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Migraine and neurodevelopmental disorders: shared mechanisms linking autism and attention-deficit/hyperactivity disorder

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ABSTRACT

Background: Migraine is a neurological disorder that often occurs alongside psychiatric and neurodevelopmental conditions. Evidence of an association between migraine and both autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) is emerging. However, the mechanisms that underlie these relationships are not fully understood. *Aim*: This narrative review concerns the evidence relating to the association between migraine, ASD, and ADHD, with a focus on their shared clinical features and the neurobiological mechanisms. *Methods*: A search was conducted on PubMed, and the reference lists of relevant publications were reviewed to identify papers published between 2020 and May 2026 that examined migraine in relation to ASD, ADHD, or neurodevelopmental disorders. Studies focused on epidemiological associations, clinical features, genetic susceptibility, and possible biological mechanisms. The relevant evidence was summarised in a narrative form. *Results*: Available evidence supports an association between migraine and both ASD and ADHD, with consistent findings concerning headache burden and sensory abnormalities. Several mechanisms have been identified, including changes in sensory processing, increased cortical excitability, neurotransmitter dysregulation, autonomic dysfunction, neuroinflammatory pathways, and sleep disturbances. Genetic studies have shown a shared susceptibility between migraine and neurodevelopmental disorders, even though the extent of the genetic overlap differs. There is also evidence of an association between maternal migraine and the child developing ADHD. *Conclusions*: Migraine and neurodevelopmental disorders may have some overlapping biological and environmental weaknesses. Further studies are required to establish causality and discover whether a better understanding of these relationships could lead to more individualized clinical management.

Keywords: migraine, autism spectrum disorder, attention-deficit/hyperactivity disorder, neurodevelopmental disorders, sensory processing

1. INTRODUCTION

Migraine as a neurological disorder

Migraine is a common neurological disorder marked by recurrent episodes of headache together with neurological and sensory symptoms. A prominent clinical symptom is a headache, but migraine is nowadays considered to be a complex neurological disorder involving changes in sensory processing, modifications in neuronal activity, and interactions between genetic, neurochemical, and environmental factors. The burden of migraine extends past individual headache attacks, as recurrent symptoms may substantially interfere with daily functioning and quality of life. The fact that it often occurs together with psychiatric and neurodevelopmental disorders has also led to increased interest in the biological mechanisms that may be common to migraine and to disorders affecting neurodevelopment and brain function (Yum & Chu, 2025).

When thinking about the connection between migraine and neurodevelopmental disorders, the sensory aspect is especially important. During a migraine attack, patients commonly experience photophobia, phonophobia, and an increased sensitivity to external stimuli, which indicates that abnormal processing of sensory information may play a role in making someone susceptible to migraines. Therefore, it is possible that the changes in sensory processing seen in some neurodevelopmental disorders are related to the mechanisms involved in migraine.

Autism spectrum disorder and attention-deficit/hyperactivity disorder as neurodevelopmental disorders

Autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) are neurodevelopmental disorders which are heterogeneous and have both genetic and environmental factors determining them. ASD is marked by persistent difficulties in social communication and interaction as well as by restricted or repetitive patterns of behavior, although abnormal sensory processing is also often noted. ADHD is defined by persistent patterns of inattention and/or hyperactivity and impulsivity and is likewise linked to a wide variety of psychiatric, neurological and somatic comorbid conditions.

The clinical features of both conditions go beyond the main symptoms upon which they are diagnosed. Alterations in sensory processing, sleep problems, emotional dysregulation and difficulties with communication can affect the way in which physical symptoms are experienced and reported. This is especially important in the case of headache disorders, since the identification and description of pain may be more difficult for some people with neurodevelopmental disorders (Vetri, 2020; Alsaad, 2024).

The association between migraine, autism and ADHD

Even though migraine is very common in the general population, its connection with neurodevelopmental disorders has attracted considerably less attention than its links with depression and anxiety. Vetri described the association between ASD and migraine as fairly unexplored, noting that only a limited number of studies had looked at their coexistence and suggesting several possibly overlapping biological mechanisms, such as abnormalities in neurotransmission, immune responses, anatomical organization and genetic susceptibility (Vetri, 2020).

It has recently been suggested that migraine might be more frequent among people with ASD, but it can be difficult to identify. Difficulties with communication and language, together with the way in which pain is described or expressed, may make headaches harder to identify, especially in children and adolescents with ASD (Alsaad, 2024).

New genetic evidence provides a more detailed understanding of the issue. Ciochetti et al., (2025) looked at the genetic relationships between ADHD, migraine, and multisite chronic pain by making use of large-scale genomic data and polygenic risk scores. They found a significant biological overlap between ADHD and multisite chronic pain, even though the connection between ADHD and migraine was not so clear. Nevertheless, the findings suggest that neurodevelopmental pathways may be involved in the association between ADHD and pain-related traits, at the same time indicating that migraine should not be assumed to share all the genetic mechanisms that are linked to both ADHD and chronic pain.

When all the available evidence is considered, it indicates that migraine may occur alongside both ASD and ADHD, but the mechanisms that underlie these associations are still not fully understood. Possible areas of overlap are sensory processing, neuronal excitability, neurotransmitter systems, immune and inflammatory pathways, sleep regulation, and genetic susceptibility. Nevertheless, the present evidence is varied, and the fact that an association is observed does not mean that it shows a common causal mechanism.

Aim of the review

The connection between migraine and neurodevelopmental disorders is therefore an area that is currently being explored in the field of neurological research. Although previous reviews have mostly looked either at migraine in relation to psychiatric comorbidities or at the association between migraine and ASD, genetic studies have started to investigate the relationship between ADHD and pain-related phenotypes.

The evidence relating to migraine, ASD and ADHD within a common mechanistic framework is still fragmented. Specifically, it is not clear whether the similarities in sensory processing, neuronal function, neurotransmission, immune regulation and genetic susceptibility indicate truly shared mechanisms or are instead due to partially independent associations. This review article is intended to summarize the existing evidence regarding the relationship between migraine, ASD and ADHD, with a special focus on any possible shared pathophysiological mechanisms. It will examine the evidence relating to sensory processing, cortical and neuronal excitability, neurotransmitter systems, autonomic and immune mechanisms, sleep and circadian regulation, and genetic susceptibility. The review will also examine the clinical implications of these associations and highlight the areas in which further research is required.

