Early Detection of Neurologic Disease: A Scoping Review

Arvin Kommu and Ollie Fegter

Abstract

Neurologic disorders include a diverse display of conditions that hinder cognitive, sensory, and motor functions, significantly contributing to global health and economic challenges. These disorders may be neurodevelopmental, neurodegenerative, or acquired over a person's lifetime, and their effects are frequently associated with psychological, social, and financial difficulties. This paper offers a scoping review of the current knowledge regarding the etiology, diagnosis, and prevention of neurologic disorders, highlighting the critical nature of early detection and intervention. Genetic and environmental factors, such as chromosomal abnormalities, exposure to teratogens, malnutrition, and hypoxic or anoxic birth events, contribute to the development of these disorders. Progression in diagnostic methods, including biomarker identification, neuroimaging techniques (e.g., MRI, PET, EEG), and genetic testing, has facilitated earlier and more accurate diagnoses, enhancing treatment and disease management. Preventative measures, such as lifestyle changes, folic acid supplementation, prenatal screenings, and, in certain instances, fetal surgery, have shown positive potential in reducing disease severity or preventing its development.

Introduction

Neurologic disorders are conditions that affect the nervous system, including the brain, spinal cord, and peripheral nerves. There are hundreds of disorders that can impact cognitive, motor, and sensory functioning. These disorders can be neurodevelopmental, such as autism spectrum disorders and cerebral palsy, neurodegenerative, such as Alzheimer's disease and Parkinson's disease, or occur across the lifetime, such as brain injuries, epilepsy, multiple sclerosis, and migraines.

Worldwide, around 3 billion people are impacted by these disorders, and their prevalence is expected to grow exponentially in low to middle-class countries over the next ten years. Consequently, neurologic disorders represent a significant portion of the worldwide health burden. This health burden comes not only directly from the diagnosis and management of neurologic conditions but also from the psychological and social consequences of chronic illness. Psychosocial factors such as mood, cognition, and interpersonal relationships are often negatively affected. For example, neurodegenerative conditions are often associated with higher rates of depression and anxiety. Traumatic brain injury and Huntington’s disease can lead to mood instability, including increased irritability, anger, or sudden mood swings. Chronic pain, memory loss, reduced mobility, language impairment, and communication difficulties are just a few of the cognitive and physical burdens resulting from these disorders. In addition to the personal health burden, neurologic disorders pose a significant economic burden worldwide. The economic burden comes through the costs of diagnosis and ongoing management and treatment, including medications and various therapies. The loss of productivity and income due to disability or decreased work capacity further worsens the financial strain on individuals and their families. For example, the average annual cost of ALS care can exceed $143,000, often forcing families to rely on savings or fundraising.

Thus, it is of the utmost importance for these disorders to be detected early to maximize the potential for prevention and treatment. Neurologic disorders are typically diagnosed through a comprehensive evaluation that includes a detailed medical history, physical and neurologic examination, and diagnostic tests, including bloodwork and neuroimaging. The purpose of this

paper is to explore, through a scoping review, which diagnostic methods and biomarkers can be used for the early detection of neurologic disorders. Subsequently, we will explore how using these tools can minimize the impact of these devastating disorders. To begin this review, we will describe the etiology of these disorders and later how to prevent and minimize the subsequent impact.

1. **How do these disorders happen/etiology (causation)?**

Genetic Factors

Genetic factors play a key role in the development of neurologic disorders. The most genetically linked neurologic disorders include: Huntington's disease, Parkinson's, ALS, and Alzheimer's. Humans have 23 pairs of chromosomes, and each pair is inherited from both mother and father. Through genetic translation and transcription, proteins are produced from the genetic code located on the chromosomes. Proteins form the internal machinery within brain cells and the connective tissue between brain cells. They also control the chemical reactions that allow brain cells to communicate with each other. Some genes make proteins that are important for the early development and growth of the infant brain. For example, the ASPM gene makes a protein that is needed for producing new nerve cells (or neurons) in the developing brain.

