

ANNUAL REPORT OF THE AUSTRALIAN GONOCOCCAL SURVEILLANCE PROGRAMME, 2011

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Abstract

The Australian Gonococcal Surveillance Programme monitors antibiotic susceptibility testing of *Neisseria gonorrhoeae* isolates in all states and territories. In 2011, the *in vitro* susceptibility of 4,133 isolates of gonococci from public and private sector sources was determined by standardised methods. Varying antibiotic susceptibility patterns were again reported across jurisdictions and regions. Resistance to the penicillins nationally was 25%, and with the exception of the Northern Territory and Tasmania, ranged from 17% in South Australia and Western Australia, to 44% in Victoria. Quinolone resistance, most at high minimal inhibitory concentration (MIC) levels, was 27% nationally (except in the Northern Territory and Tasmania), ranging from 12% in the Australian Capital Territory to 40% in Victoria. Decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more), was found nationally in 3.2% of isolates, a decrease from 4.8% in 2010. There has not been an isolate of *N. gonorrhoeae* with a ceftriaxone MIC value greater than 0.125 mg/L reported in Australia. Nationally, all isolates remained sensitive to spectinomycin. Azithromycin surveillance was performed in the Australian Capital Territory; New South Wales; Queensland; Western Australia; the Northern Territory and South Australia. Resistance was found in low numbers of gonococci, with MIC values up to 16 mg/L. The source and site of the isolates referred to the program varied by geographic location. In larger urban centres the ratio of male to female cases was high, and rectal and pharyngeal isolates were common in men. In other centres, and in rural Australia, the male to female ratio was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2012;36(2):E166–E173.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

The World Health Organization (WHO) estimates that 88 million cases of gonorrhoea (*Neisseria gonorrhoeae* infection) occur annually worldwide.¹ In Australia, the rate of gonorrhoea has increased from 35.8 per 100,000 in 2005, to 54.3 per 100,000 in 2011.² This increased rate of gonorrhoea was coupled with a global increase in the prevalence of

antimicrobial resistance (AMR) in *N. gonorrhoeae*. The potential impact of AMR on treatment outcome is a continuing and growing concern^{1,3} as effective antibiotic treatment is fundamental to disease control at the population level.³

Over time, the emergence of resistance to the penicillins, tetracyclines, macrolides and fluoroquinolone antibiotics has necessitated the removal of these agents from standard treatment regimens.⁴ This was followed by the replacement with extended-spectrum cephalosporin antibiotics (ESCs) as the recommended first line treatment for gonorrhoea in Australia and elsewhere.⁵ Unusually, but importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where extremely high disease rates persist.⁴

In large centres in urban Australia, AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.⁴ There are an increasing number of reports from overseas sources of treatment failures with orally administered ESCs.^{6,7} In Australia, oral ESCs are not available, therefore the injectable form (ceftriaxone) is recommended for use in high doses.⁵ No treatment failures have yet been reported following ceftriaxone treatment of genital-tract gonorrhoea. However there were 2 instances of failure of treatment of pharyngeal gonorrhoea after treatment with ceftriaxone 250 mg intramuscularly reported in Sydney where elimination of intercurrent genital-tract infection with the same organism was achieved.⁸ The gonococci involved both had raised minimal inhibitory concentrations (MIC values) for ceftriaxone.

Strategies for treating and controlling gonorrhoea are based on single dose regimens effecting a cure in a minimum of 95% of cases. The formulation of these regimens is reliant on data derived from continuous AMR monitoring of gonococci to the antibiotics in clinical use.^{3,9} Recently, and following the reports of treatment failures with orally administered extended-spectrum cephalosporins,^{6,7} calls have been made internationally for enhanced surveillance of all forms of gonococcal AMR in order to optimise gonococcal antibiotic treatment.^{1,10}