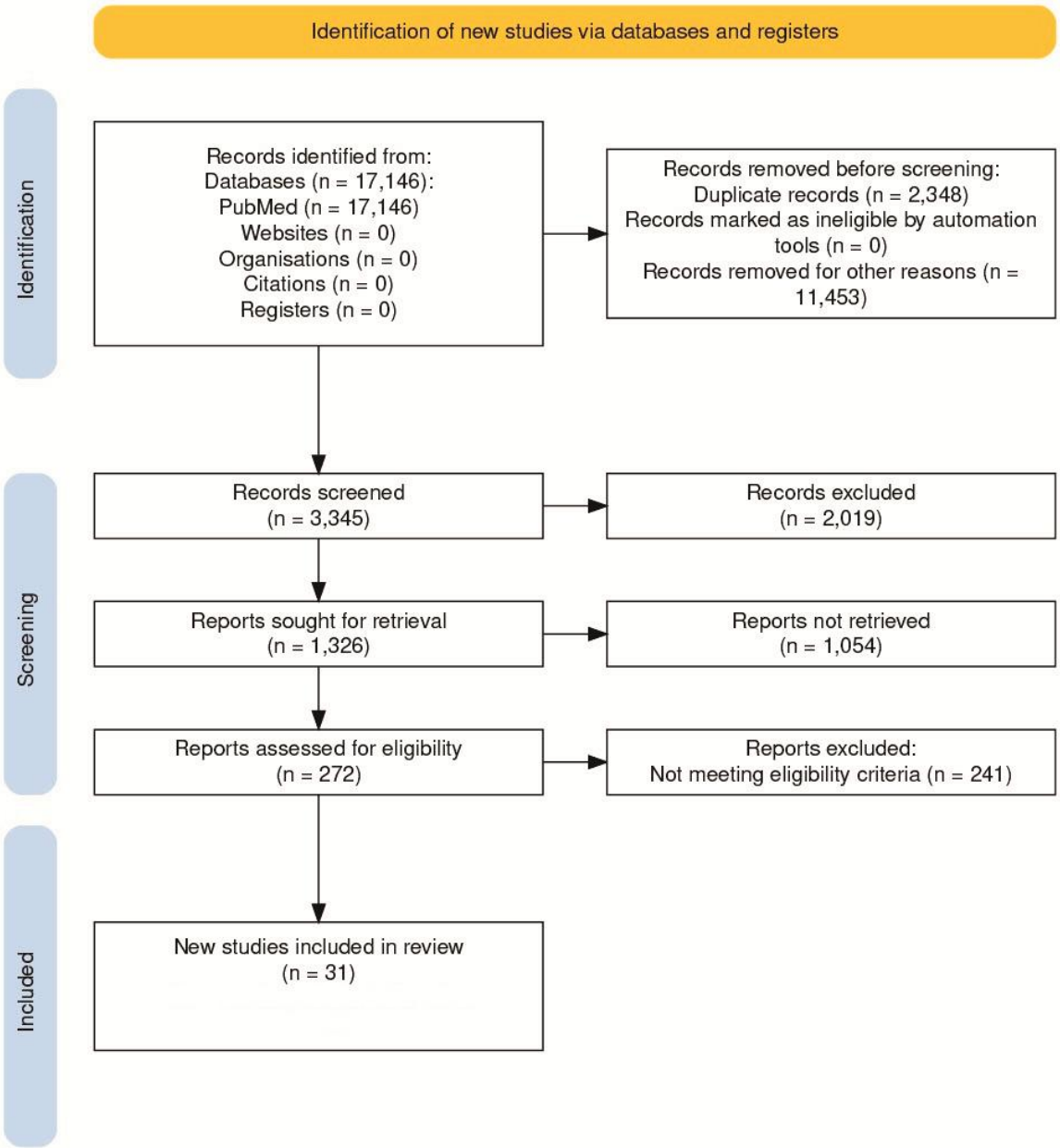

Figure 1. PRISMA flow diagram

2. REVIEW METHODS

A search for relevant studies was carried out on PubMed during the period from 2020 to May 2026 using combinations of terms associated with migraine, autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), neurodevelopmental disorders, and possible common mechanisms, and the reference lists of the relevant publications were reviewed as well. The search terms included "migraine", "autism spectrum disorder", "ASD", "attention-deficit/hyperactivity disorder", "ADHD", "neurodevelopmental disorders", "sensory processing", "cortical excitability", "autonomic dysfunction", "neuroinflammation", "neurotransmitter dysregulation", "genetic susceptibility", "sleep", and "circadian rhythm".

The research examining the association from epidemiological, clinical, genetic, or biological perspectives was taken into account. Only human studies and reviews written in English and published between 2020 and May 2026 were included. Conference abstracts, publications in a language other than English, and studies that were not related to the purpose of the review were excluded.

Relevant literature was assessed based on titles, abstracts, and full texts, and findings were synthesized narratively. The study selection process is presented in the PRISMA flow diagram (Figure 1).

3. RESULTS

Migraine and autism

Epidemiological association

The evidence currently available shows that migraine and other headache disorders are more common in people with autism spectrum disorder (ASD) than in the general population. However, the extent of this association differs from study to study and depends on the population in question and the diagnostic methods employed. A systematic review and meta-analysis of neurological disorders in autism found that migraine and headache were among the neurological conditions that occur more frequently in individuals with autism than in the general population (Pan et al., 2021).

More up-to-date data offer further evidence of this link. A large retrospective study that made use of both the US National Survey of Children's Health and an electronic health records database found that children with autism were more likely to report frequent or severe headaches than were those without autism (7.1% compared with 3.0%). Within the UCLA group as well, migraine was more common among people with autism than among those without it (3.1% versus 2.0%). Also, in individuals with autism, having either a headache or a migraine was linked to higher levels of anxiety, depression, adverse childhood experiences, and worse overall health, indicating that migraine may account for the greater burden of comorbid conditions that is already present in this group (Grant Tejada et al., 2026).

The association is also based on studies that look at autistic traits rather than at cases of clinically diagnosed ASD. In a 2025 study involving patients with migraine, autistic traits were found to be much more common than in healthy controls, with them occurring in about 35% of the migraine group as compared to 17% of the control group. The presence of autistic traits was linked to higher levels of sensory sensitivity and anxiety sensitivity and was indirectly connected to headache-related disability and psychiatric symptoms (Türkel et al., 2025).

It is important to note that recent genetic evidence indicates the association cannot always be fully accounted for by psychosocial or clinical factors. In an analysis of 337,386 participants carried out by the UK Biobank, higher polygenic scores for ASD were found to be positively linked with migraine, whether or not it was accompanied by an aura. The associations remained statistically significant even after adjusting for several possible mediators, although the effect sizes were small (Mohammad et al., 2025).

