FISIO MU

Naufal, A.F., Fadhila, N.K.N & Fitri, A.A
Fisiomu. 2026, Vol 7(2):46-61
DOI : 10.23917/fisiomu.v7i2.17426

Risk Factors For Attention-Deficit/Hyperactivity Disorder: Literature Review

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Submitted : 2026-05-09; Accepted : 2026-05-29; Published : 2026-06-01

ABSTRACT

Introduction: Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by inattention, hyperactivity, and impulsivity that affect children's social and academic functioning. ADHD has a multifactorial etiology involving interactions among biological, genetic, environmental, psychosocial, and economic factors during the prenatal and postnatal periods. This study aimed to identify the prevalence and dominant risk factors contributing to the incidence of ADHD in children based on previous research findings. **Methods**: This study employed a systematic literature review following the PRISMA guidelines and the PECO framework. Articles were searched through the PubMed and ScienceDirect databases between October and November 2025, with inclusion criteria consisting of children aged 0–18 years, observational study designs, discussion of ADHD risk factors, English language, and publications from 2015 to 2025. **Results**: A total of 28 articles met the inclusion criteria, comprising 13 prenatal phase studies (46.4%) and 15 postnatal phase studies (53.6%). Biological factors were the most dominant component (49.0%), followed by environmental (22.2%), psychosocial (13.3%), genetic (11.1%), and economic factors (4.4%). **Conclusion**: ADHD in children results from multifactorial interactions, highlighting the need for multidisciplinary approaches and multifactorial interventions beginning in the prenatal period to support prevention and early detection.

***Keywords**: ADHD, risk factors, children, prenatal, postnatal.*

ISSN 2722 – 9610
E –ISSN 2722 - 9629

INTRODUCTION

Child development encompasses physical, cognitive, and emotional aspects (Santrock, 2019). However, not all children go through the developmental stages in the same way. Some children exhibit hyperactive behavior, difficulty controlling impulses, and attention disorders. In the field of health and neurological development, this condition is known as Attention Deficit Hyperactivity Disorder (ADHD), a neurological disorder that affects the ability to manage attention and regulate behavior adaptively (Aprilia Santana et al., 2025). In Islam, variations in behavior from an early age are also shown in *QS. Yusuf: 18*, so that deviant behavior in children does not always indicate moral wrongdoing, but rather a special condition that requires attention and the right approach in education and parenting (Desmita, 2014). In addition to affecting behavior, ADHD is also closely related to children's cognitive development. Azizah et al. (2025)state that stunting has a serious impact on children's cognitive development and can increase the risk of long-term developmental disorders. According to Almadani et al. (2022), developmental disorders rooted in neurobiological processes can cause barriers in communication, decreased social interest, repetitive behavior, and low visual and fine motor attention span, all of which can impact a child's adaptive abilities in the future.

Attention Deficit Hyperactivity Disorder (ADHD) is a neurological disorder characterized by difficulty concentrating, hyperactive behavior, and impulsive tendencies (Song et al., 2025). According to the International Classification of Diseases, 10th edition (ICD-10), these symptoms appear before the age of 7, are evident in more than one environment such as home and school, and cause significant impairment in academic and social functioning (World Health Organization, 2019). In addition to being a behavioral disorder, ADHD is also associated with motor impairments, sensory integration, and postural control that affect children's functioning (Junaidin, 2024).

ADHD not only affects children's development but is also important to review its prevalence in global and national populations. Global prevalence has increased significantly, from 0.85 cases per 1,000 children in the 2017–2019 period to 2.02 cases per 1,000 children in the 2020–2023 period, or an increase of approximately 2.62 times (Song et al., 2025). Overall, the prevalence is estimated at 8%, higher in males (10%) than females (5%) (Ayano et al., 2023). National data in Indonesia is still limited and depends on local research. One study found that 21.9% of children aged 4–6 years in Gonilan Village, Surakarta, showed symptoms of ADHD, all of whom used gadgets for more than one hour per day (Naufal, et al., 2023).

Although the exact cause of ADHD is unknown, this disorder is multifactorial, involving genetic, neurobiological, and environmental factors (Thapar et al., 2012). ADHD in children can be caused by prenatal and postnatal factors (Bitsko et al., 2024). Children with ADHD parents are at higher risk, as are babies born with low birth weight (LBW) and a family history of ADHD (Ragadran et al., 2023; Rahman et al., 2021). Neurobiologically, ADHD is associated with an imbalance of the neurotransmitters dopamine and norepinephrine (Cortese, 2020). Prenatal factors such as maternal stress, exposure to cigarette smoke, alcohol, and malnutrition can affect fetal neural development (Ginting et al., 2023). Fatmarizka et al. (2023)found that physical complaints and lack of physical activity in pregnant women can affect prenatal health quality, which plays an important role in fetal development, so that suboptimal pregnancy conditions can increase a child's vulnerability to developmental disorders.Social and environmental factors that contribute to the risk of ADHD include young parental age, low economic status, ineffective parenting, lack of family support, exposure to cigarette smoke during pregnancy, exposure to heavy metals such as lead, and excessive use of gadgets from an early age (Amallia et al., 2025; Ginting et al., 2023; Russell et al., 2014; Twenge & Campbell, 2018). A systematic review of 59 longitudinal studies showed that the quality of parenting and the family environment have a significant influence on the development of children's executive function, attention, and self-regulation, and are directly related to the risk of ADHD (Claussen et al., 2024). Naufal et al. (2023)also explain that inconsistent parenting patterns and unstructured learning environments can exacerbate ADHD symptoms in children, so the quality of interaction and support from the surrounding environment plays an important role in children's behavioral development. Understanding these factors is important for early detection and rapid intervention so that symptoms do not develop more seriously (Racine, 2025). However, research still focuses on diagnosis, treatment, and therapy, while studies on risk factors in children are still limited and tend to highlight one factor separately, without looking at the relationship between factors in an integrated analytical framework.

