



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE

THE IMPACT OF NICOTINE ON HUMAN BRAIN FUNCTIONING:
NEUROBIOLOGICAL MECHANISMS, COGNITIVE EFFECTS AND
ADDICTION RISK - A NARRATIVE REVIEW

DOI

[https://doi.org/10.31435/ijitss.3\(51\).2026.6065](https://doi.org/10.31435/ijitss.3(51).2026.6065)

RECEIVED

22 July 2026

ACCEPTED

21 September 2026

PUBLISHED

28 September 2026

LICENSE



The article is licensed under a **Creative Commons Attribution 4.0 International License**.

© The author(s) 2026.

This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

THE IMPACT OF NICOTINE ON HUMAN BRAIN FUNCTIONING: NEUROBIOLOGICAL MECHANISMS, COGNITIVE EFFECTS AND ADDICTION RISK - A NARRATIVE REVIEW

Michał Kieres (Corresponding Author, Email: michalek364@interia.pl)

University of Opole, Poland

ORCID ID: 0009-0001-4763-3089

Klaudia Karolina Wójcik

University of Opole, Poland

ORCID ID: 0009-0003-0668-4466

Krzysztof Sebastian Habirek

University Clinical Hospital in Opole, Poland

ORCID ID: 0009-0004-7151-5949

Maja Maria Porążko

University Clinical Hospital in Opole, Poland

ORCID ID: 0009-0001-0248-4884

Anna Katarzyna Żurakowska-Zadrożna

University Clinical Hospital in Opole, Poland

ORCID ID: 0009-0006-0130-0576

Adrianna Krupska

Medical University of Silesia in Katowice, Poland

ORCID ID: 0009-0008-1710-1381

Julia Anna Czarnecka

Poznan University of Medical Sciences, Poland

ORCID ID: 0009-0008-2804-065X

Zuzanna Marta Marcinkowska

Medical University of Lodz, Poland

ORCID ID: 0009-0001-3563-974X

Joanna Zuzanna Gugulska

Medical University of Silesia in Katowice, Poland

ORCID ID: 0009-0007-4624-515X

Anna Kropiwnicka

Collegium Medicum, University of Zielona Góra, Poland

ORCID ID: 0009-0002-2869-369X

Aleksander Kacper Kierys

MSWiA Hospital in Wrocław, Poland

ORCID ID: 0009-0003-0810-041X

Michał Mieczysław Parus

Wrocław Medical University, Poland

ORCID ID: 0009-0000-4579-7330

ABSTRACT

Introduction: Nicotine is one of the most widely used psychoactive substances worldwide. Its effects involve multiple aspects of central nervous system functioning, influencing cognitive processes as well as mechanisms responsible for the development of addiction. In recent years, increasing scientific attention has been directed toward the impact of nicotine on the developing adolescent brain and the consequences associated with the growing popularity of novel nicotine-containing products.

Objective: The aim of this study was to analyze the current state of knowledge regarding the effects of nicotine on human brain function, with particular emphasis on neurobiological mechanisms, cognitive performance, addiction processes, and the consequences of nicotine exposure during adolescence.

Materials and Methods: A narrative review of the scientific literature was conducted using publications retrieved from PubMed, Scopus, Web of Science, and Google Scholar databases. Experimental studies, observational studies, systematic reviews, and meta-analyses addressing the effects of nicotine on the central nervous system were included in the analysis.

Results: The reviewed literature indicates that nicotine exerts its effects primarily through the activation of nicotinic acetylcholine receptors, resulting in increased dopamine release within the mesolimbic dopaminergic pathway. Short-term nicotine exposure may temporarily enhance attention, working memory, and information-processing speed. However, long-term use contributes to the development of tolerance, neuroadaptive changes, and nicotine dependence. Particularly adverse effects have been observed among adolescents, in whom nicotine may disrupt neurodevelopmental processes and increase susceptibility to addiction.

Conclusions: Nicotine has a complex impact on human brain function. Despite its potential short-term effects on selected cognitive abilities, such as improved attention and faster information processing, regular nicotine use is associated with a high risk of addiction and long-term neurobiological consequences. Preventive strategies aimed at reducing nicotine exposure among adolescents remain of particular importance for public health.

