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AMRHAI *News*

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In May, Lord Jim O'Neill published his final AMR report, which emphasized 10 key areas for urgent action and improvement. If anyone hasn't seen the report, it is available here. My secondment to his Review team was fruitful and immensely enjoyable. Elsewhere, there was a major development in Gram-negative resistance last November with the first description of plasmid-mediated resistance to colistin (and there are now three published *mcr* genes as I write this). Monitoring for colistin resistance is, however, confounded by the poor reliability of many routine antibiotic susceptibility testing (AST) methods; at present, EUCAST and CLSI only recommend microbroth dilution for colistin (see here). We found *mcr-1*-positive isolates in the UK by mining our genome data (see here), which emphasizes the importance that genomics will play in the future of AMR services. Also, through leadership of a EUCAST subcommittee, we delivered a comprehensive assessment of the potential for whole genome sequencing to replace AST for bacteria. SPOILER ALERT: it will take time, the evidence is still poor for many species! Finally, PHE's National Infection Service, of which AMRHAI is a part, is currently undergoing a major redesign with stepwise implementation scheduled to begin in 2017. So, more change is on the horizon. It's been a very busy year since our last AMRHAI Newsletter and I hope you'll enjoy this overdue issue.

Neil Woodford

Cardiac bypass heater-cooler units

The Infection Control section of AMRHAI has been part of the wider PHE investigation of bacterial contamination of cardiac bypass heater-cooler units (HCUs). A link between these machines and infections with *Mycobacterium chimaera* appearing months or years after cardiac surgery was identified by researchers in **Zurich**; they found the same species in HCU water and in the air around working machines. As the heating and cooling of blood on bypass is indirect, via an impermeable membrane, and the machines are kept well out of the sterile field, it is hypothesised that transmission is via contaminated aerosols produced by HCUs.

HCUs vary between manufacturer and model, but all have up to three joined tanks of water, filled from a common point. Water in individual tanks is either heated or cooled and circulated via replaceable flexible pipes to a cardiopulmonary bypass circuit. Even though the tanks are filled with water passed through bacteria-retentive filters, and the systems regularly emptied and disinfected, it seems inevitable that many of these unsealed systems will become polymicrobially contaminated over time. As much of this contamination will be in biofilms, it is intensely resistant to chemical disinfection and complete removal. Because the water will contract and expand as it is heated or cooled, the tanks cannot be hermetically sealed. The vigorous circulation of the water needed probably creates aerosols that can be ejected into the clinical environment via the machine's fans needed to cool compressors and motors. Such systems seem particularly prone to generating contaminated aerosols.

Guidance has been produced that, amongst other things, advises that the water in each device is microbiologically monitored; that decontamination and water change schedules advised by the manufacturers are implemented; and that the HCUs are positioned as far away from both surgical wound and exposed instruments as is practically possible. It seems as though this will remain the position until HCUs can be redesigned to minimise contamination and to allow effective decontamination of the water circuits. In the past, the same exercise was applied to the design and maintenance of endoscope washer-disinfectors, whose rinse-water circuits are also prone to bacterial contamination and resulted in improvement.

Guidance and investigation updates concerning the risk of mycobacterial infections associated with heater cooler units used in cardiothoracic surgery are available **here**.

Peter Hoffman

AMRHAI expands to include STIs

We are delighted to announce the creation of a new section within AMRHAI: AMRSTI – Antimicrobial Resistance in Sexually Transmitted Infections. AMRSTI is responsible for *Neisseria gonorrhoeae* isolates referred for identification to confirm anomalous results or for medico-legal purposes. In addition, we encourage all diagnostic laboratories to send us (i) confirmed isolates of *N. gonorrhoeae* with resistance to ceftriaxone and/or azithromycin (first-line antimicrobial therapy) for confirmation of antimicrobial susceptibility and (ii) isolates that have been related to clinical treatment failure.

The national **Gonococcal Resistance to Antimicrobial Surveillance Programme (GRASP)** and the international European Gonococcal Antimicrobial Susceptibility Programme (Euro-GASP) are both delivered by AMRSTI in collaboration with the STI/HIV department and ECDC/Orebro University, respectively.

