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Review Article

Fluoride exposure as an environmental risk factor for anxiety and depression: A narrative review

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Abstract

Fluoride is a ubiquitous environmental agent widely utilized in public health for the prevention of dental caries. Despite its benefits, chronic exposure, particularly during developmental windows of heightened vulnerability, has been associated with adverse neurodevelopmental outcomes. This narrative review evaluates the current evidence linking prolonged fluoride exposure via drinking water to anxiety and depression, synthesizing findings from both preclinical and human studies. Animal research robustly demonstrates that fluoride crosses the blood-brain barrier, accumulates in limbic structures such as the hippocampus, and promotes anxiety- and depression-like behaviors. These behavioral phenotypes are underpinned by multifaceted neurotoxic mechanisms, including mitochondrial dysfunction, sustained neuroinflammation, disruption of key neurotransmitter systems (glutamate, GABA, dopamine, and serotonin), and a critical reduction in brain-derived neurotrophic factor (BDNF), which is essential for synaptic plasticity and emotional regulation. In contrast, the human evidence remains sparse and less direct. While numerous epidemiological studies have reported associations between fluoride exposure and reduced cognitive performance or increased risk of attention-deficit/hyperactivity disorder, data on internalizing disorders are strikingly limited. To date, only a single study has directly assessed clinical anxiety and depression, finding an association with somatic symptoms but not core affective dimensions. This significant discrepancy underscores the considerable challenge in translating preclinical neurotoxicological findings to complex human mental health conditions. In light of the global prevalence of affective disorders and the substantial populations residing in endemic fluoride areas, there is an urgent need for rigorous longitudinal studies that utilize validated psychiatric instruments. Future research must prioritize investigating fluoride as a potential modifiable environmental risk factor for mental health, with specific attention to developmental timing and sex-specific susceptibilities.

Keywords: Fluoride, Neurotoxicity, Anxiety, Depression, Environmental risk factor, Neurodevelopment, Mental health

Introduction

Fluoride, a highly reactive chemical element, occurs naturally as part of compounds called fluorides, which are present in rocks, soil, air, and water in both residential and industrial areas [1]. Despite its well-established role in dental health, where it strengthens enamel and prevents tooth decay, excessive exposure to fluoride poses a significant public health problem. It is estimated that around 200 million people worldwide live in areas with endemic fluorosis [2], a condition caused by high fluoride consumption, which in regions such as parts of China, India, and Mexico often exceeds the levels recommended by the World Health Organization (WHO).

Beyond its dental and skeletal effects, emerging evidence suggests that exposure to fluoride, particularly during critical periods of development, has short- and long-term neurotoxic effects [3–

5]. Animal and human studies indicate that prenatal and perinatal exposure may be associated with loss of intelligence, hyperactivity, and mood changes. While research on cognitive impairment has advanced, there is a significant gap in understanding its impact on mental health, specifically on disorders such as anxiety and depression [3–5]. Anxiety and depression are two of the most prevalent mental disorders globally, often coexisting and sharing therapeutic approaches. The complexity of these disorders in humans, which integrate cognitive, social, and cultural dimensions, presents ethical and methodological challenges for their direct study. In this context, animal models have become indispensable tools for investigating their biological and behavioral substrates. Paradigms such as the Forced Swimming Test (FST) and Chronic Moderate Stress (CMS) allow modeling aspects such as behavioral hopelessness and anhedonia, facilitating the exploration of pathophysiological mechanisms such as HPA axis dysregulation and altered synaptic plasticity. However, it is crucial to recognize the translational limitations of these models, as they cannot capture the entirety of the human experience. Therefore, the objective of this narrative review is to synthesize and critically evaluate the available scientific evidence on the association between fluoride exposure and the onset of anxiety- and depression-related behaviors, integrating findings from preclinical studies in animal models with limited epidemiological evidence in humans. In addition, we seek to explore the proposed pathophysiological mechanisms (such as oxidative stress, neuroinflammation, and dysfunction in signaling pathways such as SIRT1/p53) to provide a comprehensive perspective on fluoride as a potential environmental risk factor for neuropsychiatric disorders.

