E. coli and “coliforms”
Resistance to multiple antimicrobials in E. coli from blood cultures is increasing; co-amoxiclav - 28.9%, fluoroquinolones - 20.9%, 3rd generation cephalosporins - 12.6%.
• A significant amount of the increasing resistance to 3rd generation cephalosporins (& fluoroquinolones) is due to the spread of CTX-M-type ESBLs (Extended Spectrum Beta-lactamases).
• Resistance in other coliforms from blood cultures remains high. Collated resistance rates for all coliforms (including E. coli) that could guide empirical therapy are co-amoxiclav – 38.1%, fluoroquinolones – 22.4%, 3rd generation cephalosporins – 19.3%, gentamicin – 6.6%, and carbapenems – 0.2%.
• Resistance rates for coliforms isolated from urinary specimens are lower than for blood culture isolates, but also continue to rise. It is notable that resistance to trimethoprim in community urinary coliforms is 30.9%. This high rate reflects selective testing in the community, and trimethoprim continues to be the first-line empirical therapy for uncomplicated UTI in the community.

Streptococcus pneumoniae
Penicillin resistance in S. pneumoniae remains stable at 4.2% which is similar to other areas of the UK.

Campylobacter spp.
Resistance to ciprofloxacin remains high at 28.1% with wide local variability and rates up to 69.8% in some areas. Resistance to erythromycin (recommended therapy) remains modest at 3.4%.

* The antimicrobial resistance report for 2008/2009 is due to be published in spring 2010.

Targeted Surveillance: Characterisation of Enterobacteriaceae (COL) resistant to 3rd Generation Cephalosporins

Introduction: Resistance to 3GC is an increasing problem, with the emergence and spread of CTX-M and plasmidic AMPC-type β-lactamases. In Wales all COL are screened for susceptibility to cefpodoxime (CPD): 6% and 12% of urinary COL isolates from the community (C) and hospitals (H) are resistant to CPD respectively. This study aims to characterise the molecular mechanisms responsible for 3GC resistance in Wales.

Materials & Methods: 524 CPD-resistant urinary COL were collected from 11 laboratories across Wales. MICs for cefoxitin, cefotaxime, ceftazidime +/- clavulanate or clavuloxacin were determined by agar dilution according to CLSI methods to determine a resistance mechanism spectrum per species.

Results: 5% of strains collected were 3GC susceptible upon confirmatory testing. CTX-M was the commonest resistance mechanism in both H and C isolates (Fig. 1), was present in a number of species, and was responsible for 64% 3GC resistance in EC (Fig. 2). 1 EC had both CTX-M and CIT and 1 K had CTX-M and FOX. 58 (17%) EC were negative for all PCRs but had an AMPC-type phenotype, suggesting resistance caused by hyper-expression of chromosomal AMPC (Fig. 2). Of these 30% originated from H and 70% from C. The 3 EBC-containing K isolates were all from a single laboratory.

Conclusions: Resistance to 3GC is established both in hospitals and the community in Wales. CTX-M is the commonest mechanism. Plasmidic AMPC beta-lactamases are emerging, especially in the community.