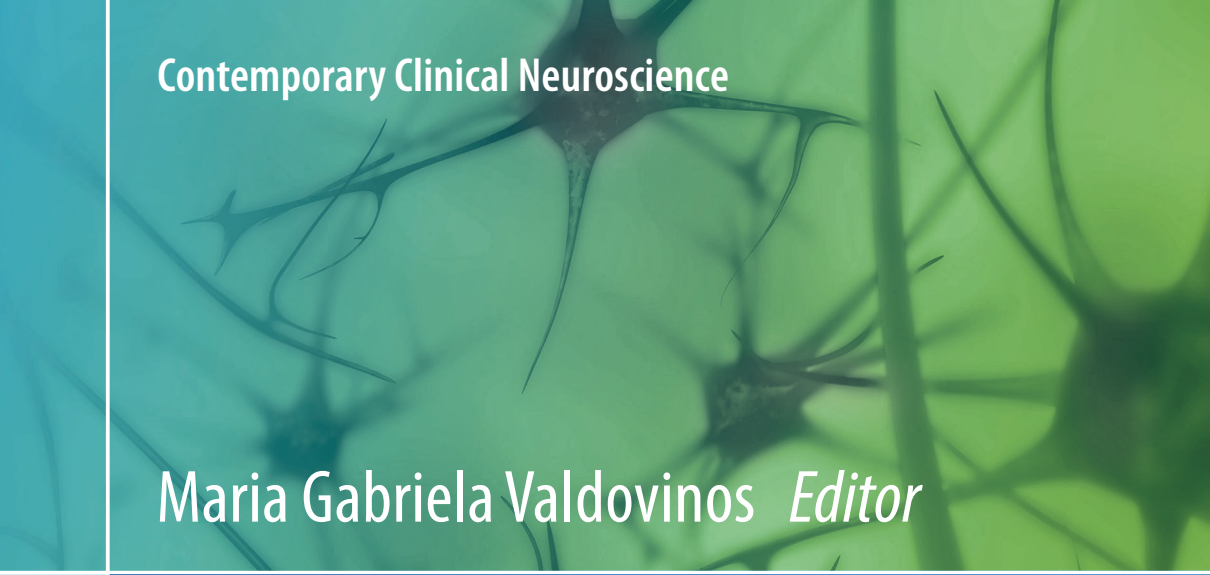

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Intellectual and Developmental Disabilities

A Dynamic Systems Approach

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Perinatal Risk and Later Intellectual and Developmental Disabilities

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Abstract Intellectual and developmental disabilities (IDD) are lifelong medical conditions that affect neurodevelopmental trajectories. Numerous risk factors have been linked to IDD, including biomedical and environmental influences. However, specific underlying etiology is not necessarily easily identified. Nongenetic pathways are dependent upon sensitive periods of neurodevelopment, including the perinatal period. In this chapter, we review nongenetic perinatal risk factors that influence fetal brain development and confer risk to later IDD diagnosis in childhood. Following this, we outline factors that mitigate risk for future offspring development of IDD. Given that environmental factors are inherently modifiable, we discuss research implications for future public health policy and advocacy.

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Intellectual and developmental disabilities (IDD) are characterized by impairments or altered developmental trajectories of intellectual and adaptive functioning (American Psychiatric Association [APA], 2013) and represent neurodevelopmental disorders that begin prior to 18 years of age. In terms of prevalence rates, 1 out of 100 children will develop an IDD following birth (Maulik et al., 2011), which can emerge from both genetic and nongenetic sources. Genetically linked IDD represent 30% to 50% of all IDD and result from various genetic abnormalities, including chromosomal alterations (e.g., trisomy 21), inherited genetic signatures (e.g., Fragile X syndrome), and/or single gene traits (e.g., Prader-Willi syndrome) (Kaufman et al., 2010; Rauch et al., 2006). Nongenetically linked IDD arise from a

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variety of risk factors or events that occur pre- and perinatally. Mechanistic pathways that connect nongenetic exposures with later IDD are less understood, despite the high prevalence. This chapter will review the current understanding of common exposures known to increase risk for IDD and elucidate key directions for future work.

IDD include a range of co-occurring neurodevelopmental conditions, such as autism spectrum disorder (autism), cerebral palsy, Down syndrome, fetal alcohol spectrum disorders, schizophrenia, and attention-deficit hyperactivity disorder (ADHD). IDD populations experience a myriad of health disparities, namely, decreased life expectancy (Anderson et al., 2013). In the United States, approximately 17% of children aged 3–17 years are diagnosed with an IDD (Cogswell et al., 2022). Data from the National Health Interview Survey suggests that the prevalence of *any* diagnosed developmental disability increased from 2019 to 2021 among children 3–17 years, from 7.40% to 8.56% in the United States (Zablotsky et al., 2023), denoting an increase in prevalence. IDD conditions manifest in the early childhood period, altering an individual's neurodevelopmental trajectory compared to their typically developing peers. The lifelong impacts associated with an IDD diagnosis underscore the need for research and healthcare support to improve the well-being of individuals with IDD and their families.