The current epidemiological evidence shows that there is an association between ASD and migraine. However, the small number of population-based studies and the differences in diagnostic methods make it impossible to estimate the extent of the comorbidity accurately.

Clinical presentation

Recognizing migraine in people who are autistic might be especially difficult since their problems with social communication, their unusual way of expressing pain, and any existing impairments in language or intelligence can interfere with the usual evaluation of the features of a headache. As a result, migraine may go undetected or may appear in the form of behavioural rather than verbally reported symptoms, especially in children and those who have more significant communication difficulties (Vetri, 2020; Alsaad, 2024).

The current body of literature shows that sensory abnormalities could be particularly important in relation to the clinical overlap between the two conditions. Sensory hypersensitivity is a feature of autism spectrum disorder and is also frequently observed in

individuals who have migraines, such as sensitivity to light, sound, smell, and other environmental stimuli. In migraine sufferers, higher levels of autistic traits have been found to correspond with greater sensory sensitivity; this sensitivity is then associated with headache-related disability and anxiety symptoms (Türkel et al., 2025).

Psychiatric comorbidity can also affect the clinical presentation, since people who have both autism and migraine may suffer from symptoms of anxiety and depression. Recent population-based data show that autistic individuals who have a headache or migraine have higher odds of both anxiety and depression than autistic individuals who do not have a headache or migraine (Grant Tejada et al., 2026).

Although the available evidence currently does not define a particular migraine phenotype that is specific to ASD, the growing understanding suggests a range of overlapping sensory, behavioural, and psychiatric features which could affect the recognition of symptoms and the extent of the illness perceived. It is important to make this distinction since autistic traits might influence the clinical expression and assessment of migraine without necessarily indicating the presence of a distinct migraine subtype.

Potential mechanisms

There are several mechanisms which have been suggested to explain the overlap between migraine and ASD. Currently the evidence points to a multifactorial model which involves altered sensory processing, changes in neurotransmitter systems, modified immune signaling, increased neuronal excitability, and a common genetic susceptibility, not instead a single common pathway (Vetri, 2020; Alsaad, 2024). One possible reason for this might be a disturbance in the balance between excitation and inhibition, along with changes in neuronal excitability. While both migraine and ASD have been linked to abnormalities in the regulation of neuronal networks, the exact direction and clinical importance of these abnormalities are still not fully understood. In ASD, altered GABAergic and glutamatergic signaling has been studied many times, and in the case of migraine abnormally high cortical excitability and problems with sensory processing are well-known areas of investigation. The mechanisms in question could provide a biological basis for the intense sensory sensitivity observed in both conditions. Another possible connection is related to the dysregulation of neurotransmitters. There has long been a link between serotonergic signaling and the pathophysiology of migraine, and it has also been looked at in the context of ASD.

Further changes involving glutamate and GABA may account for abnormal sensory processing and neuronal network regulation disorders. Vetri suggested that overlapping abnormalities in neurotransmitters could be one of several biological links between ASD and migraine (Vetri, 2020). Another possible point of convergence is to be found in the neuroimmune mechanisms.

The two disorders have both been studied in relation to altered immune signaling and neuroinflammatory processes. Yet the current evidence is still heterogeneous, and the occurrence of inflammatory abnormalities should not be taken as proof of a common causal inflammatory mechanism. In fact, attention has in recent years been given to genetic overlap. The 2025 study into polygenic scores showed that genetic susceptibility to ASD was positively linked with both migraine with and without aura, thus providing evidence of a part-shared genetic basis between the two conditions (Mohammad et al., 2025). This finding is consistent with the general notion that vulnerability in the field of neurodevelopmental vulnerability may influence later susceptibility to migraine. However, the associations were weak, suggesting that shared genetic liability is likely only one element of a multifactorial relationship. The evidence currently available is in favor of a model according to which a common neurobiological vulnerability might show itself in changes to sensory processing and neuronal excitability, genetic, neurotransmitter and immune mechanisms possibly being involved in this overlap. Nevertheless, at this stage none of these mechanisms can be regarded as definitely causal.

Migraine and attention-deficit/hyperactivity disorder

Epidemiological association

There is now evidence showing a link between attention-deficit/hyperactivity disorder (ADHD) and headache disorders, such as migraine. A systematic review and meta-analysis, which was published in 2022, showed that children with ADHD had about twice the odds of suffering a headache when compared to children without ADHD (OR 2.01, 95% CI 1.63–2.46). The overall prevalence of headache in children with ADHD was 26.6 percent, which shows that headache is a clinically important comorbidity in this group (Pan et al., 2022). Concerning migraine, more specific evidence is provided by a nationwide longitudinal study which tracked individuals with ADHD and matched controls who had no prior diagnosis of migraine. During follow-up, migraine developed in 0.6% of individuals with ADHD compared with 0.3% of controls. It was the first time that, taking into account the demographic characteristics as well as the presence of other physical and psychiatric conditions, an increased risk of developing migraine was found to be

associated with ADHD (HR 1.92, 95% CI 1.64–2.34). This association was especially clear in children and adolescents, even if it was not statistically significant in young adults (Hsu et al., 2022).

It therefore seems that the relationship is especially important in childhood and adolescence, since it is during these periods that both ADHD and migraine tend to become clinically evident. Yet the existing epidemiological evidence is not enough to establish whether ADHD itself raises the risk of migraine or whether the two conditions instead partly reflect common neurodevelopmental, genetic, or environmental susceptibility.

Genetic evidence also supports a biological relationship, but the degree of this support is less than that seen between ADHD and multisite chronic pain. Ciochetti et al., (2025) found a positive genetic relationship between ADHD and migraine, but they identified a much stronger biological overlap between ADHD and multisite chronic pain. The results indicate that migraine might be just one part of the wider association between ADHD and pain-related phenotypes rather than a condition which has a genetically distinct strong link to ADHD. Overall, current evidence supports an association between ADHD and migraine, particularly in younger populations, while the magnitude and nature of this association remain incompletely characterized.

Clinical presentation

The connection between ADHD and migraine could be affected by the fact that both conditions involve similar cognitive, behavioural, and sensory symptoms. People with ADHD usually have trouble with attention, executive functioning, and the regulation of their arousal levels. At the same time, migraine is also associated with temporary difficulties in attention and in cognitive performance. Recent studies have shown that attentional abnormalities can occur both during and between migraine attacks, which means that cognitive symptoms may be part of the general neurological profile of migraine and not merely due to ADHD (Fernandes & Gil-Gouveia, 2025). Headache can also make up a major part of the clinical burden of ADHD. Headache is common in children who have ADHD, and this association remains significant even after allowing for potential confounding factors (Pan et al., 2022). This is important from a clinical point of view since headache may be ignored when behavioral and attentional symptoms are the focus of the clinical evaluation.