The analysis of ADHD risk factors is important for early detection and prevention, but the lack of comprehensive primary research remains an obstacle. Therefore, this study uses a literature review method to identify, categorize, and analyze ADHD risk factors in children. The selection of this topic is based on the author's concern about the importance of a comprehensive understanding of ADHD risk factors as a basis for prevention, especially in elementary school children, as well as providing theoretical and practical contributions to screening, prevention, and health and education policies.

Based on this description, it can be concluded that the main issue examined in this study is the risk factors associated with the onset of ADHD in children. The study of risk factors is important because it can support early detection and prevention efforts before the disorder develops further (Racine, 2025). However, existing research has focused more on diagnosis, therapy, and behavioral intervention, while studies on risk factors are still limited and often highlight one factor in isolation without a

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E –ISSN 2722 - 9629

comprehensive analysis of their interrelationships. This study aims to identify, categorize, and analyze various ADHD risk factors based on the results of previous studies. Specifically, this study seeks to examine the prevalence of risk factors and determine the most dominant or most frequently associated factors with an increased risk of ADHD in children.

The results of this study are expected to provide academic, practical, and theoretical benefits. Academically, this study can serve as a scientific reference for students and researchers studying ADHD. Practically, the results of this study will help health workers, psychologists, educators, and parents understand the risk factors for ADHD, support early detection, and design prevention strategies. Theoretically, this study expands the literature on the etiology and prevention of ADHD and provides a basis for the development of evidence-based policies and further research.

METHODS

Type of Research

This study uses a systematic *literature review* approach aimed at identifying and analyzing risk factors for ADHD in children. This systematic approach is used because it is able to assess, evaluate, and synthesize the results of previous studies in a structured, transparent, and comprehensive manner so as to provide a strong conceptual basis and identify *research gaps* for further study (Snyder, 2019).

Retrospective Study

This study is retrospective because all data analyzed comes from previous published studies. There was no primary data collection from research subjects. The articles identified came from the PubMed and ScienceDirect databases, with publication dates between 2015 and 2025. The article search was conducted from October to November 2025.

All articles discussing risk factors for ADHD in children were analyzed retrospectively based on information listed in the primary studies, including study design, sample size, population characteristics, exposure, and key findings related to risk factors.

Literature Research

The literature search was conducted using a combination of keywords: (Attention Deficit Hyperactivity Disorder OR ADHD) AND (risk factor OR determinant OR predictor) AND (children OR pediatric). Titles and abstracts were screened first to determine relevance. Articles that met the inclusion criteria were then read in full. The data extracted included the identification of risk factors, population characteristics, research methods, and main findings. This information was integrated into a thematic synthesis to provide a comprehensive overview of ADHD risk factors in children.

Data Collection

Data collection was carried out through two main stages, namely literature search and article selection. The search was conducted on two international databases, namely PubMed and ScienceDirect, using Boolean operators to expand and clarify the search results. The selection process followed the PRISMA flow, which included the stages of article identification, title and abstract screening, eligibility assessment through full-text reading, and determination of final articles that met all inclusion criteria. Data collected from each article included study design, year of publication, sample size, type of risk factors, and research results related to ADHD incidence.

Sample Size

The sample size in this study refers to the number of articles that met the entire PRISMA selection process. The number of articles was not limited in advance; rather, all publications that met the inclusion criteria were included to ensure the breadth of coverage and richness of information analyzed. This approach aimed to strengthen the quality of data synthesis and increase the analytical power of the study.

Risk Factor Identification Process

The risk factor identification process was carried out through data extraction from each selected article. The information collected included the type of exposure or factor tested, the relationship between the exposure and the occurrence of ADHD, and the research results. This identification was based on the Population, Exposure, Comparator, Outcome (PECO) framework to ensure that each article was in line

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E –ISSN 2722 - 9629

with the focus of the study. This framework is described in more detail in Table 1.

Table 1. PECO Standards

Population	Subjects consisting of children
Exposure	Risk factors genetic, biological,environmental, psychosocial, and economic
Comparator	Children without exposure or with lower levels of lower exposure levels
Outcome	ADHD incidence

Risk factors obtained from each study were first grouped based on the phase of exposure, namely the prenatal phase and the postnatal phase. After grouping based on phase, these factors were further classified into five main categories, namely genetic, biological, environmental, psychosocial, and economic factors. This layered grouping aims to provide a clearer analysis structure and facilitate the preparation of thematic syntheses in describing the pattern of relationships between risk factors.

Criteria for Selecting Risk Factors

The selection of risk factors in this study was based on the suitability of exposure to the research objectives as described in the PECO framework. Each risk factor listed in the article must be directly relevant to the occurrence of ADHD in children and included in one of the exposure phases, namely the prenatal or postnatal phase. Risk factors were only considered if they were analyzed in primary research with an observational design and were in accordance with the child population as determined in the article selection criteria.

