



WORLD NO TOBACCO DAY: A CHALLENGE TO THE WORLD TO MAKE IT FREE FROM SMOKING

¹*Dibyendu Dutta, ¹Jaydeep Roy, ¹Soumya Chakraborty, ¹Soumya Maiti, ¹Dr. Dhruvo Jyoti Sen and
²Dr. Dhananjay Saha

¹Department of Pharmaceutical Chemistry, School of Pharmacy, Techno India University, EM: 4/1, Sector-V, Salt Lake City. Kolkata-700041, West Bengal, India.

²Deputy Director, Directorate of West Bengal Technical Education, Bikash Bhawan, Salt Lake. Sector-V, Kolkata-700091, West Bengal, India.



*Corresponding Author: Dibyendu Dutta

Department of Pharmaceutical Chemistry, School of Pharmacy, Techno India University, EM: 4/1, Sector-V, Salt Lake City. Kolkata-700041, West Bengal, India.

Article Received on 21/04/2024

Article Revised on 11/05/2024

Article Accepted on 01/06/2024

ABSTRACT

Tobacco is a plant grown for its leaves, which are smoked, chewed, or sniffed. Tobacco contains a chemical called nicotine. Nicotine is an addictive substance. Millions of people in the United States have been able to quit smoking. Tobacco leaves and the smoke generated when they are burned contain over 4 thousand chemicals, 3 the best known of which is nicotine, first isolated from tobacco leaves in 1828 by Derek Posselt and Reimann. It is the nicotine that causes smokers to become addicted to tobacco, and the chemical itself is lethal in small doses. The amount of nicotine in a cigarette varies considerably from brand to brand, but a typical cigarette contains 11.9–14.5 mg of nicotine. Smokers do not absorb all the nicotine content of the cigarette. On average, a person only absorbs 1–1.5 mg of nicotine from a single stick. Tobacco is a plant grown for its leaves, which are smoked, chewed, or sniffed. It causes you to crave a smoke and suffer withdrawal symptoms if you ignore the craving. Nicotine is a toxic substance. It raises your blood pressure and spikes your adrenaline, which increases your heart rate and the likelihood of having a heart attack.

KEYWORDS: Smoking, Nicotine, Tobacco, Nicotinic receptor, Craving, Dopamine, Acetylcholine, NMJ [neuromuscular junction].

INTRODUCTION

World No Tobacco Day (WNTD) is observed around the world every year on 31 May. The annual observance informs the public on the dangers of using tobacco, the business practices of tobacco companies, what the World Health Organization (WHO) is doing to fight against the use of tobacco, and what people around the world can do to claim their right to health and healthy living and to protect future generations.^[1] The Member States of the WHO created World No Tobacco Day in 1987 to draw global attention to the tobacco epidemic and the preventable death and disease it causes. The day is further intended to draw attention to the widespread prevalence of tobacco use and to negative health effects, which currently lead to more than 8 million deaths each year worldwide, including 1.2 million as the result of non-smokers being exposed to second-hand smoke. The day has been met with both enthusiasm and resistance around the globe from governments, public health organizations, smokers, growers, and the tobacco industry. The World Health Organization started World No Tobacco Day in 1987 to draw global attention to the preventable deaths and diseases caused by tobacco. This

year's theme, "Protecting Children from Tobacco Industry Interference," underscores the urgent need to shield youth from the detrimental influences of tobacco consumption. However, after the approval of a resolution in 1988, the world began celebrating "World No Tobacco Day" on May 31. The day marks the birth anniversary of revolutionary queen Ahilyabai Holkar in India [Ahilyabai Holkar (31 May 1725–13 August 1795), also spelled Ahalya Bai, was the Rani of Indore, within the Maratha Confederacy. She established Maheshwar (in Madhya Pradesh) as the seat of the Holkar Dynasty.^[2]

Lung diseases such as chronic bronchitis and chronic obstructive pulmonary disease, which includes obstructive bronchiolitis and emphysema. Heart disease and stroke. Ulcers of the digestive system. Osteoporosis and hip fracture. Therefore, the goals of tobacco control are to reduce tobacco-related morbidity and mortality through sustained reductions in tobacco use initiation and consumption; to reduce the exposure of non-smokers to harmful tobacco smoke; and to promote denormalization of tobacco use.



Figure-1: Ahilyabai Holkar.