Another point to consider is the possible effect of pharmacological treatment for ADHD

The systematic review and meta-analysis carried out by Pan et al., (2022) showed that some ADHD drugs were linked to a higher risk of headache when compared to a placebo, specifically mentioning atomoxetine, guanfacine, and methylphenidate. However, this does not prove that these medications cause migraine in particular, since the outcome that was examined was headache rather than migraine alone; instead, it underscores the need to tell the difference between an existing headache disorder and a side effect that occurs as a result of treatment. The clinical presentation may thus be the result of an interaction among three factors: the underlying migraine phenotype, the cognitive and behavioural characteristics associated with ADHD, and the effects of treatment. However, current evidence does not show that there is a particular migraine phenotype found only in people with ADHD.

Potential mechanisms

Various mechanisms have been suggested to account for the link between ADHD and migraine, such as changes in sensory processing, neurotransmitter signaling, cortical network function, sleep regulation, and genetic susceptibility (Yum & Chu, 2025; Ciochetti et al., 2025). Yet the evidence regarding each of these mechanisms is inconsistent, and no one particular pathway has been identified as being responsible for the association observed. The possibly overlapping mechanisms are compared across migraine, ASD and ADHD.

Shared mechanisms

Altered sensory processing

Altered sensory processing is one of the main areas in which migraine overlaps with neurodevelopmental disorders; migraine typically involves sensory hypersensitivity, particularly during an attack, and this can affect visual, auditory, olfactory and somatosensory stimuli. Neurophysiological studies carried out recently have shown that people with migraine have increased visual hypersensitivity, thus supporting the idea that there is abnormal sensory processing in the disorder (Marti-Marca et al., 2023). A similar pattern of altered sensory processing has also been recorded in autism. Sensory hyper- or hypo reactivity is considered part of the clinical features of autism and can have a substantial effect on everyday functioning. The fact that there is an overlap is especially important since sensory sensitivity is also linked to migraine-related disability. In cases of migraine, a higher level of autistic traits has been found to

correlate with greater sensory sensitivity, and sensory sensitivity may play a role in the connection between autistic traits and headache-related disability (Türkel et al., 2025). There is also evidence of altered sensory processing in ADHD.

A systematic review and meta-analysis published in 2025 that involved 30 studies and 5,374 participants revealed greater sensory abnormalities in people with ADHD in many areas, such as sensory sensitivity, sensory avoiding, low sensory registration and sensory seeking. However, a large degree of heterogeneity was found between the studies (Jurek et al., 2025). Put together, these findings indicate that changes in sensory processing are among the most reasonable explanations for the overlap in phenotypic features between migraine, ASD and ADHD. Yet sensory abnormalities are diverse and not particular to any one of these conditions; their role might thus be best seen as a common vulnerability that can play a part in the manifestation of symptoms in different disorders.

Cortical excitability

Another possible point at which migraine and neurodevelopmental disorders might converge is cortical excitability. Abnormalities in cortical responsiveness have been shown in migraine cases through the use of neurophysiological and neuroimaging techniques. Specifically, TMS-EEG studies have found altered cortical responses in people with migraine with aura, thus indicating a role for abnormal cortical excitability in the pathophysiology of migraine (Helling et al., 2023). There is also evidence of altered cortical inhibition and excitability in ASD. Investigations employing transcranial magnetic stimulation have shown abnormal responses to cortical stimulation in people with ASD, such as differences in the way motor-evoked potentials are modulated after continuous theta-burst stimulation. These results indicate altered cortical plasticity and inhibitory regulation in ASD (Jannati et al., 2021). ADHD has also been found to involve similar abnormalities. A neurophysiological study of children with ADHD showed that their short-interval intracortical inhibition (SICI)—a measure linked to GABAergic intracortical inhibition—was reduced when compared to children who develop normally. The association between SICI and cortical GABA levels also indicates that there is a role for impaired inhibitory neurotransmission in ADHD (Harris et al., 2021).

More recently, a meta-analysis of 17 studies found a significant reduction in SICI in people with ADHD. However, several other measures of cortical excitability did not show a significant difference between the ADHD group and the control groups, which suggests that the abnormality may be particular to intracortical inhibition rather than indicating generalized cortical hyperexcitability (Han et al., 2025). Taken together, these findings show that changes in cortical excitability and in inhibitory control may reflect a partly common neurophysiological feature in migraine, ASD and ADHD. Nevertheless, the specific abnormalities vary between the conditions and from one experimental paradigm to another. The evidence at present thus suggests partial convergence in the regulation of cortical networks rather than a single pattern of generalized cortical hyperexcitability.

Autonomic dysfunction

Autonomic regulation is also a possible area of overlap between migraine and neurodevelopmental disorders. In the case of migraine, changes in autonomic function have been studied by means such as heart rate variability (HRV). A cross-sectional study of patients with episodic migraine revealed a reduction in HRV, especially during migraine attacks, indicating that there is altered autonomic regulation associated with the ictal state (Zhang et al., 2021). Altered autonomic regulation has also been found in ASD. A meta-analysis published in 2020, which involved 34 studies, showed that people with ASD had significantly lower baseline HRV, especially in the measures relating to parasympathetic activity and respiratory sinus arrhythmia; reduced HRV reactivity was also noted during social stress, indicating altered autonomic flexibility in ASD (Cheng et al., 2020).

Studies that directly compared ASD and ADHD indicate that the autonomic abnormalities might not be the same in the various neurodevelopmental disorders. In children and adolescents, those with ASD exhibited higher autonomic arousal when performing an active task, while those with ADHD showed reduced autonomic arousal during resting and passive situations. These results imply that autonomic dysregulation could be a feature common to all diagnoses, although the particular pattern of this dysregulation may vary depending on the neurodevelopmental condition (Bellato et al., 2022). Put together, these findings show that changed autonomic regulation may be a possible point at which migraine, ASD, and ADHD converge. Yet the existing evidence does not prove a common autonomic abnormality affecting all three disorders. Because of the differences in the HRV measures, the experimental conditions, and the clinical populations involved, a direct comparison is difficult. Autonomic dysfunction should therefore be regarded as a potentially overlapping physiological mechanism rather than as an established common pathophysiological pathway.