Since 1981 the Australian Gonococcal Surveillance Programme (AGSP) has continuously monitored the

susceptibility of *N. gonorrhoeae* making it the longest, continuously running national surveillance system for gonococcal AMR.¹¹ The emergence and spread of penicillin and quinolone resistant gonococci in major cities in Australia has been well documented.⁴ This analysis of AMR in *N. gonorrhoeae* in Australia was derived from data collated by the AGSP during the 2011 calendar year. It provides information regarding the gonococcal isolates showing resistance to multiple antibiotics including those with decreased susceptibility to ceftriaxone.^{4,12}

Methods

Ongoing monitoring of AMR in gonococci in Australia is performed by the AGSP through a collaborative program conducted by reference laboratories in each state and territory. The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each state and territory. This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible. Both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the numbers of organisms examined are provided in order to indicate the AGSP sample size.

Gonococci isolated in, and referred to, the participating laboratories are examined for antibiotic susceptibility to the penicillins; quinolones;

spectinomycin and third generation cephalosporins; azithromycin and for high-level resistance to the tetracyclines, by a standardised methodology previously described.^{11,13} The AGSP also conducts a program-specific quality assurance (QA) program.¹⁴

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory, which collates the results and provides individual feedback to each participating laboratory. Additionally, the AGSP collects data on the gender of the patient, and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates are included in analyses.

Results

Number of isolates

There were 4,230 gonococcal isolates referred to, or isolated in, AGSP laboratories in 2011, representing 35% of the 12,118 cases of gonococcal infection notified to the Australian Government Department of Health and Ageing National Notifiable Diseases Surveillance System (NNDSS) in 2011.² This was lower than the 40%-42% referred in 2008-2010.

The source and site of infection of these isolates are shown in Table 1.

Isolate numbers in 2011 increased from those reported in 2010 in most jurisdictions: Victoria (from 913), the Northern Territory (from 448),

Table 1: Source and number of gonococcal isolates, Australia, 2011, by sex, site and state or territory

Sex	Site	State or territory								Aust
		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Male	Urethra	21	689	304	453	74	4	499	271	2,315
	Rectal	20	248	2	73	27	1	228	24	623
	Pharynx	10	201	0	37	17	1	120	17	403
	DGI	1	2	4	3	0	0	0	2	12
	Other/NS	5	5	13	4	14	0	3	6	50
	Total	57	1,145	323	570	132	6	850	320	3,403
Female	Cervix	5	135	140	204	33	0	69	121	707
	Rectal	0	8	0	2	3	0	3	0	16
	Pharynx	0	41	0	5	2	0	13	5	66
	DGI	0	0	4	4	0	0	0	7	15
	Other/NS	2	3	5	4	5	0	2	1	21
	Total	7	187	149	219	43	0	87	134	826
Total		64	1,332	472	789	176*	6	937	454	4,230

DGI Disseminated gonococcal infection.

NS Not specified

* Total includes 1 isolate with unknown sex and unknown site for South Australia.

Western Australia (from 352) and the Australian Capital Territory (from 30). There was a decrease in isolates from Queensland (from 840) and Tasmania (from 11); and similar numbers were reported from New South Wales (from 1328), and South Australia (176).

Source of isolates

There were 3,403 isolates from men (80%) and 826 (20%) from women and 1 isolate where the gender was not recorded. This male to female (M:F) ratio was 4:1, lower than in 2010 and 2009 (4.7:1 and 4.4:1 respectively). Compared with 2010, the number of isolates increased in both men (from 3,381) and women (from 719).

There were 27 isolates from disseminated gonococcal infections. Twelve were from men (0.4% of all isolates from men), which was less than in 2010 (0.7%), and 2009 (0.9%). In females, there were 15 (1.8% of all isolates from women) isolates from disseminated gonococcal infections, a decrease from the 24 isolates (3.3%) referred in 2010 but an increase from the 4 isolates (0.7%) referred from females in 2009. The infected site was not specified for 32 isolates from males and 9 isolates from females. Isolates from urine samples were regarded as genital tract isolates.