Chromosome abnormalities, including deletions, duplications, and translocations of genetic code have frequently been linked to genetic neurologic disorders. These mutations result from abnormalities in the DNA replication process. Proteins with incorrect genetic code either misfold or transform into a different protein, thus altering their intended purpose. Deletions are the loss of one nucleotide in a base pair and can result in the development of genetic disorders, such as cystic fibrosis. Two-thirds of cystic fibrosis cases, as well as the cat cry syndrome, in which children have a cry that sounds similar to a cat meowing, result from a deletion of a nucleotide during replication. Duplications are when one or more sections of DNA are produced. Overproduction of protein overwhelms the normal cellular activity, and underproduction leads to deficiencies that impair biological functions. For example, in the Charcot-Marie-Tooth disease, a duplication of the *PMP22* gene causes overproduction of myelin protein, which disrupts nerve signaling and leads to muscle weakness and sensory loss. Spinal muscular atrophy (SMA) results from a deletion or mutation in the *SMN1* gene, leading to underproduction of the SMN protein, which is vital for motor neuron survival and causes muscle weakness. A malfunctioning or altered protein can make biological systems more prone to degradation. For example, in cases of Alzheimer's disease, the buildup of misfolded amyloid-beta proteins forms plaques in the brain and hinders brain function. Mutations in genes like ASPM can trigger immune responses and inflammation in the brain, contributing to conditions like microcephaly and affecting overall brain size and function.

These disorders can be passed down from either parent and can persist through each generation (dominant) or skip generations (recessive). These dominant and recessive disorders, along with others such as polyploidy and X-linked conditions, can have various inheritance patterns. For example, mitochondrial diseases are inherited maternally. Since mothers typically pass on organelles to their offspring, including mitochondria, mutations in the mitochondrial DNA can lead to disorders that affect cellular energy production, such as Leigh syndrome or MELAS.

Environmental factors:

Prebirth environmental factors

Prebirth environmental factors, particularly teratogens, play a significant role in the development of neurologic disorders during fetal development. Teratogens are substances or conditions that can cause developmental malformations or functional deficits in a developing fetus and include drugs, medications, chemicals, certain infections, and toxic substances consumed by the mother during pregnancy or exposed to a developing fetus. One notable example is alcohol, which can lead to Fetal Alcohol Syndrome (FAS). FAS can cause abnormal facial features, reduced head and brain size, and various physical and behavioral disabilities. Medical consensus indicates there is no safe level of alcohol consumption during pregnancy. Teratogens can also increase the risk of miscarriage, preterm labor, or stillbirth.

A developing fetus can also be exposed to infection through its mother. This occurs through a process called vertical transmission, where a pathogen is passed from the mother to the fetus via the bloodstream and through the placenta. Emerging evidence suggests that a broader range of viral and bacterial infections during pregnancy can subtly alter fetal brain development, potentially leading to disorders like schizophrenia, autism spectrum disorder, and depression. Additionally, subsequent studies in humans and mouse models linked prenatal exposure to single pathogens, complex infections, and inflammatory disorders with changes in fetal brain development leading to a wide spectrum of cognitive deficits and neuropsychiatric disorders, including autism spectrum disorder.

Another factor surrounding birth that has correlation to the development of neurologic disorders is premature birth. If a baby is born too early, their brain may not be fully developed. The brain undergoes significant development during the third trimester of pregnancy, and if premature infants are born before this critical period is complete, their brains are more susceptible to injury and damage. Although in most cases, the cause of premature birth is unknown, it may occur due to infections, pregnancy complications that require early induction of labor, or other maternal stress. While neurologic syndromes are not commonly associated with all preterm births, more common symptoms/effects include: smaller size, with a head that's large compared with the body, features that are sharper and less rounded than a full-term baby's features due to a lack of cells that store fat, low body temperature, trouble breathing, and feeding problems. In the case where neurologic disorders develop, premature birth exposes the developing brain to external stimuli that differ drastically from the in-utero environment, making key neural cells like oligodendrocyte precursors and neurons highly vulnerable to stress, infection, and malnutrition. This vulnerability contributes to brain injuries such as periventricular leukomalacia, which can disrupt critical neural connections, reducing brain size, and lead to abnormalities in brain organization. Additionally, the fragile blood vessels in the brains of premature infants are prone to rupture, leading to bleeding in the ventricles.

Neurologic disorders can develop as a result of hypoxic or anoxic events that occur during birth or delivery. Anoxia refers to a complete cutoff of oxygen to the brain for a period of time, while hypoxia occurs when oxygen levels are restricted, but some oxygen flow is still present. One common cause of oxygen deprivation is umbilical cord complications. This can include prolapse, when the umbilical cord moves out of place; compression, when pressure is applied to the umbilical cord, restricting blood flow; and nuchal cord, when the umbilical cord wraps around the baby’s neck. When the oxygen supply to the brain is disrupted, it can affect the entire body.

