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First Author: Jane McHowat
Saint Louis University 1402 S Grand St Louis, MO 63104
United States
Phone: 3143236118
jane.mchowat@health.slu.edu

First Author is a: Investigator
First Author is a member of: The American Physiological Society
First Author Degree: PhD, DSc, or equivalent

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Sponsor: Jane McHowat
Sponsor Phone: 31493236118
jane.mchowat@health.slu.edu
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PAF Expression in Skin Tumors of Smokers and Never Smokers: a Potential Role in Skin Cancer Development

Jane McHowat[1], Juan Carlos Lozano[1], Patricia Missall[2]. [1]Saint Louis University, St Louis, MO, [2]Dermatology, Saint Louis University, St Louis, MO

Significant progress has been made in the management and understanding of cancer, but there remains a large disparity in cancer health within our population. One of the most well-known cancer risk factors, cigarette smoking, maintains a disproportionate prevalence in populations distinguished by socioeconomic status, race, and gender. Previous studies have demonstrated that cigarette smoking is associated with changes in phospholipase A_2 (PLA_2)-mediated pathways that are implicated in cancer development and progression. More specifically, PLA_2 activation leads to increased platelet-activating factor (PAF) synthesis, a metabolite that is increased in cancer cells. PAF accumulation is further enhanced with cigarette smoke via inhibition of PAF-acetylhydrolase, the enzyme responsible for PAF hydrolysis and inactivation. Much remains unknown about the biochemical effects of smoking on the development of skin cancers including squamous cell carcinoma (SCC) and basal cell carcinoma (BCC). However, a recent meta-analysis study demonstrated that cigarette smoking is associated with a 52% increase in the risk for SCC, but does not appear to modify the risk for BCC. In order to assess the effects of cigarette smoking in these two skin cancers, we analyzed PAF expression in skin biopsies from six smokers and non-smokers with SCC, and six smokers and non-smokers with BCC. The biopsy samples were immunohistochemically stained with antibodies specific to PAF and PAF-receptor. Immunohistochemistry revealed positive PAF and PAF-R expression with nuclear localization in all patient samples, including SCC and adjacent normal sun exposed skin. There was no significant difference between the PAF or PAF-R expression in samples of smokers when compared to nonsmokers. However, immunohistochemical analysis demonstrated a significant difference in PAF expression between SCC and BCC cells. SCC cells expressed nuclear PAF, but PAF was not detectable in BCC samples. PAF signal was also not detectable in the basal cell epidermal layer in all patient samples. These findings support a biochemical difference in SCC and BCC cells in association with PAF, which is traditionally upregulated with cigarette smoking.