KEYWORDS

Nicotine, Brain, Addiction, Dopamine, Cognitive Functions, Adolescents, Nicotinic Receptors, Neurobiology

CITATION

Kieres, M., Wójcik, K. K., Habirek, K. S., Porążko, M. M., Żurakowska-Zadrożna, A. K., Krupska, A., Czarnecka, J. A., Marcinkowska, Z. M., Gugulska, J. Z., Kropiwnicka, A., Kierys, A. K., & Parus, M. M. (2026). The impact of nicotine on human brain functioning: Neurobiological mechanisms, cognitive effects and addiction risk - A narrative review. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6065](https://doi.org/10.31435/ijitss.3(51).2026.6065)

COPYRIGHT

© The author(s) 2026. This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

1. Introduction

Nicotine is one of the most widely used psychoactive substances worldwide. It occurs naturally in tobacco plants and is delivered to the human body through conventional cigarettes, electronic cigarettes, heated tobacco products, nicotine pouches, and nicotine replacement therapy products. Despite decades of preventive and educational efforts, nicotine remains a commonly used substance and continues to represent a significant public health concern (Benowitz, 2010; World Health Organization, 2023).

In recent years, substantial changes have been observed in patterns of nicotine consumption. Although the prevalence of conventional cigarette smoking has declined in many countries, the popularity of alternative nicotine products has increased, particularly among adolescents and young adults. The rapid development of electronic cigarettes and other nicotine delivery systems has intensified scientific interest in the effects of nicotine on human brain function (England et al., 2015; World Health Organization, 2023).

Nicotine exerts its effects primarily through the activation of nicotinic acetylcholine receptors (nAChRs), which are widely distributed throughout the central nervous system. These receptors play an important role in processes related to memory, learning, attention, emotional regulation, and reward mechanisms. After entering the bloodstream, nicotine rapidly crosses the blood-brain barrier, reaching peak concentrations in the central nervous system within seconds of administration (Dani & Bertrand, 2007; Benowitz, 2010).

One of the most significant effects of nicotine is the stimulation of the mesolimbic dopaminergic pathway, commonly referred to as the brain reward system. Activation of this system increases dopamine release in the

nucleus accumbens, producing feelings of pleasure and reinforcing behaviors associated with repeated nicotine use. Prolonged nicotine exposure results in adaptive changes within the brain that may contribute to the development of tolerance, dependence, and addiction (De Biasi & Dani, 2011; Changeux, 2010).

Scientific evidence indicates that nicotine may produce both short-term beneficial effects and long-term adverse consequences. Experimental studies have demonstrated that nicotine can temporarily enhance attention, working memory, information-processing speed, and certain executive functions. Consequently, its potential therapeutic application has been investigated in selected neurological and psychiatric disorders, including Alzheimer's disease and schizophrenia (Heishman et al., 2010; Newhouse et al., 2004; Levin et al., 2006).

At the same time, chronic nicotine use is associated with numerous unfavorable neurobiological changes. Long-term stimulation of nicotinic receptors contributes to the remodeling of neuronal connections, disruption of reward system functioning, and an increased risk of addiction. Individuals dependent on nicotine frequently experience withdrawal symptoms, including irritability, impaired concentration, depressed mood, and an intensified urge to consume nicotine (Picciotto & Mineur, 2014; Prochaska & Benowitz, 2019).

Particular attention has been devoted to the effects of nicotine on the developing adolescent brain. Adolescence is characterized by intensive maturation of brain structures responsible for behavioral control, decision-making, and emotional regulation. Research findings suggest that nicotine exposure during this critical developmental period may result in long-lasting neurobiological alterations that increase vulnerability to addiction and cognitive impairment later in life (Yuan et al., 2015; Goriounova & Mansvelder, 2012; Leslie, 2020).

Given the growing popularity of novel nicotine products and the persistently high prevalence of nicotine use worldwide, further investigation into the effects of nicotine on brain function remains essential. A better understanding of nicotine's mechanisms of action may contribute to the development of more effective preventive strategies and interventions aimed at reducing the adverse health consequences associated with nicotine use.

The aim of this study is to review the current state of knowledge regarding the effects of nicotine on human brain function, with particular emphasis on neurobiological mechanisms, cognitive performance, addiction-related processes, and the consequences of nicotine exposure during adolescence.

2. Materials and Methods

2.1. Study Design

This study was conducted as a narrative review of the scientific literature concerning the effects of nicotine on human brain function. The aim of the review was to collect, analyze, and synthesize current research findings related to the neurobiological mechanisms of nicotine action, its impact on cognitive functions, the development of nicotine addiction, and the consequences of nicotine exposure during adolescence.

2.2. Literature Search Strategy

The literature review was performed using the following electronic databases:

- PubMed,
- Scopus,
- Web of Science,
- Google Scholar.
- The search process employed the following keywords and their combinations:
 - “nicotine”,
 - “brain”,
 - “nicotine addiction”,
 - “dopamine”,
 - “cognitive functions”,
 - “nicotinic acetylcholine receptors”,
 - “adolescent brain”,
 - “neurobiology of nicotine”.

The keywords were combined using the Boolean operators AND and OR to ensure a comprehensive search of publications relevant to the research topic.

