Bacteriology-2013

OMICS Group Conferences
Accelerating Scientific Discovery

International Congress on
Bacteriology & Infectious Diseases

November 20-22, 2013 DoubleTree by Hilton Baltimore-BWI Airport, USA

Ethyl alcohol metabolism by oral *streptococcus sp*. produces adherence, HPV16 entry and malignant phenotype changes in oral keratinocytes

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Abstract

Streptococcus sp. are identified in malignant and non-malignant tumors of the oral cavity and pharynx. Moreover, ethyl alcohol (ETOH) is a contributor to human oral keratinocyte (HOK) malignant transformation. Furthermore, Streptococcus sp., metabolize ETOH and synthesize acetaldehyde (AA) a DNA damaging agent to HOK. Consequently, there is an increased entry of human papilloma virus, subtype 16, HPV 16 (pseudovirus (PSV) in HOKs expressing furin convertase (FC).

In a pre-clinical assay we test this relationship to show HPV 16 targeting of HOK.

Exposure to ETOH (1.0%, v/v): Streptococci sp. (e.g., S.mutans (LT11), S. salivarius strains (110-1, 109-2, 101-7, 107-1, 101-1), S.gordonaii (V288, isogenic strain for alcohol dehydrogenase (ADH wild type, ADH null ABE)); but not Lactobacillus rhamanose (24-1, 25-2) adhere and increase expression of synedcans (1,4); APRIL and retinoid receptor □$_1$ in HOKs. Furthermore, as AA is produced; S. mutans adheres and Heparan binding protein-Histone like protein A (HlpA) is expressed with attachment eliminated by Heparanase treatment. ETOH exposure controls showed: Tetraethylthiuram disulfide (Disulfiram (1micro Molar), a acetaldehyde dehydrogenase inhibitor caused AA accumulation and enhanced PSV entry; while 4-methoxypyoralen (50 micro Molar) inhibitor of CYP2E1 and alcohol dehydrogenase activity or chloromethylketone (100 micro Molar) an inhibitor of a catalytic site for FC; depressed PSV entry. In this context, HOK biomarkers were: increased DNA damage ((bulky adduct, N^2-acetyl-deoxyguaninosine), proliferation (BrdU) and cell migration. Taken together these pre-clinical biomarkers provide a new insight into how ETOH and AA promote HPV 16 infection in human oral mucosa through a prior Streptococcus sp. infection.

Biography

Joel Schwartz has completed his D.M.D., at the age of 23 from Tufts University School of Dental Medicine; D.Med.Sc. at the age of 28 from Harvard University School of Medicine, and Oral Maxillofacial Pathology Residence at Harvard University School of Dental Medicine. Further post-graduate training was attained at Dana Farber Cancer Center. He is Professor of Oral Maxillofacial Pathology at University of Illinois at Chicago. He has published more than 100 papers in reputed journals and chapters in 16 books and serves as a reviewer for the NIH and a variety of journals.