AMRSTI is playing a leading role in investigating the high-level azithromycin-resistant *N. gonorrhoeae* outbreak and is involved in many research projects. We will be seeking to expand our work to investigate antimicrobial resistance in other STIs, to characterise bacteria causing STIs, and to expand AMRHAI's commercial work to include evaluations of new diagnostics for all bacterial STIs and new potential treatment options for *N. gonorrhoeae*. We'll keep you updated in future Newsletters. Please get in touch if you need any further information.

Rachel Pitt, Michelle Cole and Helen Fifer

Hypervirulent AND resistant *Klebsiella pneumoniae* – worrying developments

Community-acquired hypervirulent types of *Klebsiella pneumoniae*, especially clonal complex 23 of the K1 capsular type, which carry virulence genes associated with life-threatening invasive disease and typically cause liver abscesses and endophthalmitis, can affect healthy, often young, individuals. They are usually susceptible to most antibiotics, but we have received representatives of this type carrying NDM or OXA-48-like carbapenemases, with the NDM isolate (from a patient from Bangladesh) being susceptible only to colistin and tigecycline. This convergence of virulence and resistance could lead to untreatable invasive *K. pneumoniae* infections. We are not alone in observing this; KPC-positive and OXA-48-positive hypervirulent isolates have been described in the literature.

Representatives of *K. pneumoniae* ST147 (VNTR profile 5,3,5,14,14,2,4,2,1) are often associated with NDM, OXA-48, or both. We have noted virulence genes associated with hypervirulent types in a minority of isolates of this type. MinION sequence analysis of one of these suggests that it carries a large virulence plasmid carrying virulence factors such as *rmpA*, *rmpA2*, *iutA*, similar to those found in hypervirulent types. So it seems that not only are the virulent types acquiring resistance genes, but the resistant types are acquiring virulence genes. Please let us know if you suspect you have a resistant, hypervirulent isolate – we will certainly check it out!

Jane Turton

Elizabethkingia and mistaken species identity

In common with many of you, we used to rely on MALDI-ToF analysis, supported by biochemical analysis, to identify *Elizabethkingia* species, which often are identified with high scores using this method. But the recent outbreaks in the **United States** of *E. anophelis* (a species not previously included in our MALDI-ToF database), involving many deaths, prompted us to revisit some of our isolates. **Sylvain Brisse** of the Pasteur Institute, Paris - an expert on *Elizabethkingia* - showed from whole genome sequences of some of them that these were indeed *E. anophelis*, despite having originally been identified as *E. meningoseptica* by MALDI-ToF. We have therefore developed an *rpoB* sequence-based method for distinguishing species of *Elizabethkingia*. This can distinguish all the currently described species, with the exception of *E. anophelis* and *E. endophytica* (recently described from healthy wheat plants), which are very highly similar. We have also added the type strains of the missing species to our MALDI-ToF database and together with an expanded *Elizabethkingia* database kindly provided by John McQuiston who heads the CDC Special Bacteriology Reference Laboratory, Atlanta, we are investigating how reliably it can distinguish between the four species. Difficulty also arises when using 16S rRNA gene data currently available on GenBank, as many sequences are not reliable. However, with a verified 16S rRNA sequence database this improves species resolution and we are assessing the utility of all the methods above to identify *Elizabethkingia* species accurately.

Jane Turton and Julie Logan

Typing of opportunistic pathogens to aid outbreak investigations

Many isolates arrive in AMRHAI for 'typing' with insufficient or no indication of the reasons for the request, or as single isolates without other isolates of that species for comparison. We can no longer support this and **from April 2017 we will cease typing isolates submitted (singly or in multiples)** unless accompanied by clear justification, details of the comparisons sought and of the underlying question posed; we regret that 'ongoing surveillance' and 'vancomycin-resistant enterococcus' will not be considered sufficient. By introducing this streamlining to our services we hope to provide you with information for clear action. Many thanks to those who provide clear reasons – it really does help us to help you!