2.3. Inclusion Criteria

Publications were included in the review if they met the following criteria:

- articles published in peer-reviewed scientific journals,
- studies investigating the effects of nicotine on human brain function,
- experimental studies, observational studies, systematic reviews, and meta-analyses,
- publications available in English,
- studies published between 2000 and 2025.

2.4. Exclusion Criteria

The following publications were excluded from the analysis:

- non-scientific and popular science articles,
- publications without full-text access,
- case reports,
- studies not directly related to the neurobiological effects of nicotine,
- publications focusing exclusively on the somatic consequences of tobacco smoking without reference to central nervous system functioning.

2.5. Data Analysis

Following the initial selection process, a qualitative analysis of the available scientific evidence was conducted. Particular attention was paid to findings related to:

- the mechanisms of nicotine action on nicotinic acetylcholine receptors,
- the effects of nicotine on the dopaminergic system and the brain reward pathway,
- the influence of nicotine on cognitive functions,
- the neurobiological processes involved in the development of nicotine addiction,
- the consequences of nicotine exposure during adolescence.

The collected information was organized thematically and presented in the Results section, providing a comprehensive overview of the current state of knowledge regarding the effects of nicotine on human brain function.

3.1. Mechanisms of Nicotine Action in the Brain

Nicotine is a plant-derived alkaloid with potent psychoactive properties that is rapidly absorbed through mucous membranes and the pulmonary alveoli following administration. After inhalation of tobacco smoke or aerosol generated by electronic cigarettes, nicotine enters the bloodstream and crosses the blood–brain barrier within approximately 10–20 seconds, making it one of the fastest-acting psychoactive substances (Benowitz, 2010).

The primary targets of nicotine within the central nervous system are nicotinic acetylcholine receptors (nAChRs), which belong to the family of ionotropic receptors. These receptors are naturally activated by acetylcholine; however, nicotine can also bind to and stimulate them, thereby mimicking the action of the endogenous neurotransmitter (Dani & Bertrand, 2007). Nicotinic receptors are widely distributed throughout the brain, including the cerebral cortex, hippocampus, thalamus, nucleus accumbens, and ventral tegmental area (VTA), all of which play important roles in cognitive processes, emotional regulation, and reward-related mechanisms.

Particular attention has been given to receptor subtypes containing the $\alpha 4\beta 2$ and $\alpha 7$ subunits, which are responsible for many of the neurobiological effects associated with nicotine exposure. Activation of these receptors results in the opening of sodium and calcium ion channels, leading to neuronal depolarization and enhanced synaptic activity (Changeux, 2010). Consequently, numerous neurotransmitters involved in the regulation of behavior and brain function are released.

One of the most important outcomes of nicotinic receptor activation is the increased release of dopamine within the mesolimbic reward pathway. Nicotine stimulates dopaminergic neurons in the ventral tegmental area, resulting in elevated dopamine levels in the nucleus accumbens. This process produces pleasurable sensations and reinforces behaviors associated with repeated nicotine consumption (De Biasi & Dani, 2011). Consequently, this mechanism is considered a key contributor to the development of nicotine addiction.

In addition to its effects on the dopaminergic system, nicotine also influences several other neurotransmitter systems. Studies have shown that nicotine increases the release of serotonin, which is

involved in mood regulation; norepinephrine, which affects alertness and attention; glutamate, which plays a central role in learning and memory; and gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the brain (Picciotto & Mineur, 2014). This broad influence on multiple neurochemical pathways helps explain the complex effects of nicotine on human psychological functioning.

Another characteristic feature of nicotine action is the development of nicotinic receptor desensitization. Prolonged exposure to nicotine gradually reduces receptor responsiveness to stimulation, resulting in the need for higher doses of nicotine to achieve the same physiological and psychological effects (Dani & Bertrand, 2007). This process represents one of the fundamental mechanisms underlying the development of tolerance and nicotine dependence.

Neuroimaging studies further suggest that regular nicotine use may induce functional alterations in brain regions involved in impulse control, decision-making, and emotional regulation. Particularly notable changes have been observed in the prefrontal cortex, hippocampus, and amygdala, all of which play crucial roles in cognitive and emotional processing (Yuan et al., 2015).

Overall, nicotine exerts its effects on the brain primarily through the activation of nicotinic acetylcholine receptors and the modulation of multiple neurotransmitter systems. These mechanisms are responsible for both the short-term enhancement of attention and concentration and the long-term development of tolerance, dependence, and addiction observed among regular users of nicotine-containing products.

