



Annual survey of extended-spectrum β -lactamase (ESBL)-producing Enterobacteriaceae, 2011

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Up until 2005, national surveillance of extended-spectrum β -lactamase (ESBL)-producing Enterobacteriaceae (ESBL-E) was based on diagnostic laboratories referring all isolates to ESR for confirmation. This continuous surveillance ceased in 2005 and was replaced with annual surveys.

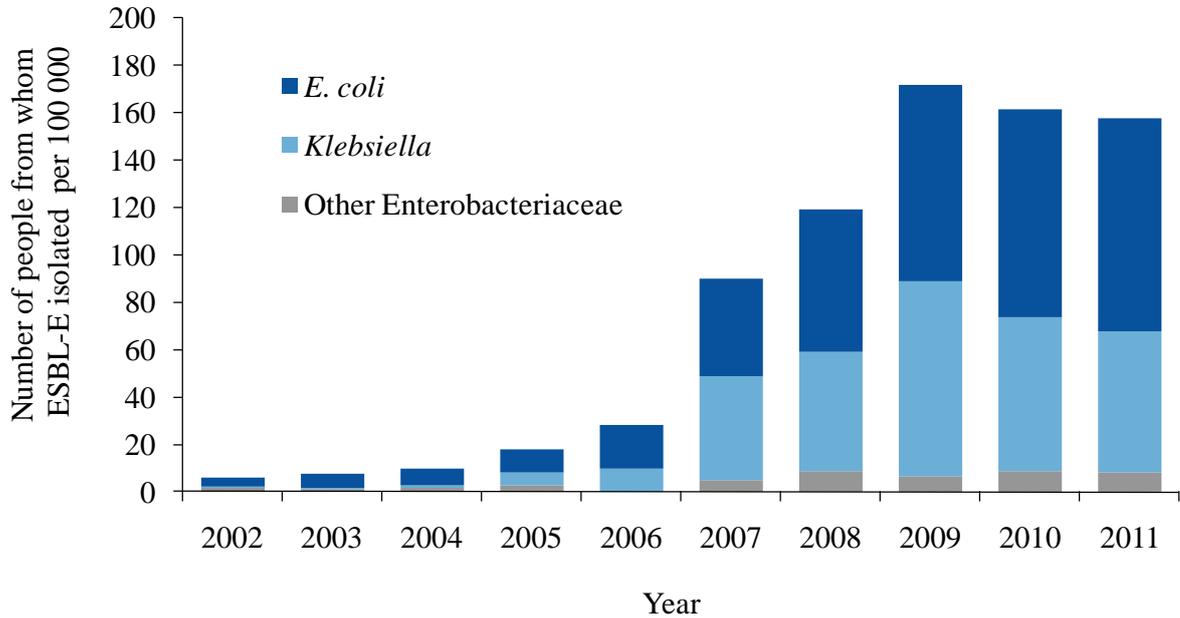
For the 2011 survey, hospital and community microbiology laboratories in New Zealand were asked to refer all ESBL-E isolated during August 2011 to ESR. Laboratories that do not test for ESBL production were asked to refer all Enterobacteriaceae resistant or intermediate to a 3rd-generation cephalosporin. The Microbiology Department, Middlemore Hospital; Microbiology Department, Hawkes Bay Hospital; and Diagnostic Medical Laboratory, Auckland, referred isolates during a 31-day period between mid-August and 31 October 2011. All remaining laboratories referred isolates during August. When referring isolates for the survey, laboratories supplied epidemiological data including patient age, geographic location, hospitalisation status, isolation site, infection or colonisation status, and if the isolate was obtained from a screen or a diagnostic specimen.

At ESR, all isolates referred for the survey were confirmed as ESBL positive by the Clinical and Laboratory Standards Institute's (CLSI's) phenotypic confirmatory disc test,¹ or a double-disc synergy test with cefotaxime, ceftazidime, cefpodoxime and cefepime as substrates.²

During the 2011 survey period, ESBL-E were isolated from a total of 578 people, which equates to an annualised incidence rate of 157.5 people with ESBL-E per 100 000 population; a small decrease on the 2010 rate of 163.7. Figure 1 shows the annual or annualised incidence of ESBL-E over the 10 years 2002 to 2011, and the distribution of ESBLs among *Escherichia coli*, *Klebsiella* species and other Enterobacteriaceae.