COTPA stands for the "Cigarettes and Other Tobacco Products (Prohibition of Advertisement and Regulation of Trade and Commerce, Production, Supply and Distribution) Act, 2003".^[3] Tobacco cultivation in India was introduced by Portuguese in 1605. Initially tobacco was grown in Kaira and Mehsana districts of Gujarat and later spread to other areas of the country. Attempt to improve Indian tobacco has begun with the establishment of the Calcutta Botanical gardens in Howrah in 1787. World No Tobacco Day 2024, celebrated on May 31st, is a global event aimed at raising awareness about the dangers of tobacco use and encouraging people to quit smoking. We now know that smoking is one cause of type 2 diabetes. In fact, people who smoke cigarettes are 30%–40% more likely to develop type 2 diabetes than people who don't smoke. People with diabetes who smoke are more likely than those who don't smoke to have trouble with insulin dosing and with managing their condition. This statistic shows the worldwide tobacco production in 2022, by country. In that year, China was the biggest tobacco producer worldwide with an amount of some 2.19 million metric tons of tobacco produced. Smoking in all public places in Bhutan became illegal on 22 February 2005. It thus became the first nation in the world to outlaw this practice outright.^[4] The Tobacco Control Act of Bhutan was enacted by parliament on 16 June 2010. Smoking can cause lung disease by damaging your airways and the small air sacs (alveoli) found in your lungs. Lung diseases caused by smoking include COPD, which includes emphysema and chronic bronchitis. Cigarette smoking causes most cases of lung cancer. Tobacco is the common name of several plants in the genus *Nicotiana* of the family Solanaceae, and the general term for any product prepared from the cured leaves of these plants. More than 70 species of tobacco are known, but the chief commercial crop is *Nicotiana tabacum*. The more potent variant *N. rustica* is also used in some countries. *Nicotiana tabacum*, or cultivated tobacco, is an annually grown herbaceous plant of the

genus *Nicotiana*. *N. tabacum* is the most commonly grown species in the genus *Nicotiana*, as the plant's leaves are commercially harvested to be processed into tobacco for human use. The plant is tropical in origin, is commonly grown throughout the world, and is often found in cultivation. It grows to heights between 1 and 2 meters (3' to 6').^[5]

Taxonomy: Kingdom: Plantae; Clade: Tracheophytes; Clade: Angiosperms; Clade: Eudicots; Clade: Asterids; Order: Solanales

Family: Solanaceae; Genus: *Nicotiana*; Species: *N. tabacum*.

Dried tobacco leaves are mainly used for smoking in cigarettes and cigars, as well as pipes and shishas. They can also be consumed as snuff, chewing tobacco, dipping tobacco, and snuff. Tobacco contains the highly addictive stimulant alkaloid nicotine as well as harmful alkaloids. Tobacco use is a cause or risk factor for many deadly diseases, especially those affecting the heart, liver, and lungs as well as many cancers. In 2008, the World Health Organization named tobacco use as the world's single greatest preventable cause of death.^[6]

Biochemistry: Nicotine releases a chemical called dopamine in the same regions of the brain as other addictive drugs. It causes mood-altering changes that make the person temporarily feel good. Inhaled smoke delivers nicotine to the brain within 20 seconds, which makes it very addictive—comparable to opioids, alcohol and cocaine. Within seconds of inhaling cigarette smoke or vape mist, or using chewing tobacco, nicotine causes the release of dopamine in the brain, which gives people a good feeling.



Figure-2: Tobacco plant, Leaves and Cigarettes.

Over time, the brain begins to crave that feeling from nicotine and people need to use more and more tobacco to get that same good feeling. Inside your brain, nicotine triggers the release of chemicals that make you feel good. As nicotine stimulates parts of your brain over and over, your brain gets used to having nicotine around. Over time, nicotine changes how your brain works and makes it seem like you need nicotine just to feel okay. Nicotine, the main active compound of tobacco, lowers the perception of pain and physical stress by reducing the amount of the neurotransmitter dopamine that is broken

down by neurons in the prefrontal cortex, a region of the brain that lies just behind the forehead. Nicotine is a dangerous and highly addictive chemical. It can cause an increase in blood pressure, heart rate, flow of blood to the heart and a narrowing of the arteries (vessels that carry blood). Nicotine may also contribute to the hardening of the arterial walls, which in turn, may lead to a heart attack. The average IQ for a non-smoker was about 101, while the smokers' average was more than seven IQ points lower at about 94, the study determined.^[7]



Figure-3: 31st May; World No Tobacco Day.