Jane Turton and Claire Perry

Ceftolozane-tazobactam reference testing

Ceftolozane-tazobactam was added to AMRHAI's antibiotic panel in July 2015, and was licensed for complicated urinary and intra-abdominal infections in November. A review of its

performance against 12-months' worth of referrals is underway but, as a pilot, we've analysed the first 10 weeks. Remember that referred isolates are sent owing to unusual resistance so you expect a lot of resistance. Nevertheless, they provide a snapshot of activity against the 'worst of the worst'.

Overall, 58.3% of referred ESBL-positive *E. coli* and *Klebsiella* were susceptible at EUCAST's 1 mg/L breakpoint *vs.* 98% of ESBL-positive *E. coli* and 81% of ESBL-positive *Klebsiella* in the **BSAC 2011-4 bacteraemia series**, which are consecutive unselected isolates. Ertapenem resistance was three-fold more frequent in ESBL producers that were ceftolozane-tazobactam-resistant than in ceftolozane-tazobactam-susceptible ESBL isolates (82.8% *vs.* 26.5%), implying that impermeability – the main source of ertapenem resistance in ESBL producers – can compromise ceftolozane-tazobactam too. MICs of 4-8 mg/L were seen for highly AmpC-derepressed *Enterobacter* (those with a cefotaxime MIC $\geq$32 mg/L); those with only partial derepression were often susceptible. Isolates with KPCs or metallo-carbapenemases were consistently resistant and, although 24/54 with OXA-48-like carbapenemase were susceptible to ceftolozane-tazobactam, 21 of these were susceptible also to unprotected ceftazidime.

The really impressive activity was for *P. aeruginosa*, where the modal MIC for the BSAC 2011-3 series was 0.5 mg/L, with 0.3% resistant (EUCAST, MIC >4 mg/L) *vs.* a 2 mg/L mode and 4.9% resistance for ceftazidime. Among 206 referred *P. aeruginosa*, 165 were susceptible to ceftolozane-tazobactam, including 41/80 isolates non-susceptible to **ALL** other penicillins, cephalosporins and carbapenems. Three-quarters of those that were resistant had metallo-, VEB, or GES β-lactamases – all uncommon in the UK. This association is so clear that, at AMRHAI's weekly review meeting, we now consider a ceftolozane-tazobactam MIC >4 mg/L – especially >16 mg/L – to predict an 'exotic' β-lactamase in *Pseudomonas*.

Since β-lactams are central to anti-pseudomonal therapy and since ceftolozane-tazobactam is the analogue most likely to be active i*n vitro* against problem strains, we anticipate that much early UK use will be microbiology-driven. As ever, off-label use should be decided on a careful risk/benefit basis. We hope that further clinical experience against *P. aeruginosa* will soon enter the public domain, as *P. aeruginosa* was poorly represented in the drug's Phase III trials. The manufacturer (MSD) has a *Pseudomonas*-relevant ventilator pneumonia trial in progress, using double the presently-licensed dose (2+1g q8h *cf.* 1+0.5g q8h) along with a **PK trial** in cystic fibrosis. So, watch this space.

David Livermore

Teicoplanin and coagulase-negative staphylococci

AMRHAI receives growing numbers of coagulase-negative staphylococci (CoNS) sent for confirmation of teicoplanin resistance. Most are from prosthetic joint infections and, most often, we confirm resistance, finding MICs of 8-32 mg/L vs. a 4 mg/L EUCAST/BSAC breakpoint.

Whilst these bacteria are a problem if teicoplanin is a hospital's preferred glycopeptide, their investigation is not public health microbiology. As early as 1990, **Vedel et al**. observed that a quarter of CoNS in Paris were teicoplanin-resistant. More recently, from 2009 to 2013, **BSAC bacteraemia surveillance** found 15-38% resistant, without trend. By contrast, vancomycin resistance rates in CoNS have remained <1%, as has teicoplanin resistance in *S. aureus*. The basis of these differences is uncertain, but may relate to whether or not teicoplanin's

lipid 'tail' can associate with the staphylococcal membrane, a mechanism not implicated for vancomycin.