The 578 ESBL-E isolates referred in 2011 comprised 329 (56.9%) *E. coli*, 219 (37.9%) *Klebsiella* species, 16 (2.8%) *Enterobacter* species, 10 (1.7%) *Citrobacter* species, 2 (0.4%) *Morganella morganii*, and 1 (0.2%) each of *Kluyvera ascorbata* and *Raoultella* species. Nineteen patients had two different ESBL-producing species and one patient had three species.

Figure 1. ESBL-producing Enterobacteriaceae incidence rates, 2002-2011



Data for 2002 to 2005 are based on continuous surveillance of all ESBL-E isolations. Data for 2006 to 2011 are annualised and based on 4-week or 1-month surveys conducted in these years. The 2006 survey only included urinary *E. coli* and *Klebsiella*, therefore the data for 2006 is not directly comparable with that for other years.

The patients from whom ESBL-E were isolated were categorised as hospital patients if they were in a healthcare facility (including emergency department, outpatient clinic or residential-care facility) when ESBL-E was isolated or had been in a healthcare facility in the previous three months. All other patients were categorised as community patients. The majority of the ESBL-E (64.4%, 362 of the 562 for whom the information reported) were isolated from patients categorised as hospital patients. A larger proportion of the ESBL-producing *Klebsiella* than *E. coli* were isolated from patients categorised as hospital patients (85.3% vs 50.2%). These proportions of hospital patients with ESBL-E are lower than recorded in previous years: for example in the 2010 survey, 83.1% of all ESBL-E, and 95.4% of ESBL-producing *Klebsiella* were isolated from hospital patients.

55.3% of the patients with ESBL-E were ≥ 65 years of age, 39.1% were 15-64 years and 5.6% were ≤ 14 years old. ESBL-producing *Klebsiella* were more likely to be isolated from older patients than ESBL-producing *E. coli*, with 63.3% of *Klebsiella* isolated from patients ≥ 65 years of age compared with 49.2% of *E. coli*.

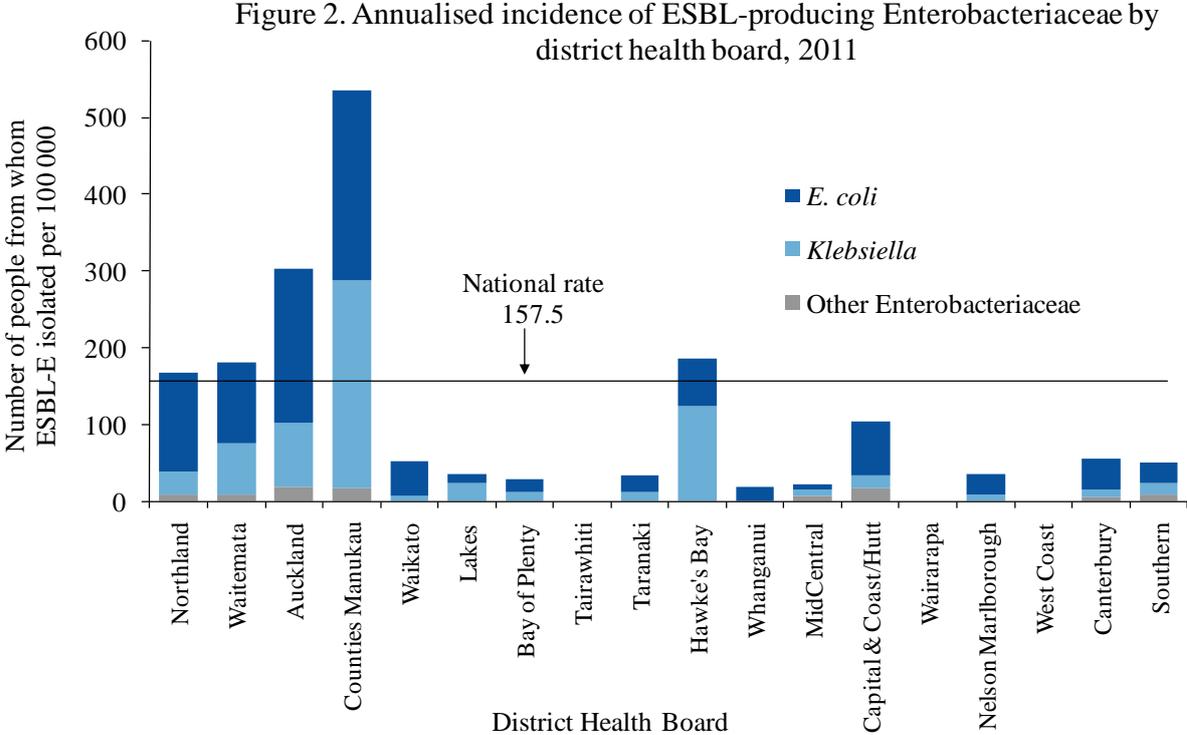
Information on whether the ESBL-E was causing infection or colonising was reported for 516 (89.3%) of the patients with ESBL-E, of whom 256 (49.6%) were considered to have an ESBL-E infection. Table 1 compares the distribution of species, hospital and community patients, and isolation sites for ESBL-E from infected sites with those from colonised sites. A larger proportion of the ESBL-producing *Klebsiella* (66.2%) than the ESBL-producing *E. coli* (39.5%) were from colonised sites. This most likely reflects the screening that occurs in hospitals as part of measures to control the transmission of these organisms, and the fact that ESBL-producing *Klebsiella* were more likely than ESBL-producing *E. coli* to be associated with hospital patients.