3.2. Nicotine, the Reward System, and Dopamine

One of the most important aspects of nicotine's effects on the brain is its influence on the mesolimbic dopaminergic pathway, commonly referred to as the reward system. This system plays a crucial role in regulating motivation, pleasure perception, and the learning of behaviors associated with rewarding outcomes. Under physiological conditions, it is activated by natural rewarding stimuli such as food consumption, physical activity, and social interactions. Similar to other addictive substances, nicotine exploits these neurobiological mechanisms, resulting in artificial stimulation of the brain reward system (De Biasi & Dani, 2011).

A key structure involved in this process is the ventral tegmental area (VTA), located within the midbrain. Dopaminergic neurons originating in the VTA project to the nucleus accumbens, prefrontal cortex, and other limbic structures responsible for emotional regulation and behavioral control. When nicotine binds to nicotinic receptors located on dopaminergic neurons, dopamine release within the nucleus accumbens increases, producing feelings of pleasure and satisfaction (Dani & Bertrand, 2007).

Elevated dopamine levels serve as a reinforcing signal, informing the brain that a particular behavior was beneficial and should be repeated. This mechanism forms the basis of conditioning processes and contributes to the development of behaviors associated with regular nicotine consumption. Each subsequent exposure to nicotine strengthens neuronal pathways linked to substance use, gradually increasing the risk of addiction (Benowitz, 2010).

Neuroadaptation represents another important component of nicotine dependence. Chronic nicotine exposure induces changes in both the number and sensitivity of nicotinic receptors, as well as alterations in dopaminergic system functioning. Over time, the brain adapts to the continuous presence of nicotine, resulting in reduced activity of the natural reward system. Consequently, individuals with nicotine dependence often derive less satisfaction from everyday activities and become increasingly reliant on nicotine as a source of pleasure (Changeux, 2010).

Long-term nicotine use also contributes to the development of tolerance. This phenomenon means that progressively larger doses of nicotine are required to achieve the same psychological effects. Tolerance results, in part, from nicotinic receptor desensitization and neurochemical adaptations occurring within multiple neurotransmitter systems (Picciotto & Mineur, 2014). As a characteristic feature of addiction, tolerance frequently leads to increased frequency of nicotine consumption and greater dependence on nicotine-containing products.

When nicotine administration is discontinued, withdrawal symptoms typically emerge. These symptoms are associated with reduced dopaminergic activity and disturbances in the neurochemical balance established during chronic nicotine exposure. Common withdrawal symptoms include irritability, anxiety, depressed mood, impaired concentration, increased appetite, and a strong craving for nicotine (De Biasi & Dani, 2011). Such symptoms represent one of the primary reasons for unsuccessful smoking cessation attempts.

Contemporary neuroimaging studies indicate that individuals with nicotine dependence exhibit altered activity in brain regions involved in impulse control and decision-making, particularly within the prefrontal cortex. These changes may impair an individual's ability to regulate substance-seeking behaviors despite awareness of the negative health consequences associated with nicotine use (Prochaska & Benowitz, 2019).

It is important to emphasize that the mechanisms responsible for reward system activation are largely independent of the source of nicotine. Conventional cigarettes, electronic cigarettes, heated tobacco products, and nicotine pouches all deliver nicotine to the brain and activate the same neurobiological pathways involved in the development of addiction (England et al., 2015).

Taken together, the available evidence indicates that nicotine's effects on the mesolimbic dopaminergic pathway constitute the primary mechanism underlying the development of addiction. Increased dopamine release, neuroadaptation, tolerance, and withdrawal symptoms collectively form the biological basis of nicotine dependence, which remains one of the most prevalent forms of addiction worldwide.

3.3. Nicotine and Cognitive Functions

The effects of nicotine on cognitive functions represent one of the most extensively investigated areas of neurobiological research. Cognitive functions encompass processes involved in the acquisition, processing, storage, and utilization of information, including attention, memory, learning, executive functions, and information-processing speed. Unlike many other psychoactive substances, nicotine exhibits stimulant properties that may lead to temporary improvements in selected cognitive abilities (Heishman et al., 2010).

One of the most well-documented effects of nicotine is the enhancement of attentional performance. Experimental studies have demonstrated that individuals exposed to nicotine achieve better results in tasks requiring sustained attention and rapid responses to external stimuli. This effect is primarily attributed to the activation of nicotinic receptors in the cerebral cortex and the increased release of acetylcholine and norepinephrine, neurotransmitters that play a crucial role in attentional processes (Newhouse et al., 2004).

A meta-analysis conducted by Heishman et al. (2010) further demonstrated that nicotine may improve working memory and information-processing speed. Individuals exposed to nicotine showed enhanced performance in tasks involving the temporary storage and manipulation of information. The authors reported that these effects were particularly evident in situations requiring intensive cognitive effort.