In addition, the risk factor selection process followed the inclusion criteria, namely articles that discussed ADHD risk factors in children, were observational primary studies, were written in English, were published between 2015 and 2025, and were available in free full-text form. Meanwhile, risk factors reported in articles were excluded if the articles met the exclusion criteria, only discussed diagnosis or therapy without mentioning risk factors, were opinion or editorial articles, individual case reports, used adult or animal subjects, or were duplicate articles or not fully accessible. Thus, only risk factors that were consistent with the PECO framework and relevant to the research objectives were included in the analysis.

Prenatal Factors

Prenatal risk factors for ADHD are mainly related to the mother's biological and health conditions during pregnancy, such as prenatal inflammation, maternal infection, exposure to drugs or chemicals, and metabolic disorders that can affect fetal development. In addition, genetic predisposition and epigenetic influences also shape the child's vulnerability in the womb. Environmental factors, pregnancy stress, and the mother's socioeconomic status can also increase these risks.

Postnatal Factors

Postnatal factors associated with ADHD include childhood health disorders such as infection, sleep disorders, extreme prematurity, allergies, brain injury, and early exposure to anesthesia. Environmental influences such as stress, pollution, cigarette smoke, as well as caregiving and psychosocial conditions also increase the risk. Family socioeconomic factors and the interaction between genetics and the environment also play a role in the emergence of ADHD symptoms after birth.

Data Recording Process

The data recording process was carried out following the PRISMA flow in its entirety, starting from the identification stage, screening, feasibility assessment, to the final article inclusion. Each article that entered the screening stage was recorded in an extraction table according to the PECO framework, which contained information about population characteristics, types of exposure, comparators, and research outcomes related to ADHD risk factors. The PRISMA approach ensures that the recording process is transparent and systematic, while the PECO framework is used to maintain consistency between the selected articles and the research objectives. All extracted data are rechecked to ensure consistency and accuracy before further analysis.

Statistical Analysis

The statistical analysis in this study was descriptive and qualitative. The reduced data were grouped based on risk factor categories and analyzed using a thematic synthesis approach. The results of the analysis were presented in the

ISSN 2722 – 9610
E –ISSN 2722 - 9629

DOI : 10.23917/fisiomu.v7i2.17426

form of narratives, tables, and charts to illustrate the relationships between factors.

RESULTS

Article searches were conducted through two databases, PubMed and ScienceDirect, with a total of 54,801 articles (PubMed = 14,873; ScienceDirect = 39,928). After the identification stage, 53,593 articles were eliminated because they did not use observational methods. During the screening process, 132 articles were eliminated because they were not in English, while 992 articles were excluded because they did not meet the specified publication year criteria. Thus, 84 articles were fully accessible for assessment of their suitability, and 56 of these were eliminated because they did not discuss ADHD risk factors. The final result left 28 articles that met the inclusion criteria and were further analyzed to identify and categorize ADHD risk factors in children.

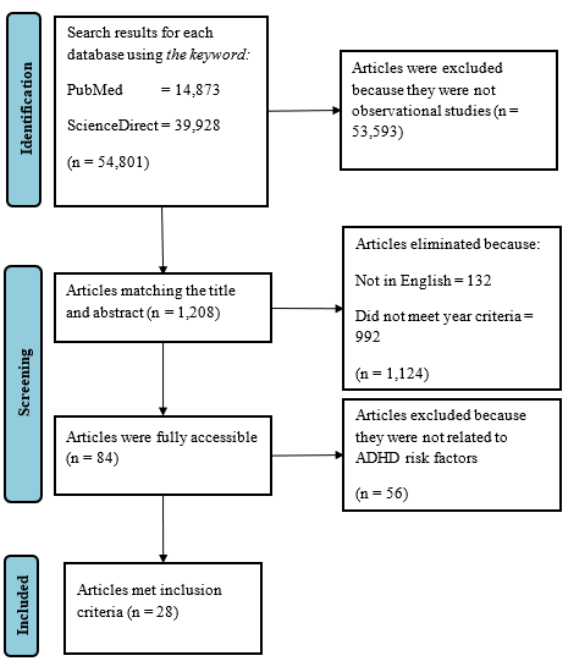

Figure 1. PRISMA Flow Diagram

A total of 28 articles screened through the PRISMA stages included observational studies published between 2015 and 2025 that examined various risk factors associated with ADHD in populations ranging from newborns to school-age children, both prenatally and postnatally. All articles were then analyzed using the PECO (Population, Exposure, Comparator, Outcome) approach as presented in Table 2 to ensure consistency between the exposure studied and the outcome of ADHD.

DISCUSSION

Based on a review of 28 scientific articles published between 2015 and 2025, the risk factors for ADHD in children are multifactorial, involving interactions between biological, genetic, environmental, psychosocial, and economic aspects.