Table 1. Comparison of ESBL-producing Enterobacteriaceae from infected and colonised sites, 2011¹

	Number (row %)	
	ESBL-E from infected sites (n=256)	ESBL-E from colonised sites (n=260)
Species:		
<i>E. coli</i>	176 (60.5)	115 (39.5)
<i>Klebsiella</i> species	67 (33.8)	131 (66.2)
other species	13 (48.2)	14 (51.9)
Isolated from:		
hospital patients ²	142 (43.6)	184 (56.4)
community patients ²	110 (61.8)	68 (38.2)
Isolation site: ³		
CSF/blood	11 (100)	0
faeces	0	230 (100)
urine	223 (91.8)	20 (8.2)
wound	10 (66.7)	5 (33.3)
other	11 (78.6)	3 (21.4)

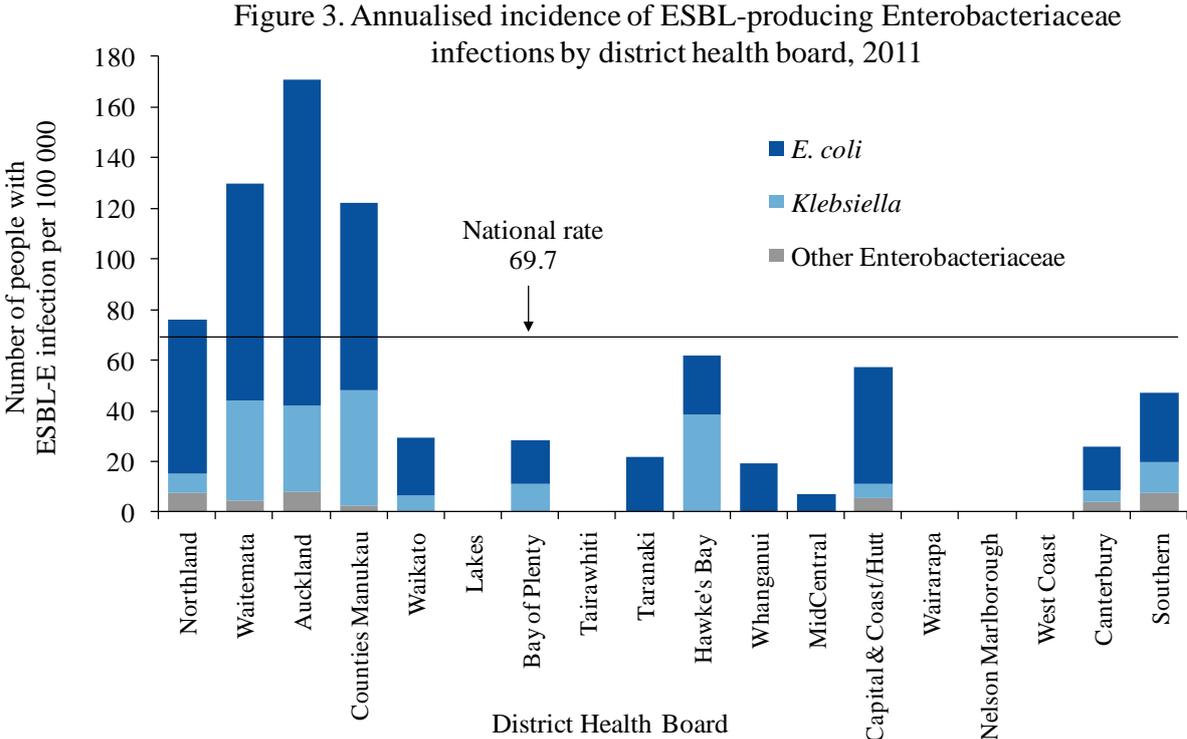
- 1 Information on whether the ESBL-E was isolated from an infected or colonised site was reported for 516 of the 578 isolates. The remaining 62 isolates are not included in the analyses in this table. Of these 62 isolates, 58 were from urine.
- 2 Patients were categorised as hospital patients if they were in a healthcare facility (including emergency department, outpatient clinic or residential-care facility) when ESBL-E was isolated or had been in a healthcare facility in the previous three months. All other patients were categorised as community patients.
- 3 Site not known for 4 isolates.