Perinatal exposures have long been shown to increase the risk for IDD in later childhood, although pathways that causally explain adverse outcomes are only beginning to be understood. Progress in this area depends on careful consideration of the multiple and diverse exposures and complex interactions that increase IDD risk via immediate, short-term, and longer-term effects that unfold across childhood. *Developmental equifinality* (Cicchetti & Rogosch, 1996) refers to the idea that exposure to diverse sets of perinatal exposures can have similar phenotypic expressions (in the case of IDD, related to cognitive, adaptive, or motor delays). Thus, identifying mechanistic pathways leading to IDD will require an understanding of how a wide range of perinatal exposures (preterm birth, infection, neurotoxin exposure, etc.) influence key developmental systems. *Developmental multifinality* (Cicchetti & Rogosch, 1996) refers to the idea that any one exposure can lead to a heterogeneous range of outcomes, which is also relevant for understanding pathways that connect perinatal exposures to IDD risk. For example, preterm birth is not only associated with risk for IDD but a wide range of neuropsychiatric disorders and/or growth delays, and despite increased risk, many children also show no long-term consequences (van Baar et al., 2005). Adding to the complexity, perinatal risk exposures often co-occur, making it difficult to disentangle the individual versus interactive influences of these adversities on developmental risk. Timing or severity of exposure, infant characteristics (male sex, low birth weight), and neuroprotective factors relate to the relative risk for IDD. Neonatal factors (male sex, low birth weight), postnatal environmental risk, and neuroprotective factors may influence the likelihood of later risk and resilience (Huang et al., 2016).

Multiple theories from various disciplines consider how and why perinatal factors may influence later IDD risk. The most prominent or widely cited theories include the "fetal programming hypothesis" (Seckl & Holmes, 2007), the "developmental programming hypotheses" (Barker, 2004; Langley-Evans, 2006, 2015), and the "developmental origins of health and disease" (DOHAD) (Barker, 1990, 2004). These models generally hypothesize that intrauterine environments, events, or injury/disturbances influence developmental trajectories at a sensitive point in development. Programmatically, these exposures influence neurocognitive, behavioral, and physical development across neonatal, infant, toddler, later childhood, and even adult stages of life. A discussion of the differences in theoretical perspective goes beyond the scope of the chapter. However, these theoretical viewpoints offer a framework for understanding how and why such a diverse set of exposures can have similar or vast consequences related to IDD (and comorbid developmental problems, which we discuss later in this chapter). For example, perinatal stressors, including nutrient or oxygen deprivation, teratogen exposure, and psychosocial adversity, are hypothesized to causally influence epigenetic and hypoxic changes in the fetal brain and body, which then influence hormones, metabolic activity, and brain development. In combination, these cascading influences predispose offspring to a spectrum of adverse outcomes. Pathways may involve myriad disturbances including altered inflammatory, glucocorticoid, or HPA axis programming (Packard et al., 2016; Xiong & Zhang, 2013), GABA signaling (Braat & Kooy, 2015; Cherubini & Ben-Ari, 2023; Deidda et al., 2014; Sgadó et al., 2011; Tang et al., 2021), and macro-level changes to the brain structure and functional organization (Bohlken et al., 2014). Further, the risk that any one exposure will lead to later IDD will depend on timing, severity, co-occurring risk factors, and neuroprotective factors (Fig. 1).

In what follows, we consider risk factors during the perinatal period that have implications for IDD in offspring. These are potential consequences of one's genetic profile, environmental exposures, and birth experiences. Some known perinatal exposures that impact developmental outcomes are term and preterm brain injury,

