# Folic acid and vitamin B12 as possible panacea against nicotine induced pancreatic β-cell apoptosis and dysfunction

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## Abstract

Cigarette smoking in regular habits affects our bodies in various ways and nicotine is the more abundant and most significant components of cigarette smoke. Epidemiological evidence strongly suggests an association between cigarette smoking and pancreatic injury. However, effects of cigarette smoking on pancreatic islets are still controversial. Impact and underlying mechanism of actions of folic acid and vitamin B12 on nicotine induced damage in pancreatic islets of rats are examined in the present study. Male Wister rats were exposed to nicotine with or without supplementation of folic acid and vitamin B12. Folic acid and vitamin B12, in combination, blunted the nicotine induced impairment in glucose tolerance, and levels of HbA1c and insulin in rats. Pro-inflammatory cytokines like TNF-α and IL-6, generation of reactive oxygen species, nitric oxide production and other oxidative stress parameters were also attenuated by folic acid and vitamin B12 in nicotine treated rats. Both, folic acid and vitamin B12 in combination also limits the nicotine induced changes in cell cycle and excessive apoptosis of the pancreatic β-cell along with altered Bcl-2, Bax, caspase-3 and caspase-9 expression and up regulation of iNOS and TNF-α. Nicotineinduced alteration in loss of mitochondrial membrane potential ($\Delta\psi m$) and release of cytochrome c also reversed by folic acid and vitamin B12 supplementation. In conclusion, folic acid and vitamin B12 protects against islet cellular oxidative stress, which is a critical step in nicotine-mediated islet injury, and improves islet cell functional status by scavenging free radicals, inhibiting the generation of pro-inflammatory mediators and apoptosis.

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