

## 6. LABORATORY SURVEILLANCE

### Laboratory-based legionellosis surveillance, 2015

Laboratory-based testing and surveillance analysed 438 notified legionellosis cases in 2015, identifying 251 cases, of which 150 met the confirmed case definition<sup>#</sup>. This compared with 135 laboratory-identified cases in 2014 (91 confirmed) and 151 cases in 2013 (110 confirmed). The year-on-year increase is significant with the HRC-funded LegiNZ study (20 May 2015 to 20 May 2016) the assumed reason for the increase. That study used molecular diagnostic testing of pneumonia cases admitted to hospital to identify patients with a *Legionella* infection. The 86% increase is probably due to historical under-diagnosis of the disease rather than an increase in disease burden.

Of the 150 cases meeting the confirmed case definition, 47 were culture-positive (all with further supporting laboratory evidence—either a positive nucleic acid amplification test (NAAT), positive serologic tests, or both). A positive *Legionella* urinary antigen test confirmed 26 more, with 17 having further supporting laboratory evidence (molecular and/or serologic). A further 77 (11 also with positive NAAT) were confirmed on serologic evidence with either a four-fold or greater rise in antibody titres (40), or antibody titre  $\geq 512$  on more than one occasion (37). Of the 101 cases meeting the probable surveillance case definition, 59 had a positive NAAT only, 25 a single positive serologic test only, and 17 with both.

Of the 251 laboratory-proven cases identified in 2015, 140 had an initial positive *Legionella* NAAT result, demonstrating the value of this method for the early detection of legionellosis cases. Clinicians are encouraged to use molecular testing of lower respiratory tract samples from suspected legionellosis cases; augmented with acute and convalescent serology for strain identification whenever culture is negative, since this often cannot be identified by the NAAT alone.

Culture isolation from lower respiratory tract continues to be the 'gold standard' in diagnosing legionellosis. Accurate diagnosis requires specific laboratory testing; it cannot be diagnosed reliably on clinical features alone. Currently, no single laboratory test offers adequate specificity and sensitivity to provide acceptable diagnostic accuracy. Optimal diagnostic accuracy and epidemiological information requires a combination of NAAT, serology and culture. Recent changes to the surveillance case definition now mean that a positive NAAT fits the confirmed case definition ([www.health.govt.nz/system/files/documents/publications/cd-manual-legionellosis-jul16.pdf](http://www.health.govt.nz/system/files/documents/publications/cd-manual-legionellosis-jul16.pdf)), whereas those diagnosed serologically with only an elevated antibody titre of  $\geq 512$  on one or more occasions, without a four-fold rise, now fit the *probable* case definition.

Reporting for 2015 used the case definition in use at that time (see Table 1 for definitions).

Table 1 shows legionellosis incidence rates calculated for each district health board where five or more cases were diagnosed. The rates ranged from 2.32 per 100,000 population in the Capital & Coast DHB to 10.46 per 100,000 population for the MidCentral DHB. The elevated incidence rate for this DHB is the result of 10 cases that were linked with a cooling tower-associated outbreak that occurred between August and October 2015.

Nationally, the most common *Legionella* species identified was *L. longbeachae* (131, 51.8%), followed by *L. pneumophila* (73, 28.9%) (Figure 3). The predominant *L. pneumophila* strain as in previous years, is still serogroup 1 (44). The remaining 48 (19.0%) cases where a causative organism was identified included *L. anisa*, *L. bozeman*, *L. dumoffii*, *L. feeleii*, *L. gormanii*, *L. jordanis*, *L. micdadei* and *L. saintelensis* infections (Figure 3).

Three recognised outbreaks occurred during 2015: one was associated with a cruise ship (2 cases infected with *L. pneumophila* sg 12); two were associated with cooling towers. The first cooling tower outbreak was traced to the Woolston industrial area of Christchurch in April 2015, and involved five cases infected with *L. pneumophila* sg 1. Using sequence-based typing (SBT) the *Legionella pneumophila* serogroup 1 strain causing this outbreak was shown to have the allele profile 12, 9, 26, 5, 3, 17, 15. The second cooling tower outbreak was traced to cooling towers at an industrial site in Pahiataua contaminated with multiple *Legionella* species and where infections with different *Legionella* species resulted: 10 cases were identified with infections directly related to exposure to the cooling towers (*L. pneumophila* sg 1 (5) and *L. longbeachae* (5)). During the outbreak case tracing, a further four cases were diagnosed serologically with strains not isolated from the towers: *L. pneumophila* sg 5 (1), *L. saintelensis* (2), and a *Legionella* species not identified (1). Using SBT the *Legionella pneumophila* serogroup 1 strain associated with this outbreak was shown to have the allele profile 12, 8, 11, 21, 40, 12, 9.

Source tracing also linked 109 legionellosis cases with exposure to compost, potting mix or other gardening activity during the incubation period. These cases included infections with *L. longbeachae* (93), *L. bozeman* (3), *L. pneumophila* (2), *L. gormanii* (2), *L. feeleii* (1), *L. micdadei* (2); and 6 where the *Legionella* species could not be identified. Of the 109, 33 had a proven link and 76 had a suspected link. For a proven link, the causative organism was isolated from composted material. For a suspected link, exposure to composted material was reported during the incubation period, but either no sampling was made or testing failed to isolate the causative organism.

Exposure to recreational waters (spa or swimming pool) was identified as the source for a further 3 cases (*L. pneumophila* sg 1 (2) and *L. anisa* (1)). Travel away from home was identified as a risk factor for a total of 10 cases: 2 domestic and 8 international (including the 2 outbreak cases linked to a cruise ship). For the travel-associated cases, the predominant infection was caused by *L. pneumophila* sg 1 (6). For the remaining 108, including 32 *L. longbeachae* and 48 *L. pneumophila*, no clear exposure source was identified.

Case numbers for *L. longbeachae* infection show a seasonal increase from early spring (September), rising to high levels in December (35). For other months of the year case numbers fluctuate between 2 and 7 a month, except for February which shows a spike to 11 (Figure 3). This pattern is associated with gardening activities, which occur

most often in spring and summer. In contrast, *L. pneumophila* infections appear to be evenly spread throughout the year, although again, as in previous years, a spike in cases was seen in April and May. The April spike coincides with the cooling tower outbreak in Christchurch, although no common source was identified for an *L. pneumophila* sg 1 cluster of 10 cases that occurred in the Greater Auckland area between March and May 2015.

ESR acknowledges the invaluable contribution of hospital laboratories at LabPlus, Middlemore, Waikato, MedLab Central, Wellington and Canterbury Health Laboratories for the molecular identification of NAAT-positive cases and provision of clinical specimens for surveillance testing, and the Public Health Service for providing environmental source tracing samples.

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**FIGURE 3. Number of legionellosis cases by *Legionella* causative agent and month, 2015**