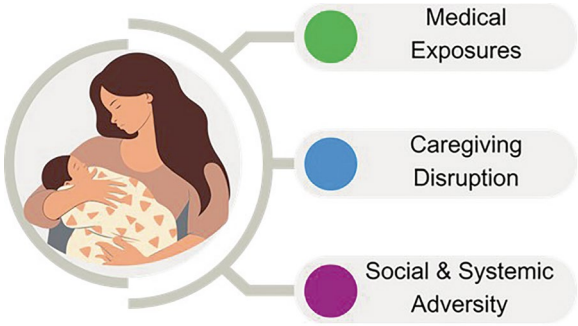

Fig. 1 Overview of perinatal factors that influence IDD risk and development

such as intraventricular hemorrhage, white matter injury, birth trauma—subpial hemorrhage, large subarachnoid hemorrhage, and perinatal stroke. Perinatal infections such as bacterial meningitis and viral meningoencephalitis present known risk factors. Other risk factors that will be addressed include infants born prematurely and infants requiring intensive neonatal interventions. First, we will define various adversities that occur during the perinatal period. Second, we will connect these exposures with various IDD that emerge in later childhood. Third, we will discuss evidence for prevention and intervention in neonatal intensive care units (NICUs) and in early development, shown to effectively mitigate risks associated with perinatal adversities and IDD in later childhood. Finally, we will discuss key gaps in understanding and addressing structural barriers that inform policy, and future directions for research.

Defining Perinatal Exposures That Pose Risk for IDD

There are many etiological pathways during brain development implicated in the perinatal environment that can consequently increase risk of IDD. We will review some common perinatal exposures that are known to undermine healthy brain development and increase risk for IDD. They are grouped into two overarching themes: medical exposures (perinatal brain injury, infection, and prematurity) and social adversities (maternal-infant separation in the NICU, and maternal mental health affecting bonding and responsiveness).

Medical Exposures

Perinatal Brain Injury The early postnatal period is a time of significant risk for brain injury and can take the form of numerous etiologies. Most commonly, 1.5 in 1000 term-born infants experience rates of hypoxic-ischemic encephalopathy (HIE) experience rates of hypoxic-ischemic encephalopathy (HIE) in 1.5 in 1000 births (Kurinczuk et al., 2010) and stroke occurs in stroke in 0.6 in out of 1000 births (Laugesaar et al., 2007). Across causes, brain injury during the perinatal period has been associated with later neurodevelopmental impairments including motor, language, and cognitive delays (Novak et al., 2018). While there is no single cause for many of these types of injury, there are multiple contributing factors that play a role in increased prevalence.

One of the most cited causes of neonatal brain injury relates to birth trauma or damage caused as a direct result of labor and/or delivery (Gupta & Cabacungan, 2021). Rather than a specific cause, birth trauma constitutes a wide range of insults

across the spectrum, from minor cuts and scrapes to severe and life-threatening damage to the brain and other organs. Of factors that make birth trauma more likely is the use of vacuums and forceps during delivery (Cieplinski & Bhutani, 1996; Linder et al., 2013) and infants who are large for gestational age, which can be common in mothers who are obese or experience gestational diabetes (Boulet et al., 2003; Persson et al. (2013); Shuffrey et al., 2023). Beyond direct mechanical causes, numerous other factors can contribute to significant brain injury, with the most common being hypoxic events. These events represent instances where insufficient oxygen reaches the brain and are associated with greater neuro developmental difficulties, especially in motor domains such as those implicated with cerebral palsy (Graham et al., 2008). Importantly, while brain injury can impact infants born at any age, it is more common in infants born prematurely due to an increased vulnerability of the immature brain to perinatal insults and a greater prevalence of prematurity-related complications with lower birth gestational age (Inder et al., 2023).

Perinatal Infections One of the other major risks for later neurodevelopmental concerns is perinatal infections, including neonatal sepsis, meningitis, and various viruses that cause high fevers and respiratory infections such as pneumonia and bronchiolitis. Due to the relatively immature immune system at birth and before, neonates are particularly susceptible to infection and vulnerable to longer-term impacts on development (Ygberg & Nilsson 2012). This is especially true for infants born prematurely who have even less mature immune responses compared to term-born infants—although differences quickly minimize between term- and preterm-born infants shortly after birth (Olin et al., 2018).

Of the most common infections, neonatal sepsis, which is an extreme response to an infection, often occurs within the first days of life and impacts as many as 2.2% of infants. Although often treatable, long-lasting neurological effects can be observed throughout childhood (Glaser et al., 2021). Like sepsis, neonatal meningitis can arise from a diverse set of causes; however, it ultimately results in severe inflammation of the meninges, the membranes surrounding the brain. Importantly, more severe cases are associated with significant motor and cognitive disabilities later in childhood (Ku et al., 2015). In both sepsis and meningitis, inflammation has been reported as the primary cause of subsequent impairments likely due to its impacts on the brain (Humberg et al., 2020). Conversely, respiratory disorders in the neonatal period are common, including pneumonia and bronchiolitis, but rather than just inflammation, these disorders can also lead to severe respiratory distress which impacts oxygenation saturation (Gill et al., 2022; Nissen, 2007). Further, studies have reported that two known viruses such as Zika (Mlakar et al., 2016) and Herpes Simplex (Fa et al., 2020) when contracted during pregnancy leading to infant in utero exposure have caused structural cortical changes. Regardless of the cause, however, the presence of neonatal infection is a significant risk factor for later neurodevelopmental impairments including cerebral palsy and intellectual disability (Rand et al., 2016; Mitha et al., 2013).

