MRSA tailors virulence mechanisms to the hospital setting

April 25 2012

(Medical Xpress) -- In the hospital environment where antibiotic usage is extremely high, it seems that healthcare associated methicillin resistant Staphylococcus aureus (MRSA) has cleverly adapted for survival.

The findings of new research by UCD scientists led by Conway Fellow, Dr Jim O'Gara from UCD School of Biomolecular & Biomedical Science indicate that the so-called 'super-bug', MRSA sacrifices virulence potential for antibiotic resistance.

When the pathogen Staphylococcus aureus becomes resistant to methicillin, it also alters the way in which it produces a biofilm. This coating by colonies of the pathogen can form on prosthetic devices implanted in patients for diagnostic or therapeutic reasons and cause infection.

Patients with implanted devices are typically in an intensive care setting in hospital and have lowered immunity. They are more susceptible to infection even from a less virulent MRSA.

Describing the study that was published earlier this month in *PLoS Pathogens*, Dr O'Gara said, "We introduced a methicillin resistance gene into pre-clinical isolates of S. aureus in the laboratory to produce a high level resistant form of the pathogen.

We worked with Professor Brendan Loftus in the UCD Conway Genomics Core to identify the genetic changes in this modified strain of

the pathogen using whole genome sequencing. These genetic changes led to biofilm development being mediated through an alternate pathway while also causing significantly reduced virulence in a murine model of device infection."

The findings indicate that MRSA has honed its arsenal of virulence mechanisms to suit the hospital environment favouring antibiotic resistant over virulence while retaining its biofilm forming capacity and using implanted medical devices in immune-compromised patients as the optimum route to infection.

Device associated infections are difficult to treat and also necessitate the removal of the device, which in itself is not a trivial procedure for the patient. Understanding the ways by which biofilms are produced is the initial challenge to developing therapeutics to treat staphylococcal biofilm infections.

This research funded the Health Research Board, IRCSET and Healthcare Infection Society (UK) was carried out in collaboration with research groups in the University of Bath, University of Nebraska Medical Centre and Harvard Medical School.

Provided by University College Dublin