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"The effect of nicotine inhalation on taste receptor gene expression and fungiform papillae density"

BACKGROUND: Smoking is one of many acquired causes of smell and taste disorders. The effect on taste may be due to cigarette smoke causing a gustatory disturbance through changing the form, quantity, and vascularization of taste buds. Nicotine's negative impact on taste buds is elicited through aversive sensory effects like oral irritation and pain as well as bitter taste. Taste buds are located on fungiform, circumvallate, and foliate papillae found on the tongue. Taste receptor cells can be stimulated by one of five basic taste qualities, which are sweet, bitter, umami (savory), salty, and sour. More recently, fat has been proposed as a sixth taste quality.

OBJECTIVES: The goal of this study is to determine the combined effects of inhaled nicotine and high fat diet (HFD) on bitter, sweet, and fat taste receptor gene expression and the effect of inhaled nicotine and HFD on the density of fungiform papillae at anterior portion of the tongue.

METHODS: Adult male C57BL/6N mice were used to investigate the combined effects of nicotine vapor inhalation and HFD on lingual taste receptor expression. The control group received room air and consumed a standard mouse chow diet. Other groups received: 1) room air and consumed a HFD, 2) nicotine via inhalation in an inhalation chamber and consumed a standard mouse chow diet, 3) nicotine via inhalation in an inhalation chamber and consumed a HFD. Following 10 weeks of inhalation exposure and diet consumption, mice were sacrificed, and tongues were harvested. Tri-reagent and a Zymo RNA kit were utilized to isolate RNA from the circumvallate papillae and followed by reverse transcriptase and qPCR. Expression of CD36 mRNA (fat), T1R2 mRNA (sweet), T1R3 mRNA (sweet), and T2R138 mRNA (bitter) was measured and normalized to cyclophilin. To determine density of the fungiform papillae, the anterior 2/3 of the tongue was stained using 0.5% methylene blue to visualize papillae. Body weight was measured, and weight gain was calculated.

RESULTS: T1R3 expression was significantly downregulated in the nicotine groups when compared to the groups receiving room air. CD36 expression did not significantly differ between nicotine and room air groups, but there was a trend toward the HFD consuming mice having higher CD36 mRNA levels ($p=.167$). There was a significant main effect of diet on body weight gain with increased body weight gain in the HFD groups. Nicotine exposure had no effect on body weight gain, T1R3 expression, and T2R138 expression. Data on fungiform papillae staining is forthcoming.

CONCLUSIONS: The results suggest that nicotine inhalation leads to a downregulation in sweet taste receptor gene expression. Further conclusions will be made once more data is available.

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