Partner services are a longstanding component of public health efforts to control sexually transmitted infections (STI). However, they have not been a consistent part of HIV prevention efforts either in high-or lower-income nations. In many areas, partner services for HIV and other STIs have been administratively separated, and the goals of partner services have usually been narrowly conceived to concentrate exclusively on the diagnosis and treatment of sex partners. This is now beginning to change. New evidence suggests that HIV PS in high income nations may be less effective at finding new cases of HIV than previously believed, but could play an important role in linkage to care. In sub-Saharan Africa, HIV PS appears to be highly acceptable and effective.

This session will focus on new opportunities in the area of HIV PS. The speaker will review the following issues: (1) data supporting the efficacy of HIV partner services as an HIV case-finding tool in both in high and low-income nations; (2) cost and cost-effectiveness data on HIV PS; (3) evidence that PS for bacterial STIs can been used to promote HIV case-finding and engagement in care among persons with previously diagnosed HIV infection; and (4) outstanding research questions related to HIV PS.

S.07 - Bacterial virulence and host response

S07.1 INSIGHTS INTO MATERNAL GONORRHOEA: HUMAN PRIMARY CERVICAL AND AMNIOCHORIONIC EPITHELIAL CELL RESPONSES TO NEISSERIA GONORRHOEAE INFECTION

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[1,2]**J L Edwards**, [1]J A Gawthorne, [1]T J Edwards. *[1]The Center for Microbial Pathogenesis, The Research Institute at Nationwide Children's Hospital, Columbus, OH, United States; [2]The Department of Pediatrics, The Ohio State University College of Medicine, Columbus, OH, United States*

Bacterial infection is widely recognised as a factor contributing to adverse pregnancy outcomes (APOs). *Neisseria gonorrhoeae* infections continue to be a universal and intractable problem. In this regard, maternal gonorrhoea increases a woman's risk for APO by 6.5-fold. Bacterial infection is thought to trigger a pro-inflammatory response that initiates those processes involved in (preterm) human parturition. The ability of gonococci to invade and transcytose amniotic sac tissues, *in vivo*, is inferred from the ability to isolate gonococci from these tissues and from amniotic fluid. However, there are currently no data to indicate how gonococcal infection can result in APO, and a physiologically relevant human model of pregnancy that is amendable to scientific analyses has hindered elucidation of factors contributing to APO. Thereby, an understanding of gonococcal infection as it relates to human pregnancy, using *human* cell models of disease, could provide new insights into the pathophysiology of gonococcal disease and of APO as they likely occur *in vivo*. To this end, we investigated gonococcal infection under conditions reflecting normal pregnancy by using primary epithelial cells derived from the human cervix (i. e. pex cells) and amniochorionic membranes (i. e. pace cells) and by altering the combined concentrations of pertinent steroid hormones. Comparative, quantitative, infection assays indicated that gonococci adhere to and invade amniochorionic cells and tissue, which was further observed to occur by a complement receptor-mediated mechanism. We demonstrate that *N. gonorrhoeae* infection of pex and pace cells elicits the differential production of nitric oxide and complement proteins, as well as the specific matrix metalloproteases, prostaglandins, and cytokines thought to participate in triggering the onset of human parturition. Hence, we provide the first direct evidence to indicate a potential link between gonococcal infection and the induction of APOs.

S07.2 THE EXTRUSION PARADIGM OF CHLAMYDIA PATHOGENESIS

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K Hybiske. *University of California, Berkeley, Berkeley, CA, United States*

Chlamydia is the most commonly reported bacterial disease in the United States, and remains the leading bacterial cause of sexually transmitted infection, responsible for approximately 90 million new STI cases annually worldwide. Of particular concern is that infections with *C. trachomatis* can lead to severe medical complications in women, such as pelvic inflammatory disease and ectopic pregnancy. Alarmingly, there remain fundamental gaps in our understanding of *Chlamydia* pathogenesis *in vivo*, for example their natural course of infection in humans and why protective immunity is not established. To help address these questions, our laboratory has been interested in determining how *Chlamydia* disseminate within the host. Our original discoveries elucidated the mechanisms by which chlamydiae exit host cells *in vitro*. Surprisingly, *Chlamydia* possess two mechanisms for cellular escape that are mutually exclusive: (*i*) Extrusion, a packaged release of *Chlamydia* in which the vacuole pinches off and exits the cell within a membrane-encased compartment; this leaves the original host cell intact, often with a residual chlamydial inclusion. (*ii*) Lysis, a destructive process that is mediated by proteases and the sequential rupture of vacuole, nuclear and plasma membranes, culminating in the release of free bacteria. The maintenance of two discrete exit mechanisms underscores the fundamental importance of this process for intracellular pathogens such as *Chlamydia*. Extrusions are novel pathogenic structures that we hypothesise confer unique means of interacting with the host's innate immune system, enabling immune evasion and promoting tissue dissemination. To this end, we have recently illuminated key characteristics of chlamydial extrusions that allow direct infection of new cells and their engulfment by professional phagocytes. Bacteria within phagocytosed extrusions are protected from macrophage killing mechanisms for at least 8 h. These results have important implications for *Chlamydia* pathogenesis *in vivo*, including dissemination, transmission and the elicitation of immune responses.

S07.3 SURVIVAL STRATEGIES OF HAEMOPHILUS DUCREYI: ROLE OF TRANSPORTERS

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S D Rinker, **M E Bauer.** *Indiana University School of Medicine, Indianapolis, IN, United States*

During human disease, Haemophilus ducreyi leads a primarily extracellular lifestyle, in which the organism is under constant pressure from the immune system. To survive in this environment, H. ducreyi expresses multiple mechanisms that counteract various antimicrobial activities of innate immunity. Key among these is secretion of LspA proteins to prevent phagocytosis, allowing H. ducreyi to reside extracellularly. When phagocytes cannot engulf bacteria, they secrete granule contents, including antimicrobial peptides (APs) such as cathelicidin and defensins, to kill the pathogens extracellularly. APs bind and destabilise cell membranes to lyse bacteria. Our laboratory is studying two transporter systems that protect H. ducreyi from human APs, including cathelicidin LL37 and beta-defensins. To prevent lethal interactions between LL37 and the inner membrane, H. ducreyi utilises the Sap (sensitive to antimicrobial peptides) transporter, which takes up periplasmic LL37 for cytoplasmic degradation. By mutagenizing structural components of the Sap transporter, we have found a direct correlation between the effectiveness of Sap-mediated LL37 resistance *in vitro* and the contribution of the transporter to virulence in humans. Further, we found that H. ducreyi