



MEETING ABSTRACT

Open Access

Nicotinic augmentation of anti-inflammatory GSK3 β signaling

David A Scott*, Richard J Lamont, Akhilesh Kumar, Huizhi Wang

From 11th Annual Conference of the International Society for the Prevention of Tobacco Induced Diseases (ISPTID)

Athens, Greece. 9-11 December 2013

Background

Glycogen synthesis kinase 3 β (GSK3 β) has been shown to be a critical mediator of the intensity and direction of the innate immune system responding to bacterial stimuli. Stimulation of the anti-cholinergic anti-inflammatory system by tobacco alkaloids (nicotine; cotinine) leads to phosphorylation and inactivation of GSK3 β and, subsequently, to immune suppression. This presentation will review the tobacco-induced dysregulation of GSK3 β signaling and provide insight into the increased susceptibility of smokers to multiple bacterial diseases, including those caused by *Mycobacterium tuberculosis*, *Legionella pneumophila*, and *Neisseria meningitidis*. The extensive ongoing efforts to exploit GSK3 β for its therapeutic potential in the control of infectious diseases will also be reviewed.

Published: 6 June 2014

doi:10.1186/1617-9625-12-S1-A15

Cite this article as: Scott *et al.*: Nicotinic augmentation of anti-inflammatory GSK3 β signaling. *Tobacco Induced Diseases* 2014 12(Suppl 1):A15.

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit



* Correspondence: dascot07@louisville.edu
School of Dentistry, University of Louisville, Louisville, Kentucky, 40292, USA

