OLSZAK, Joanna, ZALEWA, Karolina, BUDZYŃSKI, Rafał, KAMIENIAK, Anna, KLEPACZ, Natalia, CZUBA, Anna, POLESZCZUK, Mikołaj, POLESZCZUK, Karol, MILEWSKA, Alicja and KAUS, Marta. Cerebral Palsy - A Comprehensive Analysis of the Causes. Journal of Education, Health and Sport. 2025;80:59668. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.80.59668
https://apcz.umk.pl/JEHS/article/view/59668

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 22.03.2025. Revised: 25.04.2025. Accepted: 05.05.2025. Published: 07.05.2025.

Cerebral Palsy - A Comprehensive Analysis of the Causes

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Abstract

Introduction and Purpose:

Cerebral palsy (CP) is a complex motor and postural disorder resulting from damage to the developing brain. The causes of CP are diverse and can occur at various stages of a child's life—prenatal, perinatal, and postnatal. The aim of this paper is to review the scientific literature on the causes of CP, with a particular focus on the mechanisms of brain damage at each stage.
Material and methods:
A comprehensive literaturere view was conducted using the PubMed database, focusing on articles published until the end of 2024. The search included the keywords:"cerebral palsy","etiology","brain damage"and "prevention" in various combinations. Relevant studies were selected based on criteria such as etiology of cerebral palsy.
Results:
Analysis of available studies indicates that prenatal causes predominantly include genetic abnormalities, intrauterine infections, and exposure to toxins. Perinatal causes involve asphyxia, mechanical trauma, and prematurity. In the postnatal period, infections of the nervous system, traumatic brain injuries, and perinatal complications play a significant role. These causes are often interdependent, highlighting the necessity of a comprehensive approach to CP risk analysis.
Conclusion:
Understanding the multifactorial etiology of CP is essential for developing effective prevention strategies. Further research into the mechanisms of brain damage may contribute to reducing the incidence of new cases and improving the quality of life for children with CP.

Keywords: cerebral palsy, etiology, brain damage, prevention

Introduction

Cerebral palsy (CP) is a group of permanent, non-progressive movement and posture disorders, representing the most common cause of severe neurological disability in children.[1] For this reason, CP is a significant public health issue, particularly in the context of prevention.[2] This disorder occurs at a similar frequency worldwide, affecting approximately 17 million people.[3] It is estimated to occur at a rate of about 3 cases per 1,000 births.[4] This disease begins in early childhood.[5][6] The term "cerebral palsy" is a collective designation that reflects the inherent

diversity of this condition.[3] It encompasses all non-progressive, permanent movement and posture disorders resulting from damage occurring in the developing brain of the fetus and infant.[7]The causes of cerebral palsy are complex, involving both genetic and environmental factors, making awareness of their interplay crucial for effective diagnosis and treatment. CP can manifest in various clinical forms and display different neuropathological patterns visible in brain imaging. This disorder is associated with multiple deficits, which may include intellectual disability, speech and language difficulties, as well as motor impairments.[8]The main types of cerebral palsy include: spastic hemiplegia, affecting one side of the body; spastic diplegia, involving both lower limbs; spastic quadriplegia, where movement limitations affect all limbs; as well as extrapyramidal and dyskinetic palsy, characterized by uncontrolled, involuntary movements. Additionally, ataxic palsy is marked by difficulties in maintaining balance and coordinating movements.[9] Spasticity is the most common form, occurring in approximately 80% of children with CP. Motor impairments can lead to secondary complications such as pain, hip dislocation, balance issues, hand dysfunction, or equinus deformity.The diagnosis of CP is primarily based on clinical assessment; however, magnetic resonance imaging (MRI) can help identify brain damage, especially when the cause of symptoms is unclear. To assess the degree of motor disability and therapy effectiveness, tools such as the Gross Motor Function Classification System are used.[10] The discussed symptoms affect a child's daily functioning, including learning ability, communication, and independent movement. Therefore, a thorough neurodevelopmental assessment is crucial to identify associated deficits and develop an individualized treatment plan.[8] Moreover, CP often coexists with other developmental disorders, such as intellectual disability, autism, epilepsy, or vision problems.[3] The diagnosis of CP does not specify particular pathological or etiological characteristics, encompassing both genetic and environmental factors.[11] The search for risk factors for cerebral palsy remains an ongoing area of research. Frequently analyzed factors include low gestational age and adverse events during pregnancy, childbirth, and the neonatal period. However, it is equally important to consider preconception factors, such as maternal health before pregnancy, as well as postnatal factors that may impact a child's neurological development after birth.[12] In some cases, the cause of CP may remain unclear, further complicating the diagnostic process.[8] Understanding the pathophysiology of CP plays a key role in developing effective protective and therapeutic strategies. Unfortunately, despite growing awareness of the significance of this disorder, there are