Antibiotic susceptibility patterns

In 2011, 4,133 of the 4,230 gonococcal isolates (98%) remained viable for antibiotic susceptibility testing. These were examined by the AGSP reference laboratories for susceptibility to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics), azithromycin, spectinomycin and for high level resistance to tetracycline. As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

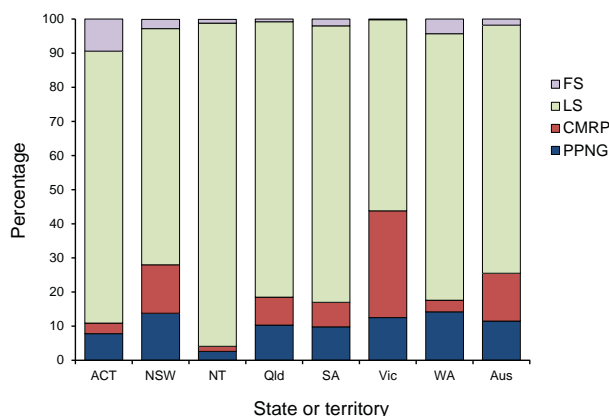
The categorisation of gonococci isolated in Australia in 2011 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to treatment with the penicillin group of antibiotics (penicillin, ampicillin and amoxicillin with or without clavulanic acid), are caused by penicillinase-producing *N. gonorrhoeae* (PPNG) and/or *N. gonorrhoeae* that are chromosomally resistant to penicillin (CMRP). Resistance in the PPNG group results from the production of beta-lactamase, and in the CMRP group by the aggregation of chromosomally-controlled resistance mechanisms.⁴ Chromosomal

resistance is defined by a MIC to penicillin of 1 mg/L or more.^{4,13} The MIC is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions. Infections with gonococci classified as fully sensitive (FS: MIC \leq 0.03 mg/L) or less sensitive (LS: MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Nationally, of those gonococci available for susceptibility testing 1,053 (25%) were penicillin resistant by one or more mechanism in 2011, a further decrease in the proportion of isolates resistant to this group of antibiotics recorded in 2010 (29%); 2009 (36%) and 2008 (44%). In 2011, there were 579 (14%) CMRP and 474 (11%) PPNG identified. In 2010 there were 17% CMRP and 12% PPNG demonstrating that the decrease in penicillin resistance nationally in 2011 was predominantly due to chromosomally mediated resistance, as was the case in 2008 and 2009; whereas in 2010 there was a decrease in the proportion of gonococci with both chromosomally mediated resistance and penicillinase production.

The proportion of all gonococcal isolates that were penicillin resistant was highest in Victoria with 44% (CMRP 31%: PPNG 13%) and New South Wales with 28% (CMRP 14%: PPNG 14%) (Figure 1).

Figure 1: Penicillin resistance of gonococcal isolates, Australia, 2011, by state or territory



- FS Fully sensitive to penicillin.
 LS Less sensitive to penicillin.
 CMRP Chromosomally mediated resistant to penicillin.
 PPNG Penicillinase-producing *Neisseria gonorrhoeae*

Proportions of resistant gonococci were lower than those reported in 2010 in New South Wales, Queensland, Western Australia, and South Australia. In Victoria, the proportions were essentially unchanged from 2010. There were 2 CMRP

identified in the Australian Capital Territory and 5 PPNG; and in Tasmania there were no CMRP and 3 PPNG. In the Northern Territory, there were 19 penicillin-resistant gonococci: 7 CMRP and 12 PPNG giving a total of 4.2% of strains that were penicillin-resistant in 2011; a higher proportion than in 2010 (3.6%), but similar to the proportion in 2006 to 2009 (4.2% in 2009; 3.9% in 2008; 4.1% in 2007; 4.6% in 2006).

