Background
Acute otitis externa (AOE) is a common inflammatory condition affecting the external ear that occasionally presents with persistent, severe pain, which may be unresponsive to first-line therapy and require assessment and treatment in the hospital setting.

Aims
To identify the microorganisms responsible for cases of otitis externa presenting to Wellington Hospital, New Zealand, over a five-year period between 2007 and 2011. We also aim to evaluate current management of this condition and to recommend future treatment options.

Method
A five-year retrospective study, with data obtained from case notes and electronic records for all patients presenting with otitis externa to Wellington Hospital between 2007 and 2011.

Results
Of 347 cases identified, 144 were included in the study. *Pseudomonas aeruginosa* (*P. aeruginosa*) was the most common organism (46.5 per cent), while *Staphylococcus aureus* (*S. aureus*) was the second most common (31.9 per cent). Most patients received appropriate topical treatment. However, a significant number were treated with systemic antibiotics alone without adverse outcomes.

Conclusion
*Pseudomonas aeruginosa* is the most common microbe causing acute otitis externa in patients that require hospital level management in Wellington, New Zealand. In most cases, patients received appropriate topical therapy; however, it appears a large number received systemic antibiotic therapy without topical treatment. We recommend broad-spectrum topical antimicrobial therapy in all patients with uncomplicated AOE and culture-sensitive topical treatment with consideration of systemic antimicrobials for severe AOE requiring hospital admission.

Key Words
Acute otitis externa; microbiology; epidemiology

What this study adds:

1. What is known about this subject?
The aetiology, treatment, and organisms responsible have been described in overseas publications. Information regarding the epidemiology and management of this condition in New Zealand is not available.

2. What new information is offered in this study?
The microbiology and treatment in a New Zealand centre has not previously been described. This may be influenced by local population idiosyncrasies such as swimming habits, bacterial colonisation, health-seeking behaviours; prescribing practices of health practitioners; and pharmaceutical subsidies for the various available antimicrobials.

3. What are the implications for research, policy, or practice?
Primary and emergency physicians can be better informed in making decisions to treat or refer patients.
Background

Acute diffuse otitis externa and otomycosis are inflammatory conditions involving the external auditory canal and are usually associated with bacterial and fungal infections of the skin and subcutaneous tissue. Many factors contribute to the risk of developing otitis externa, including diabetes, pre-existing dermatitis, bacterial colonisation, exposure to external factors (swimming, trauma), and previous infections. The current gold-standard treatment is topical antibiotics in the form of ear drops, which may also contain topical steroids. Systemic antibiotics are reserved for severe or resistant cases such as patients that are systemically unwell, suffering complications (e.g., abscess, etc.), are immunosuppressed or have complete canal stenosis. The main aim of this study was to identify the microorganisms responsible in patients with clinical otitis externa requiring hospital level care in the Wellington region of New Zealand. We also set out to evaluate current management and determine whether appropriate treatment is being prescribed.

Normal management of uncomplicated acute otitis externa should consist of appropriate analgesia and keeping the ear absolutely dry. Broad-spectrum topical antimicrobials are to be prescribed if the external auditory canal is patent. If the canal is occluded by debris, aural toilet should be performed prior to topical agents being applied. Inflammatory occlusion of the canal should be treated with aural toilet, ear wick insertion, and topical antimicrobials. Patients with complications of otitis externa, e.g., pinna cellulitis, or those not responding to topical therapy should be started on systemic antimicrobials and be considered for acute hospital admission (Figure 1).

A literature review revealed no published data on the microbiology of otitis externa in New Zealand. There are several studies published overseas that have identified the major microbes responsible. Roland et al. published a large prospective study in the United States showing Pseudomonas aeruginosa (P. aeruginosa) (38 per cent) being the most prominent microbe, followed by Staphylococcus epidermidis (9 per cent), and Staphylococcus aureus (S. aureus) (7 per cent). They recommend using a broad-spectrum antibiotic that covers P. aeruginosa (e.g., ciprofloxacin) due to the wide range of microbes isolated in their study. A prospective British study published in 2010 identified pathogens in acute otitis externa cases referred to the acute otolaryngology clinic at a large teaching hospital over a six-month period. Out of 144 cases, P. aeruginosa was isolated in 45.1 per cent of cases while S. aureus was isolated in 9 per cent. Their recommendations were for treatment with topical agents that contained polymixin B, gentamicin, or ciprofloxacin due to the relative resistance of Pseudomonas to other antibiotics. A Cochrane systematic review concluded that topical treatments alone are sufficient to treat uncomplicated otitis externa; the choice of topical treatment does not significantly alter outcomes, with the only exception being that acetic acid was not as effective as antimicrobial agents. We aim to identify the major pathogens responsible for otitis externa presentations to a New Zealand hospital and to determine if current practice is consistent with the Cochrane review recommendations.