Laboratories should **not** submit CoNS to AMRHAI for investigation of teicoplanin resistance. Treatment should be guided by results of local testing with gradient strips, though it should be remembered that in prosthetic device infections CoNS grow as biofilms, which are likely to be far more recalcitrant than suggested by any susceptibility test. **From April 2017, AMRHAI will stop testing teicoplanin against CoNS, unless specifically requested, in which case it will be done as a charged service.** We remain happy to receive and test any *S. aureus* suspected of teicoplanin resistance as well as any staphylococci, regardless of species, suspected of resistance to vancomycin, daptomycin, linezolid, tigecycline, oritavancin or dalbavancin.

David Livermore

Challenges of quinolone susceptibility testing in non-typhoidal *Salmonella*

The EUCAST clinical breakpoint for ciprofloxacin against Enterobacteriaceae is 0.5 mg/L. **Guidelines** further advise that "There is clinical evidence for ciprofloxacin to indicate a poor response in systemic infections caused by *Salmonella* spp. with low-level ciprofloxacin resistance (MIC >0.06 mg/L). The available data relate mainly to *Salmonella* Typhi, but there are case reports of a similarly poor response with other *Salmonella* species". Whilst high-level quinolone resistance due to mutations within the chromosomal genes (*gyrA, gyrB, parC and parE)* are easily detected by screening with nalidixic acid (30 μg) or ciprofloxacin (5 μg) discs, neither approach will detect low-level resistance (ciprofloxacin MICs 0.125-1 mg/L), which is due to *aac(6')-Ib-cr*, *qepA*, *oqxAB* or variants of *qnr*, which collectively constitute plasmid-mediated quinolone resistance (PMQR) determinants.

The most reliable phenotypic method for inferring likely PMQR is to determine ciprofloxacin MICs by a dilution method. As this is impractical in most laboratories, EUCAST advises use of a pefloxacin (5 μg) disc and **Skov and colleagues** reported that these discs produce zone diameters of 6-26 mm (EUCAST method) for PMQR isolates vs. >26 mm for wild-type isolates. Ciprofloxacin and norfloxacin are the only fluoroquinolones that possess the side-chain targeted by the enzyme encoded by *aac(6')-Ib-cr*; pefloxacin is not affected and so will not detect this resistance mechanism. However, our WGS data indicate that *aac(6')-Ib-cr* is very rare in *Salmonella* (more so than in other Enterobacteriaceae). All treatment failures should have MICs ratified by the reference service.

Gauri Godbole and Robert Hill

Launch of WGS in AMRHAI

The costs and infrastructures involved in whole genome sequencing (WGS) mean that we will need to implement WGS in AMRHAI, as elsewhere in PHE's National Infection Service, in a step-wise fashion. Thanks to a host of bioinformaticians and scientists in Colindale, **we are all set to replace some of our molecular-based *S. aureus* reference services with WGS**. In the first instance, we will focus on services where WGS will enhance national public health surveillance, and enable the consolidation of complex workflows alongside time and cost efficiencies for AMRHAI.

We will use WGS to characterise MRSA from cases of invasive disease (bacteraemia), referrals for detection of antimicrobial resistance (AMR) genes, toxin gene profiling etc.

During the summer you will start to see some reports stating that the lineage, AMR and toxin gene profiles have been derived from WGS as opposed to PCR-based data. If you have any queries, please contact Angela Kearns.

Angela Kearns, Bruno Pichon, Michel Doumith

A case of mis-identification?: Expansion of the *S. aureus*-related complex

A recent **paper** describes the identification of two novel species of *S. aureus*: ***S. argenteus*** (from humans) and ***S. schweitzeri*** (from non-human primates in Africa). WGS studies have shown they form part of the **"*S. aureus*-related complex",** but are distinct phylogenetically.