If a doctor suspects that a newborn has been deprived of oxygen at birth, the Apgar score becomes a crucial assessment tool. The Apgar score evaluates a newborn’s overall

health within its first five minutes, based on factors such as skin color, reflexes, muscle tone, breathing, and heart rate. For newborns with a low Apgar score, immediate intervention is crucial to limit systemic neurologic damage. In addition to an Apgar score, doctors can monitor newborns for signs of oxygen deprivation by evaluating whether the baby presents with weak reflexes, poor muscle tone, or seizures. Infants with mild brain injuries due to restricted oxygen supply often respond well to treatment and recover without complications. However, moderate to severe brain injuries can have long-term effects on a child's developing brain and body, potentially leading to neurologic disorders such as cerebral palsy, autism, and seizures. Cognitive and behavioral issues, including ADHD, learning disabilities, and lower IQ scores, may also arise. Additionally, developmental delays in speech, motor skills, and nutritional deficiencies due to feeding difficulties can occur. The most effective way to prevent oxygen-related injuries is through close monitoring of both mother and child during pregnancy and delivery. Any signs of distress should be addressed immediately, and if complications arise, healthcare providers should act promptly to ensure the best possible outcome for the newborn.

Lifestyle factors such as malnutrition, poor diet, and lack of physical activity all contribute to the development of neurologic disorders as well. Essential nutrients like omega-3 fatty acids, iron, and B vitamins are critical for brain development and function. If an individual is unable to consume these nutrients regularly through meals and supplements, they may be more prone to developing neurologic disorders. Deficiencies can lead to cognitive impairments, delayed myelination, and an increased risk of neurodevelopmental disorders such as ADHD and learning disabilities. Physical movement promotes neuroplasticity, brain oxygenation, and myelination. Nutrient levels, influenced by factors like illness, malnutrition, or supplementation, are essential for the nervous, cardiovascular, and immune systems, and any imbalances can lead to various clinical effects. An example of a disorder caused by malnutrition is Wernicke's encephalopathy, a neurologic disorder caused by thiamine (vitamin B) deficiency and typically associated with anorexia nervosa and alcohol use disorder. Another example is scurvy, caused by vitamin C deficiency, which can lead to neurologic

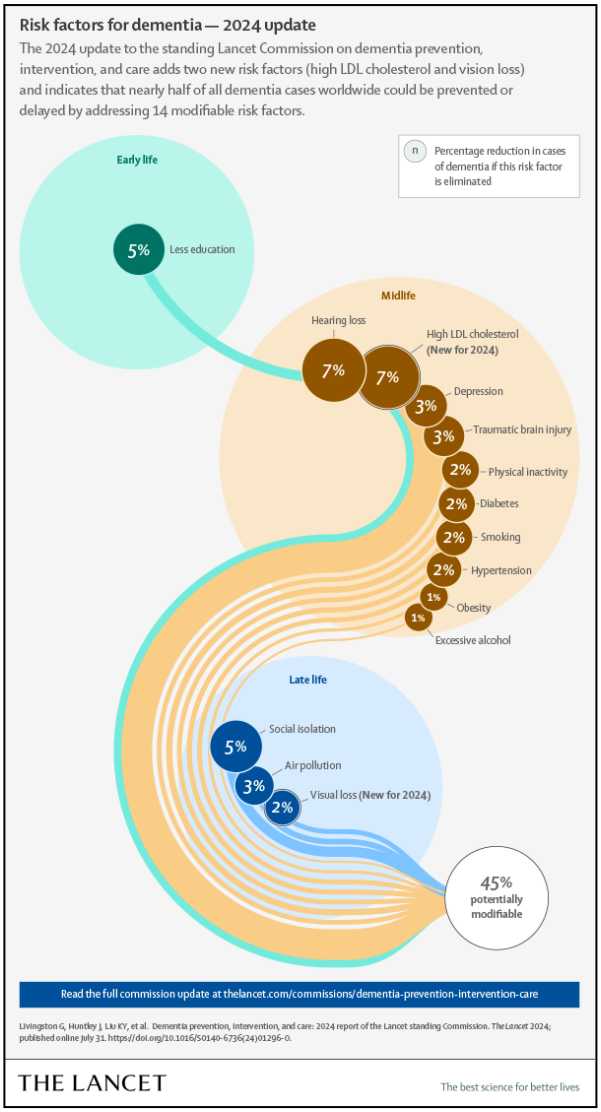

Figure 1. Risk Factors for Dementia (Lancet, 2024)

