Review Article

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A Narrative Review on the Role of Nicotine in Modulating HPA Axis Dynamics: Insights into Stress, Anxiety, and Peer Pressure

Dentistry Section

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ABSTRACT

India is the second-largest tobacco consumer and third-largest tobacco producer in the world. Stress is considered an important factor associated with nicotine dependency. Stress increases cortisol secretion in the bloodstream. Stress affects the pathway that sends signals from brainstem nuclei to the hypothalamic-pituitary axis and the release of adrenocorticotropic hormone and cortisol in mammals. This comprehensive review elucidates the intricate relationship between cigarette smoking and the Hypothalamic-Pituitary-Adrenal (HPA) axis. Additionally, it delves into the psychological factors, including peer pressure, depression, and anxiety, that influence an individual's propensity to engage in smoking behaviour. Prolonged nicotine use impacts the expression of numerous essential neuropeptides in the brain, including Arginine Vasopressin (AVP) and Corticotropin-Releasing Factor (CRF). Nicotine produces transient euphoric effects and transient elevation of cortisol levels. Patients with anxiety/panic disorders have a greater tendency to depend on nicotine consumption, and peer pressure also plays a key role in the increase in smoking among young adults. This review attempts to close this gap by examining how nicotine affects HPA function and its consequences for metabolic control, stress-response modulation, and addiction biology. Comprehending this connection is essential for developing treatment plans to reduce nicotine-related health problems and improve smoking cessation techniques. In conclusion, nicotine intake indirectly modulates the hypothalamic-pituitary-adrenal axis activity and transiently alters cortisol secretion, contributing to stress-response dysregulation and nicotine dependence.

Keywords: Addiction, Cortisol, Hypothalamus, Psychological factors, Tobacco

INTRODUCTION

Worldwide, tobacco smoking is the leading preventable cause of death and disability. An astounding 8.7 million individuals die each year as a result of this risk factor. Additionally, it has been reported that 1.3 million of these fatalities were non smokers, including infants and newborns. Secondhand smoke exposure poses a significant risk to women and children in particular. In underdeveloped nations, tobacco use has a disproportionately large negative impact on health [1]. Between 2005 and 2030, it is predicted that tobacco use in these nations will claim the lives of almost 40 million individuals [2]. India is ranked third worldwide in terms of tobacco production and ranks second in terms of tobacco consumption [3]. In addition to billions of dollars in directly associated health expenses, this causes almost one million fatalities annually in India (roughly one-sixth of all tobacco-related deaths globally) [3]. An analysis conducted recently found that 11.6% of Indians used tobacco. For people between the ages of 30 and 69, smoking is causally linked to 1 in 5 fatalities for men and 1 in 20 deaths for women [4]. In 2023, Many nations have made tremendous progress in reducing tobacco use and adopting measures that promote health, as evidenced by the World Health Organisation (WHO) report on the global tobacco epidemic [5]. The National Family Health Survey (NFHS-5) estimates that 46.9% of Indian men and women who are 15 years of age or above consume any form of tobacco. Even after starting various policies and methods to control cigarette smoking, there is a rise in the deaths of patients who smoke [6]. It is widely accepted that smoking cigarettes is an addictive disorder that is characterised by physical dependency and tolerance. Humans and laboratory animals self-administer intravenous nicotine under a variety of circumstances, suggesting that nicotine is the main addictive substance in cigarettes. Nicotine dependence is determined by various factors like socioeconomic status, demographic group, stress levels, depression, and anxiety [7]. Males from lower-class and more economically disadvantaged communities are more prone to smoking in India [8]. Regarding states, the eastern region, spanning from West Bengal to the northeastern regions, has a higher tobacco use rate than the national average [9]. One of the major factors responsible for nicotine dependency is stress. Stress increases cortisol secretion in the bloodstream. Stress affects the pathway that sends signals from brainstem nuclei to the HPA axis and the release of adrenocorticotropic hormone and cortisol in mammals [9,10]. The present review outlines the connection between smoking cigarettes and the HPA axis. The second is how the individual was persuaded to smoke by peer pressure and other psychological conditions like despair and anxiety. In contrast to many previous literature reviews that have considered nicotine and the HPA axis as distinct topics or offered only broad insights, the current review highlights their interrelationship. This comprehensive approach could offer a deeper understanding of their relationship.