Neuroinflammation

Neuroinflammatory mechanisms are also a possible point of convergence between migraine and neurodevelopmental disorders. In the case of migraine, there is growing evidence indicating that neuroinflammatory and neuro-glial processes play a role in the activation and regulation of the trigeminovascular system. A systematic review of the role of glial cells in migraine found evidence of neuroinflammation and neuro-glio-vascular interactions in the pathophysiology of migraine. However, much of the existing evidence is still based on preclinical studies (Zhang et al., 2023). Neuroinflammatory mechanisms have also been studied in great detail in relation to ASD. A systematic review of microglia-mediated neuroinflammation showed that there is evidence of microglial activation in a number of different areas of the brain in people with ASD. However, the number of studies available and the variety of methods used prevented definite conclusions from being reached. Further recent evidence has also outlined changes in innate immune function, including interactions between circulating myeloid cells and brain-resident microglia, which suggests that immune–neural interactions may play a role in at least some individuals with ASD (Hughes et al., 2023).

Similar mechanisms have been proposed in ADHD. Reviews have pointed out links between ADHD and higher levels of oxidative stress, inflammatory processes, and changes in immune-related pathways. The suggested mechanisms are inflammatory cytokine signaling, genetic susceptibility related to inflammatory pathways, and the possible effects of inflammatory exposure during early life on brain development (Corona, 2020).

When combined, these findings indicate that neuroinflammatory signaling might be a partially shared biological feature in migraine, ASD, and ADHD. Yet, the evidence varies greatly between the conditions and comes from a diverse range of clinical, molecular, and experimental studies. In the case of migraine, most of the mechanistic evidence relates to neurogenic inflammation and to interactions between neurons and glial cells, while in ASD and ADHD the evidence tends to focus on systemic immune dysregulation, microglial activation, or inflammatory susceptibility. Hence, the current evidence suggests that neuroinflammation could be an overlapping mechanism rather than indicating a single common inflammatory pathway underlying all three disorders.

Neurotransmitter dysregulation

There is also another possible point at which migraine and neurodevelopmental disorders overlap concerning neurotransmitter systems. As serotonergic signaling has a well-established role in the pathophysiology of migraine, it has likewise been connected with ADHD and other psychiatric comorbidities. Recent evidence shows that serotonin may be involved both in the neurobiology of ADHD and in the occurrence of many somatic comorbidities, such as migraine (Yum & Chu, 2025; Faraone et al., 2025).

Dopaminergic signaling could also help to explain the similarity between migraine and ADHD. Since dopamine plays a role in pain modulation, sensory processing, and autonomic reactions in migraine, it is also a key element in the neurobiology of ADHD (Yum & Chu, 2025; Ciochetti et al., 2025).

Neurotransmitter signaling alterations could therefore play a role in the presence of common clinical features such as sensory hypersensitivity, difficulties with attention, and changes in pain processing. Yet it is unlikely that neurotransmitter abnormalities indicate a single common mechanism since serotonergic and dopaminergic systems interact with other biological pathways, such as cortical excitability and neuroinflammatory signaling, and their effects may vary depending on the disorder and clinical state.

When put together, the current evidence indicates that dysregulation of neurotransmitters may represent a potentially overlapping biological mechanism connecting migraine with neurodevelopmental disorders, especially via serotonergic and dopaminergic pathways. However, the evidence available does not prove that there is a single abnormality of a neurotransmitter which serves as a common causal mechanism for migraine, ASD and ADHD.

Genetic susceptibility

There is some of the strongest evidence from a genetic standpoint indicating that there is partial overlap in susceptibility between migraine and neurodevelopmental disorders. In a large cohort from the UK Biobank, polygenic risk scores for autism spectrum disorder are positively linked with both migraine without aura and migraine with aura, which suggests that the genetic vulnerability associated with autism spectrum disorder may play a role in making someone susceptible to migraine (Mohammad et al., 2025).

There is also genetic overlap between ADHD and migraine. More recent studies have found genetic signals in common between ADHD and different pain conditions, such as migraine, indicating that neurodevelopmental pathways may play a role in making people susceptible to pain-related disorders (Ciochetti et al., 2025). ASD and ADHD show a large degree of overlap in their common genetic variation. A summary of the main genetic findings is given in Table 1.

Table 1. Shared genetic links between migraine, ASD, and ADHD with key susceptibility genes and effect estimates.

Genetic Link	Evidence	Key Genes/Loci
ASD ↔ Migraine	ASD polygenic scores positively associated with migraine, migraine with aura and migraine without aura; OR 1.04–1.05	Polygenic risk; no single locus identified (Mohammad et al., 2025)
ASD ↔ ADHD	37.5% of ASD-associated SNPs colocalized with ADHD SNPs; 19.6% of ADHD-associated SNPs colocalized with ASD SNPs; bidirectional genetic association	INSM1, PAX1**;** shared SNPs (Peyre et al., 2021; Baranova et al., 2022)
ADHD ↔ Migraine	Positive genetic correlation and polygenic overlap, but substantially weaker than ADHD–multisite chronic pain	Shared variants/loci; neurodevelopmental pathways (Ciochetti et al., 2025)
Migraine ↔ ASD: ion-channel biology	Familial hemiplegic migraine genes overlap with genes/pathways implicated in ASD; direct evidence for the same variants causing both conditions remains limited	CACNA1A, ATP1A2, SCN1A (Vetri, 2020; Yum & Chu, 2025)

In a genomic study of ASD and ADHD, 37.5% of the SNPs associated with ASD at a p-value of less than 1×10^{-6} were found to colocalize with ADHD SNPs, and 19.6% of the SNPs associated with ADHD colocalized with ASD SNPs. The analysis revealed shared genetic variants and genes, such as INSM1 and PAX1, suggesting that there is a partially shared genetic basis between the two neurodevelopmental disorders (Peyre et al., 2021). Bidirectional Mendelian randomization analyses also showed that a genetic predisposition to ASD was linked with an increased risk of ADHD and that a genetic predisposition to ADHD was linked with an increased risk of ASD (Peyre et al., 2021). The same applies to Baranova et al., who also found bidirectional genetic relationships, indicating that the two disorders share a common genetic architecture and offering evidence that a genetic vulnerability to one condition raises the risk of the other (Baranova et al., 2022).

It has been shown that both migraine and neurodevelopmental disorders may have a common biological origin through ion-channel genes, since the genes CACNA1A and SCN1A have been associated with both migraine and ASD; this indicates that abnormalities in the function of calcium and sodium channels could be responsible for the shared symptoms (Yum & Chu, 2025).

One cannot conclude that migraine, ASD, and ADHD are due to the same genetic mechanism just because there are common genetic variants; genetic susceptibility is most likely affected by developmental and environmental factors, which in turn give rise to a range of clinical symptoms. Therefore, the current evidence supports a model that involves some overlap in genetic vulnerability rather than a single common genetic pathway.