Methods: Narrative Review Approach

To meet the objective, an extensive, albeit non-systematic, literature search was conducted in the PubMed, Scopus, and Web of Science databases, considering articles published up to September 2025. The search strategy combined key terms such as (Fluoride) AND (Anxiety OR Depression OR Depressive behavior) AND (rodent OR mouse OR rat OR human). The selection of literature was guided by the following principles: priority was given to original studies and reviews that evaluated fluoride exposure and its relationship to behavioral outcomes of anxiety and depression or to relevant neurobiological mechanisms. The synthesis of information was qualitative, organizing the evidence thematically to provide a logical and critical overview, identifying trends, consistencies, and gaps in the current literature.

Research in Animal Models

It is known that fluoride can cross the blood-brain barrier. Long-term exposure to fluoride affects the central nervous system through various mechanisms. This exposure can affect structures such as the hippocampus, causing behavioral changes and mood disorders.

Studies on rats exposed to sodium fluoride determined that the adverse effects on learning ability and memory are sex- and dose-specific, being more significant in male rats [6]. Conversely, maternal exposure to low concentrations of sodium fluoride (5–10 mg/L) reduced anxiety-related behaviors in young and adult female rats compared to young male offspring. It also caused an increase in hyperactivity in adult offspring born to fluoride-exposed mothers [7]. Studies on animal models have suggested a link between fluoride exposure and an increased risk of ADHD (Attention Deficit Hyperactivity Disorder), which is especially prevalent in males [6,8,9]. Our working group in a previous work [10] examined the effects of experimental fluoride poisoning on anxiety and depression symptoms in animal models from 2013 to 2025. The analysis revealed a significant pro-depressive effect in the tail suspension test (TST) with an SMD of 1.53 ($p<0.001$). Additionally, a clear dose-response relationship was recorded ($\beta=0.069$ per ppm, $p=0.0004$). Males appear particularly susceptible to fluoride's effects on pro-depressive symptom expression ($\beta=2.68$, $p=0.010$) and decreased IQ. These findings suggest that critical exposure windows may vary by sex. However, it should be noted that the existing information regarding sex differences in animals exposed to fluoride consumption is still very limited, so further research is needed to elucidate any potential relationships in this field. On the other hand, it has been reported that fluoride consumption affects neurotransmitter levels, [11] such as those of glutamate and histamine. An increase in these neurotransmitters has been recorded after exposure to fluoride, while levels of acetylcholine and dopamine decrease. Similarly, exposure to fluorides has been reported to cause behavioral changes in mice, suggesting an imbalance between nervous system excitation and inhibition, depending on the dose and duration of fluoride exposure [12]. Exposed mice exhibit higher serotonin concentrations and higher levels of fluoride in the brain compared to unexposed mice after exposure [13]. Serotonin (5-hydroxytryptamine [5-HT]) is a neurotransmitter strongly associated with depression and anxiety development [14]. Inadequate levels of 5-HT in the brain have led to the use of 5-HT1A agonists, 5-HT1 antagonists, and 5-HT2 antagonists in the treatment of anxiety disorders [15]. Exposure to fluoride has been associated with increased excitation in the hippocampus, memory impairment, and behaviors linked to anxiety and depression in adult mice [16,17]. This may be due to fluoride crossing the blood-brain barrier and causing maladaptive changes in the hippocampus [12]. Based on these studies, it has been suggested that fluoride may cause behavioral changes associated with anxiety and depression by altering serotonin levels and/or affecting specific brain regions in response to exposure (e.g., hippocampal excitation) [12]. Other studies in rats have reported neuroanatomical abnormalities, including swollen mitochondria, disrupted myelin sheaths, enlarged axons, and vacuolated Schwann cells [11]. A parallel set of observations was made in mouse cells. Cells that had been exposed to fluoride for a period of 24 hours demonstrated an increase in cell swelling, agglutination, vacuole formation, edema, and loss of synapses when compared to the control group. The release of the neurotransmitter glutamate was disrupted, and cellular structural damage occurred, thereby supporting the hypothesis of fluoride-induced neurotoxicity [18]. The study of molecular mechanisms in psychiatric disorders has recently highlighted the role of sirtuins, a family of NAD^+-dependent deacetylases. Among these, Sirtuin 1 (SIRT1) has been identified as a key player in neuronal proliferation and has emerged as a promising therapeutic target for depression and anxiety. This scoping review examines the complex interaction between fluoride and SIRT1, exploring its context-dependent nature and potential relevance to mental health. The extant evidence suggests that fluoride does not invariably act as a SIRT1 activator or inhibitor; its effect is contingent on cell type, dose, and exposure duration. In peripheral cells, such as ameloblasts, fluoride has been shown to induce an adaptive response that upregulates SIRT1 expression, thereby counteracting oxidative stress [19,20]. In contrast, in neuronal and glial models, fluoride has been observed to inhibit SIRT1 activity, resulting in increased p53 acetylation, mitochondrial dysfunction, and apoptosis [21].

