MARJEN
EDUCATION SERVICES
ABN 87 170 789 310

Margaret Jennings (B.App.Sc.), 33 Stanley Avenue, Eltham, Victoria 3095
Tel/Fax: (03) 9439 2436 Mobile: 0404 088 754 Email: marjenes@optusnet.com.au

www.marjenes.com.au

2019 July TOPIC # 6 – Some relevant research findings from a recent microbiology (ASM) conference

Presented in June in Adelaide - the role of the gut microbiome in health and disease, inflammation in fungal infections in the immunocompromised as a predisposition to further infection and tolerance of S. aureus to chlorhexidine.

1. Dr Alan Landay Rush University Medical Centre, Chicago has found that in many HIV patients who had good CD4 T cell recovery and viral suppression there still existed persistent level of immune activation and inflammation. Viral rebound almost always occurs when therapy is interrupted and inflammation is reduced on initiation of therapy but still persists in viral suppressed patients. He found that changes in the gut microbiome in HIV subjects lead to persistent systemic inflammation especially with the loss of short chain fatty acid producing bacteria. He found that critical changes in the tryptophan catabolic pathway and its pro inflammatory profile contributes to non HIV co morbidities **especially cardiovascular disease diabetes and neurocognitive outcomes.** He concluded with a discussion of the current therapeutic paradigm for modifying the host microbiome with pre or probiotics and the prospective of fecal transplants in HIV. In addition he discussed repurposing drugs from other therapeutic areas such as diabetes.

2. Professor Luigina Romani University of Perugia Luigina Romani's field is antifungal immunity — her major interest is understanding its mechanism that might lead to the activation of protective and non-protective adaptive immunity. Fungal commensals coexist in a complex milieu of bacteria within the human body. Severe and recurrent infections tend to manifest more frequently within immunocompromised hosts and microbial dysbiosis, suggesting that the host/microbe environment could significantly influence the infection, the generation of colonization resistance and protective tolerance and the efficacy of therapyShe introduced the concept of protective tolerance, the basis of immunotherapy, the use of dendritic cells to develop fungal vaccine, the pathogenic role of inflammation in fungal infections, the discovery of tryptophan metabolites as potential antifungal strategies and, more recently, the use of functional genomics and metagenomics for predictive medicine. She prosed that the microbiota itself ought be researched for vaccination and therapy in fungal infections because the mechanistic role of gut microbes and their microbial metabolites underlying human infections and diseases remains unknown.

3. Collateral Damage: Biocide Use and the Co-Selection of Multidrug Resistant Staphylococcus aureus Glen P Carter et al - University of Melbourne, Parkville etc. S. aureus causes a variety of illnesses, ranging from skin and soft tissue infections through to pneumonia, endocarditis and bloodstream infections. Infection prevention and control (IPC) is critical for reducing the rates of S. aureus infection, with the use of biocidal agents such as chlorhexidine gluconate (CHG) being an important component of current IPC programs. Recent international guidelines recommend the universal use of biocides for skin decolonisation in "high risk" hospital patients to prevent HCAIs, including those caused by S. aureus. This has led to concerns about possible "collateral damage" associated with the increasingly widespread and indiscriminate use of biocides such as CHG in our hospitals. Of particular concern is the possibility that CHG use might be associated with the emergence of antimicrobial resistance (AMR). Here we have used whole genome sequencing and Markov network analyses to determine the collateral antimicrobial resistances associated with biocide tolerance genes in S. aureus. These analyses clearly demonstrate the genetic potential for biocide- mediated co-selection of AMR in S. aureus. Furthermore, using a combination of in vitro testing and clinically relevant skin infection models, we provide compelling experimental evidence to show that the use of biocides, including CHG, can rapidly coselect for the emergence of multidrug resistant S. aureus isolates.

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Microbiologist and Infection Control Consultant/Educator