complications such as irritability, depression, and neuropathy, alongside its effects on connective tissue and immune function. Thiamine is crucial for several neuronal metabolic processes, and its depletion can result in neuronal cellular death and pathological lesions in various regions of the brain. Along with a lack of proper nutrition, an inactive lifestyle, especially in early childhood, may slow motor development and cognitive function and increase the risk of obesity, cardiovascular disease, type 2 diabetes, osteoporosis, and neurodegenerative disorders such as Alzheimer's disease later in life. In addition, physical inactivity inhibits neurogenesis (the formation of new neurons) by reducing circulation and the availability of growth factors, which are crucial for brain cell growth and repair.

Improving vascular (heart) health is the most effective method for promoting brain health. The brain relies on a constant supply of oxygen and nutrients delivered through the bloodstream. Any condition that affects the heart and blood vessels such as hypertension (chronically elevated blood pressure), atherosclerosis (the buildup of fats, cholesterol, and other substances in and on the artery walls), or Type 2 diabetes mellitus (a chronic metabolic condition characterized insulin resistance due to hyperglycemia) can impair cerebral blood flow, increasing the risk of cognitive decline and neurologic disorders.

A significant number of modifiable risk factors for dementia are vascular risk factors, meaning that improving heart health can also protect cognitive function (See Figure 1). Some key vascular risk factors that contribute to dementia include high blood pressure, which damages small blood vessels in the brain and increases the risk of stroke, as well as high cholesterol, which leads to plaque buildup in arteries, reducing blood flow to the brain. These risk factors are modifiable through lifestyle changes, and managing them can significantly reduce the likelihood of developing neurodegenerative diseases like the ones mentioned earlier. To improve heart health through lifestyle changes, it is important to focus on a heart-healthy diet, engage in regular physical activity, maintain a healthy weight, abstain from smoking, manage stress, and get enough sleep. Prioritizing fruits, vegetables, and whole grains while limiting sodium can lower blood pressure and cholesterol, as these foods are rich in nutrients and fiber. Also, the CDC and the Physical Activity Guidelines for Americans recommend that individuals should get at least 30 minutes of exercise at least five days a week (150 minutes a week) in order to improve overall health. After reviewing methods for early detection of neurologic disease, this paper will continue to explore preventative measures that individuals can take in the future.

2. Diagnosis, Methods, and Biomarkers For Early Detection

The first step in preventing or limiting the effects of neurologic disorders is early detection and timely diagnosis. Diagnosis for rare neurologic disorders can often take time, with some individuals seeing multiple providers and waiting years for a definitive diagnosis. In recent years, differential diagnosis has been aided by advancements in the fields of biomarkers, neuroimaging, and karyotyping.

Biomarkers are measurable biological indicators such as proteins, genes, or imaging patterns that signal the presence or progression of a disease. They can be detected through blood tests, genetic analysis, saliva, or neuroimaging, helping identify individuals at risk for neurologic disorders before symptoms arise. Biomarkers can be detected both in vitro and after birth. In vitro biomarkers for neurologic disorders are molecules or substances detected inside or outside the fetus’ body, like blood, amniotic fluid, or cerebrospinal fluid (CSF). These can

indicate the presence, severity, or progression of a neurologic condition. For example, amniotic fluid can be tested for neurologic disorders such as neural tube defects or developmental disorders like Down Syndrome or Fragile X Syndrome. Biomarkers drawn from blood after birth can include biological fluids like blood, urine, and cerebrospinal fluid (CSF; fluid that circulates around the brain and spinal cord) and can provide valuable diagnostic information. Biomarkers help in identifying at-risk individuals, reducing disease variability in clinical studies, tracking disease progression, and serving as targets for clinical trials. The implementation of biomarkers able to detect amyloid and tau proteins in CSF has facilitated an earlier and more accurate diagnosis of Alzheimer's disease.