Data on the place or mode acquisition were available for 86 (18%) infections with PPNG. Forty-one (8.6%) of the infections with PPNG were acquired locally, and 45 (9.4%) were acquired by overseas contact. These external contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand, The Philippines, Indonesia and Vietnam the most numerous. Additionally, disease was also reportedly acquired in Malaysia, Papua New Guinea, Singapore, Malaysia and, more widely, Ireland and the United States of America.

Ceftriaxone

From 2001 onwards, low numbers of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06–0.125 mg/L) have been found in Australia. The proportion has increased incrementally with the data from recent years showing a rise from 0.6% in 2006; 0.8% in 2007; 1.1% in 2008; 2.0% in 2009; to 4.8% in 2010. In 2011, a decreased proportion of isolates with decreased susceptibility to ceftriaxone was observed nationally: 134 of 4,129 (3.2%). There has not been an isolate of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L reported in Australia.

In Victoria, 50 of 937 isolates (5.3%) had decreased susceptibility to ceftriaxone; 58 of 1,322 (4.4%) from

New South Wales; 2 of 64 (3.1%) from the Australian Capital Territory; 18 of 789 (2.8%) from Queensland; 3 of 454 (0.7%) from Western Australia; and 2 of 470 (0.4%) from the Northern Territory. There were no isolates with decreased susceptibility to ceftriaxone reported from Tasmania.

In 2011, there was a decrease in the number of gonococci with decreased susceptibility to ceftriaxone compared with 2010, in all jurisdictions with the exception of the Northern Territory and Tasmania, as shown in Table 2.

Spectinomycin

Again in 2011, all isolates from all jurisdictions were susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by jurisdiction. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MIC values are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.¹³

Nationally in 2011, 1,132 of gonococci examined (27%) had some level of resistance to quinolones (QRNG), a further decrease in proportion nationally of quinolone resistance from the 35% in 2010; 43% in 2009 and 54% in 2008. The majority of QRNG found in 2011 (1,099; 97%) had resistance at a higher level i.e. MICs \geq 1 mg/L and many of these had MIC levels of 8–64 mg/L, the same proportion reported in 2010.

Table 2: Number and per cent of gonococcal isolates with decreased susceptibility to ceftriaxone, Australia, 2009 to 2011, by state or territory

State or territory	2009		2010		2011	
	n	%	n	%	n	%
Australian Capital Territory	2	5.3	2	6.7	2	3.1
New South Wales	16	1.7	74	5.6	58	4.4
Northern Territory	1	0.2	1	0.2	2	0.4
Queensland	10	1.8	26	3.2	18	2.3
South Australia	9	5.3	19	11.6	1	0.7
Tasmania	0	0.0	0	0.0	0	0.0
Victoria	17	2.2	52	5.7	50	5.3
Western Australia	9	3.1	17	5.2	3	0.7
Australia	64	2.0	191	4.8	134	3.2

MIC value 0.06–0.125 mg/L

Figure 2: Percentage of gonococcal isolates less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2011, by state or territory



LS QRNG, MIC 0.06–0.5 mg/L

R QRNG, MIC 1 mg/L or more

In Victoria, 378 (40%) of all isolates examined were QRNG (2010: 44%); in New South Wales there were 453 (34%) (2010: 40%); in South Australia there were 37 (24%) (2010: 41%); in Western Australia there were 90 (22%) QRNG (2010: 40%); in Queensland there were 144 (18%) (2010: 28%); and in the Australian Capital Territory there were 12 (18%) (2010: 60%). In the other jurisdictions the numbers of QRNG remained low and essentially unchanged from 2010; with 16 (3.5%) in the Northern Territory and 2 QRNG from Tasmania.

Information on country of acquisition of QRNG was available for 167 (14.8%) of the 1,132 cases. Ninety-seven of these (8.6%) were acquired locally and 50 (3.7%) infections were acquired overseas with Thailand, The Philippines and Indonesia the most numerous. Additionally, disease was also reportedly acquired in Hong Kong, Singapore, Vietnam and South Africa.