Method

All cases coded as otitis externa by the medical record stream that presented to the Emergency Department, the Ear Nose and Throat (ENT) surgery outpatients clinic, or were admitted to Wellington Hospital were included in the study. Of these, all cases where microbiology samples were not taken were excluded from the audit. Cases of pinna cellulitis, pre- or post-auricular abscesses without external auditory canal involvement were also excluded as these were considered complicated cases. Their management would normally consist of systemic intravenous antibiotics without topical therapy and surgical drainage of any collection (Figure 2).

Results

The Medical Coding Department identified a total of 347 otitis externa presentations at Wellington Hospital between January 2007 and December 2011. Sixty-one of these were excluded as there was an additional diagnosis made at the time (e.g., pinna cellulitis or an abscess); 85 cases were identified and excluded as being recurrent presentations (42 represented to the ENT clinic and 43 to the Emergency Department); and 57 cases were considered minor infections and did not have microbiology samples taken by the treating clinician (excluded). Five cases were incorrectly coded as “swimmer’s ear” (acute diffuse otitis externa). However, these were patients presenting to the Emergency Department with ear pain, tinnitus, or blocked ears after swimming without any signs of infection, i.e., discharge or erythema (excluded). The remaining 144 cases were included in the audit. Sixty-two (43 per cent) were males and 82 (57 per cent) were females (Table 1).
Table 1: Presenting age distribution

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–5</td>
<td>14</td>
</tr>
<tr>
<td>6–15</td>
<td>11</td>
</tr>
<tr>
<td>16–30</td>
<td>27</td>
</tr>
<tr>
<td>31–45</td>
<td>51</td>
</tr>
<tr>
<td>46–65</td>
<td>26</td>
</tr>
<tr>
<td>&gt;65</td>
<td>15</td>
</tr>
</tbody>
</table>

Ethnicity
Seventy-five were NZ European, 33 Maori, 15 Pacific, and 21 belonged to other ethnic groups. Ninety-one cases presented with their first episode, while 53 had previous history of otitis externa. The majority of cases were unilateral, however, 19 had bilateral disease. Ninety-six cases were managed as outpatients, while 48 required admission.

Microbiology
Of the 144 cases, 18 patients had no growth from the swab. Ninety-one cases had a single organism identified and 35 cases had multiple organisms. *P. aeruginosa* was identified in a total of 67 samples (46.5 per cent), 41 of these were as a single organism and 26 were in combination with another pathogen. *S. aureus* was identified in a total of 46 cases (31.9 per cent), 26 as a single organism, and 20 in combination.

Of the 35 cases with multiple organisms grown, 12 samples had both *S. aureus* and *P. aeruginosa* growth. Other significant contributors to infection (either as a single organism or in combination with another) are as follows: yeast (not *Candida*)—14 cases (9.7 per cent); *E. coli*—six cases (4.1 per cent); *Enterococcus* species—six cases (4.1 per cent); *Candida albicans*—five cases (3.4 per cent); *Streptococcus agalactiae*—five cases (3.4 per cent) (Table 2).

Our audit also revealed that Sofradex ear drops were the most common first-line agent with 60 cases receiving this as initial treatment (41 per cent). Ciprofloxacin ear drops were second with 56 cases being initially treated (38.9 per cent). Maxitrol was initially used in eight cases (5.5 per cent), while Locorten-Vioform was initially used in six cases (4.1 per cent).

Twenty-three cases (15.7 per cent) required a second topical agent due to failure of the initial treatment. Ciprofloxacin drops were used as a second-line agent in 12 cases (8.3 per cent), while Sofradex was used as a second-line agent in four cases (2.7 per cent). The most notable finding was the widespread use of oral (systemic) antibiotics. Eleven cases (7.5 per cent) had systemic oral antibiotics as first-line agents, while an additional 47 cases (32.6 per cent) received concurrent topical and oral systemic treatment. Four cases (2.7 per cent) had severe systemic symptoms and received intravenous antibiotics. The most common oral agent used was ciprofloxacin with 28 cases (19.4 per cent); 15 received oral amoxicillin-clavulanate (10.4 per cent); and 10 received oral flucloxacillin (6.9 per cent).

Discussion
Acute otitis externa is a common disease affecting a significant proportion of the population. The prevalence of otitis externa varies between regions with a yearly rate of four per 1,000 in the US, 10 per 1,000 in the UK, and 12 per 1,000 in the Netherlands.\(^6\) It is seen in all age groups and is five times more common in swimmers.\(^6\) Our results do not identify any particular demographic group within the Wellington catchment as being at a significantly increased risk.