Additionally, advances in neuroimaging techniques have proven to be essential in early diagnosis, treatment monitoring, and improving our understanding of neurologic disorders. Neuroimaging refers to a variety of techniques used to visualize either the structure or function/activity of the brain and nervous system. This allows scientists and doctors to see inside the brain non-invasively, which can aid in medical diagnosis, surgical planning, research, and treatment monitoring. Table 1 provides an overview of some common neuroimaging techniques.

Table 1. Neuroimaging Techniques

Imaging Technique	Brief description	Neurologic disorders that benefit from neuroimaging biomarkers	Pros	Cons
MRI	Uses magnetic fields and radio waves to produce detailed images of brain structures.	Brain tumors, multiple sclerosis (MS), stroke, traumatic brain injury (TBI), Alzheimer's disease	High-resolution images, no radiation, excellent soft tissue contrast	Expensive, slow, not suitable for some patients with metal implants or claustrophobia
CT	Uses X-rays to produce cross-sectional images of the brain.	Stroke, head trauma, hydrocephalus, brain hemorrhages	Fast, widely available	Radiation exposure, less detailed soft tissue contrast than MRI
PET	Uses radioactive tracers to detect metabolic and molecular brain activity.	Alzheimer’s, Parkinson’s, epilepsy, brain tumors	Detects early functional changes, good for tracking disease progression	Expensive, involves radiation, limited availability

fMRI	Measures brain activity by detecting changes in blood oxygen levels.	Epilepsy, brain mapping before surgery, stroke recovery, depression	Non-invasive, shows real-time brain function, high spatial resolution	Sensitive to motion, slower than EEG, indirect measure of neural activity
EEG	Measures electrical activity in the brain using scalp electrodes	Epilepsy, sleep disorders, encephalopathy, brain death diagnosis	High temporal resolution, portable, non-invasive, inexpensive	Poor spatial resolution, limited depth detection, sensitive to noise

Table 1 Legend. ***MRI = magnetic resonance imaging, CT = computed tomography, PET = positron emission tomography, fMRI = functional magnetic resonance, EEG = electroencephalogram***

Another way we can prevent the impact of neurologic disorders is through the analysis of genes and karyotyping to look at a patient's chromosomes. As described above, neurologic disorders can stem from mutations in genes, so analyzing genetic makeup throughout numerous stages of life can be beneficial. By identifying these mutations early, during stages like parental screening, in vitro fertilization, or postnatal testing, we can enable early interventions, lifestyle planning, and treatments that delay progression of the disease. Karyotyping is a diagnostic technique that pairs and orders all the chromosomes in a genome, allowing doctors to detect large-scale genetic abnormalities such as aneuploidies (ex. trisomy 21), deletions, duplications, translocations, and inversions. By using standardized staining procedures to highlight structural features, karyotypes provide valuable insights into birth defects and genetic disorders. Genetic testing, on the other hand, can detect smaller mutations in individual genes. Table 2 presents some notable advantages and limitations of different methods of genetic testing and karyotyping.

Table 2. Advantages and Limitations of Genetic Testing Methods.

Method	Description	Advantages	Limitations
Karyotyping	Visual examination of stained chromosomes to identify large-scale chromosomal abnormalities, deletions, duplications, or rearrangements.	- Low cost - Detects large chromosomal abnormalities (e.g., trisomy 21) - Widely available - Wide view of genome	- Cannot detect small mutations (<5–10 Mb) - Lower resolution than molecular methods

Non-Invasive Prenatal Testing	Sequencing of the entire DNA to detect mutations in both coding and non-coding regions.	- Comprehensive detection of known and new mutations - High resolution - Data can be reanalyzed later	- High cost - Longer turnaround time - Requires advanced interpretation - May detect incidental or uncertain findings
Whole Genome Sequencing	Analyzes cell-free fetal DNA in maternal blood to screen for common chromosomal abnormalities.	- Safe and non-invasive - Can be done as early as 10 weeks - High accuracy (>99%) for trisomy 21, 18, and 13 - Reduces anxiety with early, reliable results	- Positive results require confirmation (e.g., amniocentesis) - Limited to common conditions - Small risk of false positives/negatives - High cost and health insurance may not pay for it if the pregnancy is considered low-risk
Ultrasound Imaging (NT & Anatomy Scans)	Non-invasive imaging using sound waves to visualize internal structures such as organs, nerves, blood vessels, and fetal anatomy.	- No ionizing radiation - Portable and real-time guidance - Visualizes nerves and vessels - Improves the safety and accuracy of procedures - Useful in pediatrics - Faster onset and longer duration of nerve blocks	- Lower resolution at greater depths - Bone blocks imaging - Operator-dependent and requires training - Limited in detecting genetic or molecular issues
Fetal Surgery	In-utero surgical procedure to correct select structural abnormalities before birth.	- Can improve long-term outcomes - Prevents further fetal damage - Increases chances of survival in severe conditions - Enhances organ development in some cases	- High maternal and fetal risk - Preterm labor, membrane rupture, bleeding, infection - Requires specialized surgical teams - Only for specific, severe conditions

