



James Mahony

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Biography

James Mahony is currently working as a Professor Emeritus in Pathology and Molecular Medicine at University of Toronto, Canada. He is teaching with in the faculty of health sciences includes medical microbiology/infectious diseases and pathology residency training programs, graduate course in clinical virology (MS763) and medical sciences. He completed his fellowship in Microbiology at American Academy of Microbiology as well as in Canadian College of Microbiology. He has decorated his carrier with several publication with local, international, industrial collaboration with Drs Mark Loeb, Jenny Johnstone, Marek Smieja, Peter Timms (Brisbane), Phil Hansbro (Newcastle, Australia), Lee Ann Campbell (Seattle), Theo Moraes (Toronto) and Luminex Molecular Diagnostics, Qiagen, Pro-L. The major focus area of his research is the pathophysiology of acute respiratory infections caused by specific viruses (influenza, RSV) and bacteria (*Chlamydia pneumoniae*, *P aeruginosa* and *C difficile*). One of the major focuses of his laboratory is the development of new antimicrobial agents for both respiratory viruses and bacteria. In addition to the development of novel therapeutics the other focus of his clinical research is in the areas of diagnostics.

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INTRANASAL VACCINATION WITH THE TYPE III SECRETION SYSTEM (T3SS) ANTIGEN BD584 REDUCES BOTH VAGINAL SHEDDING OF *CHLAMYDIA TRACHOMATIS* AND ASSOCIATED UPPER GENITAL TRACT PATHOLOGY

C*hlamydia trachomatis* infections are the most prevalent sexually transmitted bacterial infections in the world. The WHO has estimated that there are 131 million new cases every year and recently it has been shown that prior Chlamydia infection is associated with increased risk of ovarian cancer. With up to 90% of women and 50% of men having asymptomatic infections many infections go undiagnosed and untreated leading to complications in women including pelvic inflammatory disease (PID), tubal factor infertility and ectopic pregnancy. Public health programs, including screening, partner identification and treatment have failed to curb infection rates indicating the need for an effective vaccine. We have shown previously using a mouse challenge model that vaccination with the BD584 antigen protected mice against *C muridarum* and reduced both bacterial shedding in the vagina and upper genital tract (UGT) pathology. We have now extended these findings to investigate whether BD584 protects against *C trachomatis* infection. C57BL/6 mice were vaccinated intranasally with BD584 and CpG adjuvant (BD584/CpG) then challenged intravaginally with *C trachomatis*. BD584 vaccination elicited serum neutralizing antibody, vaginal antibody and cell-mediated immune responses consistent with a Th1 polarized immune response (INF γ , IL-17 and IgG2a/c:IgG1 antibody ratio). Vaccinated mice had reduced vaginal shedding and reduced UGT pathology (uterine horn dilation and hydrosalpinx) providing evidence that vaccination can protect against late sequelae following resolution of a *C trachomatis* infection. We will also present data on a novel delivery method for the BD584 vaccine involving genetically engineered bacteria. We are currently investigating the efficacy of the BD584 vaccine in a second animal model and if the vaccine is effective in the piglet model then these results would strengthen the rationale for the use of BD584 T3S proteins in a human vaccine and a phase I human trial.