still significant gaps in its early detection, treatment, and prevention. However, with advancements in biomarker research, there is hope that our knowledge of the mechanisms underlying CP will expand, paving the way for the development of new therapeutic methods.[3]

Prenatal Causes

Cerebral palsy (CP) can have various causes, including prenatal factors that play a key role in its development. The most important ones include preterm birth, multiple pregnancies, intrauterine growth restriction (IUGR), and maternal infections. Increasing research also points to the significance of genetic predispositions, which may influence these risk factors. The latest genetic analyses show that in approximately 28–31% of CP cases, a genetic basis can be identified, with most cases associated with single nucleotide variants and a smaller proportion linked to copy number variants. Advancing genetic diagnostics opens new perspectives in understanding CP etiology and potentially tailoring treatments to individual patient needs.[11] Discoveries regarding the genetic causes of CP began appearing in the literature as early as the 1990s.

However, it was not until 2015 that the first study on systematic genome sequencing of individuals with CP was published. Since then, the number of scientific reports has steadily increased, pointing to both inherited genetic variants in familial CP cases and de novo variants in sporadic cases. This highlights the significant role of rare, strong genetic changes in CP pathogenesis. Identified genes show considerable heterogeneity, which is not surprising—they often overlap with other neurodevelopmental disorders such as intellectual disability, epilepsy, and autism, as well as movement disorders like hereditary spastic paraplegias, dystonia, and ataxia.[3] Although current analyses are limited by the small number of studied patients, high genetic heterogeneity, and a lack of validation studies, researchers have managed to identify genes associated with pathways regulating neurological development and neuronal connectivity. Similar to other neurodevelopmental disorders such as autism or intellectual disability, the genomic architecture of CP is likely highly complex. While we are only beginning to understand the impact of genetic factors on this condition, further research could provide valuable insights into CP neurobiology and reveal new therapeutic targets.[13] Current CP findings can be

classified based on effect size and allele frequency in the population, which are inversely correlated. Most identified genes follow Mendelian inheritance patterns, meaning they contain rare variants with significant impact, such as CTNNB1 and FBXO31. There is also evidence of variable penetrance for some variants, such as F5, which can lead to diverse phenotypes.