The IQs of young men who smoked more than a pack a day were lower still, at about 90. Nicotine has been shown to improve attention, learning, and memory. Nicotine, the primary cigarette component, disrupts sleep by causing disturbances when used before bedtime and due to night-time cravings. It also leads to irregular circadian rhythms, snoring, and obstructive sleep apnoea. Nicotine is the chemical that makes cigarettes addictive. But it is not responsible for the harmful effects of smoking, and nicotine does not cause cancer. People have safely used nicotine replacement therapy (NRT) to stop smoking for many years. The research suggests nine out of 10 people falsely believe nicotine is very harmful to their health, when in fact it is no more dangerous than the caffeine in a cup of coffee. For example, nicotine influences a wide variety of cognitive functions such as attention, learning and memory and has been found to improve cognitive impairment in disease-induced cognitive disorders, including Alzheimer's disease (AD),

Parkinson's disease (PD), age-associated memory impairment, age-associated memory impairment. Turns out, nicotine itself is a problem. It significantly lowers sperm count, sperm motility, sperm viability, and increases the percent of sperm with abnormal morphology. It also reduces the weight of the testes, testosterone level, and testicular antioxidant status.^[8]

Nicotine is a naturally produced alkaloid in the nightshade family of plants (most predominantly in tobacco and *Duboisia hopwoodii*) and is widely used recreationally as a stimulant and anxiolytic. As a pharmaceutical drug, it is used for smoking cessation to relieve withdrawal symptoms. Nicotine acts as a receptor agonist at most nicotinic acetylcholine receptors (nAChRs), except at two nicotinic receptor subunits (nAChR α 9 and nAChR α 10) where it acts as a receptor antagonist.

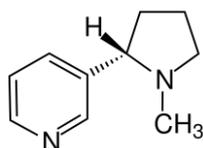


Figure-4: Nicotine Structure.

Chemistry: IUPAC: 3-[(2S)-1-methylpyrrolidin-2-yl]pyridine; CAS: 54-11-5. Nicotine stimulates the adrenal glands to release adrenaline, which stimulates the central nervous system and increases blood pressure, respiration, and heart rate. Nicotine increases dopamine levels, which affect the brain pathways that control reward and pleasure. Nicotine increases dopamine levels, which affect the brain pathways that control reward and pleasure. Continued nicotine exposure results in long-term brain changes, including addiction to the drug and increased susceptibility to addiction to other substances.^[9]

Pharmacology: Nicotine is a constituent of tobacco smoke that exerts its psychoactive effects via binding to nicotinic acetylcholine receptors (nAChRs) in brain. Human genetic studies have identified polymorphisms in nAChR genes, which predict vulnerability to risk for tobacco dependence. The binding of nicotine and ACh to nicotinic receptors cause a conformational change that either opens or closes the receptors' ion channels, thereby changing the receptors' functional state. Before binding agonist, the receptor is in the resting state and is non-functional. Nicotine is a nonselective nicotinic receptor agonist, acting at nicotinic receptors of the autonomic ganglia, at the neuromuscular junction, and in the brain. Nicotine acts through a family of nicotinic acetylcholine receptors (nAChRs), which are widely expressed throughout the central nervous system (CNS) and periphery, including regions of the hypothalamus, a part

of the brain that mediates homeostatic regulation of hunger, satiety, food intake, and energy expenditure. Nicotinic receptors are responsive to the agonist nicotine, while muscarinic receptors are responsive to muscarine. The two receptors differ in function as ionotropic ligand-gated and G-protein coupled receptors, respectively. Nicotine mainly shows its action through specific nicotinic acetylcholine receptors located in brain. It stimulates presynaptic acetylcholine receptors thereby enhancing ACh release and metabolism. Dopaminergic system is also stimulated by it, thus increasing the concentration of dopamine in nuclear accumbens. Many nicotinic receptors appear to modulate neurotransmitter release through excitatory mechanisms. Presynaptic receptors likely provide a feedback mechanism on transmitter release. Such presynaptic action affects the release of acetylcholine, dopamine, noradrenaline, serotonin, γ -aminobutyric acid, and glutamate. Nicotinic antagonists that block the receptor include mecamylamine, dihydro- β -erythroidine, and hexamethonium. Nicotine releases a chemical called dopamine in the same regions of the brain as other addictive drugs. It causes mood-altering changes that make the person temporarily feel good. Inhaled smoke delivers nicotine to the brain within 20 seconds, which makes it very addictive—comparable to opioids, alcohol and cocaine. Stimulation of a nicotinic ACh receptor causes depolarization and activation of L-type Ca^{2+} channels in rat pinealocytes.^[10]

