SENSITIVE AND SPECIFIC DETECTION OF CARBAPENEMASE-PRODUCING CARBAPENEM RESISTANT *ENTEROBACTERIACEAE* IN THE ROUTINE MICROBIOLOGY LABORATORY WITH MODIFIED HODGE TEST AND CARBANP TEST

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Carbapenemase-producing carbapenem resistant *Enterobacteriaceae* (CP-CRE) are of major clinical and public health concerns due to the highly transmissible nature and the lack of therapeutic options. Rapid and reliable detection of CP-CRE in the routine clinical microbiology laboratory is critical for the timely optimisation of therapy and implementation of infection control measures. This study aims to evaluate the performance of the various phenotypic methods that are available in our laboratory.

312 carbapenem intermediate or resistant *Enterobacteriaceae* isolates were tested for carbapenemase production using the modified Hodge test (MHT), the CarbaNP test, the MAST Carbapenemase Detection Test (MASTDISCS D70C) and the KPC/MBL and OXA-48 Kit (ROSCO Diagnostica). The phenotypic test results were compared against molecular detection of the carbapenemase genes via polymerase chain reaction (PCR).

In our laboratory, the MHT had a sensitivity of 95.8% and a specificity of 87.7%. The CarbaNP test had a sensitivity of 96.6% and a specificity of 93.7%. Combining the MHT and CarbaNP test improved the sensitivity to 100%, with a positive predictive value of 97% and a negative predictive value of 100%. In conclusion, the combination of MHT and CarbaNP test represents the most sensitive and cost effective methods of CP-CRE phenotypic detection in our laboratory.

CHARACTERISATION OF A STAPHYLOCOCCAL TYPE IB PARTITIONING SYSTEM IN GRAM-POSITIVE HOSPITAL PATHOGENS

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Strains of methicillin-resistant *Staphylococcus aureus* (MRSA), coagulase-negative staphylococci (CNS), and vancomycin-resistant enterococci (VRE) that are resistant to multiple antimicrobial agents, are a major cause of hospital-acquired infections around the world and are responsible for significant morbidity and mortality. These Gram-positive pathogens commonly contain multiresistance plasmids that encode partitioning systems, which increase their segregational stability. Such systems contribute to the prevalence and spread of these plasmids, and effectively maintain resistance in the absence of selection. Three types (I-III) of partitioning systems are recognised based on centromere structure and type of motor/DNA binding protein.

Putative Type Ib partitioning systems which have been identified on resistance plasmids from clinical staphylococcal and enterococcal isolates, contain candidate centromere-like site(s), and *parA* and *parB* genes that are predicted to encode a deviant Walker ATPase motor protein and a ribbon-helix-helix DNA binding protein, respectively.

The overall aim of this study is to further characterise these Type Ib partitioning systems. In context of the staphylococcal system, chloramphenicol acetyltransferase assays have shown that the putative ParB protein regulates *par* operon transcription. Additionally, live-cell imaging via fluorescence microscopy has shown that a ParA-GFP fusion protein, which is functional in the absence of wild-type ParA, localises to the bacterial nucleoid.

RISK ASSESSMENT OF HUMAN INFECTION BY H9 INFLUENZA VIRUSES USING AN EXPLANT SYSTEM

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Aims: Humans are susceptible to many strains of influenza viruses including H9N2 which at present is the most widespread and prevalent influenza virus in poultry found in Asia. The aim of this study was to determine the ability of H9 viruses isolated from humans and poultry to replicate in human lung and bronchus explants.

Methods: Eight strains of avian and human H9 influenza virus were used: A/Duck/Shantou/2030/2001 (H9N1), A/Chicken/Hong Kong/NT449/2007 (H9N2), A/Chicken/Hong Kong/YU341/2008 (H9N2) and A/Chicken/Hong Kong/SSP117W/2009 (H9N2); and A/Hong Kong/1073/99 (H9N2), A/Hong Kong/2108/2003 (H9N2), A/Hong Kong/2269955/2008 (H9N2), and A/Hong Kong/464419/2009 (H9N2), together with a rgD190E mutant. Two reverse genetics viruses with surface H9 and internal H1N1 genes were also used. Fresh human lung and bronchial explants were used as previously published.

Results: Productive replication in bronchus *ex vivo* cultures was observed with 2269955/08 and 464419/09 while lung *ex vivo* cultures supported the replication of all human and avian strains. The rgD190E mutant showed no difference in replication at the bronchial epithelium but appeared to show a greater degree of infection and replication in lung.

Conclusion: The human explant system should be considered as a model for H9 infection and may reduce the need for ferrets and mice in risk assessment models.

PRIMARY AMOEBIC MENINGOENCEPHALITIS IN NORTH QUEENSLAND – THE DIAGNOSTIC CHALLENGES OF *NAEGLERIA FOWLERI*

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