Prematurity and Related Complications Premature birth is one of the most common risk factors for later IDD. While most infants are born at term around 40 gestational weeks, approximately 1 in 10 births are infants born preterm before 36 gestational weeks (Osterman et al., 2023; Walani, 2020). While those rates have remained consistent over the past two decades (Blencowe et al., 2012; Gyamfi-Bannerman & Ananth, 2014), advancements in neonatal care have dramatically increased the survival rate (Bell et al., 2022; Younge et al., 2017) and also the age at which ex utero survival for extremely premature infants is possible. However, there is consistent evidence that infants born preterm are at greater risk for a wide range of developmental delays and impairments, including pronounced sensorimotor deficits (Cabral et al., 2016; Hee Chung et al., 2020), language delays (Barre et al., 2011; Woodward et al., 2009), and broad cognitive impairments (Hee Chung et al., 2020; Kerr-Wilson et al., 2012) alongside higher rates of neurodevelopmental disorders (Bhutta et al., 2002; Joseph et al., 2017; Larroque et al, 2008; Sucksdorff et al., 2015). IDD is a potential risk factor for very prematurely born babies, as studies have found that prematurity may cause future development of neurobehavioral needs related to lower IQ, inattention, learning problems, and mental health problems (Perlman, 2001; Hack & Taylor, 2000).

The preterm period, between birth and term equivalent age, represents a particular period of neuro-vulnerability. The period typically comprising the late second through third trimesters is a period of rapid brain development. During this period, in utero fetuses experience rapid increases in brain volume (Andescavage et al., 2017), strengthening of the brain's white matter tracts (Jaimes et al., 2020), and development of the earliest functional proto-networks (Turk et al., 2019; van den Heuvel & Thomason, 2016). Perturbations to these developmental processes likely influence later neurodevelopmental sequelae. In terms of impact on the developing brain, preterm birth has been associated with structural and functional alterations that are unique and not related to focal brain injury. For example, prematurity has been connected with specific reductions in cortical and subcortical gray matter, diminished cortical gyrification, and delayed maturation in gray and white matter structures (Ball et al., 2012; Keunen et al., 2012; Rathbone et al., 2011; Shimony et al., 2016; Volpe, 2009; Thompson et al., 2007). Recent work indicates that premature birth influences connectivity patterns across the whole brain (Doria et al., 2010; Smyser et al., 2016; Scheinost et al., 2017) and has a pronounced effect on connectivity that supports thalamo-cortical circuitry, both structurally (Ball et al., 2013, 2014) and functionally (Doria et al., 2010; Smyser et al., 2010; Toulmin et al., 2015). Reductions in inter-hemispheric connectivity (Smyser et al., 2010, 2013) and altered lateralization of language regions (Kwon et al., 2015) have also been associated with premature birth. Recent work shows that prematurity may increase connectivity between superior parietal regions and motor areas, which may explain risk for developmental coordination problems that emerge later in life (Eyre et al., 2021). These disruptions in long-range and intrahemispheric connectivity seem to be

candidate pathways that increase risk for long-term cognitive deficits and IDD risk that emerges in later childhood. White matter injury related to intraventricular hemorrhage is also common in high-risk preterm babies and can manifest as diminished inter and intrahemispheric connectivity on functional levels (Grotheer et al., 2023; Omidvarnia et al., 2015; Smyser et al., 2013).

IDD risk may stem from myriad maturational differences and injuries to the developing brain of prematurely born children. The most prevalent differences and injuries include perinatal brain injury, co-occurring medical diagnoses that alter circulatory or pulmonary function, and changes arising from development occurring in the extrauterine rather than intrauterine environment (Polglase et al., 2014; Sarda et al., 2021; Volpe, 2009; Yates et al., 2021). Beyond those injuries, however, many infants experience comorbid medical conditions in tandem with their premature birth. While all forms of medical comorbidities are of concern in preterm infants for later neurodevelopment in particular are those that can impair either blood flow to the brain or the oxygenation of blood being carried to the brain. These can include brief complications such as a short-term hypoxic event or longer-term medical diagnoses. Among the list of common co-occurring medical conditions in premature infants, those such as patent ductus arteriosus (PDA) or bronchopulmonary dysplasia (BPD) are associated with neurodevelopmental outcomes in childhood (Gudmundsdottir et al., 2021; Hee Chung et al., 2020; Schmidt et al., 2003). Even with infants without significant injury or comorbidities, altered developmental trajectories can be observed. One likely explanation is the stark difference between the intrauterine and extrauterine environments. The intrauterine environment radically differs from the extrauterine, especially for younger-born infants who are admitted to the NICU. During intrauterine development, fetuses are in a heavily shielded environment protected from noxious stimuli such as bright lights, loud noises, and intense tactile experiences. Daily life in a NICU is significantly more sensorially intense with loud noises—ranging from constant machine hum to alarm alerts, which can reach as loud as 85 decibels (Slevin et al., 2000; Williams et al., 2007)—and bright, intense lights (White, 2020), which have been associated with poorer outcomes (Ream & Lehwald, 2018). Efforts to reduce sensory stimulation and stress in the NICU are burgeoning, which may reduce risk for atypical development.