Sleep and circadian rhythm

Sleep disturbances are an additional possible area in which migraine and neurodevelopmental disorders may overlap. A systematic review and meta-analysis of 42 studies showed that adults who had both ASD and ADHD exhibited impaired sleep when compared to healthy controls, involving a longer sleep-onset latency, lower sleep efficiency, more frequent awakenings, and a poorer perceived sleep quality (Lugo et al., 2020).

Sleep disturbances are also often linked with migraine. A systematic review showed a consistent relationship between migraine and sleep disorders, even though the direction of this association and the mechanisms involved are still complex and not fully understood (Tiseo et al., 2020). Likewise, more recent systematic evidence based on objective evaluations of sleep has similarly revealed changes in sleep efficiency, sleep fragmentation, and arousal patterns in people who have migraine. However, there is a great deal of variation among the studies (Munday et al., 2026).

It appears that sleep could be a clinically significant factor linking migraine with both ASD and ADHD. Damage to sleep might make people more susceptible to migraine attacks and exacerbate the symptoms, while the symptoms of migraine themselves may interfere with sleep. In the case of neurodevelopmental disorders, poor sleep may also account for problems with attention, sensory regulation and daily functioning.

Circadian regulation might thus be another possible element of this overlap. Yet the present evidence points more to an association between sleep disturbances and these disorders than to a specific common circadian mechanism. It follows that sleep abnormalities should be regarded as a potentially modifiable clinical factor and as a possible contributor to the shared symptom burden rather than as an established common pathophysiological mechanism.

Maternal migraine and offspring neurodevelopment

Emerging evidence suggests that migraine in mothers may be associated with neurodevelopmental outcomes in their offspring. A large population-based study of a Danish cohort found that children of mothers with migraine had a slightly increased risk of psychiatric disorders, including pervasive developmental disorders and behavioural and emotional disorders with onset usually occurring in childhood and adolescence (Wang et al., 2021). The risk was still low overall, and possible explanations include genetic susceptibility, stress during pregnancy, or medication use. However, not all research agrees, and other studies have found no strong association between maternal migraine and neurodevelopmental problems in children. There is an increasing amount of evidence suggesting that migraine in parents, particularly in mothers, could be connected to the neurodevelopmental outcomes of their children. A study based on a population sample that included 22,747 children whose parents had migraine showed that these children were at an increased risk of being diagnosed with ADHD if one of their parents had migraine. When the sex of the parent was considered individually, the association was found in the case of maternal migraine but not with paternal migraine, indicating the possibility of a developmental or familial factor that is specific to mothers. Yet the same study found no significant association between parental migraine and ASD (Li et al., 2024).

More recent evidence has looked into whether the link between maternal migraine and offspring ADHD could be due to a direct effect in the womb. Luo et al. found an association between maternal migraine in the first trimester and ADHD traits in the children when they were seven years old, as well as a connection between the mothers' genetic tendency to suffer from migraine and the offspring's ADHD traits (Luo et al., 2026).

It should be pointed out that Mendelian randomization provided only weak evidence of a direct causal effect of the mother's genetic susceptibility to migraine on the child's ADHD; these findings suggest that the epidemiological association observed could in part be due to common genetic or familial susceptibility rather than result from a direct causal mechanism in utero (Luo et al., 2026).

When put together, the current evidence indicates that maternal migraine may point to a higher neurodevelopmental vulnerability in children, especially concerning ADHD. Yet the existing data do not show that maternal migraine during pregnancy directly causes ADHD; the relationship is therefore another instance of the complex interaction between genetic susceptibility, developmental factors, and environmental exposures.

4. DISCUSSION

Main findings and interpretation

This review shows that migraine is linked to both autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). Still, the strength and character of the evidence vary between the two neurodevelopmental disorders. The connection between migraine and ASD is backed by epidemiological, clinical, and genetic evidence, while the evidence relating to ADHD is also indicative, though it seems more varied. Importantly, the existing literature does not support the idea that there is a specific migraine pattern found only in people with ASD or ADHD. On the contrary, the neurodevelopmental features may affect how migraine symptoms are experienced, expressed, and recognized.

The results also indicate that the link between migraine and neurodevelopmental disorders cannot be accounted for by a single biological pathway. Possible areas of overlap include sensory processing, cortical excitability, autonomic regulation, neurotransmitter systems, inflammatory signaling, sleep, and genetic susceptibility. Nevertheless, the amount of evidence varies greatly among these mechanisms, and a great many of the supposed similarities are based on indirect comparisons between different groups of subjects rather than on studies that have looked at all three conditions at the same time.

The genetic evidence also supports partial biological convergence. There is an association between the polygenic liability for autism spectrum disorder and migraine, and there is a considerable degree of genetic overlap between autism spectrum disorder and ADHD. By contrast, the genetic link between ADHD and migraine is less clear than the relationship between ADHD and multisite chronic pain. These results contradict a straightforward model according to which migraine, autism spectrum disorder and ADHD share the same genetic basis. Rather, the evidence available is more in line with the notion of partially overlapping susceptibility interacting with disorder-specific developmental, biological and environmental factors.

The present evidence tends to support the idea that there may be common elements of biological vulnerability between migraine and neurodevelopmental disorders without this indicating that they are manifestations of the same underlying disorder.

Shared mechanisms: convergence without a single pathway

The mechanistic findings discussed in this study indicate a convergence at a number of levels of nervous-system function. Changes in sensory processing and in cortical excitability might be among the most relevant areas of overlap. Sensory hypersensitivity is a prominent feature in migraine. It is also typical of ASD, and neurological studies point to disturbances in both cortical inhibitory and excitatory processes in ASD and ADHD. The similarities between the conditions thus offer a reasonable basis for understanding how sensory stimuli and changes in cortical processing might lead to symptoms in different disorders. Yet the particular neurophysiological abnormalities found in each disorder are not the same, and so they should not be seen as proof of a single common cortical abnormality.

