done at the Public Health Agency of Canada since 2010. In 2017, information about *emm* types and antimicrobial resistance was available for 2417 (97%) of 2486 invasive GAS cases reported to the Canadian Notifiable Diseases Surveillance System.[4] *Emm*1 has been the most prevalent *emm* type in Canada since 2010, representing 449 (18%) of 2473 isolates tested in 2017.

In 2017, the Public Health Agency of Canada began an invasive GAS genomics initiative to support surveillance and outbreak investigations, and this collection now includes a wide distribution of *emm*, antimicrobial-resistance, and multilocus-sequence types. The collection is comprised of approximately 3000 genomes from cultures isolated since 2010 from all regions of Canada. We did a core SNP phylogenetic analysis at the Public Health Agency of Canada on 178 Canadian invasive GAS *emm*1 genomes and representative $M1_{UK}$ (n=16), $M1_{intermediate}$ (n=5), and $M1_{global}$ (n=17) genomes described by Lynskey and colleagues.[1] 17 (10%) Canadian isolates clustered with the representative $M1_{UK}$ genomes (appendix). The 17 Canadian genome sequences had the same 27 key SNPs, including the transcriptional regulator RofA mutations Met197Ile, Phe198Val, and Asp370Asn, as were found in the $M1_{UK}$ genomes. The Canadian $M1_{UK}$ isolates were collected between February, 2016, and June, 2019, and were broadly distributed across Canada.

Further investigations, including sequencing of additional historical *emm*1 isolates, proteomic studies, and a review of epidemiological data on clinical manifestations, disease severity, and risk assessments, are planned. The successful international dissemination of the $M1_{UK}$ clone with enhanced invasive potential supports the call for improved surveillance activities, both nationally and globally, to monitor the spread of such clones and provide a rapid response and support for public health interventions.

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Against the trend: a decrease in scarlet fever in New Zealand

We read with interest the study by Nicola Lynskey and colleagues[1] that the seasonal rise in scarlet fever in England in 2016, and the concomitant rise in invasive *Streptococcus pyogenes* (group A streptococcus) disease, was associated with an *emm*1 strain ($M1_{UK}$) with invasive potential. Scarlet fever notifications have risen in England since 2014, and the incidence has increased throughout Asia (China, Hong Kong, and Singapore) since 2009.[2] Apart from the 2016 emergence of $M1_{UK}$, contemporary scarlet fever outbreaks have been polyclonal and associated with multiple *emm*-types. Earlier this year, Walker and colleagues[3] reported the isolation of an epidemic *emm*12 GAS strain from scarlet fever patients in Australia.

Scarlet fever is not notifiable in New Zealand and hence population surveillance data are not routinely available. We extracted data on International Classification of Diseases for scarlet fever (code A38) from the national hospitalisation database between 2009 and 2018 for children aged 15 years and younger. We included all admissions with the specified code in any diagnosis column, and found 98% of cases coded as scarlet fever in the primary diagnosis column on discharge. By contrast with other settings, scarlet fever admissions decreased in 2012 and remained low for 4 years, before returning to baseline admission levels in 2016 (figure).

The observed decrease in scarlet fever hospital admissions coincides with a reduction in first-episode acute rheumatic fever incidence

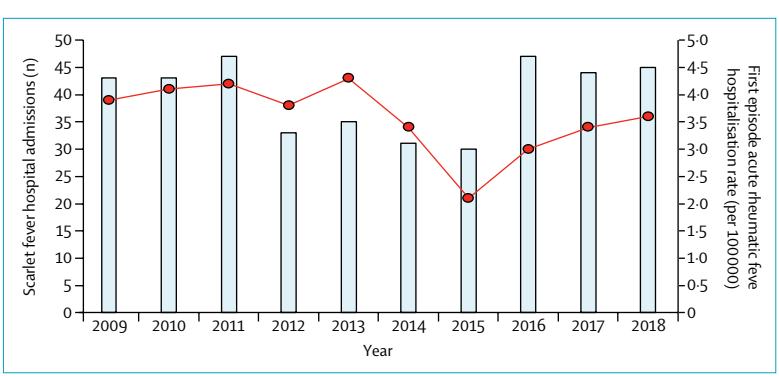

Figure: **Annual number of hospital admissions for scarlet fever in children aged 15 years and younger and hospitalisation rate for acute rheumatic fever in New Zealand, 2009–18**