Another important area of investigation concerns the effects of nicotine on learning processes. Nicotinic receptors are abundantly expressed in the hippocampus, a brain structure responsible for the formation of new memories and the consolidation of long-term memory. Activation of these receptors may enhance synaptic plasticity and facilitate the formation of new neuronal connections, thereby supporting learning and memory-related processes (Levin et al., 2006).

Due to these observed cognitive effects, nicotine has been investigated as a potential therapeutic agent in certain neurological and psychiatric disorders. The literature describes the possible application of nicotinic receptor agonists in the treatment of Alzheimer's disease, schizophrenia, and attention-deficit/hyperactivity disorder (ADHD). Some studies suggest that stimulation of nicotinic receptors may partially compensate for cognitive deficits associated with these conditions (Newhouse et al., 2004). Nevertheless, the potential therapeutic benefits do not justify the use of nicotine as a cognitive enhancer because of its substantial addictive potential.

It should also be emphasized that the beneficial effects observed following nicotine administration are generally short-lived. Among regular users of nicotine-containing products, improvements in concentration and memory may not necessarily reflect genuine enhancement of cognitive abilities but rather the temporary alleviation of withdrawal symptoms resulting from declining nicotine levels in the body (Benowitz, 2010). In such cases, the administration of nicotine merely restores cognitive functioning to pre-withdrawal levels.

An increasing body of evidence also points to the negative consequences of chronic nicotine exposure on cognitive performance. Long-term nicotine use may induce functional alterations within the prefrontal cortex, a brain region responsible for planning, decision-making, and behavioral control. Individuals with nicotine dependence may exhibit impaired executive functioning, increased impulsivity, and difficulties regulating behavioral responses (Picciotto & Mineur, 2014).

Particularly adverse consequences may occur among individuals who begin using nicotine during adolescence. During this developmental period, the brain undergoes intensive maturation, and disruptions in neuroplastic processes may result in long-lasting cognitive alterations. Research findings suggest that nicotine exposure at a young age may increase the risk of memory deficits, impaired concentration, and learning difficulties later in life (Yuan et al., 2015).

Overall, nicotine may temporarily enhance selected cognitive functions, particularly attention, working memory, and information-processing speed. These effects result from the activation of nicotinic receptors and the modulation of neurotransmitter systems involved in cognitive processing. However, chronic nicotine use is associated with the risk of adverse neurobiological changes and the development of addiction, which substantially limits the potential benefits of its stimulant effects.

3.4. Development of Nicotine Addiction

Nicotine addiction is a chronic disorder characterized by the compulsive use of nicotine despite awareness of its harmful health consequences. According to contemporary neurobiological models, addiction develops as a result of complex interactions among alterations in the brain reward system, learning processes, memory mechanisms, and environmental and genetic factors (Benowitz, 2010).

The development of nicotine addiction begins with the repeated activation of nicotinic acetylcholine receptors by nicotine. Continuous stimulation of these receptors leads to increased dopamine release within the mesolimbic reward pathway, reinforcing behaviors associated with nicotine consumption. Over time, the brain begins to associate specific situations, locations, emotions, and environmental cues with the pleasurable effects of nicotine, resulting in the formation and consolidation of nicotine-related habits (De Biasi & Dani, 2011).

An important component of addiction development is neuroadaptation. In response to chronic nicotine exposure, changes occur in both the number and functioning of nicotinic receptors. Paradoxically, unlike many other receptor systems, prolonged nicotine exposure may increase the number of certain nAChR subtypes, particularly $\alpha 4\beta 2$ receptors. This phenomenon, known as receptor upregulation, represents one of the biological mechanisms responsible for maintaining nicotine dependence (Changeux, 2010).

As addiction progresses, tolerance to nicotine develops. This process involves the gradual adaptation of the body to the presence of nicotine, requiring higher doses or more frequent nicotine administration to achieve the same effects. Tolerance affects both the psychological and physiological responses to nicotine. Consequently, individuals with nicotine dependence often increase the number of cigarettes smoked per day or the frequency of use of other nicotine-containing products (Picciotto & Mineur, 2014).

Another characteristic feature of nicotine addiction is the occurrence of withdrawal symptoms following cessation of nicotine use. These symptoms typically emerge within a few hours after the last dose and reach their peak intensity during the first days of abstinence. Common withdrawal symptoms include irritability, anxiety, depressed mood, impaired concentration, sleep disturbances, increased appetite, and an intense desire to consume nicotine, commonly referred to as nicotine craving (Prochaska & Benowitz, 2019).

Learning and memory processes also play a crucial role in maintaining nicotine addiction. Research indicates that cues associated with nicotine use can activate the same brain regions that are stimulated during direct nicotine consumption. The sight of a cigarette, the smell of tobacco smoke, or social situations associated with smoking may trigger strong neural responses and increase the risk of relapse even after prolonged periods of abstinence (De Biasi & Dani, 2011).