Additionally, rarer mutations in genes like COL4A1 and COL4A2 are associated with significant phenotypic variability, particularly in terms of neurological disorder severity, including structural brain anomalies, strokes, epilepsy, and motor impairment. This suggests a pleiotropic influence of these genes on symptom diversity. Only one genome-wide association study (GWAS) has analyzed CP as a polygenic disorder, identifying a single nucleotide polymorphism (SNP) in GRIK4 that reached genome-wide significance among individuals with spastic CP. Preliminary evidence also suggests the existence of monogenic forms of CP, particularly in families with more than one affected member, implying Mendelian inheritance. Identified recurrent de novo mutations in multiple different genes, mostly damaging, are a common cause of sporadic CP. There are also cases of genetic variants with a broad phenotypic spectrum, such as COL4A1, COL4A2, and F5, possibly due to their multifactorial nature. Some genes, such as KANK1, ADD3, and AP complexes, have a well-documented role in familial CP. Among CP cohorts, one of the most frequently identified variants is a pathogenic mutation in CTNNB1, accounting for 2.6–4% of genetically diagnosed cases. CTNNB1 encodes β-catenin, a key protein in brain development involved in cell migration and the WNT signaling pathway, which regulates cell proliferation and differentiation during central nervous system development. Patients with CTNNB1 syndrome exhibit a typical clinical phenotype, including facial dysmorphisms, microcephaly, motor, language, and cognitive impairments, as well as behavioral abnormalities such as autistic traits or aggressive behaviors. Interestingly, the WNT/β-catenin pathway is also regulated in oligodendrocyte progenitors in the white matter lesions of term neonates who have experienced severe hypoxic-ischemic encephalopathy. Its dysregulation leads to arrested maturation and failed remyelination in animal models, suggesting a potential convergence point between monogenic and traumatic causes of CP. Additionally, the discovery of recurrent de novo mutations in RHOB and FBXO31 genes in unrelated individuals with similar clinical phenotypes suggests a monogenic basis for CP. One of the most common genetic variants in CP involves mutations in the COL4A1 and COL4A2 genes, which have long been linked to cerebrovascular diseases. These genes encode the alpha-1 and alpha-2 chains of type IV collagen, which are

crucial for the vascular basement membrane. Variability in these genes correlates with significant heterogeneity in symptoms, ranging from structural brain anomalies to epilepsy and motor impairment. Early brain development is a crucial period vulnerable to gene-environment interactions, which can influence neuroplasticity in the long term. Consequently, COL4A1 variability is suggested to be a potentially modifiable risk factor, and cesarean section for fetuses with mutations in this gene may reduce the risk of perinatal brain hemorrhage. CP is also associated with non-genetic risk factors, such as preterm birth, multiple pregnancies, intrauterine growth restriction (IUGR), maternal infections, and perinatal asphyxia. Infants with IUGR show brain structure alterations, including reduced gray matter volume, affecting oxygen delivery to the forebrain and cerebellum, thereby disrupting development. Moreover, babies with a birth weight below the 10th percentile have a twofold increased risk of CP. Among term newborns, the risk of CP is significantly higher in twin pregnancies compared to singleton pregnancies. In multiple pregnancies, additional factors such as low birth weight, monochorionicity, and twin-to-twin transfusion syndrome (TTTS) also increase CP risk. TTTS, by causing uneven blood distribution between twins, leads to severe multi-organ changes that can negatively affect brain development. CP is reported in 30–40% of children who survive TTTS, and the risk is seven times higher in donor twins.[11] Although prematurity and hypoxic-ischemic injury are well-documented risk factors for CP, in up to one-third of children with this condition, no classic causes are identified.[13] In addition to well-known child-related risk factors such as periventricular leukomalacia or intraventricular hemorrhage, maternal and paternal factors also significantly influence CP development, including diabetes, drug abuse, and seizure disorders. In high-income countries, the increasing trend of delayed parenthood has prompted researchers to examine the impact of parental age on CP risk. Most studies have focused on older maternal age, but a recent case-control study also highlighted the significance of paternal age as a risk factor.[12] Research indicates that lower gestational age is a significant risk factor for cerebral palsy (CP).[14] Among maternal factors, medical conditions such as chorioamnionitis play a key role, demonstrating a positive correlation with CP. However, the impact of preeclampsia remains inconclusive—some analyses suggest its association with CP, while others do not confirm this link. Low birth weight most frequently exhibits a positive correlation with CP risk, while findings regarding male sex are inconsistent. Furthermore, the combination of male sex with prematurity or low birth weight further increases the likelihood of CP.[15] Other significant risk factors

include intrauterine exposure to infection or maternal fever during labor, ischemic stroke, and congenital anomalies. While each of these factors, if severe enough, can independently lead to CP, their cumulative effect most often exceeds the body's adaptive capacity and results in neurological damage.[16] Studies also indicate that in low-income countries, CP risk varies and is higher than that observed in high-resource countries. Contributing factors include a higher prevalence of maternal HIV infection during pregnancy. Modifying these factors should be considered a potential strategy for reducing the prevalence of this disorder.[17]

