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Alpha-enolase, a key glycolytic enzyme is up-regulated in cervical cancer cells exposed to reference-research cigarette and E-cigarette

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Cervical cancer is the third most common cancer in women worldwide. Cigarette smoking is considered to be a common risk factor for developing cancer of the cervix. A recent meta-analysis showed the risk of squamous cell cervical cancer doubled in women who currently smoke. However, because of the complex composition of cigarette smoke, the detailed path of physiological mechanisms is not fully understood. We decided to analyze the effects of soluble cigarette smoke on the viability of cervical cancer cells and proteomically explore its effect on the expression of various proteins in cervical cancer cells. Exposure of cervical cancer He-La cells to soluble reference-research cigarette smoke extract caused an increase in expression of a number of proteins, as identified by proteomic analysis. Alpha-Enolase, a glycolytic enzyme, known to be involved in tumorigenesis and metastasis, was significantly higher in cells exposed to soluble cigarette smoke extract when compared to untreated control cells. Alpha-enolase is a hypoxia-responsive gene; hence we explored the HIF pathway. We found a concomitant increase in Hif-1 α expression in He-La cells exposed to cigarette smoke extract through suppression of prolyl 4 hydroxylase activities. It is possible that cigarette smoke could be inhibiting prolyl 4 hydroxylase activities to stabilize and enrich Hif-1 α which in turn, is activating α -enolase. Pathway enrichment analysis of the interacting proteins of enolase resulted in glycolytic pathways, supporting enolase function in a cell. Induction of ENO1 genes might provide a survival advantage to cells under hypoxia and acidic environments generated by cigarette smoke. Simultaneously, we analyzed E-cigarettes (nicotine) effect on cervical cancer cells. E-cigarettes are being used as alternatives to traditional cigarettes to facilitate smoking cessation. Our ongoing study reveals that e-cigarettes also induce alpha-enolase levels in He-La cells to the same extent or higher than traditional cigarettes. Further studies on the role of enolases in smoking-induced progression of tumorigenesis needs to be evaluated.

Biography

Wasia Rizwani completed her PhD in Biochemistry in 2006 from Osmania University, Hyderabad, India. She has done her Post-doctoral fellowship and MD at Anderson-Orlando. She started her career as Research Scientist/Independent Investigator, Osmania University. Currently she is working as Principal Investigator in Osmania University, Hyderabad.

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