Contemporary neuroimaging studies have further demonstrated that chronic nicotine use affects the functioning of the prefrontal cortex, a brain region responsible for impulse control and decision-making. Altered activity within these areas may reduce an individual's ability to regulate substance-seeking behaviors and may hinder successful smoking cessation attempts (Yuan et al., 2015).

Genetic factors also contribute significantly to the development of nicotine addiction. It is estimated that the heritability of susceptibility to nicotine dependence ranges from approximately 40% to 70%. Particular attention has been devoted to genes involved in nicotinic receptor function and nicotine metabolism. Individual genetic differences may partly explain why some individuals develop nicotine addiction more rapidly than others despite similar levels of exposure (Benowitz, 2010).

In recent years, increasing attention has been directed toward the role of novel nicotine products, including electronic cigarettes, heated tobacco products, and nicotine pouches. Although these products are often perceived as less harmful than conventional cigarettes, they still deliver nicotine to the brain and activate the same neurobiological pathways responsible for addiction development. Consequently, their use may contribute to the maintenance of nicotine dependence, particularly among younger users (England et al., 2015).

In summary, nicotine addiction results from complex neurobiological processes involving activation of the reward system, neuroadaptation, the development of tolerance, and the occurrence of withdrawal symptoms. These mechanisms reinforce nicotine-seeking behaviors and make smoking cessation a significant biological and psychological challenge.

3.5. Effects of Nicotine on the Developing Adolescent Brain

The effects of nicotine on the developing adolescent brain constitute one of the most intensively studied areas of contemporary neurobiological research. Scientific interest in this topic has increased considerably due to the growing popularity of electronic cigarettes and other nicotine-containing products among adolescents and young adults. Unlike the mature adult brain, the adolescent brain undergoes extensive structural and functional development, making it particularly vulnerable to the effects of psychoactive substances, including nicotine (Yuan et al., 2015).

Adolescence is characterized by dynamic changes within the central nervous system. During this period, neuronal networks are reorganized, unnecessary synaptic connections are eliminated through the process of synaptic pruning, and the prefrontal cortex undergoes substantial maturation. These developmental processes are essential for the acquisition of fully developed cognitive and emotional capacities, including planning, impulse control, risk assessment, and decision-making. However, exposure to nicotine may disrupt these processes and lead to long-lasting neurobiological consequences (Goriounova & Mansvelder, 2012).

Experimental studies have demonstrated that nicotine interacts with developing nicotinic acetylcholine receptors, which play a critical role in brain maturation. Excessive stimulation of these receptors during adolescence may result in abnormal neuronal network organization and alterations in neurotransmitter systems involved in attention, memory, motivation, and emotional regulation (Dani & Bertrand, 2007).

Particular attention has been devoted to nicotine's effects on the dopaminergic system. During adolescence, the brain reward system exhibits heightened sensitivity to reinforcing stimuli. Consequently, young nicotine users may experience stronger rewarding effects than adults. Enhanced activation of the dopaminergic pathway may accelerate the development of addiction and promote the consolidation of nicotine-related behaviors (Yuan et al., 2015).

Research findings also indicate an association between nicotine use during adolescence and impaired cognitive functioning. Individuals who initiate nicotine use at a young age more frequently exhibit difficulties with attention, working memory, and executive functions. Some studies suggest that these impairments may persist into adulthood, even after nicotine use has been discontinued (Goriounova & Mansvelder, 2012).

Another important issue concerns the impact of nicotine on adolescent mental health. An increasing number of studies report associations between regular nicotine use and a higher risk of anxiety symptoms, mood disturbances, and emotional difficulties. Although these relationships are multifactorial in nature, alterations in dopaminergic and serotonergic neurotransmission are believed to contribute significantly to the observed effects (Picciotto & Mineur, 2014).

A further public health concern is the so-called "gateway effect," according to which early nicotine exposure may increase susceptibility to the use of other psychoactive substances later in life. Research suggests that nicotine-induced neurobiological changes may enhance brain sensitivity to the rewarding effects of alcohol, cannabinoids, and other addictive substances (Yuan et al., 2015).

In recent years, the rapid increase in electronic cigarette use among adolescents has become a major concern. These products are often perceived as less harmful than conventional cigarettes, which may lead to an underestimation of the risks associated with nicotine exposure. Nevertheless, regardless of the delivery method, nicotine acts on the same receptors and neurobiological pathways involved in addiction development (England et al., 2015).

From a public health perspective, preventive measures aimed at reducing nicotine exposure among adolescents are of particular importance. Health education programs, regulations governing the sale of nicotine-containing products, and initiatives promoting healthy lifestyles may contribute to reducing the number of young individuals who begin using nicotine.

