[Does nicotine add to the carcinogenic strain of tobacco smoke?]

BACKGROUND: It is accepted that nicotine in tobacco smoke causes addiction via nicotinic acetylcholine receptors in the central nervous system. For a long time, the tumorigenic potential of smoking was attributed to compounds other than nicotine. However, more recently data have accumulated which suggest that nicotine may add to the cancer risk by stimulating cellular growth via non-neuronal acetylcholine receptors, by suppressing apoptosis, and by inducing angiogenesis not only in atheromatous plaques but also in tumors. In the present study the possible direct genotoxic effects of nicotine on DNA were investigated in human target cells of carcinogenesis in the upper aerodigestive tract.

PATIENTS AND METHODS: Human nasal mucosa, lymphatic tissue of the palatine tonsils, supraglottic epithelium of the larynx, and peripheral lymphocytes were exposed to rising concentrations of nicotine. DNA damage was investigated by alkaline single-cell microgel electrophoresis (Comet) assay. Cytotoxicity was assessed by trypan blue exclusion.

RESULTS: Nicotine induced dose-dependent DNA damage in all cell types at low cytotoxic concentrations that allowed viabilities well above 80%. The lowest nicotine concentrations eliciting a significant increase in DNA migration were 1 mM for tonsillar cells and 0.25 mM for all other cell types.

CONCLUSION: Nicotine induces genotoxic effects in human
target cells of carcinogenesis in the upper aerodigestive tract at relevant concentrations. Thus, nicotine may contribute directly to tumor initiation resulting from smoking.