Folic Acid Supplementation	Daily intake of folic acid (400–800 mcg) before and during early pregnancy to prevent neural tube defects; also used to treat folate deficiency and lower homocysteine	- Reduces risk of neural tube defects (e.g., spina bifida, anencephaly) - Inexpensive and widely available - Added to many foods - Also helps treat folate deficiency and high homocysteine	- Must be taken before/during early pregnancy for full effect - High doses may mask B12 deficiency or increase seizure risk - Possible side effects (e.g., nausea, skin reactions) - Limited benefit if taken too late

3. What can be done to prevent or minimize a disorder?

As previously mentioned, the early detection of neurologic disorders is crucial for enhancing patient outcomes and alleviating the overall disease burden. Progress in diagnostic techniques, including biomarkers identified through blood fluid analysis, neuroimaging methods, genetic testing, and karyotyping, enables earlier and more precise identification of neurologic conditions. Timely diagnosis not only allows for prompt intervention but also helps in connecting patients with suitable specialists, such as genetic counselors and neurologists, thus reducing the often lengthy diagnostic journey.

Lifestyle choices, such as maintaining a balanced diet rich in essential nutrients, engaging in regular physical activity, ensuring adequate sleep, and participating in cognitive activities that stimulate the mind, contribute to brain health and can decrease the likelihood of developing certain neurologic conditions. Avoiding teratogens and harmful exposures during critical developmental periods, particularly during pregnancy, is essential for preventing fetal brain injuries and developmental issues. Routine prenatal screenings, including anatomy ultrasound scans and genetic testing techniques like the ones described earlier in the paper, facilitate the early identification of potential disorders, allowing for a more beneficial strategy of limitation.

In summary, preventing and minimizing neurologic disorders requires a comprehensive approach that integrates early diagnostic advancements with modifiable lifestyle factors and medical interventions. Through these combined efforts, we can reduce the incidence and severity of neurologic diseases, improving quality of life for affected individuals, and reducing the societal and economic burden these disorders impose.