The available evidence demonstrates that adolescence represents a period of heightened vulnerability to the effects of nicotine. Exposure to nicotine during this critical stage of development may disrupt neurodevelopmental processes, impair cognitive functioning, increase the risk of emotional and mental health problems, and accelerate the development of addiction. Therefore, protecting young people from exposure to nicotine-containing products remains one of the major challenges of contemporary public health.

4. Discussion

The present literature review demonstrates that nicotine exerts a multifaceted influence on human brain function. Its mechanisms of action involve the activation of nicotinic acetylcholine receptors, modulation of multiple neurotransmitter systems, and alterations in brain structures responsible for cognitive processes, emotional regulation, and addiction development. The available evidence confirms that nicotine induces significant neurobiological changes following both short-term and long-term exposure.

One of the most important findings emerging from the reviewed studies is that the effects of nicotine extend beyond its addictive properties. Numerous experimental investigations have shown that nicotine may temporarily enhance selected cognitive functions, including attention, working memory, and information-processing speed (Heishman et al., 2010; Newhouse et al., 2004). These effects are primarily attributed to nicotinic receptor activation and increased neurotransmitter release within neural pathways involved in cognitive performance.

However, it should be emphasized that the potential cognitive benefits associated with nicotine use are generally short-lived and do not outweigh the risks associated with regular nicotine consumption. As tolerance and biological dependence develop, adaptive changes occur within the nervous system that may contribute to impaired behavioral control and the maintenance of addictive behaviors (Benowitz, 2010; De Biasi & Dani, 2011).

The reviewed literature consistently indicates that the mesolimbic dopaminergic pathway plays a central role in the development of nicotine addiction. Repeated activation of the reward system reinforces nicotine-seeking behaviors and contributes to the emergence of withdrawal symptoms following cessation. These mechanisms closely resemble those observed in other forms of substance dependence, highlighting the strong addictive potential of nicotine (Changeux, 2010; Picciotto & Mineur, 2014).

Particularly important are the findings concerning the effects of nicotine on the developing adolescent brain. Current evidence suggests that nicotine exposure during adolescence may induce long-lasting structural and functional alterations in the brain, increasing vulnerability to addiction and cognitive impairment later in life (Yuan et al., 2015). Given the rapidly increasing popularity of electronic cigarettes among young people, these findings have substantial implications for preventive strategies and public health policies.

Attention should also be directed toward the changing landscape of nicotine-containing products. Although many modern nicotine delivery systems are marketed as less harmful alternatives to conventional cigarettes, current evidence indicates that nicotine itself continues to affect the same neurobiological pathways involved in addiction development (England et al., 2015). Therefore, reducing exposure to tobacco combustion products does not eliminate the risks associated with nicotine-induced alterations in brain function.

Several limitations of the present review should be acknowledged. A considerable proportion of the available evidence regarding nicotine's mechanisms of action is derived from experimental models and observational studies, which may limit the ability to establish definitive causal relationships. Furthermore, methodological differences among studies, including variations in study populations, nicotine dosage, and methods used to assess cognitive performance, may influence the interpretation and comparability of findings.

Future research should focus on the long-term consequences of using emerging nicotine products, particularly among adolescents and young adults. Further investigation is also warranted regarding the relationship between nicotine use and mental health, neurodegenerative processes, and the potential application of knowledge concerning nicotinic receptors in the development of novel therapeutic approaches.

In conclusion, the available scientific evidence indicates that nicotine exerts complex effects on brain function. Although nicotine may temporarily improve certain cognitive abilities, long-term exposure is associated with a substantial risk of addiction and adverse neurobiological consequences. Of particular concern are the effects of nicotine exposure during adolescence, which may have lasting implications for health, cognition, and overall functioning throughout life.

5. Conclusions

Based on the conducted literature review, the following conclusions can be drawn:

1. Nicotine directly affects brain function through the activation of nicotinic acetylcholine receptors and the modulation of multiple neurotransmitter systems, including the dopaminergic, serotonergic, and noradrenergic pathways.
2. One of the primary mechanisms underlying nicotine's effects is the stimulation of the mesolimbic reward pathway, resulting in increased dopamine release and reinforcement of nicotine-seeking behaviors.
3. Short-term nicotine exposure may temporarily enhance selected cognitive functions, including attention, working memory, and information-processing speed.
4. Long-term nicotine use leads to adaptive changes within the nervous system, contributing to the development of tolerance as well as psychological and physiological dependence.
5. Withdrawal symptoms that emerge following nicotine cessation constitute a major obstacle to successful smoking cessation and contribute significantly to relapse.
6. The adverse effects of nicotine are particularly pronounced during adolescence, when the brain is undergoing intensive development. Nicotine exposure during this period may result in long-lasting neurobiological alterations and increased susceptibility to addiction and cognitive impairment.
7. Regardless of the method of nicotine delivery—whether through conventional cigarettes, electronic cigarettes, heated tobacco products, or nicotine pouches—nicotine activates the same neurobiological mechanisms involved in addiction development.
8. In light of current scientific evidence, preventive measures aimed at reducing nicotine exposure, particularly among children and adolescents, should constitute an essential component of public health strategies.
9. Further research is needed to evaluate the long-term effects of emerging nicotine products on brain function and mental health outcomes.