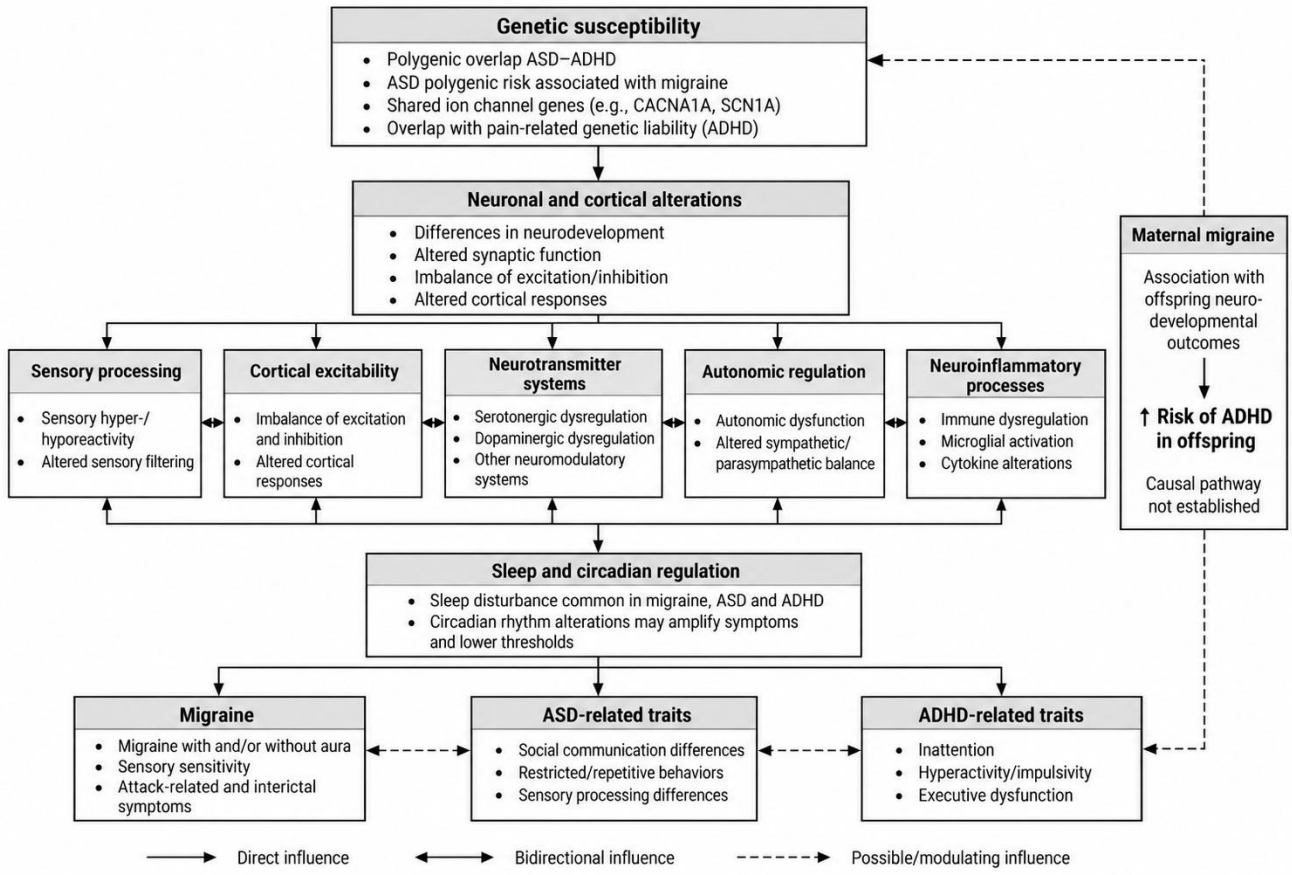

Figure 2. Proposed model of shared mechanisms linking migraine, ASD and ADHD.

There is also the possibility of convergence through autonomic regulation. It has been noted that autonomic function is altered in both ASD and ADHD, and likewise autonomic and trigeminovascular mechanisms are associated with migraine. Yet the results vary depending on the experimental conditions and the clinical populations involved. This variation indicates that autonomic dysfunction may act as a modulating factor rather than serving as a uniform biomarker common to all three conditions. There are also other possible links involving neurotransmitter and inflammatory pathways. The fact that serotonergic and dopaminergic signaling are

connected with migraine and ADHD, and that abnormalities relating to inflammation and the immune system have been investigated in both ASD and ADHD, means that these mechanisms may interact with cortical excitability, sensory processing, and autonomic regulation rather than acting independently. It therefore becomes clear that the biological overlap should be viewed as a network of interacting processes rather than as a collection of separate mechanisms.

The genetic evidence supports this explanation. The fact that there is a shared polygenic susceptibility together with overlapping ion-channel pathways implies that changes in neuronal excitability may play a role in the vulnerability seen in these disorders. On the other hand, since the polygenic associations have only a small effect size and there is limited evidence for the same causal variants, the shared genetic results should not be taken as indicating a common monogenic cause.

There is also a clinically important connection in this area. Since sleep disturbances are common in people with migraine, ASD and ADHD, they may affect sensory regulation, attention, daily functioning and the likelihood of experiencing a migraine. That said, the evidence for a particular shared circadian mechanism is less strong than the evidence for sleep disturbances themselves. Sleep should thus be seen as both a possible factor increasing the severity of symptoms and as a clinical aspect that may be modified.

When put together, these findings lend support to a model involving partial mechanistic convergence. Although the same biological systems may affect more than one disorder, their contribution, direction, and clinical expression can vary from one individual to another. This model is more in line with the present evidence than the idea of a single common pathophysiological pathway. A conceptual model that integrates the proposed shared mechanisms connecting migraine, ASD and ADHD is given in Figure 2.

Clinical implications

The results have several consequences for clinical practice. Migraine might be overlooked in people with ASD, especially when communication difficulties, unusual ways of expressing pain, or other coexisting sensory abnormalities make it hard to carry out a standard headache evaluation. In these cases, any deviations from an individual's normal behavioral and sensory patterns could offer useful contextual information, even though general behavioral changes should not be taken automatically as signs of migraine. A summary of the main areas of clinical overlap between migraine, ASD and ADHD is given in Table 2.

Table 2. Clinical overlap between migraine, ASD and ADHD

Clinical feature	Migraine	ASD	ADHD	Potential clinical overlap
Sensory hypersensitivity	Photophobia, phonophobia, osmophobia, tactile sensitivity	Hyper-/hyporeactivity to sensory stimuli	Sensory sensitivity may occur	Increased sensory burden and possible overlap in sensory processing
Attention and cognition	Transient cognitive difficulties during attacks	Executive and cognitive difficulties may occur	Core attentional and executive difficulties	Cognitive symptoms may complicate assessment of migraine and ADHD/ASD
Sleep disturbance	Common and associated with migraine burden	Frequently reported	Frequently reported	Sleep disturbance may amplify symptoms across conditions
Autonomic symptoms	Nausea, vomiting and autonomic symptoms are common	Autonomic dysregulation has been reported	Autonomic alterations have been described	Potential shared autonomic component
Pain communication	Headache is the defining symptom	Pain may be expressed atypically or communicated with difficulty	Pain may be less readily recognized in the context of attentional/behavioral symptoms	Migraine may be under-recognized
Psychiatric comorbidity	Anxiety and depression commonly coexist	Psychiatric comorbidities are frequent	Psychiatric comorbidities are frequent	Greater overall symptom burden and diagnostic complexity

In people with ADHD, the clinical picture can be complicated by the fact that symptoms relating to attention and those relating to cognition occur together during migraine attacks. Moreover, headaches can appear as an adverse reaction to certain ADHD medications. It therefore follows that a careful assessment of the timing of the headaches in relation to the start of treatment and to when the medication is taken may help in telling apart an existing headache disorder from symptoms that have emerged as a result of treatment.

