

CIGARETTE SMOKE EXPOSURE UPREGULATION OF PGE₂ EXPRESSION IN THE BLADDER

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Cigarette smoking remains the leading cause of preventable death and disease in the United States. Despite known risks, 42.1 million Americans continue to smoke, making research into tobacco-related pathologies relevant and necessary. We previously discovered that cigarette smoke exposure leads to tumor progression in the breast via platelet activating factor (PAF) and other phospholipase A₂ (PLA₂) metabolic pathways. These results led us to examine the PLA₂ metabolite, prostaglandin E₂ (PGE₂). We observed the expression of PGE₂ in mouse bladder and human heart tissue from nonsmokers and long-term smokers via immunohistochemistry. The urothelium of mouse bladder revealed a significant increase in expression of PGE₂ when compared to the nonsmokers via immunohistochemical analysis. In addition, we examined PGE₂ expression in cardiac biopsy patients and found no significant increase in expression. Our results suggest that while cigarette smoking may play a role in cancer of the breast and bladder, it may not have significant consequences in other pathologies such as heart disease. Our future studies aim at investigating other members of the PLA₂ family in bladder for similarities to tumorigenesis and progression in the breast. Overall, we have concluded for the first time that cigarette smoke exposure increases PGE₂ expression in the bladder following exposure. This provides a potential therapeutic target for cigarette smoked-induced tumorigenesis and progression.