In conclusion, nicotine is a substance that exerts complex effects on the central nervous system. Despite its potential short-term stimulatory effects on selected cognitive functions, regular nicotine use is associated with a substantial risk of addiction and long-term neurobiological consequences, particularly among young individuals.

Author's contribution:

Conceptualisation: Michał Kieres

Methodology: Michał Kieres, Klaudia Karolina Wójcik

Software: Aleksander Kacper Kierys, Adrianna Krupska

Check: Anna Kropiwnicka, Anna Katarzyna Żurawska- Zadrożna, Joanna Zuzanna Gugulska

Formal analysis: Krzysztof Sebastian Habirek, Michał Mieczysław Parus, Maja Maria Porażko

Investigation: Julia Anna Czarnecka, Zuzanna Marta Marcinkowska

Resources: Michał Kieres, Klaudia Karolina Wójcik, Krzysztof Sebastian Habirek, Maja Maria Porażko, Zuzanna Marta Marcinkowska

Data curation: Anna Kropiwnicka, Anna Katarzyna Żurawska- Zadrożna, Joanna Zuzanna Gugulska, Michał Kieres, Klaudia Karolina Wójcik, Michał Mieczysław Parus

Writing –rough preparation: Michał Kieres, Klaudia Karolina Wójcik, Krzysztof Sebastian Habirek, Maja Maria Porażko, Zuzanna Marta Marcinkowska, Anna Kropiwnicka

Writing –review and editing: Michał Kieres, Klaudia Karolina Wójcik

Project administration: Michał Kieres

REFERENCES

1. Benowitz, N. L. (2010). Nicotine addiction. *New England Journal of Medicine*, 362(24), 2295–2303.
2. Changeux, J. P. (2010). Nicotinic receptors and nicotine addiction. *Comptes Rendus Biologies*, 333(5–6), 421–425.
3. Dani, J. A., & Bertrand, D. (2007). Nicotinic acetylcholine receptors and nicotinic cholinergic mechanisms of the central nervous system. *Annual Review of Pharmacology and Toxicology*, 47, 699–729.
4. De Biasi, M., & Dani, J. A. (2011). Reward, addiction, withdrawal to nicotine. *Annual Review of Neuroscience*, 34, 105–130.
5. England, L. J., Bunnell, R. E., Pechacek, T. F., Tong, V. T., & McAfee, T. A. (2015). Nicotine and the developing human: A neglected element in the electronic cigarette debate. *American Journal of Preventive Medicine*, 49(2), 286–293.
6. Goriounova, N. A., & Mansvelder, H. D. (2012). Short- and long-term consequences of nicotine exposure during adolescence for prefrontal cortex neuronal network function. *Cold Spring Harbor Perspectives in Medicine*, 2(12), a012120.
7. Heishman, S. J., Kleykamp, B. A., & Singleton, E. G. (2010). Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacology*, 210(4), 453–469.
8. Levin, E. D., McClernon, F. J., & Rezvani, A. H. (2006). Nicotinic effects on cognitive function: Behavioral characterization, pharmacological specification, and anatomic localization. *Psychopharmacology*, 184(3–4), 523–539.
9. Newhouse, P. A., Potter, A., & Singh, A. (2004). Effects of nicotinic stimulation on cognitive performance. *Current Opinion in Pharmacology*, 4(1), 36–46.
10. Picciotto, M. R., & Mineur, Y. S. (2014). Molecules and circuits involved in nicotine addiction: The many faces of smoking. *Neuropharmacology*, 76, 545–553.
11. Prochaska, J. J., & Benowitz, N. L. (2019). Current advances in research in treatment and recovery: Nicotine addiction. *Science Advances*, 5(10), eaay9763.
12. World Health Organization. (2023). *WHO report on the global tobacco epidemic 2023: Protect people from tobacco smoke*. World Health Organization.
13. Yuan, M., Cross, S. J., Loughlin, S. E., & Leslie, F. M. (2015). Nicotine and the adolescent brain. *The Journal of Physiology*, 593(16), 3397–3412.
14. Leslie, F. M. (2020). Unique, long-term effects of nicotine on adolescent brain. *Pharmacology Biochemistry and Behavior*, 197, 173010.