Results *Treponema pallidum* binds both the rounded and spread morphologies of activated platelets via a polar tip structure, maintaining a firm tether under fluidic conditions. A lack of interaction between heat-killed *T. pallidum* and platelets confirmed specificity and identified heat-labile *T. pallidum* surface components as mediators of this interaction. Viability assays illustrated *T. pallidum* retained viability in platelet rich plasma for >3 days under these conditions.
Conclusion The demonstration in this study of (1) prolonged *T. pallidum* survival within human platelet rich plasma and (2) *T. pallidum*-platelet interactions indicates that platelets do not exhibit a direct antimicrobial effect on *T. pallidum* and that *T. pallidum* mediates a strong and specific interaction with human platelets. These findings may reveal a novel mechanism of host survival employed by this elusive pathogen.

O06.2 INITIAL INTERACTIONS OF HERPES SIMPLEX VIRUS WITH HUMAN SKIN DENDRITIC CELLS

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Introduction HSV2 initially infects the stratified squamous epithelium of the anogenital mucosa prior to entering nerve endings, resulting in lifelong latent infection of neurons in the dorsal root ganglia. We have recently reported that topical application of HSV-1 to the inner surface of human foreskin explants, simulating *in vivo* infection, infects epidermal Langerhans cells (LCs) which then emigrate into the dermis. Here they formed large cell clusters with dermal dendritic cells (DCs). HSV-expressing LC fragments were observed inside the dermal DCs/macrophages.
Methods To define the mechanism of this interaction, we isolated LCs and dermal DCs from large human abdominal skin specimens by flow sorting. LCs were infected with HSV2 and co-cultured with dermal DCs.
Results All infected LCs developed apoptosis and fragments of them were observed within the dermal DC cytoplasm. HSV infected LCs expressed several chemokines as RNA and protein, with corresponding receptors expressed on dermal DC subsets. These DCs also expressed several phagocytic/apoptotic receptors for phosphatidylserine. In genital herpes lesions the selective contact of CD8 T cells with one of three dermal DC subsets was observed. The distribution of CD4 T cells and contact with these DC subsets is eventually being studied.
Conclusion Thus, we conclude that a viral antigen relay takes place whereby HSV infected LCs undergo apoptosis and are taken up by dermal DCs by phagocytosis for subsequent antigen presentation, probably via different pathways for CD4 and CD8 T cells. As dendritic cells are key targets for the new generation of vaccine adjuvants these studies define potential cellular targets for mucosal vaccines.

O06.3 ANALYSIS OF THE *TREPONEMA PALLIDUM* PROTEOME FOR EVIDENCE OF HOST PROTEIN MIMICRY; IDENTIFICATION OF A MECHANISM FOR BACTERIAL PERSISTENCE AND ESTABLISHMENT OF LATENCY DURING SYPHILIS INFECTION?

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Introduction The causative agent of syphilis, *Treponema pallidum*, is a highly invasive pathogen that can establish lifelong latency. Experimental evidence generated by our laboratory demonstrates a subset of *T. pallidum* proteins exhibits mimicry of host proteins, a strategy that may be used by *T. pallidum* to evade detection by the immune system and establishment of latency. Here we analysed all *T. pallidum* proteins of unknown function to assess the complete repertoire of potential host protein mimics expressed by this stealthy and highly successful pathogen.
Methods Amino acid sequences of 327 functionally unannotated protein-coding genes from *T. pallidum ssp. pallidum* (Nichols strain) were submitted to the protein fold recognition server, Phyre2. For each *T. pallidum* protein, the 20 top-ranked template matches and structural models were obtained. To identify potential *T. pallidum* host protein mimics, we analysed the source organism and functions of all high-confidence template proteins used for modelling (confidence scores/90%; alignment coverage/10%).
Results High-confidence structural predictions were generated for 51% of *T. pallidum* proteins with no assigned function (167/327). Analysis of these 167 functionally unannotated proteins identified a range of *T. pallidum* proteins predicted to adopt structural folds similar to domains from host proteins central to the processes of homeostasis and self-recognition, including Toll-like receptors, extracellular matrix components, and proteins involved in cell-signalling, complement and blood coagulation pathways.
Conclusion Our analyses have identified a complement of potential host protein mimics within *T. pallidum*. This novel finding will provide significant insight into *T. pallidum* virulence mechanisms for mediating host attachment and subverting host recognition, thereby aiding establishment of persistent infection. Our results also illustrate the power of molecular modelling for enhancing our understanding of microbial pathogenesis and disease establishment for bacterial pathogens with unique proteomes.

O06.4 HIGHER LEVELS OF A CYTOTOXIC PROTEIN, VAGINOLYSIN, IN LACTOBACILLUS-DEFICIENT COMMUNITY STATE TYPES IN THE VAGINAL MUCOSA

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Introduction Bacterial cytotoxic proteins, such as vaginolysin (VLY) produced by *Gardnerella vaginalis*, are thought to be virulence factors that *in vitro* alter cell integrity and local immunity. VLY may play a significant role in bacterial vaginosis (BV), therefore we assessed whether *G. vaginalis* dominant