Perinatal Causes

Cerebral palsy (CP) is often associated with prematurity and perinatal asphyxia. Approximately 50% of CP cases occur in children born prematurely.[3] Premature birth remains a major risk factor for CP.[18] Studies have shown that 43.7% of children with CP were born prematurely, with the highest risk among infants born before the 28th week of pregnancy. Only 19.5% of children born at term had CP. Another risk factor for premature births is the use of assisted reproductive technologies (ART), which double the incidence of CP, mainly due to the higher number of preterm births associated with ART.[11] Premature infants are particularly vulnerable to brain damage caused by factors such as hypoxia, growth restriction, infections, or inflammatory reactions that can occur during pregnancy or birth.[3] Among the main perinatal risk factors for CP are extremely low birth weight (ELBW) and very low birth weight (VLBW), preterm birth, neonatal encephalopathy, preeclampsia, an Apgar score below 4 in the first minute, multiple pregnancies, infections, and inflammatory conditions.[19][20] Perinatal asphyxia was long considered the main cause of cerebral palsy (CP), and interventions such as cesarean section and electronic fetal heart monitoring were suggested as preventive measures.
However, despite the significant increase in cesarean sections in recent years, no decrease in CP

incidence has been observed. Research suggests that less than 10% of CP cases are associated with acute perinatal asphyxia, and most CP cases have their origin before birth. A surprising finding from recent studies indicates higher effectiveness of genetic diagnostics among children with CP who experienced perinatal asphyxia, compared to children with CP without asphyxia. This may suggest that perinatal hypoxia is not always the result of events during birth, but could be a secondary effect of earlier susceptibility, which may partly have a genetic basis.[11] Severe birth complications, such as placental abruption, umbilical cord prolapse, or uterine rupture, also significantly increase the risk of cerebral palsy (CP). Fortunately, these are rare events, often leading to neonatal death, so their contribution to the overall number of CP cases remains small.[16] In one study conducted in two major centers specializing in cerebral palsy in Japan, MRI scans and outcomes of children with CP, born at term and over three years old, were analyzed. The etiology of cerebral palsy in these children was classified based on imaging as: perinatal ischemic stroke (PIS), brain dysgenesis (CD), middle cerebral artery infarction (MCAI), and deep gray matter infarction (DGMI). In the case of PIS, three types were distinguished: periventricular venous infarction (PVI) and two types of arterial infarctions. The studies aimed to determine the relationship between types of brain damage and early symptoms such as hemiparesis, as well as long-term health outcomes, including motor function (e.g., age of walking onset), intellectual development, and epilepsy. The results showed that the majority of children with CP had PIS, with PVI being the most common cause in children born at term. It turned out that the type of brain damage, especially PVI, was closely associated with the onset of early hemiparetic symptoms, as well as with long-term functional outcomes. This study highlights the importance of perinatal factors, including ischemic strokes, which can lead to severe brain damage, and thus to the development of cerebral palsy.[21] Numerous pieces of evidence indicate that cerebral palsy rarely results solely from issues related to perinatal care.

Increasingly, research suggests that genetic factors play a significant role in its etiology. Similar to many other neurological disorders, CP exhibits a complex inheritance model that can be described as multifactorial. This means both etiological and genetic heterogeneity, as well as complex interactions between genetic predispositions and environmental factors. These discoveries are changing the current understanding, emphasizing the need for further research.[22]