Currently, the evidence is not enough to warrant the development of a separate algorithm for the treatment of patients who have ASD or ADHD. Standard methods of treating migraines should continue to form the foundation of treatment, and clinical care will have to take into account individual variations in communication, sensory tolerance, sleep, adherence, and the ability to identify triggers. At this time, there is not enough evidence to establish whether one's neurodevelopmental status systematically affects the way they respond to specific migraine therapies.

These factors lend more weight to having a greater clinical awareness rather than leading to the establishment of a separate diagnostic category. The evidence currently available does not show the existence of a distinct neurodevelopmental subtype of migraine.

Maternal migraine and developmental implications

Women who have migraines while pregnant could be at risk. It has been shown that maternal migraines are associated with an increased likelihood of complications, including preeclampsia, preterm birth, and low birth weight. These difficulties can influence the child's development both before and after birth. Further studies are required to understand the effect of maternal migraines on long-term development fully.

The fact that maternal migraine is linked to offspring having ADHD gives the connection between migraine and neurodevelopment a new aspect. Evidence from population-based data indicates that maternal migraine is linked to a higher risk of ADHD in the children. In contrast, a similar association with paternal migraine or with offspring having ASD has not been consistently shown.

Although this finding may be important, it should be interpreted with caution since a maternal-specific association does not on its own prove that there is a direct effect within the uterus. It is possible that shared genetic susceptibility, familial characteristics, environmental exposures, and other types of confounding factors could all have contributed to the association that has been observed. Thus, the evidence currently available indicates that maternal migraine is a relevant developmental association rather than showing that there is a causal prenatal pathway. It will be necessary for future studies that combine longitudinal follow-up with genetic and familial approaches to be able to tell the intrauterine effects apart from those that are inherited or caused by environmental factors.

Limitations and challenges

The following limitations of the present evidence should be taken into account. Firstly, a large number of the studies currently available are cross-sectional, retrospective, or based on registries. While such designs can identify associations, they offer only limited insight into the question of temporal relationships and causality.

Secondly, there is a great deal of heterogeneity in the way both migraine and neurodevelopmental disorders are assessed. The various studies differ in their use of formal diagnoses, screening tools, and the dimensional measures of autistic or ADHD traits, and the way in which headaches are phenotyped also varies from study to study, which hampers direct comparisons of the results.

Thirdly, migraine, ASD, and ADHD are very infrequently looked at together in the same group of people. In most studies, migraine is examined in relation to either ASD or ADHD, which means that the proposed shared mechanisms are usually deduced by comparing the results from different populations. As a result, the strength of the conclusions about true biological convergence is reduced.

The methodological quality and source of the mechanistic evidence also differ. Certain findings are based on neurophysiological or molecular measurements, while others come from reviews or preclinical research. Specifically, when drawing conclusions about neuroinflammation and glial mechanisms from experimental models and applying them to human disease, such evidence should be interpreted with caution.

The fact that there is a genetic overlap does not signify causation. It could be because of pleiotropy, because of correlated biological pathways, or because of a more general vulnerability in neurodevelopment. Similarly, the association between a mother's migraines and the outcomes of her children does not by itself show us whether the effects take place in the womb or are instead the result of familial or genetic confounding.

Future research

Future studies should go beyond showing that migraine is linked with ASD and ADHD and should instead aim at discovering the mechanisms and clinical consequences of this overlap; it would be especially useful to carry out prospective longitudinal studies that include people with migraine, ASD and ADHD all within the same research program.

Greater standardization of migraine phenotyping and neurodevelopmental assessment would increase the comparability of studies. Genetic methods, such as polygenic risk analyses and Mendelian randomization, could further elucidate the common susceptibility and possible causal pathways. At the same time, functional studies are required in order to find out how the identified genetic variants affect neuronal excitability, sensory processing and neurotransmission. It might be possible to find out whether the mechanisms that have been identified separately in ASD, ADHD, and migraine actually converge in the same people by combining studies that use objective measures of cortical excitability, autonomic function, sleep, and inflammatory signaling. Furthermore, maternal migraine also needs to be investigated using longitudinal family-based and genetically informed designs.

It is now necessary to carry out treatment studies which include people with ASD and ADHD to find out whether the neurodevelopmental features affect the response to migraine treatment, tolerance of it, adherence to it, or the profile of adverse effects. This type of research could in the end help to enable more individualized methods of diagnosing and managing migraine.

5. CONCLUSION

Migraine seems to be more commonly linked with both autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD), although the degree of evidence varies between the two disorders. Even though the current evidence does not indicate the presence of a particular migraine subtype specific to neurodevelopmental disorders, differences in sensory processing, communication, and cognitive functioning might affect the way migraine is expressed and recognized in these groups.

The connection between migraine, ASD, and ADHD is probably due to a several factors. Altered sensory processing, increased neuronal excitability, dysregulation of neurotransmitters, problems with autonomic function, sleep disturbances, and inflammatory pathways may all be responsible for the associations that have been observed, and recent genetic studies have provided evidence for some degree of shared susceptibility. Yet there is a great difference in the strength of the evidence among the various mechanisms, and no one biological pathway has been identified as the common cause of all three conditions. The growing body of evidence relating to mothers having migraines and the neurodevelopment of their children still highlights the complexity of these connections. The fact that there is an association between a mother's migraines and her child developing ADHD does not mean that this involves a direct causal effect during the time in the womb and might instead be due to shared genetic or familial susceptibility.

More longitudinal, genetic, and mechanistic studies are needed to establish the direction and causal links of these associations. A better understanding of migraine in people who have ASD and ADHD could lead to improved recognition and clinical management. In general, gaining an understanding of the relationship between migraine and neurodevelopmental disorders might offer insights into common biological vulnerabilities and at the same time promote more individualized methods of diagnosis and care.

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Conflict of interest

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Data and materials availability

No new data were created or analyzed in this study. Data sharing is not applicable to this article.

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