References

1. Miller, Gabrielle F., et al. “Costs of Nonfatal Traumatic Brain Injury in the United States, 2016.” *Medical Care*, vol. Publish Ahead of Print, 28 Jan. 2021, https://doi.org/10.1097/mlr.0000000000001511 .
2. Thakur, Kiran T., et al. “Neurological Disorders.” *PubMed*, The International Bank for Reconstruction and Development / The World Bank, 2016, www.ncbi.nlm.nih.gov/books/NBK361950/ .
3. Alcolea, Daniel, et al. “Blood Biomarkers in Neurodegenerative Diseases: Implications for the Clinical Neurologist.” *Neurology*, U.S. National Library of Medicine, 25 July 2023, pmc.ncbi.nlm.nih.gov/articles/PMC10435056/ .
4. Medline Plus. “Health Risks of an Inactive Lifestyle.” *Medlineplus*, National Library of Medicine, 1 Sept. 2017, medlineplus.gov/healthrisksofaninactivelifestyle.html .
5. National Institute of Neurological Disorders and Stroke. “Brain Basics: Genes at Work in the Brain | National Institute of Neurological Disorders and Stroke.” *Www.ninds.nih.gov*, July 2010, www.ninds.nih.gov/health-information/patient-caregiver-education/brain-basics-genes-work-brain .
6. Mannino MC;Cassidy MB;Florez S;Rusan Z;Chakraborty S;Schoborg T; “Mutations in Abnormal Spindle Disrupt Temporal Transcription Factor Expression and Trigger Immune Responses in the Drosophila Brain.” *Genetics*, U.S. National Library of Medicine, pubmed.ncbi.nlm.nih.gov/37831641/
7. Bouyssi-Kobar, Marine, et al. “Third Trimester Brain Growth in Preterm Infants Compared with in Utero Healthy Fetuses.” *Pediatrics*, vol. 138, no. 5, Oct. 2016, p. e20161640, https://doi.org/10.1542/peds.2016-1640 .
8. Ganguly, Prabarna. “Deletion.” *National Human Genome Research Institute*, 2020, www.genome.gov/genetics-glossary/Deletion.
9. Cleveland Clinic. “Teratogens: Effects, Types, Risks & Prevention.” *Cleveland Clinic*, 2022, my.clevelandclinic.org/health/articles/24325-teratogens.
10. “Sera PreTRM.” *Sera PreTRM*, 3 Nov. 2022, www.pretrm.com/for-moms/healthy-pregnancy-blog/premature-delivery/ashleys-preterm-birth-story-the-psychological-effects-of-premature-birth/ .
11. Mayo Clinic. “Premature Birth.” *Mayo Clinic*, 22 Mar. 2024, www.mayoclinic.org/diseases-conditions/premature-birth/symptoms-causes/syc-20376730.
12. “Oxygen Deprivation.” *Birth Injury Guide*, www.birthinjuryguide.org/causes/oxygen-deprivation/.
13. Brown, Thomas M. “Neuropsychiatric Scurvy.” *Psychosomatics*, vol. 56, no. 1, Jan. 2015, pp. 12–20, https://doi.org/10.1016/j.psym.2014.05.010
14. Tsalamandris, Gabriela, et al. “The Role of Nutrition in Neurological Disorders.” *Nutrients*, vol. 15, no. 22, 7 Nov. 2023, pp. 4713–4713, www.ncbi.nlm.nih.gov/pmc/articles/PMC10674646/, https://doi.org/10.3390/nu15224713.
15. Medline Plus. “Health Risks of an Inactive Lifestyle.” *Medlineplus*, National Library of Medicine, 1 Sept. 2017, medlineplus.gov/healthrisksofaninactivelifestyle.html.
16. Novak, Iona, et al. “Early, Accurate Diagnosis and Early Intervention in Cerebral Palsy.” *JAMA Pediatrics*, vol. 171, no. 9, 1 Sept. 2017, p. 897, pubmed.ncbi.nlm.nih.gov/28715518/, https://doi.org/10.1001/jamapediatrics.2017.1689.

17. Soler-Casas, A, et al. “The Impact of Prenatal Diagnosis on the Prevention of Chromosomal Mental Retardation. Chromosomal Alterations That Can Be Detected by Prenatal Diagnosis.” *Revista de Neurologia*, vol. 42 Suppl 1, July 2006, pp. S27-32, pubmed.ncbi.nlm.nih.gov/16506129/.
18. Aymé, S, et al. “Prenatal Diagnosis in France.” *European Journal of Human Genetics : EJHG*, vol. 5 Suppl 1, 1997, pp. 26–31, pubmed.ncbi.nlm.nih.gov/9101175/.
19. Thompson, Paul M, et al. “Imaging Genomics.” *Current Opinion in Neurology*, vol. 23, no. 4, Aug. 2010, pp. 368–373, https://doi.org/10.1097/wco.0b013e32833b764c.
20. World Health Organization. “Over 1 in 3 People Affected by Neurological Conditions, the Leading Cause of Illness and Disability Worldwide.” *World Health Organization*, 14 Mar. 2024, www.who.int/news/item/14-03-2024-over-1-in-3-people-affected-by-neurological-conditions--the-leading-cause-of-illness-and-disability-worldwide.
21. Ossenkoppele, Rik, et al. “Tau Biomarkers in Alzheimer’s Disease: Towards Implementation in Clinical Practice and Trials.” *The Lancet Neurology*, vol. 21, no. 8, Aug. 2022, pp. 726–734, https://doi.org/10.1016/s1474-4422(22)00168-5.
22. “Neural Tube Defects.” *Www.hopkinsmedicine.org*, www.hopkinsmedicine.org/health/conditions-and-diseases/neural-tube-defects.
23. Baschat, Ahmet Alexander. “Preventing and Treating Birth Defects: What You Need to Know.” *Www.hopkinsmedicine.org*, www.hopkinsmedicine.org/health/treatment-tests-and-therapies/preventing-and-treating-birth-defects-what-you-need-to-know.
24. National Institute of Neurological Disorders and Stroke. “Brain Basics: Genes at Work in the Brain | National Institute of Neurological Disorders and Stroke.” *Www.ninds.nih.gov*, July 2010, www.ninds.nih.gov/health-information/patient-caregiver-education/brain-basics-genes-work-brain.
25. Pittman, Alan, and John Hardy. “Genetic Analysis in Neurology.” *JAMA Neurology*, vol. 70, no. 6, 1 June 2013, p. 696, https://doi.org/10.1001/jamaneurol.2013.2068 . Accessed 18 Mar. 2019. al-Haddad, Benjamin J. S., et al. “The Fetal Origins of Mental Illness.” *American Journal of Obstetrics and Gynecology*, vol. 221, no. 6, 1 Dec. 2019, pp. 549–562, www.sciencedirect.com/science/article/pii/S000293781930777X , https://doi.org/10.1016/j.ajog.2019.06.013.
26. van Campen, Julia. “Karyotype — Knowledge Hub.” *GeNotes*, 17 Aug. 2022, www.genomicseducation.hee.nhs.uk/genotes/knowledge-hub/karyotype/ .
27. O’Connor, Clare. “Karyotyping for Chromosomal Abnormalities.” *Nature.com*, 2008, www.nature.com/scitable/topicpage/karyotyping-for-chromosomal-abnormalities-298/.
28. tess. “All about Genetic Sequencing – TESS Research Foundation.” *TESS Research Foundation*, 20 Mar. 2023, www.tessresearch.org/genetic-sequencing/.
29. “NIPT Test – Everything You Need to Know.” *Testmottagningen*, 17 Oct. 2024, www.testmottagningen.se/en/artiklar/kvinnohalsa/nipt-test-fordelar-nackdelar-och-vad-du-bor-tanka-pa/ .
30. PFCLA. “Pros and Cons of Preimplantation Genetic Screening (PGS).” *Pacific Fertility Center Los Angeles*, 15 Apr. 2021, www.pfcla.com/blog/preimplantation-genetic-screening-pgs-pros-and-cons .