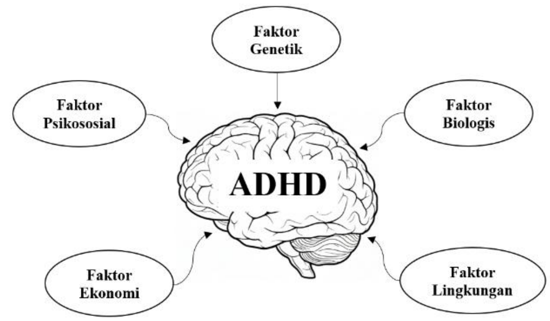

Figure 2. Mind Map of ADHD Risk Factors

Further analysis shows that biological factors are the most dominant component, with 22 occurrences (49.0%), followed by environmental factors (10 occurrences, 22.2%), psychosocial (6 occurrences, 13.3%), genetic (5 occurrences, 11.1%), and economic (2 occurrences, 4.4%). These results indicate that ADHD disorders are more often associated with biological and environmental mechanisms, both of which play a role in the prenatal and postnatal phases, while psychosocial and economic factors tend to reinforce postnatal symptoms. These findings are more clearly seen in Figure 3, which shows the prevalence of each ADHD risk factor and compares their direct contributions.

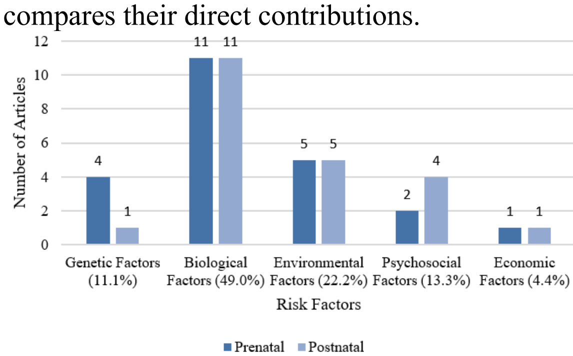

Figure 3. Prevalence of ADHD Risk Factors

The review results show that 13 studies (46.4%) examined risk factors in the prenatal

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phase, which included various biological, genetic, environmental exposures, and maternal health conditions during pregnancy. Prenatal risk factors found in various studies include prenatal inflammation (Takahashi et al., 2023), changes in microRNA expression (Dypås et al., 2025), maternal prenatal depression (Tusa et al., 2025), maternal exposure to infections such as CMV and Mycoplasma (Borbye-Lorenzen et al., 2025), and antibiotic exposure during pregnancy (Holmgaard et al., 2024). Other factors include exposure to methylparaben (Baker, Wu, et al., 2020), iodine imbalance (Abel et al., 2017), intrahepatic cholestasis of pregnancy (Chen et al., 2024), and extreme prematurity (Leviton et al., 2018). Additional factors include a high polygenic risk score (Takahashi et al., 2023), genetic pleiotropy (Koller et al., 2024), ADHD symptom-related polygenic scores (Piazza et al., 2024), and prenatal paracetamol exposure (Baker, Lugo-Candelas, et al., 2020). All these prenatal factors are further explained through the mechanisms depicted in the underlying process in Figure 3.

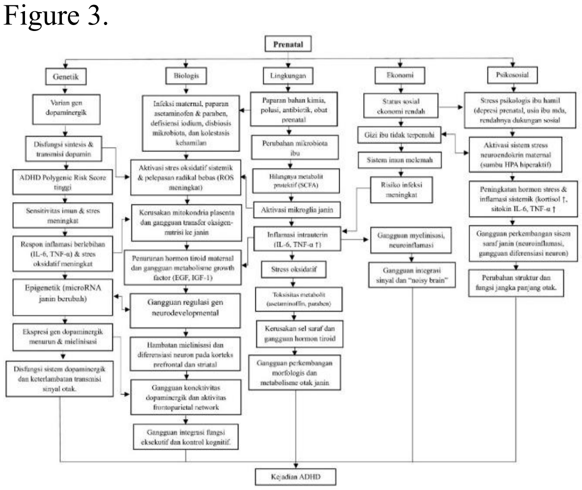

Figure 4. Prenatal Underlying Process

Meanwhile, 15 studies (53.6%) focused on the postnatal phase, covering various factors such as loud snoring (Joseph et al., 2025), COVID-19 lockdown stress (Hernández-Lorca et al., 2023), changes in gut microbiota (Ahrens et al., 2024), extreme prematurity (Haile et al., 2024), and exposure to general anesthesia (Ing et al., 2017, 2020). Other factors include a history of meningitis (Hadzic et al., 2017), increased cases during the COVID-19 pandemic (Song et al., 2025), psychological distress in gender dysphoria (Nunes-Moreno et al., 2022), and OSAHS sleep disorders (Wu et al., 2017). Additional findings include biomarkers of traumatic brain injury (Wilkinson et al., 2017), systemic inflammation in preterm infants (Allred et al., 2017), atopic dermatitis (Lee et al., 2016), poor glycemic control in type 1 diabetes (S. Liu et al., 2021), and the psychosocial conditions of refugee children during the pandemic (Ünver & Perdahlı Fiş, 2022). All of these postnatal factors are further described in the underlying process in Figure 4.