The results of this audit have clearly shown *P. aeruginosa* and *S. aureus* as major causative agents of otitis externa cases presenting to Wellington Hospital. The data is consistent with the findings of several other studies identified in the literature.\(^2,4,5\) Overall, *P. aeruginosa* was found in 46.5 per cent of patients, while *S. aureus* was found in 31.9 per cent. They were found concurrently in 8.4 per cent of cases, and again, this proves consistent with information published in the literature.\(^2\)

In New Zealand, treatment of otitis externa with antimicrobials usually comprises either Sofradex drops (dexamethasone + framycetin + gramicidin) or Ciproxin HC (ciprofloxacin + hydrocortisone). Other agents used include Kenacomb (gramicidin + nystatin + neomycin), Maxitrol ointment (neomycin + polymixin B + dexamethasone), and Locorten-Vioform (clioquinol + flumethasone). The choice of first-line treatment depends on clinician preference and experience, financial considerations of the patient, and previous patient resistance to antibiotics. Many primary care clinicians will treat otitis externa without microbiology results as most will resolve with the above treatments. It is difficult to determine which patients will be refractory to topical treatment. However, it seems logical that patients requiring assessment and management in the hospital setting should all have microbiology swabs obtained as these cases are at higher risk of complication and antibiotic resistance.
The New Zealand Pharmaceuticals Management Agency (PHARMAC) is the government agency responsible for determining which medications are subsidised for use in New Zealand. The cost of each drug to the patient depends on the subsidy provided by PHARMAC for that particular drug (Table 3).

### Table 3: Cost of the most commonly prescribed pharmaceuticals

<table>
<thead>
<tr>
<th>Drug</th>
<th>Subsidy</th>
<th>†Drug cost to patient (per pack)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sofradex (8ml)</td>
<td>Partially subsidised</td>
<td>$8.23</td>
</tr>
<tr>
<td>Ciproxin HC (10ml)</td>
<td>Not subsidised</td>
<td>$28.12</td>
</tr>
<tr>
<td>Kenacomb (7.5ml)</td>
<td>Fully subsidised</td>
<td>$0.00</td>
</tr>
<tr>
<td>Maxitrol eye drops (5ml)</td>
<td>Fully subsidised</td>
<td>$0.00</td>
</tr>
<tr>
<td>Locorten-Vioform (7.5ml)</td>
<td>Fully subsidised</td>
<td>$0.00</td>
</tr>
</tbody>
</table>

† Pharmacy dispensation fee is not included in patient costs and prices may vary between pharmacies in New Zealand.

The treatment of uncomplicated acute otitis externa, as recommended by the Cochrane Research Group, consists of appropriate analgesia along with broad-spectrum topical antimicrobial agents. Systemic treatment should be reserved for patients with complications or systemic symptoms. The main advantages of topical therapy over systemic therapy are: higher concentrations of antibiotic can be achieved through topical treatment when compared to systemic treatment; topical therapy has minimal systemic effects or side effects; and prevents formation of resistant organisms in other parts of the body. Our audit concluded that the vast majority of cases received appropriate topical therapy with either Sofradex or Ciproxin. However, a significant number of simple cases, i.e., those without significant occlusion of the external auditory canal, were given first-line oral systemic treatment in addition to topical therapy. Five cases received oral antibiotics without concurrent topical treatment. There was no reported failure of treatment or other complication reported in these cases.

In total, 40 per cent of cases involved in the audit received oral antibiotics as part of their treatment. This number far exceeds our initial expectations and may be due to prescriber preference or lack of awareness of current otitis externa prescribing recommendations.

All of the four cases that received intravenous antibiotics were admitted to the hospital with signs of systemic infection; i.e., fever > 38.0°C with or without tachycardia. Their treatment was appropriate given the severity of their respective infections. Of the 11 cases that received first-line oral systemic antibiotics, eight were initiated by Emergency Department staff and only one had a documented reason for this choice. The other three cases were started on systemic treatments by the ENT registrar due to concerns regarding systemic infection.

A limitation of this study is that a large number of eligible participants had to be excluded as microbiology swabs were not taken at the time of presentation. Hence, the results may be skewed towards cases that were considered severe enough for swabs, and therefore may have overrepresented certain more aggressive pathogens. Given the need for secondary care, we would recommend all patients have microbiology samples taken. These cases are likely to represent more complicated infections and may be caused by resistant organisms.

The audit covered both emergency presentations and specialist ENT clinic reviews—because these involve providers from different specialties, their investigative practices, knowledge base and treatments are likely to differ. Another factor that may confound the data is that Ciproxin HC ear drops are not subsidised in New Zealand by PHARMAC, while Sofradex drops are. Thus, patient affordability may have played a role in the choice of medication prescribed.

### Conclusion

Acute otitis externa presentations to Wellington Hospital are most frequently caused by *P. aeruginosa* and *S. aureus*. These findings are consistent with internationally published data. A large number of patients were prescribed systemic antibiotics as a first-line treatment. Current best practice for treatment of uncomplicated acute otitis externa should be restricted to analgesia and broad-spectrum topical antimicrobials with or without steroids. A targeted therapy may be used once a microbiological diagnosis is made.