Postnatal Causes

Cerebral palsy (CP) is a neurological disorder that can have various causes, including those occurring after the birth of the child, i.e., postnatal causes. These factors lead to damage to the developing brain in infancy or early childhood, causing permanent motor and postural disorders. One of the most common postnatal risk factors for CP is central nervous system (CNS) infections, such as meningitis and encephalitis. These infections can be caused by bacteria (e.g., *Streptococcus pneumoniae*, *Neisseria meningitidis*) or viruses (e.g., herpes virus, enteroviruses). Inflammation and immune response mechanisms can lead to brain swelling, neuronal necrosis, and white matter damage, which negatively affect the child's development.[23] Some studies have shown that elevated levels of inflammatory proteins, measured in the blood of extreme preterm infants in the first days after birth, are prognostic for the occurrence of cerebral palsy (CP) at 24 months of age. Specifically, prolonged elevation of proteins such as tumor necrosis factor-α, interleukin-8, TNF-α receptor-1, interleukin-6, E-selectin, and insulin-like growth factor-binding protein-1. Additionally, the presence of at least four elevated proteins previously associated with cognitive impairment and microcephaly increases the likelihood of developing both diparesis and hemiparesis. Thus, monitoring the levels of inflammatory proteins in the first two weeks of life in preterm infants can serve as an important indicator of the risk of CP development.[24] Another significant cause is mechanical brain injuries, which can occur as a result of traffic accidents, falls, or injuries related to child abuse (e.g., shaken baby syndrome).
These injuries can lead to intracranial hemorrhages, hypoxia, and secondary inflammatory and degenerative changes that impair the functioning of the nervous system.[25] Intraventricular hemorrhage and periventricular leukomalacia are the main pathological changes observed in preterm infants who develop spastic cerebral palsy.[9] Postnatal brain hypoxia is also an important postnatal factor in the development of CP. It can occur as a result of cardiac arrest, prolonged seizures, meconium aspiration into the lungs, as well as severe lung diseases in preterm infants, such as bronchopulmonary dysplasia. Insufficient oxygen supply to the brain leads to the death of nerve cells, particularly in areas responsible for motor control, such as the motor cortex and basal ganglia. Metabolic disorders, such as severe neonatal hypoglycemia or

hyperbilirubinemia, may also play a significant role in the pathogenesis of CP. Prolonged low glucose levels can lead to brain damage, especially in areas sensitive to energy deficiencies, while high bilirubin levels can cause toxic damage to the basal ganglia, leading to neurological symptoms, including spasticity and athetoid involuntary movements. In summary, postnatal causes of cerebral palsy primarily include infections, injuries, hypoxia, metabolic disorders, as well as tube feeding, mechanical ventilation, and dopamine administration at six months of age.[25] Early diagnosis and treatment of these conditions can reduce the risk of permanent brain damage and minimize the consequences for the child's motor function.

Summary

Understanding the etiology of cerebral palsy (CP), encompassing both genetic and environmental factors as well as their interactions, is crucial for improving therapeutic possibilities and implementing personalized medical care. Contemporary research highlights the increasing role of interactions between genes and environmental factors, which may explain the diversity of symptoms and disease progression. In-depth analysis of CP causes can contribute to the adjustment of preventive and therapeutic measures, enabling a more individualized approach to patients. This allows for the optimization of medical and rehabilitative interventions, such as occupational therapy, physical therapy, and speech therapy, which in the long term can improve the quality of life for individuals affected by CP.[11] Risk factors for CP, arising from the interaction of maternal, birth-related, and child-related factors, are particularly prominent in low-income countries. Many of these factors could be effectively minimized through improvements in prenatal and perinatal care. Appropriate medical support during pregnancy and the postpartum period, including early detection of risks and preventive interventions, could significantly reduce the risk of CP. Accurate patient selection and the use of advanced technologies, such as high-throughput genetic platforms, play a key role in the diagnostic process for CP. Precisely identifying target genes and using rigorous clinical interpretation criteria allow for more accurate diagnosis of the disease's causes. With next-generation sequencing technology, it is possible to identify rare mutations that may influence the development of CP, which could contribute to new treatment methods. Studies suggest that next-generation sequencing should be considered a fundamental diagnostic tool for patients with cryptogenic CP, especially in cases where the

etiology remains unclear. This approach can significantly contribute to the development of personalized therapy methods and improve patient care.[26]

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