Garry M Walsh, J Clin Cell Immunol 2014, 5:5
http://dx.doi.org/10.4172/2155-9899.S1.017

OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference and Exhibition on
Clinical & Cellular Immunology
September 29-October 01, 2014 DoubleTree by Hilton Baltimore-BWI Airport, USA

From neonate to adult: The role of environmental agents on airway epithelial cell function

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Immunologically mediated inflammation undoubtedly plays a central role in the airway inflammation characteristic of asthma but such inflammation alone does not explain many of the characteristic pathological features of asthma. There is increasing evidence that the epithelium of the asthmatic airways is structurally and functionally abnormal and critically contributes to the inflammatory response and re-modeling of asthmatic airways. In asthmatics the airway epithelium secretes a variety of cytokines, chemokines and growth factors (e.g., PGE2, IL-8, IL-1, IL-6 eotaxin, RANTES, TNF-α, GM-CSF, TGFα/β) with increased responses to pathogenic and environmental stresses (e.g., air pollution, viral infection, oxidative stress) compared to healthy subjects. We have a program of work that is examining the effect of a number of environmental agents (house dust mite antigen, side stream tobacco smoke, nanoparticles) on the secretory function of cultured airway epithelial cells from neonates, children and adults with or without asthma. We are also examining the modulating effects of established asthma therapy such as the leukotriene antagonist Montelukast. This presentation will cover the background to our studies and present data on our key findings to date.

Biography

Garry M Walsh gained his PhD (1992) from National Heart & Lung Institute, University of London, U.K. His work on the pathogenesis of respiratory and allergic disease spans 30-years with an emphasis on the role played by airway epithelial cells and eosinophils. He has over 160 peer-reviewed articles, invited editorials, reviews and book chapters. His publications have been cited over 3,800 times (h-index 37) and has been an invited speaker and/or chairman at over fifty international meetings. He is the Editor-in-Chief of *Therapeutics and Clinical Risk Management; Clinical Medicine: Therapeutics,* and the *Journal of Cell Death* and also a member of the editorial boards of 16 further journals.

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