See **Online** for appendix

in New Zealand, where Māori and Pasifika children have unacceptably high acute rheumatic fever rates. In 2012, a nationwide Rheumatic Fever Primary Prevention programme[4] was launched, which was an unparalleled 5-year effort to reduce acute rheumatic fever by treating *S pyogenes* pharyngitis in children at high risk. Programme coverage peaked in 2014, with more than 250 schools and 53 000 children enrolled, when a reduction in acute rheumatic fever was observed. However, this reduction was not sustained, and from 2016 onwards the incidence increased back to pre-programme levels. Despite this waning efficacy, our scarlet fever data suggest that the Rheumatic Fever Primary Prevention programme had a broader effect on serious *S pyogenes* disease in children in New Zealand. This effect was probably a consequence of intensive sore throat treatment and widespread public health awareness campaigns within the programme.

Worryingly, our data suggest that acute rheumatic fever rates are an indicator for scarlet fever in New Zealand. With acute rheumatic fever increasing, and our proximity to countries with epidemic scarlet fever, public health authorities need to be prepared for a scarlet fever outbreak. In a setting where *S pyogenes* incidence is already one of the highest in the world,[5] there is an urgent need to increase surveillance efforts and detect *S pyogenes* strains with invasive potential.

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Should we fear gonorrhoea?

Marcus Chen and colleagues[1] tested solithromycin for the treatment of uncomplicated genital gonorrhoea. They were unable to show non-inferiority of this relatively new macrolide antibiotic compared with standard treatment with ceftriaxone and azithromycin.

Similar to other molecules in development, solithromycin was largely expected to treat gonorrhoea in the context of global fear of antibiotic resistance. Indeed, *Neisseria gonorrhoeae* has been classified by the US Centers for Disease Control and Prevention as a threat with a high emergency level.[2] According to their 2013 report, 30% of *N gonorrhoeae* strains are resistant to antibiotics.

Instead of searching for new antibiotics, we should be looking at what we already know. Some old oral antibiotics with few side-effects could be investigated for the treatment of gonorrhoea.[3] An interesting alternative to new molecules could be fosfomycin trometamol. This molecule has recently been tested in a randomised trial.[4] 62 men received a single dose of fosfomycin trometamol on days 1, 3, and 5 after diagnosis (confirmation of Gram-negative diplococci on Gram strain of urethral secretion), and were compared with 61 men receiving standard treatment. 60 (97%) of 62 patients in the fosfomycin trometamol group were clinically and microbiologically cured 7 days after intervention, with no relapse at day 14. In France, this treatment is available at a cost of €4·66 a day and should be explored instead of new and costly molecules.

Information about the spread of multidrug-resistant *N gonorrhoeae* should be treated with caution. Antibiotic choice in each hospital should be ruled by local ecology and not by international communication. As an example, in October, 2019, we did data extraction for all strains of *N gonorrhoeae* cultivated in our laboratory at Institut hospitalo-universitaire Méditerranée infection (Marseille, France) using NexLabs (V01.32.S50; Technidata, Montbonnot Saint Martin, France). Between January, 2010, and September, 2019, 214 strains of *N gonorrhoeae* were isolated from clinical samples (mainly from the emergency and gynaecology departments). Of 170 strains with available antibiograms, no strain was resistant to ceftriaxone, and 101 (57%) strains were resistant to fluoroquinolones. According to these data, ceftriaxone (the reference treatment) is efficient in Marseille for the treatment of gonorrhoea. In case resistance should emerge, fosfomycin trometamol could be added to the standard treatment. This association has been tested in vitro with success against *N gonorrhoeae*.[5]

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