Journal of Infection (2005) **50**, e1–e37

Abstracts of the Eleventh Conference of the Federation of Infection Societies, 2004

***These authors have been awarded a Young Investigator Award**

0163-4453/$30.00
doi:10.1016/j.jinf.2005.01.012

INVITED SPEAKERS' ABSTRACTS

S02

WHAT'S NEW RESPIRATORY VIROLOGY

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The last 5 years has seen an explosion in this field. New pathogens have been discovered, new diagnostic tests have been introduced, new vaccines are being trialed and antiviral drugs have been showing some success. The human metapneumovirus was initially thought to be as important as RSV but it is now thought to be not as common as RSV and that disease is milder. A recent observation indicates that HuMPV is usually only severe as part of a mixed infection. It is unusual in adults, though can cause outbreaks in the elderly. Coronavirus has hit the headlines in the form of SARS, but another example, NL63, was described this year. It is too early to tell if it is important, but it does account for a small proportion of previously undiagnosed respiratory tract infections. More information is becoming available on rhinoviruses; not only are they the commonest precipitating factor for asthma, but they are also found in exacerbations of COPD and are increasingly recognised as a significant cause of pneumonia. This includes the immunosuppressed. Influenza continually hits the headlines; South East Asia has had an epidemic of avian influenza which hopefully will not set up transmission chains in humans. Point mutation in influenza viruses have now been strongly associated with increased virulence in humans. On the diagnostic front, nucleic acid testing is becoming predominant and many laboratories expect to have stopped culturing respiratory viruses within 2–3 years. Nucleic acid testing is more rapid, significantly more sensitive and allows a greater number of pathogens to be detected than either culture or direct fluorescence. A full respiratory screen, including staff costs, is approximately 9 euros. Cidofovir is showing efficacy in adenovirus infections in the immunocompromised, although reducing immunosuppression is probably more efficacious. A new monoclonal antibody against RSV has its place. Vaccines are in trials for RSV and parainfluenza 3.

S04

CHANGING FACE OF EXTENDED-SPECTRUM β-LACTAMASES

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Until recently, extended-spectrum β-lactamases have largely comprised mutant TEM and SHV enzymes from nosocomial pathogens, principally *Klebsiella* spp. This pattern is now changing, with the rapid dissemination of CTX-M enzymes, a group of ESBLs previously prevalent only in South America. CTX-M ESBLs evolved via the escape of chromosomal β-lactamase genes from *Kluyvera* spp. to mobile DNA. Over 35 variants are known, split into 4 or 5 clusters. Different variants are proliferating in different parts of the world: CTX-M-2 in Argentina and Israel: CTX-M-9 in Spain, CTX-M-14 in China and CTX-M-15 in Europe. Despite this geographic variation, the consistent pattern is for *Escherichia coli* to be the main host and for CTX-M enzymes to occur in community as well as nosocomial isolates. Most producers are from urinary infections, but some are from bacteraemias. Prior to 2000, CTX-M producers were unrecorded from the UK but, in the past 18 months, ARMRL has received over 500 referred *E. coli* isolates with CTX-M enzymes from over 75 UK labs. These represent only a fraction of all producers: one Trust alone has had >350 infections due to producers. Most referred isolates have CTX-M-15 β-lactamase but a few have CTX-M-9 or other types. About 33% belong to one major strain (designated A) and a further 33% to four other strains, with all these five being of serotype 025 and maybe having a common ancestor; the remaining producers are clonally diverse. About 25% of producers are from GP patients, many of them elderly, with underlying disease and recent hospital contact. All are multi-resistant, with consistent susceptibility only to carbapenems, nitrofurantoin and fosfomycin; strain A is also susceptible to gentamicin. The spread of CTX-M enzymes has forced re-thinking of ESBL detection methods. Ceftazidime resistance -previously advocated a single indicator of likely ESBL producers-is inconsistent among CTX-M producers and it is additionally necessary to test cefotaxime. Alternatively, isolates may be screened for cefpodoxime resistance. Isolates resistant to any of these cephalosporins should then have confirmatory tests performed, seeking synergy between the indicator cephalosporin and clavulanic acid. The spread of CTX-M enzymes into community *E. coli* means that isolates from GP samples also need screening by these methods, not just nosocomial isolates.

S05

FIVE RECENT RANDOMISED CONTROLLED TRIALS THAT HAVE CHANGED THE WAY WE MANAGE SEVERE SEPSIS

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The Surviving Sepsis Campaign represents an international collaboration of 13 critical care, infectious disease and nursing organisations. The aim of the campaign is to increase awareness and improve outcome in severe sepsis. Earlier this year, more than 50 aspects of the management of the septic patient were reviewed with the aim of providing practical guidelines.[1]

This presentation focuses on five of the guidelines that have been shown in randomised controlled trials to reduce mortality.

1. Early Goal Directed Therapy (EGDT):[2] Earlier work showed that supra-normal haemodynamic and oxygen delivery goals applied late in the septic process had no impact on survival. Rivers and colleagues used 'normal' goals, applied for 6 h from admission to hospital. Patients with septic shock were randomised to receive either standard resuscitation end points (CVP 8–12 mmHg and MAP >65 mmHg) or standard end points in addition to the target of a mixed venous oxygen saturation of >70%. This was achieved using dobutamine +/− transfusion. The in-hospital mortality was 30.5% in the EGDT group and 46.5% in the control group.
2. Activated Protein C:[3] The PROWESS trial was stopped after interim analysis because of the survival advantage demonstrated for drotrecogin alfa (activated). Twenty-eight day mortality was 24.7% in the group allocated to receive drotrecogin and 30.8% in the control group.
3. Ventilation with Low Tidal Volumes:[4] The ARDS Network Trial was also stopped early as it demonstrated a lower mortality rate in those patients ventilated with tidal volumes of 6 ml/kg, compared against a more traditional 12 ml/kg (31% vs. 39.8%). Over-stretch of relatively normal alveoli is known to cause release of inflammatory cytokines and a perpetuation of the lung injury process.
4. Moderate Dose Corticosteroids:[5] Although high doses of corticosteroids are known to increase mortality, this multicentre RCT showed that synacthen non-responsive patients with septic shock fared better with low dose steroids compared against placebo.
5. Tight Control of Blood Sugar.[6] This study showed a nearly 4