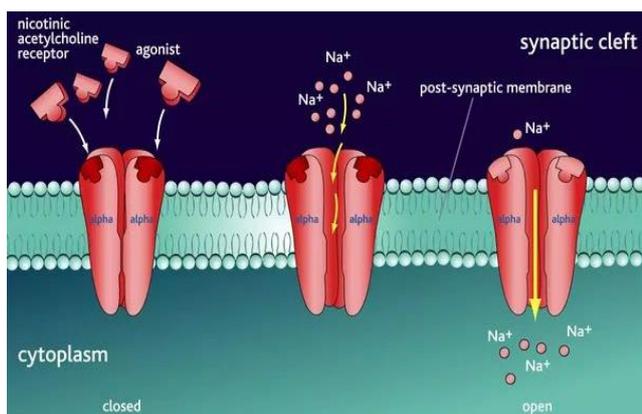
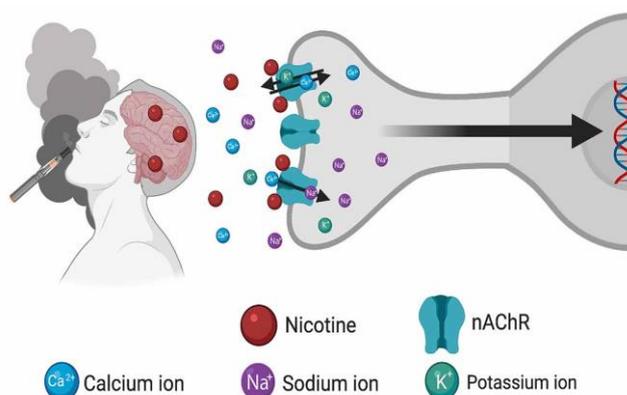


Figure-5: Nicotinic receptor.

Nicotine binds to the acetylcholine receptors (nAChRs) in the brain and influences the cerebral metabolism by stimulating these receptors. The stimulation of presynaptic nAChRs on the neurons increases the transmitter release as well as the metabolism. The initiation of nicotine abuse involves the mesolimbic dopamine system, which contributes to the rewarding



sensory stimuli and associative learning processes in the beginning stages of addiction. Nicotine binds to neuronal nicotinic acetylcholine receptors (nAChRs), which come in a diverse collection of subtypes. Stimulation of central nAChRs by nicotine results in the release of a variety of neurotransmitters in the brain, most importantly dopamine. Nicotine causes the release of dopamine in the

mesolimbic area, the corpus striatum, and the frontal cortex. Midbrain dopaminergic (DA) neurons are governed by an endogenous cholinergic system, originated in the mesopontine nuclei.^[11] Nicotine hijacks nicotinic acetylcholine receptors (nAChRs) and interferes with physiological function of the cholinergic system. Actually they are receptors for the same single neurotransmitter, acetylcholine. The different types of receptors are named for the drugs that bind best with the receptors. Nicotine binds better than other drugs to the ionotropic ACh receptor, hence, following convention, the receptor is named nicotinic. There are two properties of nicotinic AChRs which suggest that these receptors could take part in other physiological interactions at synapses. The first is the ability of the nicotinic AChR channel to conduct Ca^{2+} ions. The second is that nicotinic AChRs can show steady-state activation at low agonist concentrations. Nicotine binds to nicotinic receptors in the brain, augmenting the release of numerous neurotransmitters, including dopamine, serotonin, norepinephrine, acetylcholine, gamma-aminobutyric acid, and glutamate. Cigarette smoke has other psychoactive properties apart from nicotinic receptor stimulation. Nicotine that gets into your body through cigarettes activates structures normally present in your brain called receptors. When these receptors are activated, they release a brain chemical called dopamine, which makes you feel good. Nicotine increases dopamine levels, which affect the brain pathways that control reward and pleasure. Continued nicotine exposure results in long-term brain changes, including addiction to the drug and increased susceptibility to addiction to other substances. Nicotine competitively binds to nicotinic cholinergic receptors. The binding of the agonist to the nicotinic receptor triggers off a conformation change of the architecture of the receptor, which opens the ionic channel during a few milliseconds. This channel is selective for cations (especially sodium). Activation of excitatory receptors, such as nicotinic acetylcholine receptors (nAChRs), one is known to depolarize cells and activate voltage-gated ion channels.^[12] Direct nicotinic receptor antagonists competitively block activity at the NMJ and result in weakness or muscle paralysis. Nicotine is a dangerous and highly addictive chemical. It can cause an increase in blood pressure, heart rate, flow of blood to the heart and a narrowing of the arteries (vessels that carry blood). Nicotine may also contribute to the hardening of the arterial walls, which in turn, may lead to a heart attack. Nicotine is a dangerous and highly addictive chemical. It can cause an increase in blood pressure, heart rate, flow of blood to the heart and a narrowing of the arteries (vessels that carry blood). Nicotine may also contribute to the hardening of the arterial walls, which in turn, may lead to a heart attack. A key function of nicotinic receptors is to trigger rapid neural and neuromuscular transmission. Nicotinic receptors are found in: The somatic nervous system (neuromuscular junctions in skeletal muscles). The sympathetic and parasympathetic nervous system (autonomic ganglia). From a systems