Social Input and Caregiving Adversities

Thus far, this chapter has focused on medical hazards that cause IDD. However, burgeoning evidence over the last decades has connected psychosocial adversity, and toxic stress, with risk for IDD and other neurodevelopmental and mental health problems. The experience of extreme stress prior to conception through the postpartum period is increasingly shown to shape developmental outcomes. Effects can occur independently or interactively with other stressors. We focus on two common

stressors that infants are likely to face in the NICU or immediately following birth which can increase the risk for IDD in later childhood.

Separations and Disruptions in Caregiving Following typical birth, newborns spend much of their time in close contact with their primary caregiver, which serves to support the regulatory capabilities of the infant and promote bonding. Infants cared for in the NICU are often deprived of the same level of close physical contact with primary caregivers, which is increasingly shown to act as an additional stress exposure at this formative phase of life. Duration of separations may last up to weeks or months for the highest risk cases, adding an additional challenge to healthy development on top of myriad other stressors related to medical procedures, overstimulating conditions, pain, and instability. Not surprisingly, stress exposure during NICU, including separations from caregivers, has been linked with altered functional connectivity and decreased brain size in infancy (Smith et al., 2011) and risk for IDD or related neurodevelopmental delays when in toddlerhood (Chau et al., 2013). Painful procedures in the NICU have been linked to altered early brain development. This may be more relevant to the section above on preterm birth—explaining some of the within-group variance in outcomes among babies born preterm (Brummelte et al., 2012). Later in this chapter, we will discuss growing support for interventions that promote close physical contact and minimal separation between mothers and their babies cared for in the NICU.

Systemic Factors, Poverty, and Institutional Racism

Mental illness and poverty can exacerbate the challenges associated with IDD by the limited access to healthcare, education, and support services, in addition to adverse environmental conditions that can affect brain development. Addressing poverty and its associated disparities is critical for improving outcomes for individuals with IDD. Factors that provide support can often buffer against ill effects. By offering affordable services and collaborative treatment with medical professionals, educational systems, and family members, it is possible to provide early diagnosis and intervention, improving long-term outcomes for these individuals. Specialized resources and quality education in policy reformation not only scaffold the development and learning of those affected but also contribute to breaking the cycle of poverty that can exacerbate rates of IDD.

Racism and Discrimination Racism and discrimination escalate liability for IDD risk. Disability status including IDD is known to impact socioeconomic status, affecting SES outcomes such as educational achievement, income, and employment (Gage et al., 2021). Individuals with IDD demonstrate higher unemployment rates than their nondisabled peers and employment opportunities are, on average, lower paying (Quierós et al., 2015). Systemic, institutional, and personal experiences of discrimination, such as those related to one's sexual orientation/identity, ethnicity, and religious and cultural practices, can marginalize women and increase stress dur-

ing pregnancy. For example, women predominantly from Black and Latinx communities report higher at-risk adverse mental health outcomes associated with systematic racism and preterm birth are reportedly higher for these communities (Alhusen et al., 2016; Bower et al., 2018, 2023). From the extant literature, social inequities demonstrate strong relations with an individual's IDD status.

Poverty Approximately 719 million people around the world live in impoverished conditions, with the COVID-19 pandemic recently exacerbating this issue (CDC, 2023). Poverty exerts a significant and detrimental effect, particularly on those with IDD. Epidemiological research has consistently demonstrated an association between poverty and IDD across countries, with the prevalence of IDD higher among those in lower socioeconomic positions (Emerson, 2007). Restricted access to healthcare services due to poverty can result in delayed diagnosis and treatment, hindering early intervention efforts. This limitation coupled with reduced awareness and opportunities for educational enrichment can compound the challenges faced by those with these disorders (Graham, 2005). Furthermore, inadequate nutrition and food insecurity can impair healthy brain function and development (Prado & Dewey, 2014), potentially worsening the impact of these disorders and escalating the incidence of IDD (Rose-Jacobs et al., 2008). Cross-sectional research from the US National Survey of Children's Health found that among preterm children, children living in households that could not afford nutritious meals were more likely to have a learning disability compared to households who could afford nutritious meals (Okoli et al., 2022). The chronic stressors and adverse environmental factors prevalent in impoverished households may increase the severity of IDD (Hertzman & Boyce, 2010).