MATERIALS AND METHODS

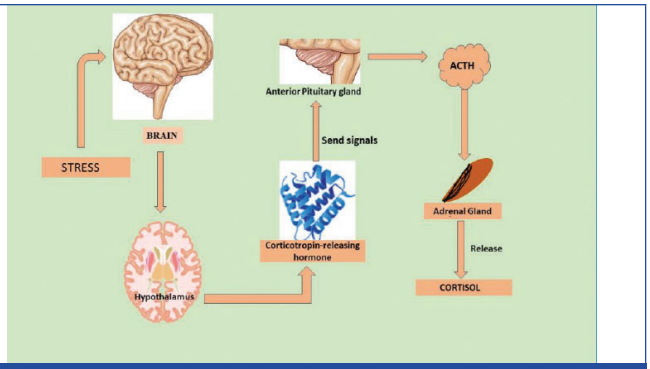
Search Strategy

The present narrative review was conducted using a comprehensive literature search of PubMed, Scopus, Google Scholar, and Web of Science databases for articles published between 2000 and 2025. Keywords used for the search included 'nicotine', 'hypothalamic-pituitary-adrenal axis', 'HPA axis', 'stress', 'cortisol', 'anxiety', 'peer pressure', and 'smoking behaviour'. Original research articles, review articles, and relevant observational and experimental studies published in English were included. Articles lacking relevance to nicotine-mediated neuroendocrine mechanisms or behavioural associations were excluded.

Hypothalamus Pituitary Adrenal Axis and Stress

The sympathetic nervous system, which includes the hypothalamus, the pituitary gland, and the adrenal axis, connects the brain to the body's extremities. The hypothalamus, pituitary, and adrenal axis

are important parts of the neuroendocrine system that control how the body responds to stimuli that disturb the body's homeostatic balance, whether physical or psychological. Emerging as "the general adaptation syndrome," the typical adaptive reaction of an individual to extended stress entails hormonal and Central Nervous System (CNS) interactions [10]. Adrenocorticotropic hormone is released by the anterior pituitary in reaction to danger, while corticotropin-releasing hormone is released by the hypothalamus. In response, the adrenal glands release glucocorticoids, with the predominant one being cortisol in humans [Table/Fig-1] [11].



[Table/Fig-1]: Hypothalamus-pituitary-adrenal-axis and stress.

The inhibitory action of these glucocorticoids then signals the system to cease the stress response. Reproduction, growth, immune response, energy expenditure, and homeostasis are all influenced by low glucocorticoid levels. However, in a high-stress environment, disruptions to the HPA axis (e.g., chronic elevation or blunting of glucocorticoids) can have several detrimental effects, including immune system and inflammatory dysregulation, metabolic syndrome, hypertension, cardiovascular disease, and deficits in cognitive function [12].

How are Stress, HPA Axis, and Smoking Interrelated

Stress and addiction have been linked in several research studies. Many viewpoints emphasise the vital impact that stress plays. Studies have shown a connection between smoking and both acute and chronic stress, showing this association at several stages of the addiction process, including initiation, maintenance, and relapse to cigarette smoking [10,13]. Two biological stress systems of interest are the HPA axis, which can be evaluated by measuring salivary and serum/plasma cortisol concentrations, and the Autonomic Nervous System (ANS), which is frequently investigated by analysing catecholamine levels and cardiovascular responses [14]. Acute nicotine exposure and psychological stress have strikingly balanced effects on neuronal pathways involved in both reward and stress regulation. "Acute nicotine administration," which is short-term nicotine use, is linked to HPA axis activation [15]. More specifically, nicotine releases Corticotropin-Releasing Hormone (CRH) via binding to cholinergic receptors in the hypothalamus and locus coeruleus. Subsequently, the adrenal glands release cortisol, and the pituitary releases Adrenocorticotropic Hormone (ACTH), increasing both cortisol and ACTH overall [16]. Studies involving human smokers have indicated a continuous rise in cortisol levels after smoking two or more cigarettes [17-19]. Additionally, it seems that the dose-dependent consequences of smoking include heart rate changes, acute nicotine-induced HPA activation, and favourable subjective effects [Table/Fig-2] [20,21].

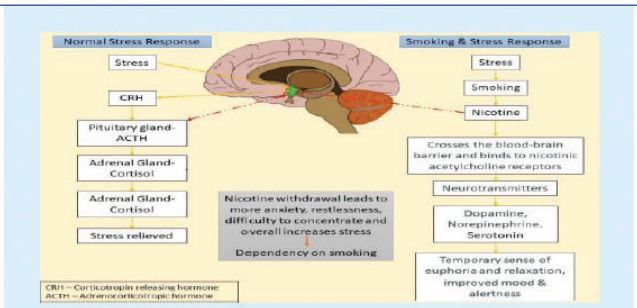
Effects of Smoking on the HPA Axis

Previous studies have shown that smoking cigarettes causes alterations in vasopressin levels in the hypothalamus. It has been demonstrated that nicotine dependence stimulates the HPA axis [22-26]. Rats were administered 100, 200, or 500 mg/kg of nicotine intraperitoneally (i.p.). Similarly, showed the same effect, i.e., a dose-dependent activation of the HPA axis [27]. The authors report that although the highest