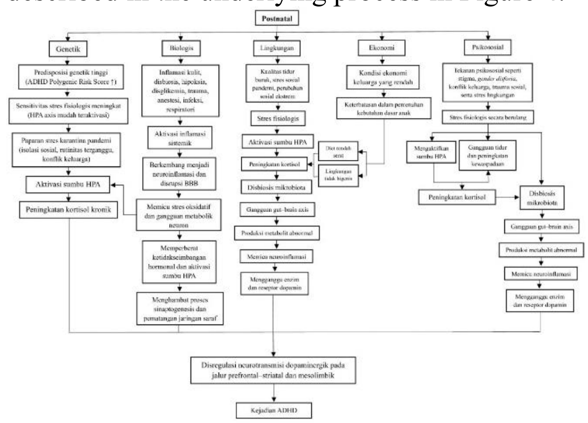

Figure 5. Postnatal Underlying Process

The series of mechanisms in the underlying process indicate that ADHD incidence is influenced by the interaction of various factors acting during both the prenatal and postnatal phases. Therefore, the next section elaborates in more detail on the contribution of each genetic, biological, environmental, psychosocial, and economic factor found in this study.

Prenatal Phase

Various risk factors are at work from early pregnancy and form the basis of a child's neurodevelopmental vulnerability. In the prenatal phase, internal factors, including genetics, maternal and fetal biological conditions, hormonal regulation, inflammation, and epigenetic processes, together with external factors, including environmental conditions, chemical exposure, psychosocial stress, and economic circumstances during pregnancy, interact to determine the quality of fetal brain development before entering the postnatal phase. Based on genetic contributions, genetic factors play a major biological role in shaping an individual's vulnerability to ADHD. A total of five articles (11.1%) identified genetic

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E –ISSN 2722 - 9629

contributions. Based on four prenatal studies and one postnatal study, it was found that the expression of genes that regulate neurotransmitter systems, inflammation, and nerve development has a significant influence on the emergence of ADHD symptoms.

In the prenatal phase, research by Takahashi et al. (2023)explains that the interaction between the polygenic risk score (PRS) for ADHD and increased levels of inflammatory cytokines such as IL-6 and TNF-α during pregnancy can increase the risk of ADHD in children. This mechanism reinforces the concept of gene–environment interaction, where genes act as a basic predisposition, while prenatal inflammation acts as a catalyst for phenotype expression. These findings are supported by research by Dypås et al. (2025),which identified 19 types of microRNA (miRNA) in umbilical cord blood that can predict ADHD diagnosis with high accuracy. Changes in miRNA expression affect genes that regulate dopaminergic synapse formation, neuron differentiation, and circadian rhythms, suggesting an epigenetic mechanism that bridges the relationship between genetics and the intrauterine environment.

Furthermore, Koller et al. (2024)identified the CADM2 rs62250713 gene variant, which not only plays a role in regulating ADHD impulsivity but also correlates with a tendency toward substance use disorders. This polymorphism confirms the existence of genetic pleiotropy that affects the brain's reward system. Research by Piazza et al. (2024) also shows that ADHD polygenic scores have a strong association with core symptoms of inattention and impulsivity, reinforcing the hypothesis that a combination of small gene variants can cumulatively form a characteristic neuropsychological pattern from the fetal stage.

Furthermore, when viewed from a biological perspective, this factor is the most dominant category and is found in 22 articles (49.0%), making it the aspect most frequently associated with the onset of ADHD and the broadest dimension covering various aspects, ranging from inflammation, hypoxia, chemical exposure, to endocrine disorders. Eleven prenatal studies showed that changes in maternal physiology can cause neurotoxic effects on the fetus, while eleven postnatal studies confirmed that metabolic dysfunction, infection, and early inflammation also contribute to the risk of ADHD.

In the prenatal phase, research by Borbye-Lorenzen et al. (2025)confirms that high IgG levels against Mycoplasma pneumoniae and Cytomegalovirus in the blood of newborns increase the risk of ADHD (OR 1.30). This is due to the activation of fetal microglia, which causes chronic neuroinflammation. Furthermore, Holmgaard et al. (2024)found that the use of penicillin antibiotics during pregnancy reduces epidermal growth factor and sTNF-RI levels in neonates, which play an important role in neuronal maturation.

Research by Chen et al. (2024)found that maternal intrahepatic cholestasis increases the risk of neurodevelopmental disorders, including ADHD, especially when onset occurs in the first trimester. The proposed mechanism is bile acid toxicity to neuronal differentiation. Abel et al. (2017) confirmed that iodine deficiency or excess causes maternal thyroid dysfunction, which interferes with fetal brain myelination. These findings are consistent with Baker, Wu, et al. (2020),who showed that exposure to methylparaben can cause maternal hypothyroidism, preterm birth, and ADHD risk in children. A study by Z. Y. Liu et al. (2024)added that chronic hypoxia due to congenital heart defects causes cognitive deficits through brain tissue hypoxia. Baker, Lugo-Candelas, et al. (2020)found that prenatal exposure to paracetamol alters frontoparietal brain connectivity, providing evidence of a link between pharmacological toxicity and attention circuit dysfunction.

Additionally, Tusa et al. (2025) demonstrated that prenatal depression increases maternal cortisol, which disrupts the HPA axis and increases the risk of ADHD in children. Dypås et al. (2025) reinforced these findings through epigenetic mechanisms, in which miRNA expression reflects prenatal hormonal stress. Takahashi et al. (2023) also showed that prenatal inflammation amplifies genetic effects on ADHD risk. Leviton et al. (2018) added that extreme prematurity, mechanical ventilation, and prenatal infection increase ADHD risk through

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impaired neurovascular maturation and chronic inflammatory status. Thus, these eleven articles describe that prenatal biological processes work through three main mechanisms: maternal and fetal inflammation, endocrine and metabolic dysfunction, and hypoxia and toxic exposure. The combination of these three causes permanent structural and functional changes in the fetal brain.