Consequences of Perinatal Risk Related to IDD

Thus far, we have discussed how environmental risks during the perinatal period can consequently cause IDD, reviewing common perinatal exposures that are known to undermine healthy brain development. Below, we focus on IDD, mapping the phenotypes related to an IDD, with implications in a facet of developmental domains—cognitive function, adaptive function, motor disabilities, learning disabilities, and language delays. We also discuss the association with risk for autism, attention disorders, and other known psychiatric comorbidities.

Defining Intellectual and Developmental Disabilities

Diagnostic criteria for IDD are defined by multiple organizations and bodies, including the International Classification of Disease (ICD-11) from the World Health Organization (WHO), the American Association on Intellectual and

Developmental Disabilities (AAIDD; Schalock et al., 2010), and the APA Diagnostic Statistical Manual (5th ed., text rev.; *DSM-5-TR*; American Psychiatric Association, 2022). Definitions across these organizations define IDD based on three criteria involving (1) cognitive functioning including intellectual abilities such as reasoning, problem-solving, memory, and learning; (2) adaptive functioning, encompassing the practical skills required for everyday life, such as communication, self-care, social interactions, and the ability to live independently; and (3) a manifestation of symptoms of intellectual and adaptive disability occurring in childhood. The severity of intellectual developmental disorder (intellectual disability) is categorized into different levels through the DSM-5-TR, ranging from mild, moderate, severe, and profound. Diagnosis is usually made in childhood, based on standardized assessments and clinical evaluations. The process is often expensive and quite involved, so there are likely many individuals, especially with mild IDD, who go undiagnosed. IDD may occur in isolation or in combination with a number of diagnoses or impairments, including psychomotor disability, language delay, autism, developmental coordination disorder, ADHD, and other forms of developmental psychopathology including depression, anxiety, psychotic disorders, and somatic conditions. Throughout childhood, children with IDD often show various learning difficulties (in math, reading, etc.) and neurocognitive difficulties in memory, attention, and executive function. Below, we review the various functional domains associated with IDD understood to be influenced by perinatal adversities.

Cognitive Disability Intelligence is measured by different conventional psychometric instruments, such as the Kaufman Assessment Battery for Children (Singer et al., 2012), Wechsler intelligence scales (Wechsler, 2014), and Stanford-Binet intelligence scales (Roid & Pomplun, 2012), among others, which result in the intelligence quotient (IQ). IQ, measured typically as a standard score with an average of 100 (standard deviation of 15), allows for comparison of the performance of a child with established age-graded norms. IQ tests measure cognitive skills. Although the definition of intelligence and the use of psychometric intelligence tests are controversial (Eysenck, 1971; Ganuthula & Sinha, 2019), most psychologists agree that intelligence involves a combination of many mental processes to learn from situations, apply knowledge and skills, and think abstractly. While some research has indicated that the age of reaching developmental milestones was associated with IQ in later years (Murray et al., 2007), early language skills seem to be more predictive of later IQ than other domains (Peyre et al., 2017).

Motor Disabilities A wide range of IDD entails delays or difficulties in gross motor functions such as sitting or standing, fine movements such as grabbing an object using an index finger and thumb, and general motor coordination necessary for complex systematized movements such as walking or running (Biotteau et al., 2020; Lucas et al., 2016). Rather than a single diagnosis, motor disabilities constitute a large umbrella of disorders, which in early life include cerebral palsy, ataxia, tremors, and various tic disorders. Many motor disorders, such as cerebral palsy, are thought to arise prior to birth, although diagnosis often does not occur until at least

around 6-months of age (Novak et al., 2017). Typically, diagnosis occurs once early motor milestones (such as rolling from front to back, sitting independently for brief periods, or passing toys from one hand to another) do not develop when expected. Early interventions are often thought to be key in mitigating the long-term impacts of motor disorders, with earlier intervention yielding more efficacious outcomes throughout the lifespan (Hadders-Algra, 2021). For example, premature infants are frequently referred for motor interventions prior to formal diagnosis (Spittle et al., 2015).