Initially thought to be largely restricted to remote communities in Australia, *S. argenteus* has now been described in Asia, America and Europe. It is associated with a similar spectrum of disease to "classical" *S. aureus* and may be methicillin-resistant (*mecA*$^{+}$). *S. argenteus* lacks the carotenoid pigment that gives *S. aureus* its characteristic golden colour, so colonies appear white. Using conventional laboratory methods (Gram stain, coagulase, DNase, commercial agglutination, MALDI-TOF MS, Vitek, Phoenix, etc.), *S. argenteus* will be mis-identified as *S. aureus.*

S. argenteus is more reliably identified using molecular techniques such as MLST and *spa* typing. As reported **here**, we have noted a number of *S. argenteus* among the isolates referred to us as *S. aureus* (MSSA and MRSA). We have therefore updated our database to include this species, so you may receive reports from us stating "*S. argenteus*" as opposed to "*S. aureus*".

Angela Kearns and Sharla McTavish

Molecular characterisation of linezolid resistance in Gram-positives

Resistance to linezolid is rare (<1% of Gram-positive isolates), but is observed in enterococci and staphylococci. Until recently, linezolid resistance amongst isolates referred to AMRHAI was usually attributed to mutations (most commonly G2576T) within the chromosomal genes encoding 23S rRNA.

Back in **2012**, transferrable linezolid resistance conferred by *cfr* gene was first reported in the UK in staphylococci and enterococci from humans. *cfr* encodes a rRNA methyltransferase, which modifies the 23S rRNA causing resistance to phenicols, lincosamides, oxazolidinones, pleuromutilins and streptogramin A (PhLOPS phenotype). More recently, **Wang and colleagues** identified *optrA*, which encodes a plasmid-mediated ABC transporter that confers resistance to oxazolidinones and phenicols, in *Enterococcus faecium* and *E. faecalis* in humans and animals. *cfr* and *optrA* are a greater public health concern than mutational linezolid resistance since infection prevention and control measures may not successfully contain the linezolid resistance as these genes may transfer to other strains, species and genera.

Until recently, characterisation of linezolid resistance in AMRHAI involved detection of the G2576T 23S rRNA mutation and screening for the *cfr* gene. We have now implemented a multiplex PCR to detect both *cfr* and *optrA* genes, and this will be performed on isolates that AMRHAI confirms to be linezolid resistant. This multiplex will allow AMRHAI to monitor the emergence of plasmid-mediated linezolid resistance. Non-structured retrospective screening

has already identified the *optrA* gene in *E. faecalis* referred by two UK laboratories, leading to recent issue of a National Resistance Alert.

Danièle Meunier and Katie Hopkins

How to cap an escalating service for molecular detection of carbapenemases?

PHE's Toolkits for the management and control of carbapenemase-producing Enterobacteriaceae (CPE) focus on the early detection of patients colonised or infected with CPE in order to prevent onwards transmission. AMRHAI uses an **in-house RT-PCR** to screen for carbapenemase genes in all Enterobacteriaceae isolates submitted for investigation of carbapenem resistance (and in non-fermenters where presence of a metallo-carbapenemase is suspected). However, **with the increasing availability and use of commercial carbapenemase detection tests in NHS and private laboratories, the confirmation of carbapenemase production in Gram-negative bacteria is rapidly becoming a method for diagnostic microbiology laboratories rather than for reference laboratories.**

We have noted from the submission forms that accompany suspected carbapenem-resistant bacterial isolates referred to AMRHAI that an increasing number of diagnostic laboratories are implementing either in-house or commercial assays for detection of carbapenemase genes. We ask laboratories that use one of the **three commercial assays** that AMRHAI have previously evaluated, or those that have sufficient data to demonstrate that their in-house molecular assay demonstrates equivalent sensitivity and specificity to AMRHAI's in-house RT-PCR, to consider whether molecular detection of carbapenemase genes by AMRHAI is still required. We have already been in contact with some of you and have mutual agreement that AMRHAI will take in-house 'PCR positive' results at face value (we will still investigate those found negative locally in order to rule out presence of rare carbapenemases). If you no longer require AMRHAI confirmation of your local laboratory PCR results please notify me of your decision via **email**. Reducing the time we spend confirming your local PCR results will free up our staff time to follow up those isolates that require extra work to rule out an underlying carbapenemase and to pursue genuine reference investigation of carbapenemase producers.

