

Can Waterpipe Smoking cause Oral Cancer?

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The World Health Organization (WHO) study group on Tobacco Product Regulation had suggested a striking similarity between waterpipe smoking (WPS) and cigarette smoking. Several well-established carcinogens present in cigarettes like polycyclic aromatic hydrocarbons, volatile aldehydes, phenols, and heavy metals were also noted in WPS.¹⁻³ Further, the period of one WPS session is significantly higher than smoking one cigarette. This increased exposure time causes a potentially increased intake of smoke and other chemical components of WPS.² The difference in the dosage of WPS and cigarette smoking is so significant that the WHO has equated 1 WPS session to smoking more than 100 cigarettes.³

Sajid et al⁴ estimated the carcinoembryonic antigen (CEA) levels among waterpipe smokers and nonsmokers. The WPS users had a mean CEA level of 3.58 ± 2.61 ng/mL in comparison to 2.35 ± 1.15 ng/mL in nonsmokers. Mamtani et al⁵ published a meta-analysis in 2016 analyzing the relationship between cancer and WPS. The meta-analysis showed a strong correlation between WPS and cancer of the lung, head and neck, and esophagus. A systematic review conducted by Awan et al⁶ observed WPS to have a strong correlation with lung cancers, but other forms of cancers were found to show a relatively weak to no association. At present, the association between WPS and

oral cancer is poorly documented. Most studies exploring the effects of WPS and oral cancer have failed to account for the concomitant use of cigarette, tobacco, and alcohol.⁷ Thus, as these studies were biased, it was not possible to access WPS as an independent risk factor for oral cancer.⁸

It is well established that cigarette smoking can cause malignant changes in epithelial cells and several studies have shown cigarette and WPS to share several carcinogens.¹ In addition to this, as stated earlier, WHO has equated 1 WPS session to smoking 100 cigarettes or more.³ Based on the above data, it can be hypothesized that WPS is potentially carcinogenic to the oral mucosa. The future research goals in WPS include conduction of multicenter case-control and prospective studies with adequate sample size to test this hypothesis. To record exposure times to allow estimation of dose-response. Further, it is vital to conduct *in vivo* and *in vitro* studies to elicit the molecular changes in the oral mucosa from chronic exposure to WPS. Decoding the molecular biology of WPS induced carcinogenesis will provide early diagnostic markers and vital therapeutic targets.

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