Regarding environmental factors, this aspect plays a major role in mediating and modulating the relationship between genetics and biology in the development of ADHD. This factor was found in 10 articles (22.2%), ranking second after biological factors. Based on the review results, there were ten articles discussing environmental aspects, with five studies focusing on the prenatal phase and five others on the postnatal phase. These factors include chemical exposure, infection, economic stress, and social conditions that affect the development of the child's nervous system.

In the prenatal phase, a number of studies show that exposure to the environment of pregnant women can disrupt fetal neurogenesis directly or indirectly through hormonal and immunological mechanisms. Research by Baker, Wu, et al. (2020)revealed that exposure to methylparaben, a common endocrine-disrupting compound in consumer products, is correlated with maternal hypothyroidism and an increased risk of ADHD in children. This mechanism is explained by disruption of the hypothalamic-pituitary-thyroid axis, which causes thyroid hormone imbalance in the fetus, thereby inhibiting myelination and neuron differentiation.

These findings are in line with research by Abel et al. (2017), which shows that both iodine deficiency and excess in pregnant women can increase the risk of ADHD in children. This imbalance in micromineral nutrition indicates that environmental factors such as maternal nutritional intake have a significant influence on fetal brain development, particularly through thyroid hormones, which are highly sensitive to changes in iodine levels.

In addition to nutritional factors, pharmacological exposure has also been shown to be risky. A study by Baker, Lugo-Candelas, et al. (2020)found that high levels of acetaminophen in fetal meconium were associated with an increased risk of ADHD in childhood, partly mediated by impaired connectivity of the frontoparietal network of the brain. This shows that even medications that are generally considered safe during pregnancy can have long-term effects on fetal neural development if used excessively.

The study by Holmgaard et al. (2024)also highlights that prenatal antibiotic use is associated with decreased levels of epidermal growth factor (EGF) in neonates, which has implications for suboptimal neuronal maturation processes. Meanwhile, Leviton et al. (2018)expanded on these findings by highlighting the effects of intrauterine infection, prematurity, and low social environment, which increase fetal vulnerability to oxidative stress and chronic inflammation. The overall evidence from these five studies shows that an unhealthy prenatal environment, whether due to chemical exposure, nutritional disorders, or infection, can cause significant biological changes to fetal brain development and increase the risk of ADHD later in life. An optimal prenatal environment with balanced nutrition, free of toxins, and stable social-emotional conditions is key to early prevention.

In addition, psychosocial factors also play an important role in strengthening and modulating the genetic and biological expression of ADHD. A total of six articles (13.3%) identified psychosocial factors as one of the risks for ADHD, especially those related to maternal stress and the child's social conditions. Of these articles, two discussed the influence of stress and maternal mental health during the prenatal phase, while the other four highlighted the child's social and psychological conditions in the postnatal phase. The overall evidence shows that psychological stress originating from the social and emotional environment contributes to neurobiological dysfunction relevant to ADHD, particularly through the mechanisms of stress hormone dysregulation, increased inflammatory- , and impaired social relationships.

In the prenatal phase, research by Tusa et al. (2025)provides strong evidence that maternal depression during pregnancy correlates with an increased risk of ADHD in children. Prenatal affective disorders cause increased cortisol levels and dysfunction of the hypothalamic–pituitary–

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adrenal (HPA) axis, which can interfere with the development of the fetal prefrontal cortex and limbic system. Chronically high cortisol during pregnancy affects the regulation of the neurotransmitters dopamine and serotonin, which are the two main chemical systems disrupted in ADHD. This maternal stress also increases the release of proinflammatory cytokines such as IL-6 and TNF-α, which cross the fetal blood-brain barrier, exacerbating the hormonal effects on synapse formation and neuron myelination.

In addition to maternal depression, research by Leviton et al. (2018)also highlights the role of other prenatal psychosocial factors such as socioeconomic stress, young maternal age, smoking habits, and pre-conception obesity. These factors describe the chronic social stress experienced by mothers before and during pregnancy, which then contributes to an increased risk of ADHD in premature children. This study shows that the effects of socioeconomic stress do not only manifest through lifestyle behaviors, but also work at the biological level through increased systemic inflammation and cortisol regulation disorders. Thus, both studies show that the prenatal phase is a critical period in which the mother's emotional and social stress can leave permanent biological traces on the fetal nervous system.

Finally, economic factors also make an important contribution. These factors are often considered an indirect social context, but they have a significant influence on the emergence of ADHD through both biological and psychosocial pathways, with findings from two articles (4.4%) showing that differences in the prenatal and postnatal phases affect their mechanism of action on ADHD risk. this factor acts as a contextual determinant that exacerbates the impact of other risk factors.

In the prenatal phase, research by Leviton et al. (2018)found that low socioeconomic status of the mother correlates with an increased risk of premature birth, neonatal mechanical ventilation, and exposure to intrauterine infection. These three conditions contribute to an increased risk of ADHD in children through biological stress and inflammatory mechanisms. Poor economic conditions are also associated with a lack of access to optimal nutrition during pregnancy, which can disrupt the mother's thyroid and cortisol hormone homeostasis and impact fetal nervous system development. Thus, the mother's economic status during the prenatal period plays a dual role as an indicator of psychosocial stress and as a biological determinant through its influence on nutrition and environmental exposure.