Language and Communication Disabilities Language and communication disabilities represent a broad umbrella of symptoms and disorders that are primarily marked by difficulty expressing oneself and can include delays in reaching normative speech milestones and difficulties engaging in age-appropriate expressive language to the absence of speech entirely (APA, 2013). By the age of 3, it is believed that language and communication disabilities affect approximately 6% of children (Boyle et al., 1996). Beyond the direct issues associated with communication difficulties, speech and language disabilities at early ages can often be an early indicator of later concerns. Toddlers with language and communication difficulties are at greater risk for later developmental concerns including cognitive delays and intellectual disabilities (Marrus & Hall 2017), broader social impairments (St Clair et al., 2011), and elevated risk for psychiatric disorders (Snowling et al., 2006) including autism spectrum disorder (Luyster et al., 2007). Using advanced neuroimaging techniques, such as electroencephalogram (EEG), magnetic resonance imaging (MRI), and functional near-infrared spectroscopy (fNIRS), neural signatures of complex interactions of brain systems have been implicated in language development (Kuhl, 2010). These neuroimaging techniques are also used to investigate the neural correlates of nonverbal communication skills in infants and children, including those with difficulties in nonverbal communication (e.g., interpreting social cues, facial expressions; Bayet & Nelson, 2019).

Learning Disabilities Learning disabilities (LDs) with rates that suggest a global impact on about 5% of school-aged children, encompass several disorders that affect the acquisition, retention, comprehension, or application of verbal and nonverbal information, and interfere with specific aspects of school achievement (Lagae, 2008). The DSM-5 categorizes LDs into three major academic domains: reading, writing, and mathematics, with difficulties lasting for at least 6 months (APA, 2013). Specific LDs include dyscalculia, dysgraphia, and dyslexia, the latter accounting for approximately 80% of LDs (Shaywitz et al., 1998). While IQ may correlate with LD, it is important to note that they are separate constructs. LDs can mimic or be comorbid with other neurodevelopmental disorders, making it imperative to distinguish symptoms for proper diagnosis. While early detection can be challenging, typical detection occurs around third grade, after the child is in the academic setting for several years. Family history seems to be the best parameter to select those at risk. Once diagnosed, it is essential for the treatment to encompass a

collaborative effort, involving the family, school, medical professionals, and psychologists (Cortiella & Horowitz, 2014).

Related Neurodevelopmental Conditions Autism, attention disorders, and schizophrenia can co-occur with IDD, implicating various neurological domains such as cognitive flexibility, social impairment, attention, memory, and emotion regulation. Symptoms emerge over time, due to similar pathways (changes in gray and white matter, ventricular enlargement) or distinct pathways and processes that are altered during brain development. Associations with perinatal risk are reviewed in the section that follows.

Autism IDD frequently co-occur with autism spectrum disorder, which is a pervasive developmental condition characterized by impaired communication and social interaction as well as restricted, repetitive patterns of behavior, interests, or activities. The term "autism" is used to refer to the autism spectrum, which affects approximately 1 in 36 children (Maenner et al., 2023). Population-based case-control studies have provided evidence that autism is related to adverse pregnancy and delivery processes (Larsson et al., 2005). While there is an overlap between risk factors for ID and autism, not all autistic individuals are also diagnosed with ID. Perinatal risk factors implicated in the subsequent development of autism and overlap with IDD include the following: *Maternal autoimmune and inflammatory disorders* are also associated with a child receiving an autism diagnosis (Meltzer & van de Water, 2017; Chen et al., 2016). *Premature birth* (Allen et al., 2020; Crump et al., 2021) and/or *low birth weight* (Lampi et al., 2012; Talmi et al., 2020) are also at an increased likelihood for autism; similarly, *low APGAR scores at birth* have been linked to subsequent autism diagnosis (Modabbernia et al., 2019). Large cohort studies have found that *advanced maternal* and *paternal age* are independently associated with a higher likelihood of offspring receiving an autism diagnosis (Sandin et al., 2016). Additionally, recent studies have found a link between *a family history of psychiatric problems* and a subsequent diagnosis of autism in offspring (Xie et al., 2019). Taking all these findings together, it is important to explore these factors as well as those discussed in this chapter as potential risks for a co-diagnosis of autism and IDD.

Attention-Deficit/Hyperactivity Disorder (ADHD) Attention-deficit/hyperactivity disorder (ADHD) is characterized as a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with daily functioning or development (APA, 2013). Youth diagnosed with ADHD often experience symptoms related to deficits in executive function and challenges with behavioral self-regulation (APA, 2013). While not all children with ADHD have a co-diagnosis of IDD, the same perinatal experiences conferring risk for IDD are linked to attention disorders, including prenatal stress, exposure to toxicants and teratogens, and maternal mental health depression disturbance. These developmentally manifest as symptoms of inattention and/or hyperactivity-impulsivity, often in combination with other disorders (Wiggs et al., 2016).