### References


PEER REVIEW
Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST
The authors declare that they have no competing interests.

FUNDING
No sources of funding to declare.

ETHICS COMMITTEE APPROVAL
Formal ethics approval not required as per Wellington Regional Ethics Committee guidelines as no new information was collected, personal details remained confidential, and treatment regimen was not changed. Refer to: http://www.otago.ac.nz/research/proposals/otago004486.html
Figure 1: Flow chart outlining the treatment of uncomplicated acute otitis externa

Patient with acute otitis externa. Treat with analgesia and advise about keeping ear dry.

Any signs of canal occlusion

No

Yes

Treat with broad-spectrum topical antimicrobial agent

Occluded by debris? Or Occluded by inflammation and oedema?

Debris

Treat with aural toilet and then with broad-spectrum topical antimicrobial agents

Oedema

Treat with aural toilet, ear wick placement and topical antimicrobial agents. Consider systemic therapy
Figure 2: Inclusions and exclusions

347 presentations identified as otitis externa between January 2006 and December 2011.

- Excluded due to additional diagnoses
  - 24 excluded due to Pinna Cellulitis
  - 30 excluded due to abscess
  - 2 excluded due to non-infective cause of inflammation
  - 5 excluded due to swimmer’s ear

286 presentations identified as uncomplicated

- Excluded due to recurrent presentation
  - 43 excluded due to repeat presentation to the emergency department
  - 42 excluded due to repeat presentation to the acute ENT clinic

201 presentations identified as uncomplicated cases presenting for the first time

- Excluded for clinical reasons
  - 57 excluded due to the treating clinicians considering the case mild; adequately treated in the community; and not requiring microbiological diagnosis

144 presentations fulfil both inclusion and exclusion criteria and were included in the audit
Table 2: Frequency of cultured pathogenic organisms in included cases of otitis externa

<table>
<thead>
<tr>
<th>Single organisms</th>
<th>Number</th>
<th>%</th>
<th>Multiple Organisms</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>P. aeruginosa</em></td>
<td>41</td>
<td>28.4</td>
<td><em>P. aeruginosa</em> + <em>S. aureus</em></td>
<td>12</td>
<td>8.3</td>
</tr>
<tr>
<td><em>S. aureus</em></td>
<td>26</td>
<td>18.1</td>
<td><em>P. aeruginosa</em> + yeast</td>
<td>6</td>
<td>4.2</td>
</tr>
<tr>
<td>Yeast (not Candida)</td>
<td>7</td>
<td>4.9</td>
<td><em>P. aeruginosa</em> + enterococcus</td>
<td>4</td>
<td>2.8</td>
</tr>
<tr>
<td><em>C. albicans</em></td>
<td>3</td>
<td>2.1</td>
<td><em>P. aeruginosa</em> + <em>S. agalactiae</em></td>
<td>3</td>
<td>2.1</td>
</tr>
<tr>
<td><em>Proteus mirabilis</em></td>
<td>2</td>
<td>1.4</td>
<td><em>S. aureus</em> + <em>S. pyogenes</em></td>
<td>2</td>
<td>1.4</td>
</tr>
<tr>
<td><em>Streptococcus pyogenes</em></td>
<td>2</td>
<td>1.4</td>
<td><em>S. aureus</em> + group G streptococcus</td>
<td>2</td>
<td>1.4</td>
</tr>
<tr>
<td><em>Streptococcus pneumoniae</em></td>
<td>1</td>
<td>0.7</td>
<td><em>S. aureus</em> + <em>E. coli</em></td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Schwanella putrificiens</td>
<td>1</td>
<td>0.7</td>
<td><em>E. coli</em> + aspergillus</td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Group G streptococcus</td>
<td>1</td>
<td>0.7</td>
<td>Group G streptococcus + yeast</td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td><em>Haemophilus influenzae</em></td>
<td>1</td>
<td>0.7</td>
<td><em>S. aureus</em> + <em>S. agalactiae</em></td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Beta haemolytic streptococcus</td>
<td>1</td>
<td>0.7</td>
<td><em>S. aureus</em> + <em>E. coli</em> + <em>S. agalactiae</em></td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>1</td>
<td>0.7</td>
<td><em>P. aeruginosa</em> + <em>C. albicans</em></td>
<td>1</td>
<td>0.7</td>
</tr>
<tr>
<td>Enterobacter agglomerans</td>
<td>1</td>
<td>0.7</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td><em>E. coli</em></td>
<td>1</td>
<td>0.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspergillus</td>
<td>1</td>
<td>0.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acinobacter</td>
<td>1</td>
<td>0.7</td>
<td></td>
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</tbody>
</table>