



# Nicotine is Insufficient as a Carcinogen, It's Functions as a Tumor Promoter on Purpose

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## Abstract

Nicotine constitutes approximately 0.6~3.0% of the dry weight of tobacco. Like anything that enters the body, nicotine is also metabolized. Therefore, any activity that increases your metabolic rate can help speed up the clearance of nicotine. Nicotine also promotes cancer growth, angiogenesis, and neovascularization. Thereby, nicotine impeding apoptosis, promoting tumor growth and activating growth factors. This article provides a brief description of nicotine is unusual in comparison to most drugs, as its concentration profile changes from induced tumor growth to cytotoxic with increasing doses. The concentration of nicotine stimulated cell growth correspond to low concentration was needed, while high concentration was cytotoxic.

**Keywords:** Carcinogen; Nicotine; Tumor growth; Cytotoxic; Apoptosis

## Introduction

Tobacco smoking lead to DNA damage. Cigarette smoke contains more than 6,000 components [1]. Smoking is the leading risk factor for lung cancer. Secondhand smoke exposure or air pollution appears to be the primary underlying cause of cancer. Exercise is a good way to increase the rate of metabolism. Exercise improves heart rate and increases the rate of metabolism and burning of heat [2]. For people who have many years of smoking, it is important to start exercising. Make sure to drink plenty of water because nicotine is soluble in water, so drinking water helps to excrete the substance through the urine. Vitamin A is also helpful in removing nicotine from the body because it also has the effect of speeding up the metabolism [3]. Because nicotine tends to destroy vitamin C in the body, it is important to supplement it after quitting smoking. Nicotine is highly addictive. It follows that nicotine is associated with cancer in humans [4]. Therefore, effects on chemotherapeutics by several malignant cell lines, nicotine in concentrations as low as 1 $\mu$ M decreased. Because of treatment with 1 $\mu$ M nicotine significantly enhanced the frequency of formation of micronuclei, although a higher nicotine concentration. Nicotine acts as tumor growth promoted resistance to apoptosis leading to carcinogenesis [5]. Nicotine is concomitant dual effects on anti-apoptosis and genotoxic activity.

Nicotine is unusual in comparison to most drugs. With increasing dosages, its profile changes from stimulant to sedative. At very

high doses it dampens neuronal activity [6]. This phenomenon is as well-known "Nesbitt's paradox". Micronuclei are characterized in the cancerous cells have some sort of DNA damage. Micronuclei body is a small body can be seen in a newly divided daughter cell. Micronuclei body increased is usually an indication of increased DNA damage or mutation [7]. The mechanisms leading to the formation of Micronuclei body are chromosome breakage and disturbance of the chromosome-segregation system, which represents an irreversible DNA damage. This mechanism responsible for the genotoxic effects caused by nicotine [8]. Effects of nicotine on angiogenesis have been demonstrated for lung tumor cells. Reports had also been demonstrated in H157 lung cancer, where nicotine significantly increased the size and number of tumors in the lung [9]. On the other hand, at high nicotine concentrations (>1 $\mu$ M) with consistent cytotoxic effects and appeared to be due to direct cell kill. Cellular cytotoxicity was associated with inhibition of DNA synthesis, not stimulation of DNA synthesis. This is the main way that micronuclei are formed [10]. Micronuclei can also be spontaneously formed as a byproduct of inhibition of DNA synthesis. This mechanism to micronuclei formation is by a double-strand break DNA, creating a separate linear fragment lead to formation of a micronucleus. Micronuclei are small [11].

These extranuclear bodies that are formed during mitosis from lagging chromosomes. This results in parts of the cell senses extra chromosomes, the cell can attempt to remove the extra chromo-

somes in another cell membrane, separate from the other normal chromosomes being broken off and enveloped as an extra nucleus in one of the daughter cells [12]. Nicotine is an important component in tobacco. Among various subtypes of nicotinic receptors, homopentamers of  $\alpha 7$ nAChR can bind nicotine with highest affinity and mediate multiple effects of nicotine in lung cancer [13]. nAChRs expressed on lung carcinoma or mesothelioma form a part of an autocrine-proliferative network facilitating the growth of neoplastic cells. Target drugs as a form of molecular medicine, targeted therapy blocks the growth of cancer cells by interfering with specific targeted molecules needed for carcinogenesis and tumor growth [14,15]. Nicotine could induce the proliferation of a variety of lung carcinoma cell lines, but there is no evidence that nicotine itself provokes cancer. Nicotine alone is generally accepted as a tumor promoter, but not a tumor initiator in carcinogenesis [16,17]. Nicotine can prevent apoptosis induced by various agents in NSCLC.

## Conclusion

The concentrations of nicotine promote cell proliferation correspond to the low concentrations, while high concentrations are cytotoxic. During nicotine in concentrations as low as  $1\mu\text{M}$ , nicotine activates cell migration, proliferation, survival, and anti-apoptotic effects exerted, in contrast, modulation chemotherapeutics on several different malignant cell lines. This phenomenon which nicotine-mediated inhibition of apoptosis may contribute to observed in normal and transformed cells derived from the pathogenesis of tobacco use-related cancer use as well as decrease the efficacy of cancer therapies.

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## Conflict of interest

The authors do not have any conflict of interest in the manuscript.

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