S. No.	Author and Year	Title of the study	Inferences
1.	Chatzi G et al., [20] (2024)	Is social disadvantage a chronic stressor? Socioeconomic position and HPA axis activity among older adults living in England.	At a social disadvantage, cortisol levels increased, and a significant number of participants were smokers.
2.	Chen Y et al., [21] (2024)	Deficient sleep, altered hypothalamic functional connectivity, depression, and anxiety in cigarette smokers.	Altered hypothalamic connectivity with deficient sleep in adults who smoke. Elevated hypothalamic functional connectivity with the somatosensory cortex was related to worse sleep quality and greater severity of depression and anxiety symptoms in smokers.

[Table/Fig-2]: Studies conducted proving evidence of the interrelationship between stress, HPA axis, and smoking [20,21].

nicotine concentration produced the maximum corticosterone response, a higher dosage of nicotine causes a biphasic response, with a corticosterone peak occurring immediately after injection for the first 15 minutes, followed by a second and higher peak for 20 minutes. There was just one peak visible in the ACTH response [28]. The HPA responses to nicotine were eliminated by hypophysectomy [29]. Several studies have demonstrated that individuals who have acquired an addiction to alcohol, nicotine, or illegal substances have dysregulated HPA axis functioning, which is frequently measured using cortisol in saliva [30-33]. Adults who are dependent on drugs such as alcohol, nicotine, or other substances exhibit persistent activation of the HPA axis, and similar patterns are observed during periods of nicotine withdrawal on the HPA axis [Table/Fig-3,4] [34,35-38].



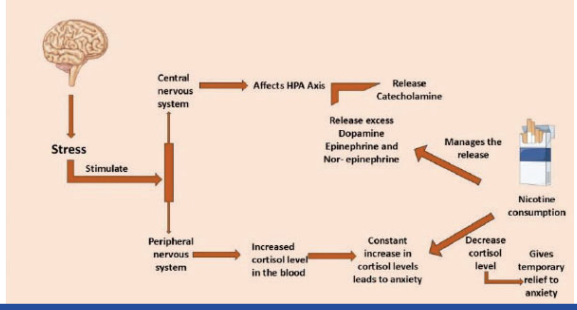
[Table/Fig-3]: Comparison of normal stress response, smoking-induced stress response.

S. No.	Author/Year	Title of the research	Inference
1.	Mendelson JH et al., [35] (2005)	Effects of low- and high-nicotine cigarette smoking on mood states and the HPA axis in men.	Increase in ACTH, heart rate, and elevation in HPA axis after smoking cigarettes.
2.	Gentile NE et al., [36] (2011)	Sexually diergic HPA responses to single-dose nicotine, continuous nicotine infusion, and nicotine withdrawal by mecamylamine in rats.	The study demonstrated that the HPA axis responds to stimulation by single doses of nicotine /cigarettes.
3.	Rao U et al., [37] (2009)	Contribution of HPA activity and environmental stress to vulnerability for smoking in adolescents.	In over 150 adolescents followed from 6 months to 5 years, higher basal cortisol levels (measured using nocturnal free urinary cortisol and salivary cortisol levels) correlated with an increased risk of smoking initiation and smoking persistence. suggesting a dose-dependent effect of nicotine or a threshold response to nicotine in activating the HPA axis
4.	Buchmann AF et al., [38] (2010)	Cigarette craving increases after a psychosocial stress test and is related to cortisol stress response but not to dependence scores in daily smokers.	A significant increase in cortisol concentrations was found following exposure to the stressor in daily smokers, occasional smokers, and nonsmokers, and the magnitude of the cortisol increase in the daily smoker group was lower than in the occasional smokers and nonsmoker groups. The release of cortisol is directly related to the activation of HPA axis.

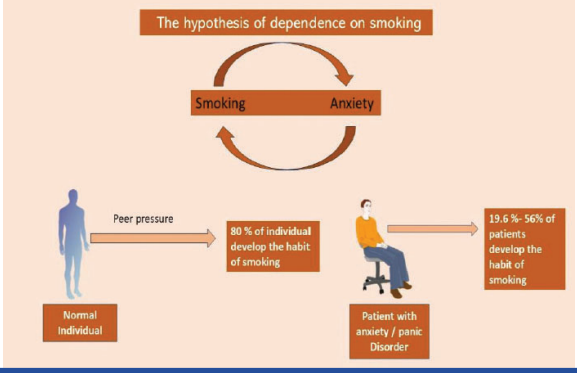
[Table/Fig-4]: Studies that show the effect of smoking on the HPA axis [35-38].