Recent studies have further delineated the disruptions in SIRT1-BDNF signaling and SIRT1-dependent mitochondrial biogenesis in neural cells exposed to fluoride [22–24]. Despite these compelling preclinical findings, the translation to human mental health remains limited. To date, there have been no clinical studies that have established a direct correlation between environmental fluoride exposure and SIRT1 activity in the human brain, nor have there been any studies that have demonstrated a link between environmental fluoride exposure and psychiatric disorders. Consequently, while the fluoride-SIRT1 axis offers a valuable model for understanding molecular mechanisms of neurotoxicity, future research must focus on human biomonitoring studies and the SIRT1-BDNF-PGC-1α axis to bridge the gap between environmental exposure and neuropsychiatric dysfunction. Furthermore, it has been proposed that the glycogen synthase kinase 3β (GSK-3β)/β-catenin signaling pathway may be impaired by fluoride exposure, potentially resulting in neuronal death or apoptosis [25]. It has been hypothesized that sodium fluoride, in a dose-dependent manner, impedes neurogenesis in rats via this specific pathway. When high doses of fluoride were administered to rats, GSK-3β activity was induced, resulting in reduced ß-catenin signaling, which decreased in the nucleus and had reduced cytoplasmic expression. This phenomenon could be interpreted as a sign of neurotoxicity [26].

Human Studies: Cognitive Function and Prior Analyses

A body of scientific evidence suggests that exposure to fluoride, particularly during critical stages of development, may act as a neurotoxic agent in humans, with both cognitive and behavioral implications. In the cognitive sphere, research has centered on its potential impact on IQ. Systematic reviews and meta-analyses consistently conclude that higher fluoride exposure is associated with lower IQ in children [27–30], who are more vulnerable than adults to its neurotoxic effects. Subsequent research, including that by the renowned toxicologist Philippe Grandjean in 2019, has reinforced these findings, even suggesting that fluoride levels considered safe in drinking water could affect neurodevelopment. Beyond the realm of cognition, research has established a correlation between fluoride exposure and mental health concerns, as well as neurobehavioral conditions. Epidemiological studies have associated elevated fluoride levels in drinking water with a higher prevalence of ADHD diagnoses and symptoms of hyperactivity and inattention. In this regard, prenatal exposure emerges as a period of particular vulnerability, potentially triggering delayed behavioral effects [29]. However, the extant evidence on mental health is more limited. While there is a preponderance of research in animal models demonstrating a link between fluoride exposure and anxiety and hyperactivity, there is a paucity of research in human subjects. A study conducted in humans found an association with somatization symptoms, but not with anxiety or depression, which is unexpected given the typical comorbidity of these conditions. The mechanisms proposed for this neurotoxicity include mitochondrial damage (with dysfunction in biogenesis and loss of structure), neuroinflammation, alterations in neurotransmitters, and disruption of key signaling pathways. Furthermore, a discrepancy in outcomes based on gender has been documented, with male subjects exhibiting a more pronounced response, characterized by a substantial decline in IQ and an increased prevalence of somatization symptoms [31].