Azithromycin

In 2011, data on azithromycin susceptibility was available from all states and territories except Victoria. Overall, nationally, the proportion of isolates exhibiting resistance (azithromycin MIC ≥ 1 mg/L) was low (1.1%) and the maximum MIC value recorded in Australia to date is 4 mg/L. MIC levels in azithromycin resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

High-level tetracycline resistance

High-level tetracycline resistance *N. gonorrhoeae* (TRNG) is used as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever, used for treatment of gonorrhoea in Australia. Despite the lack of use of this antibiotic group, the proportion of TRNG detected continued to increase between 2006 and 2009 (12% to 21%), and was stable in 2010 (21%). In 2011, there was a decrease in TRNG nationally with 733 of 4,129 isolates (18%) reported.

TRNG were present in all jurisdictions, with the highest proportion in Western Australia (107 TRNG, 26%); the Northern Territory (114 TRNG, 25%); Queensland (149, 19%); New South Wales (236, 18%) and South Australia (24, 16%). Lower proportions were present in Victoria (95 TRNG, 10%) and in the Australian Capital Territory there were 6 TRNG (9%) and there were 2 TRNG in Tasmania.

Discussion

The WHO recommendations for standardised treatment regimens for gonorrhoea are based on data from epidemiological surveillance of the distribution and extent of AMR in gonococci.³ An antimicrobial resistance rate of 5% or more in gonococci isolated in a general population is the 'threshold' for removal of an antibiotic from treatment schedules and substitution with another, effective, agent.^{3,15} Programs such as the AGSP are conducted to determine the proportion of antimicrobial resistance in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.^{3,5,9,13,15}

Surveillance producing quality AMR data, on a sufficient and representative sample of isolates,^{3,13,15} is a pivotal part of a strategy for disease control; however the *in vitro* growth requirements and the fastidious nature of *N. gonorrhoeae* can complicate this process. In 2011, the strains examined by the AGSP were sourced from the public and private health sectors, constituting a comprehensive sample (35% of all notifications nationally) that meets these requirements, in spite of the increasing use of nucleic acid amplification testing (NAAT) for diagnosis of gonorrhoea in Australia, which does not provide AMR testing. The AGSP distributes reference panels for use in internal quality control and provides an External Quality Assurance Programme^{14,16} required for validation of gonococcal AMR data.

The overall number of gonococcal strains examined by the AGSP in 2011 (4,133) was higher than the number examined in previous years, but the proportion of isolates received from notified cases in Australia was lower than that examined in the

years 2008 to 2010, a reflection of the increase in the number of cases and the increase in NAAT testing. Isolate numbers in 2011 increased from those reported in 2010 in most jurisdictions except Queensland and Tasmania, and were unchanged in New South Wales and South Australia.

In 2011, 25% of gonococci nationally were resistant to the penicillins, and 27% to the quinolone antibiotics. These proportions were again reduced from those reported nationally in 2008 to 2010 whereas previously they had been increasing each year since 2003.⁴ In 2011, there were decreased numbers of gonococci with both chromosomally mediated resistance to penicillin and penicillinase production, but predominantly chromosomally mediated resistance. Aggregated data have shown a predominant clone of CMRP coupled with high-level quinolone resistance circulating with increasing frequency annually since 2003.^{4,12} It is possible that the continued reduction in resistance to both penicillin and the quinolones seen since 2008 continues to reflect a 'clonal shift' in gonococcal isolates.

The proportion of gonococci with high-level tetracycline resistance decreased in 2011 in Australia where there is a low level of exposure to these antibiotics.⁴

Evidence of the 'rural-urban divide'⁴ in gonococcal resistance was maintained, (Figures 1 and 2) underscoring the need for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions. Some remote areas with high disease rates continue to be able to use penicillin-based treatments, but effective use of this inexpensive and acceptable treatment is contingent on vigilant monitoring of resistance patterns.