Association between Anxiety/Panic Disorder and Nicotine Consumption- How Does The HPA Axis Play a Role?

Three non exclusive hypotheses might account for the link between smoking and anxiety. Either smoking causes anxiety, or anxiety causes smoking, or third, there may be a common susceptibility factor that affects both smoking and anxiety rates [35-36]. There is evidence that those who experience higher levels of anxiety are also more inclined to smoke [37]. Multiple ideas have been proposed to elucidate this phenomenon, including the use of cigarettes as a self-medication method for decreasing anxiety and the underlying vulnerability of those with anxiety to start smoking as a result of peer pressure. Finally, numerous shared vulnerability factors have been identified that may increase the likelihood of both smoking and increased anxiety [38,39]. For example, lower Socioeconomic Status (SES) is associated with both increased smoking behaviours and anxiety. The current rates of daily smoking among people with panic disorder have varied from 19% to 56% throughout research. Rates of current daily smoking among persons with panic disorder are often higher than those among individuals without psychiatric disorders and with particular forms of other mental health issues when comparison groups have been used [40]. For instance, according to McCabe RE et al., (2004), 40.4% of people with panic disorder who were seeking therapy also smoked. Still, this percentage was 19.6% among individuals with social phobia and 22.4% for those with obsessive-compulsive disorder who were also seeking treatment for their anxiety issues. These results are consistent with epidemiological study data [Table/Fig-5a,b] [41].



[Table/Fig-5a]: Association between anxiety and nicotine consumption.



[Table/Fig-5b]: Hypothesis on consumption of nicotine; Normal Individual consuming nicotine due to peer pressure; Patients with anxiety/panic disorder develop habit of smoking.

Correlation between Peer Pressure and Smoking

The primary factors contributing to the initiation of smoking among students are peers, parental influence, and media. Peer pressure is considered a major contributing factor in the early initiation of smoking among adolescents, particularly in secondary school [42--47]. Numerous additional research studies have established a correlation between student smoking behaviours and family smoking. Teenagers have easy access to cigarettes if their parents/ guardians are indulging in the habit of smoking. Students are influenced to use tobacco from an adolescent age by social, environmental, psychological, and genetic variables. High levels of peer pressure have an impact on the usual developmental stage of adolescents and can influence risk-taking behaviours, including drug use [14]. An estimated 80% of the one billion teenage smokers worldwide, particularly in low- and middle-income nations, experience illness and death from tobacco use. This is particularly pertinent for college and high school kids who are dealing with significant health issues like stress. It is often acknowledged that young people's early tobacco experimentation and propensity to continue smoking are significantly influenced by their peer group [42]. A study conducted in 2006 showed a strong relationship between smoking and peer pressure in school students; the smoking level increases with increased peer pressure and decreases with decreased peer pressure [43]. smoking by peers, and to a lesser extent by parents and family, has a strong influence on the uptake of smoking in male adolescents attending schools. Previous studies concluded that parents who smoked increased the likelihood that their adolescents would also smoke, whereas adolescents who had strong parental monitoring were less likely to engage in smoking behaviour [44,45]. According to a cross-sectional study done in Baghdad in 2018, students are more likely to interact with many peer smokers over many years, which may help to explain the effect of peer pressure, both from cigarettes and shisha, on students who smoke [46]. Other researchers have found that long-term friendships with smokers have the potential to influence non smokers to try smoking, which is a result of both the influence of peer pressure and psychological disorders that develop during adolescence [45-49].

Limitation(s)

The present narrative review has certain limitations. The included studies varied in methodology, population characteristics, and nicotine exposure assessment, which may limit generalisability. As a non systematic review, selection bias and publication bias cannot be excluded. Additionally, the reviewed evidence primarily demonstrates associative rather than causal relationships between nicotine exposure, HPA axis modulation, stress, and psychological factors.

CONCLUSION(S)

The HPA axis is a key regulator of the body's stress response, and nicotine has a substantial effect on it. An effective stimulant for HPA axis activation is nicotine. ACTH and corticosterone are released as a result of this. Prolonged nicotine use impacts the expression of numerous essential neuropeptides in the brain, including AVP and CRF. These alterations may contribute to increased stress sensitivity among smokers. Patients with anxiety/panic disorders have a greater tendency to depend on nicotine consumption and peer pressure also plays a key role in the increase in smoking among young adults.

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