Postnatal Phase

The postnatal period is an important stage in the course of ADHD risk, as neurodevelopmental vulnerabilities that began to form during the prenatal phase continue and are further influenced by various biological, environmental, psychosocial, and economic exposures after birth. In this phase, risk is influenced by a combination of internal factors, including biological vulnerability, inflammation, stress regulation, and neural development processes, as well as external factors, including home environment, social pressure, and family economic status. Thus, prenatal factors do not stand alone but interact dynamically with postnatal conditions in shaping the clinical manifestation of ADHD. In this phase, this vulnerability does not cease, but continues to be modulated by various risk factors that work simultaneously throughout the child's development.

In this phase, genetic factors continue to play a role as biological vulnerabilities that modulate children's responses to various stressors after birth. In the postnatal phase, one article shows that genetic predispositions formed during the prenatal period remain active and influence neuropsychological development during childhood. Research by Hernández-Lorca et al. (2023)shows that children with high genetic predisposition (high PRS) experienced a significant increase in ADHD symptoms during the pandemic quarantine period. This condition illustrates a continuous gene-environment correlation, in which individuals with genetic vulnerability are more sensitive to psychosocial stress. These results reinforce the view that genetics not only determine the basic potential for disorders, but also modulate physiological responses to environmental stressors throughout development.

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This vulnerability is then reinforced by various biological factors that emerge after birth. In the postnatal phase, eleven articles show that biological factors continue to maintain and reinforce the vulnerability that has been established since the prenatal period. If prenatal maternal physiological changes pose an early risk, then the postnatal phase maintains that risk through inflammation, early infection, metabolic dysfunction, intermittent hypoxia, and changes in the microbiota. These biological processes disrupt the ongoing maturation of the nervous system, facilitating the emergence or worsening of ADHD symptoms in childhood. Research by Allred et al. (2017)confirmed that increased IL-6R and TNF-α cytokines during the first month of life correlate with ADHD symptoms at age 10. Wilkinson et al. (2017)showed that high NSE and low sNCAM levels post-trauma are associated with long-term attention deficits.

Lee et al. (2016)confirmed that chronic skin inflammation increases the risk of ADHD by almost threefold, mainly due to sleep disturbances and excessive immune activation. S. Liu et al. (2021)showed that chronic dysglycemia increases brain oxidative stress and disrupts dopaminergic neurotransmission. Two other studies by (Joseph et al., 2025; Wu et al., 2017)reveal that intermittent hypoxia due to sleep disorders causes dopamine- e dysregulation and increased cortisol, exacerbating hyperactivity.

Furthermore, Ing et al. (2017, 2020) concluded that exposure to general anesthesia under the age of five increases the risk of ADHD due to the neurotoxic effects of GABAergic agents on synaptogenesis. Haile et al. (2024)showed that chronic respiratory disorders and neonatal morbidity decrease attention function, while Ahrens et al. (2024)demonstrated that early gut microbiota dysbiosis affects central nervous system regulation through the gut–brain axis.

Finally, Hadzic et al. (2017)reported that children who experienced bacterial meningitis in their first year of life had a thirty-fold higher risk of ADHD compared to controls, demonstrating the destructive impact of early CNS infection on neural tissue. The consistency of findings from these eleven studies confirms that postnatal biological factors work through systemic inflammatory pathways, metabolic disorders, and sustained hypoxic stress. These biological changes prolong the effects of prenatal exposure and facilitate the clinical expression of ADHD in childhood and adolescence.

After biological factors, postnatal environmental influences also play a major role in maintaining existing vulnerability. In the postnatal phase, five articles show that the environment continues to play an important role in maintaining and modulating vulnerability that has been established since the prenatal period. After birth, factors such as sleep quality, home conditions, social stress, and changes in routine also affect stress regulation and cognitive function, which can trigger or exacerbate ADHD symptoms. Research by Joseph et al. (2025) shows that poor sleep environment factors, such as loud snoring due to poor room air quality or noise pollution, can increase hyperactive behavior through impaired brain oxygenation. These findings are reinforced by research by Hernández-Lorca et al. (2023), which shows that environmental stress during quarantine triggers an increase in ADHD symptoms, especially in children with a high polygenic risk score (PRS). The interaction between genetic factors and social pressure confirms that children with biological vulnerability are more sensitive to changes in routine and social isolation. Prolonged stress during lockdown is suspected to disrupt dopamine regulation and the HPA axis, thereby exacerbating the clinical manifestations of ADHD.

Additionally, Ahrens et al. (2024)found that exposure to an unhygienic home environment and a low-fiber diet is associated with gut microbiota dysbiosis, which then affects children's cognitive function through the gut–brain axis mechanism. Studies (Song et al., 2025; Ünver & Perdahlı Fiş, 2022)highlight that drastic changes in the social environment during the pandemic, mainly due to isolation, social restrictions, and the shift to online learning, are very strong postnatal environmental factors in triggering new ADHD symptoms or worsening symptoms in children who were previously biologically vulnerable.