The emergence and spread of gonococci with decreased susceptibility to the later generation cephalosporin antibiotics also referred to as ESCs has been documented in the AGSP reports.¹² These gonococci have also been found in rapidly increasing numbers in the WHO Western Pacific Region.¹⁷ In 'urban' Australia, the injectable agent ceftriaxone is now the standard treatment for gonorrhoea in public sector clinics, with the recommended dosage being 500 mg by intramuscular injection. This dose is higher than the 250 mg dose that is more commonly used throughout the Western Pacific Region¹⁸ but 500 mg is the smallest volume vial currently available in Australia.

Decreased susceptibility to the ESCs has been accompanied by increasing numbers of reports of treatment failures with the orally administered antibiotics in this group.^{7,18,19} To date, there have been

no strains of *N. gonorrhoeae* reported in Australia with a ceftriaxone MIC value greater than 0.125 mg/L; nor substantiated reports of treatment failure in genital tract gonorrhoea following ceftriaxone therapy. However, concerns are escalating locally and globally as increasing proportions of strains with decreased susceptibility are reported.⁵

In Australia in 2011, the proportion of gonococcal isolates with decreased susceptibility to the ESCs (3.2%) was lower than the 4.8% reported in 2010 but has markedly increased from the proportion reported in 2009 (2%). Surveillance to monitor *N. gonorrhoeae* with elevated MIC values coupled with sentinel site surveillance in high risk populations is critically important to inform therapeutic strategies and to detect instances of treatment failure. Sentinel site surveillance programs involve patient follow-up and test of cure (TOC) cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. This is currently being conducted in a very limited number of settings in Australia, and needs to be expanded throughout all jurisdictions as a matter of priority.

The mechanisms of resistance responsible for the MIC increases to ceftriaxone in gonococci include the presence of 'mosaic' *penA* genes in gonococci with raised ESC MIC values. The *penA* gene encodes penicillin binding protein 2 (PBP2), the major site of action of ceftriaxone, and mosaic PBP2 are altered to reduce this activity. Additional gene polymorphisms that affect antibiotic access to the organism complement these PBP2 changes and further increase ESC MICs.⁵ Of recent interest has been an extension of a study from 2001 to 2005 on the dynamics of the spread of mosaic PBP2-containing gonococci (mPBP2-GC) in Australia. Initial investigations suggested that mPBP2-GC found locally were also present in Hong Kong (where they were associated with treatment failure with an oral ESC, ceftibuten),⁷ and Japan.²⁰ Continuing studies in 2007 and 2008 showed that the subtypes of the mPBP2-GC present in Australia had altered markedly and that these strains had increased as a proportion of all gonococci tested.²⁰ Also of relevance have been local studies that showed that other non-mosaic lesions in *penA* were also responsible for increases in ceftriaxone MIC values similar to those found in mosaic PBP2 containing gonococci.²¹ These lesions were single nucleotide polymorphisms that represented mutations occurring in the *penA* of *N. gonorrhoeae*. This contrasted with the mosaic *penA* alteration that results from acquisition of 'foreign' DNA by the gonococcus.²² However, not all increases detected in ESC MIC levels can be explained by the molecular mechanisms described so far. This poses difficulties in developing reliable laboratory methods for the detection of ESC 'resistant' gonococci.

All gonococcal isolates tested in Australia in 2011, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci were found to be resistant to azithromycin in 2011. Recently, the United States Centers for Disease Control and Prevention and United Kingdom guidelines have moved to recommend a dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection.^{23,24} Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas. MIC levels in azithromycin-resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

The continued emergence and spread of anti-microbial resistance in *N. gonorrhoeae* is a global public health issue, and the evolution of this resistance is complex, and requires attention to both disease control strategies and rational use of antibiotics.^{10,25,26} Critically, disease control strategies and the understanding of the global scope of AMR are informed by surveillance programs of AMR nationally and internationally. Continuing commitment and vigilance to surveillance of AMR in *N. gonorrhoeae* means that maintenance of culture-based systems is crucial whilst surveillance is based on testing of gonococcal isolates.

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