Please note we would still appreciate if you referred your locally-confirmed carbapenemase-producers to us (using the web-based **Electronic Reporting System (ERS)**) as this will help to inform our understanding of the national epidemiology of these isolates. The ERS was upgraded in July with new features including the ability for laboratories to record local molecular test results and is the only method currently available for this locally-generated molecular data to be captured and inform regional and national trends. Following feedback from laboratories and Trusts over the past 12 months a reporting tool to enable users to produce reports for their laboratories and/or Trusts has also been added. Further details on the ERS upgrade can be found **here**.

Katie Hopkins and Dean Ironmonger

***Bartonella* clinical sample testing**

With the suspension of the *Bartonella* serology testing service provided by the Respiratory and Vaccine Preventable Bacteria Reference Unit (RVPBRU) there is still a requirement for *Bartonella* testing. The provision of 16S rRNA PCR and gene sequencing provided by the

Bacterial Identification Section, AMRHAI is available for testing normally 'sterile site' samples, such as tissue samples eg cyst, lymph node, aortic tissue etc., but **not** from **blood/serum**. For solid samples (eg tissues), please provide a small fingernail size piece of tissue in a sterile container and do not add any water or buffers; for liquid samples please provide ~ 200 µl (min 100 µl) in a sterile container. All samples must have the **M1 Microbial Identification Request Form** completed, ensuring the following: sample type is indicated as clinical, the source as other (eg lymph node tissue, aortic tissue, pus), the **safety question** is answered and a **separate** request form is used for each sample to be tested. Guidance for submission is detailed in the **PHE Bacteriology Reference Department User Manual**.

Julie Logan

Building capacity for antimicrobial stewardship in low and middle income countries

Nan Shetty represented AMRHAI's WHO Collaborating Centre for Reference & Research on Antimicrobial Resistance and Healthcare Associated Infections as **a trainer and mentor** at the WHO Training Course on Antimicrobial Resistance Surveillance and Stewardship, Acibadem Medical University, Istanbul, 9-12 November 2015.

For the past 2 years Nan has worked with WHO in several low and middle income countries (LMIC) to build capacity in setting up quality management systems to detect antimicrobial resistance and put in place clinical antimicrobial stewardship programmes. She has also worked with LMIC in Central Asia and Eastern Europe to facilitate developing National Action Plans to combat antimicrobial resistance as mandated by the World Health Assembly.

Training was provided to AMR focal points from countries in Central Asia and Eastern Europe (largely Russian speaking and with simultaneous translation into Russian). The training addressed the underlying rationale and basic principles of antimicrobial stewardship through a mix of short didactic presentations and interactive case discussions. Ways to both improve individual prescribing practices for common infections, as well as introduction of techniques to implement an institutional antimicrobial stewardship program, were presented.

Topics covered included: basic principles of antimicrobial therapy, common inpatient antimicrobial stewardship programme interventions, adapting an international/national guideline into an institution-specific clinical pathway, laboratory processes used to detect resistance and resistance mechanisms with clinical interpretation, and use of standardized EUCAST methodology so data are quality assured and comparable across countries.

Our WHO Collaborating Centre has played a leading role through the expertise of many of its staff members to build capacity and help reduce the emergence of AMR.

Nandini Shetty

And finally …AMRHAI publications.

I usually end our Newsletters by providing readers with a rather self-indulgent list of our publications since the last issue. If interested, you can find them by searching our senior staff (see below) on PubMed etc.

Suffice to say that AMRHAI continues to be hugely prolific, with David Livermore and me both listed in **Thomson Reuters Highly Cited Researchers 2015**. Thanks to my entire AMRHAI team and to our many collaborators who help to make this possible.

Neil Woodford

AMRHAI senior staff …for when you need advice.

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