perspective, nicotinic receptors have a role in directly stimulating not only postsynaptic neurons but also other functions. For example, nicotinic receptors are located on the blood vessels and can modulate blood flow. Nicotine has many effects on central nervous system activity. Nicotine has both positive and negative reinforcing properties, meaning that the drug enhances the activity of brain reward circuits (positive reinforcement) while attenuating the activity of brain aversion circuits during withdrawal (negative reinforcement). Nicotine exposure also leads to changes in levels of the neurotransmitters, the brain's chemicals, such as dopamine and serotonin and so can rewire your brain so it becomes more addicted to nicotine and the release of dopamine that comes with it.^[13]

CONCLUSION

May 31st was chosen to mark the anniversary of the WHO's constitution coming into effect. World No Tobacco Day plays a vital role in: Educating the public: Raising awareness about the severe health consequences of tobacco use, including cancer, heart disease, stroke, and lung diseases. The Member States of the World Health Organisation designated May 31 as World No Tobacco Day in 1987, to draw attention to the global tobacco epidemic, and the preventable death and disease it causes." This year, World No Tobacco Day calls on us all to protect children from the interference of the tobacco industry. World No Tobacco Day 2024, celebrated on May 31st, is a global event aimed at raising awareness about the dangers of tobacco use and encouraging people to quit smoking. The World Health Organization started World No Tobacco Day in 1987 to draw global attention to the preventable deaths and diseases caused by tobacco.

REFERENCES

1. Wakefield MA, Loken B, Hornik RC. Use of mass media campaigns to change health behaviour. *Lancet*, 2010; 376: 1261e71.
2. Abroms LC, Maibach EW. The effectiveness of mass communication to change public behavior. *Annu Rev Public Health*, 2008; 29: 219e34.
3. Christakis NA, Fowler JH. The collective dynamics of smoking in a large social network. *N Engl J Med.*, 2008; 358: 2249e58.
4. Asbridge M. Public place restrictions on smoking in Canada: assessing the role of the state, media, science and public health advocacy. *Soc Sci Med.*, 2004; 58: 13e24.
5. Doll R, Peto R, Boreham J, et al. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ*, 2004; 328: 1519.
6. Alday J, Murukutla N, Cedillo C, et al. Smoke-free Sao Paulo: a campaign evaluation and the case for sustained mass media investment. *Salud Publica Mex*, 2010; 52: S216e25.
7. Durkin SJ, Biener L, Wakefield MA. Effects of different types of antismoking ads on reducing disparities in smoking cessation among

- socioeconomic subgroups. *Am J Public Health*, 2009; 99: 2217e23.
8. Gagne L. The 2005 British Columbia smoking cessation mass media campaign and short-term changes in smokers attitudes. *J Health Commun.*, 2008; 13: 125e48.
 9. Grigg M, Waa A, Kawenata Bradbrook S. Response to an indigenous smoking cessation media campaign. It's about whanau. *Aust NZ J Public Health*, 2008; 32: 559e64.
 10. Niederdeppe J, Fiore MC, Baker TB, et al. Smoking-cessation media campaigns and their effectiveness among socioeconomically advantaged and disadvantaged populations. *Am J Public Health* 2008; 98: 916e24.
 11. Perusco A, Poder N, Mohsin M, et al. Evaluation of a comprehensive tobacco control project targeting Arabic-speakers residing in south west Sydney, Australia. *Health Promot Int.*, 2010; 25: 153e65.
 12. Richardson AK, Green M, Xiao HJ, et al. Evidence for truth (R) the young adult response to a youth-focused anti-smoking media campaign. *Am J Prev Med.*, 2010; 39: 500e6.
 13. Dr Dhruvo Jyoti Sen; Non-oxygenated alkaloid vaping lowers stress and creates mind to impress: *European Journal of Pharmaceutical and Medical Research*, 2024; 11(2): 636-643.