In addition to the environment, psychosocial factors also have a sustained

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contribution in the postnatal phase . In the postnatal phase, four articles show that psychosocial factors continue to influence the vulnerability that has been formed since the prenatal period. If maternal stress previously contributed to the early risk of ADHD, then after birth, social pressure, stigma, trauma, and family conditions play a role in maintaining or worsening children's stress regulation dysfunction. These factors can disrupt emotional development and impulse control, thereby reinforcing the expression of ADHD symptoms in childhood. Research by Nunes-Moreno et al. (2022)found that children with gender dysphoria had twice the risk of being diagnosed with ADHD compared to the control group. These results are explained by the concept of minority stress theory, in which social pressure and stigma of minority identity produce chronic stress that triggers HPA axis dysregulation and increased cortisol, similar to the pattern of prenatal maternal stress. This chronic stress response can worsen executive dysfunction and impulse control in individuals who already have underlying neurobiological vulnerability.

The research by Ünver & Perdahlı Fiş (2022)expands on these findings in an extreme social context. Refugee children who experienced social dislocation, loss, and limited mental health services during the pandemic showed a significant increase in cases of externalizing disorders, including ADHD. Living conditions in environments with repeated trauma, cultural changes, and limited access to services contributed to attention disorders through repeated activation of the physiological stress system, which ultimately inhibited the development of prefrontal neural connectivity.

Additionally, the study by Song et al. (2025)provides a broad population perspective on the impact of social stress due to the pandemic. During the lockdown period, there was a significant increase in the incidence of ADHD, not only in children but also in adolescents and young adults. Researchers attribute this to increased academic pressure, social isolation, and disruption of daily routines, all of which induce chronic psychosocial stress and sleep disturbances. This situation reflects how large-scale social environmental factors can amplify the expression of ADHD symptoms at the population level.

A recent study that further enriches this understanding is Ahrens et al. (2024). Although primarily focused on biological factors, this study also found a significant association between family psychosocial status and the composition of children's gut microbiota. Children from families with high levels of socioeconomic and emotional stress tend to have more severe microbial dysbiosis patterns, which affect neuroactive metabolites such as short-chain fatty acids that play a role in prefrontal cortex function. These findings suggest that psychosocial factors not only work in the emotional realm but also indirectly affect biological systems through the gut–brain axis pathway.

Finally, postnatal economic conditions also reinforce the risk of ADHD through various interrelated pathways. In the postnatal phase, findings from one article show that family economic conditions after birth remain an important determinant that reinforces the risk of ADHD. If in the prenatal phase economics affect maternal health and the quality of the uterine environment, then after birth, economic limitations have an impact on diet, environmental hygiene, cognitive stimulation, and access to health services, which indirectly maintain the biological and psychosocial vulnerability of children to ADHD. Research by Ahrens et al. (2024) shows that the economic conditions of families after birth are closely related to variations in dietary patterns, environmental hygiene, and the composition of children's gut microbiota. Children from low-income families have a higher proportion of pathogenic bacteria and lower levels of short-chain fatty acids, which negatively impact the development of the central nervous system. Economic inequality also affects access to cognitive stimulation and health services, thereby reinforcing the biological effects on attention and impulse control.

CONCLUSION

Based on a review of 28 observational articles published between 2015 and 2025, this study confirms that ADHD in children is a neurodevelopmental condition that arises from multifactorial interactions between biological,

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genetic, environmental, psychosocial, and economic aspects that occur from the prenatal to postnatal phases. Biological factors have been shown to be the most dominant component, particularly through inflammatory mechanisms, metabolic disorders, hypoxia, and toxic exposure that affect neural maturation. Genetic factors play a role as a basis for vulnerability that is then modulated by environmental exposure, while psychosocial and economic factors reinforce the manifestation of symptoms through the effects of chronic stress, family instability, microbiota dysbiosis, and limited access to health care and cognitive stimulation. These findings indicate that the risk of ADHD does not stem from a single factor, but is formed from a series of interrelated processes that impact the development of the central nervous system, particularly the prefrontal area that regulates attention, impulse control, and executive function.

RECOMMENDATIONS

There are several limitations to this study that should be noted. This study is still limited in its use of data that is largely observational and cross-sectional, so that cause-and-effect relationships and changes in risk over time cannot be described in depth. In addition, variations in the quality of reports, population heterogeneity, and differences in risk factor measurement methods between articles mean that the results must be interpreted with caution. Several important aspects, such as family conditions, parenting patterns, and social environment variations, were not fully covered in all of the articles analyzed, so the potential for contextual bias still exists.

Based on these limitations, several suggestions can be made. For future researchers, it is recommended to develop longitudinal studies covering longer periods of time to see more comprehensive patterns of ADHD risk development from the prenatal to postnatal periods. The use of data covering biological, environmental, and psychosocial aspects is expected to provide a more comprehensive understanding of the interaction between factors in the development of ADHD. In addition, future research needs to consider variations in family conditions, parenting patterns, and the environment in which children grow up, so that the findings can be more relevant and easily applied in prevention and treatment efforts for at-risk children.

ACKNOWLEDGEMENT

Sincere appreciation is also extended to the Physiotherapy Study Programme, Faculty of Health Sciences, Muhammadiyah University of Surakarta, for their support and facilitation. In addition, the author gratefully acknowledges the Institute for Research and Innovation of Muhammadiyah University of Surakarta and the Ministry of Research, Technology, and Higher Education (Kemenristek Dikti) for their funding and support, which made this research possible.

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