Impact on Mental Health: Anxiety and Depression

While research in animal models has demonstrated a consistent correlation between fluoride exposure and behaviors indicative of anxiety and depression [9,32], epidemiological evidence in humans is limited and findings are less conclusive. The most frequently cited cross-sectional study that directly addresses this issue was conducted by Malin and Till [33]. A team of researchers recently conducted a study that analyzed data from the US National Health and Nutrition Examination Survey (NHANES). The objective of the study was to investigate the association between urinary fluoride levels and symptoms of depression and anxiety. The researchers' findings were unexpected, as they revealed no significant association between fluoride exposure and symptoms of anxiety or depression. However, a statistically significant association was reported between higher scores on somatization and the presence of psychological origin in physical complaints. This result is particularly intriguing given the high comorbidity that typically exists between somatization, anxiety, and depression, suggesting that the mechanisms of action of fluoride on mental health may be specific and warrant further investigation [33]. A more recent study in China explored this relationship in a different way and found no direct association between urinary fluoride levels and depression in adults. However, their analysis indicated that fluoride exposure could interact with or be mediated by other factors, such as thyroid function, which in turn is linked to mood disorders. This finding suggests the potential for indirect and multifaceted relationships [34]. In contrast to the paucity of studies in adult populations, research in child populations is virtually nonexistent. This finding is of particular significance in the context of existing literature, given the recognized vulnerability of the developing brain to neurotoxins and the prevalence of anxiety disorders during childhood and adolescence. In summary, the evidence in humans linking fluoride exposure to anxiety and depression is limited and inconclusive, contrasting with the solid findings in animals. The extant literature on the subject is inconclusive, as the studies conducted thus far have been unable to establish a direct and consistent association. However, the studies do suggest the possibility of effects on related dimensions, such as somatization, or indirect mechanisms. The necessity for additional longitudinal studies, particularly in child and adolescent populations, is paramount. These studies must utilize precise exposure biomarkers and validated diagnostic tools to evaluate mental health. According to the World Health Organization (WHO), depression and anxiety disorders are projected to affect approximately 5% and 3.6% of the global population in 2023, respectively. These conditions are a significant cause of disability globally, and suicide, often linked to them, was one of the top 20 causes of death in 2020 [35].

In summary

A limited number of studies have examined the association between mental health conditions, such as anxiety and depression, and prolonged exposure to fluoride in drinking water. Fluoride has been demonstrated to exert beneficial effects on the human body, including the suppression of dental caries. However, it has also been observed that this chemical accumulates in brain tissue over time [31,32,34]. Moreover, exposure to fluoride has been shown to influence brain systems that are critical for mood regulation, including:

1. BDNF expression is reduced. A critical factor in promoting neuronal survival, whose decrease has been associated with the neurotrophic hypothesis of depression.
2. It has been demonstrated to modify the equilibrium between GABA and glutamate within the nervous system. It has been demonstrated that the expression of enzymes and transporters involved in the synthesis of GABA (the main inhibitory neurotransmitter) is decreased, resulting in a disruption of the equilibrium of excitation and inhibition within the brain has been demonstrated to be associated with anxiety.
3. The substance has been demonstrated to exert an effect on the serotonergic system. An increase in the expression of the inhibitory 5-HT1A receptor is observed, which could reduce the activity of serotonin, a key neurotransmitter in mood disorders.

In this regard, evidence suggests that exposure to fluoride may predispose individuals to the manifestation of behaviors related to anxiety and depression, mainly through the disruption of neurochemicals involved in synaptic plasticity (BDNF), excitatory/inhibitory balance (GABA/Glu), and serotonin and dopamine signaling. The following text is intended to provide a comprehensive overview of the subject matter.

These findings in animal models are consistent with human studies reporting a correlation between fluoride levels and anxiety symptoms.

Conclusions

1. The available scientific literature suggests that exposure to fluoride during the prenatal and perinatal periods may result in adverse neurodevelopmental outcomes, including cognitive impairment, hyperactivity, and mood disturbances. However, there is a paucity of research on the emotional and mental impacts in comparison to cognitive outcomes.
2. Fluoride has been demonstrated to induce mitochondrial damage, manifesting in a decline in mitochondrial DNA, a disruption in biogenesis, and a deterioration in mitochondrial structure. Mitochondrial function has been identified as a critical factor in neurodevelopment. Consequently, any disruption in mitochondrial function may result in neurodevelopmental and mental disorders, including depression, anxiety, and ADHD.
3. The impact of fluoride appears to be more pronounced in males, particularly with regard to diminished IQ and somatization symptoms. The critical windows of exposure exhibit variation according to sex.
4. While research conducted on animal models has established a correlation between fluoride exposure and symptoms of anxiety and depression, only a limited number of studies have been conducted on human subjects to investigate these effects. These studies have identified associations between fluoride exposure and somatization symptoms, though not with anxiety or depression.
5. A multitude of mechanisms have been postulated to explain the phenomenon of fluoride-induced neurotoxicity, including the induction of mitochondrial damage, neuroinflammation, alterations in neurotransmitters, and disruption of various signaling pathways.
6. A paucity of research exists on the effects of fluoride on mental health during childhood and adolescence, as well as on compounds such as fluorosilicic acid, which is the most commonly used in water fluoridation.

Recommendations

Further research is necessary to enhance our understanding of the impact of fluoride on mental and cognitive health, while accounting for confounding variables such as socioeconomic status, parental intelligence, and additional chemical exposures.

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