Figure 2 shows the incidence of ESBL-E in each district health board (DHB). The highest annualised incidence rates, and rates above the national rate of 157.5 per 100 000 population, occurred in the Counties Manukau (535.3 per 100 000), Auckland (302.2), Hawke’s Bay (184.9), Waitemata (180.3) and Northland (166.9) DHBs.



Data for the Capital & Coast and Hutt District Health Boards (DHBs) is combined as ‘Capital & Coast/Hutt’, and data for the Canterbury and South Canterbury DHBs is combined as ‘Canterbury’.

Some of the apparent differences in ESBL-E rates between DHBs evident in Figure 2 could be due to differences in screening policies between DHBs. Figure 3 shows the annualised DHB incidence rates for ESBL-E that were isolated from infections only. The five DHBs with the highest rates of ESBL isolations (Counties Manukau, Auckland, Hawke’s Bay, Waitemata and Northland, Figure 2) also had the highest rates of ESBL-E infection (Figure 3), although Auckland and Waitemata DHBs had higher ESBL-E infection rates than Counties Manukau DHB, which had the highest overall rate of ESBL-E isolations.



Data for the Capital & Coast and Hutt District Health Boards (DHBs) is combined as ‘Capital & Coast/Hutt’, and data for the Canterbury and South Canterbury DHBs is combined as ‘Canterbury’.

The proportions of the ESBL-E isolates that would be categorised as ESBL screen positive, cefotaxime resistant and ceftazidime resistant, on the basis of interpreting cefotaxime and ceftazidime zones of inhibition according to the 2011 CLSI standards,¹ are shown in Table 2. 98.2% of the ESBL-producing *E. coli* and *Klebsiella* isolates were categorised as cefotaxime resistant, but only 49.8% of these isolates were categorised as ceftazidime resistant, presumably due to CTX-M-type ESBLs being prevalent. Similarly, 90.0% of the species other than *E. coli* and *Klebsiella* were categorised as cefotaxime resistant, but only 53.3% were ceftazidime resistant.

The ESBL-producing *E. coli* and *Klebsiella* isolates that were ceftazidime resistant or intermediate were tested for plasmid-mediated AmpC β -lactamases. Four (1.2%) of the 329 ESBL-producing *E. coli* had a plasmid-mediated AmpC β -lactamase: three CIT types and one DHA type. Six (2.7%) of the 219 ESBL-producing *Klebsiella* had a plasmid-mediated AmpC β -lactamase: one CIT type and five DHA types.

Table 2. Cefotaxime and ceftazidime susceptibility of ESBL-producing Enterobacteriaceae, 2011

Species	Number (%) of ESBL-producing Enterobacteriaceae (n=578)							
	Cefotaxime				Ceftazidime			
	S ¹	I ¹	R ¹	Screen positive ²	S	I	R	Screen positive ²
<i>E. coli</i> and <i>Klebsiella</i> n=548	3 (0.5)	7 (1.3)	538 (98.2)	546 (99.6)	145 (26.5)	130 (23.7)	273 (49.8)	442 (80.7)
Other species n=30	1 (3.3)	2 (6.7)	27 (90.0)	30 (100.0)	8 (26.7)	6 (20.0)	16 (53.3)	24 (80.0)

1 S, susceptible; I, intermediate; R, resistant; based on cefotaxime and ceftazidime zone diameters interpreted according to the 2011 CLSI interpretive standards (see reference 1 below).

2 ESBL screen positive according to the 2011 CLSI interpretive standards, that is, cefotaxime zone diameter ≤ 27 mm, ceftazidime zone diameter ≤ 22 mm.

References

- 1 Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing; twenty-first informational supplement. Wayne (PA): CLSI; 2011. CLSI document M100-S21.
- 2 Jarlier V, Nicolas MH, Fournier G, et al. Extended-broad spectrum β -lactamases conferring transferable resistance to newer β -lactam agents in Enterobacteriaceae: hospital prevalence and susceptibility patterns. Rev Infect Dis 1988; 10: 867-78.