent ¹	E. faecium			E. faecalis
	vanA n=96 ²	vanB n=50	All n=146	– vanB n=8 ³
ampicillin	97.9	100	98.6	0.0
ciprofloxacin	97.9	100	98.6	87.5
gentamicin high-level	61.5	26.0	49.3	100
nitrofurantoin	10.4	4.0	8.2	0.0
quinupristin/dalfopristin	1.0	0.0	0.7	100^{4}
streptomycin high-level	83.3	68.0	78.1	12.5
teicoplanin	84.4	0.0	55.5	0.0
tetracycline	58.3	18.0	44.5	87.5
multiresistant ⁵	96.9	90.0	94.5	87.5

Table 2. Resistance among VRE referred to ESR, 2008

1 Ampicillin, ciprofloxacin, gentamicin, linezolid and teicoplanin susceptibilities were determined by Etest MICs. Nitrofurantoin, quinupristin/dalfopristin, streptomycin and tetracycline susceptibilities were determined by disc testing. All isolates were susceptible to linezolid.

2 Includes isolates of two different strains from the same patient.

3 All *E. faecalis* isolates had the vanB genotype.

5 Resistant \geq 3 classes of antibiotics in addition to glycopeptides (quinupristin/dalfopristin resistance not included for *E. faecalis*).

⁴ *E. faecalis* are intrinsically resistant to quinupristin/dalfopristin.