31. Roberts, Steve. “Ultrasound-Pros and Cons.” *European Society for Paediatric Anaesthesiology*, 2018, www.euroespa.com/science-education/specialized-sections/espa-pain-committee/us-regional-anaesthesia/ultrasound-pros-and-cons/.
32. “Fetal Surgery | Boston Children’s Hospital.” *Www.childrenshospital.org*, www.childrenshospital.org/treatments/fetal-surgery.
33. Meara Withe. “10 Conditions Doctors Assess with an EEG.” *Medicalnewstoday.com*, Medical News Today, 19 June 2023, www.medicalnewstoday.com/articles/10-conditions-diagnosed-with-an-eeg#sleep-disorders .
34. Radiology Info. “Magnetic Resonance, Functional (FMRI) - Brain.” *Radiologyinfo.org*, 2018, www.radiologyinfo.org/en/info/fmribrain.
35. Krans, Brian. “Brain PET Scan.” *Healthline*, Healthline Media, 2 Nov. 2018, www.healthline.com/health/brain-pet-scan#purpose.
36. Johns Hopkins Medicine. “Computed Tomography (CT or CAT) Scan of the Brain.” *John Hopkins Medicine*, 2024, www.hopkinsmedicine.org/health/treatment-tests-and-therapies/computed-tomography-ct-or-cat-scan-of-the-brain.
37. Cleveland Clinic. “Brain MRI: What It Is, Purpose, Procedure & Results.” *Cleveland Clinic*, 9 May 2022, my.clevelandclinic.org/health/diagnostics/22966-brain-mri.
38. Gottesman, Rebecca F., and Sudha Seshadri. “Risk Factors, Lifestyle Behaviors, and Vascular Brain Health.” *Stroke*, vol. 53, no. 2, 10 Jan. 2022, https://doi.org/10.1161/strokeaha.121.032610.
39. “6 Lifestyle Changes to Improve Your Heart Health.” *Tallahassee Memorial Health*, 2017, www.tmh.org/healthy-living/blogs/healthy-living/6-lifestyle-changes-to-improve-your-heart-health.
40. Livingston, Gill, et al. “Dementia Prevention, Intervention, and Care: 2024 Report of the Lancet Standing Commission.” *The Lancet*, vol. 404, no. 10452, 1 July 2024, www.thelancet.com/journals/lancet/article/PIIS0140-6736(24)01296-0/fulltext, https://doi.org/10.1016/s0140-6736(24)01296-0.