Schizophrenia Childhood-onset schizophrenia is a psychiatric disorder that affects emotions, temperament, and behavior. A child with this disorder has aberrant behavior and emotional manifestations, such as psychotic symptoms (i.e., having strange ideas, thoughts, or feelings that are not based on reality; Keepers et al., 2020). Schizophrenia is not typically diagnosed in pediatric populations younger than age 12. Often, the psychotic symptoms start in adolescence and are more common in males earlier in development (Albert & McCaig, 2015). Schizophrenia is often linked to IDD, and they are not mutually inclusive; nonetheless, a schizophrenia diagnosis does not necessarily imply an IDD. Schizophrenia is also a consequence of genetic and environmental exposure, discussed in this chapter. The causes of schizophrenia are well known and include brain pathology, genetics, environmental factors, and gene-environmental interactions. The DOHAD model provides robust evidence toward a neurodevelopmental model, wherein developmental complications as early as the late first or early second trimester lead to the activation of pathologic neural circuits during adolescence or young adulthood (Fatemi & Folsom, 2009).

Prevention and Intervention

So far, our review has focused on the risk for IDD stemming from multiple adverse exposures and experiences. It is equally important to emphasize how positive environments or interventions can mitigate risk for IDD and support healthy development in the short and long term. Considering the significant neuroplasticity during the neonatal period, early interventions prove to be the most effective in mitigating potential risk factors for IDD (Cioni et al., 2016). Therapeutic input can range from basic application of neuroprotective practices that support brain health during fetal and perinatal phases of development, to higher-level nutritional interventions, to supporting bonding and reducing stressful separations between caregivers and infants around the time of birth. A nonexhaustive list of interventions that will be reviewed below includes kangaroo care, nutritional interventions, family-integrated care, pain reduction techniques, and medical prevention.

Medical, Pain Management, and Nutritional Intervention

Invasive Medical Intervention While any infant may receive early medical interventions, infants born preterm and those who are term born with genetic or congenital disorders are at greater likelihood of receiving both noninvasive and invasive medical procedures. For all infants, major interventions such as surgeries during the perinatal period can confer significant risks for later neurodevelopment, while even noninvasive procedures can confer risk for some infants, such as those born preterm.

For all infants, major invasive procedures are typically only undertaken when it is deemed medically essential given the particular vulnerability of long-term impacts on later development. For example, infants born with congenital heart disease are often at risk for serious health outcomes without surgical interventions. However, it's becoming increasingly apparent that receiving those life-saving interventions often is associated with an elevated risk for later neurodevelopmental impairments (Howell et al., 2019). Importantly, this effect is not limited to cardiac disorders, as surgical interventions for noncardiac disorders see similar increases in neurodevelopmental knock-on effects in childhood (Moran et al., 2019). There are many perioperative factors across cardiac and noncardiac disorders that may account for these observed differences. First, vulnerability to brain injury remains a persistent concern throughout the perinatal period with surgery often associated with subsequent injury related to hypoxia and inflammation (Abella et al., 2015; Pironkova et al., 2017). Moreover, administration of anesthetics during surgery is associated with greater apoptosis of nervous system cells and greater risk for neurodevelopmental delays (Sanders et al., 2013).

NICU-Based Pain Reduction Techniques Infants born at younger ages often experience extended NICU stays and, even among the healthier infants, often experience repeated exposures to painful events such as heel sticks to collect routine blood draws. These can add up quickly; however, very preterm infants can often experience over 100 skin break procedures over the standard course of care before they are discharged, with some reaching as high as 600 (Carbajal et al., 2008; Cong et al., 2017). While these heel sticks, venipuncture, and similar skin break procedures are routine in adults, they may pose a significantly larger risk in younger-born preterm infants. Specifically, pain sensation pathways are considered mature by 20 weeks gestation (Anand & Hickey, 1987; Fitzgerald, 1993), while the pain modulatory response is not fully developed until after birth and nociceptive blocking signaling may not be available until nearing term age (Fitzgerald, 1991). As a result, earlier-born infants may be at unique risk of insufficiently moderate pain response. Importantly, the relationship between pain exposure and neurodevelopment has been consistently observed in preterm infants, with greater exposures associated with broad neurodevelopmental impairments during infancy and early childhood (Valeri et al., 2015; Vinall et al., 2014).

Pain reduction techniques in the NICU include strategies to minimize the discomfort experienced by premature infants during medical procedures, such as intubation or needle pricks. While these techniques do not directly target IDD, reducing pain and stress during the NICU stay may have an indirect effect on long-term neurodevelopment. Studies have shown that interventions such as sucrose administration or nonpharmacological measures, such as facilitated tucking and gentle human touch, can reduce pain and stress in preterm infants (Stevens et al., 2016). Extending this finding, one RCT has also shown that skin-to-skin care can reduce biobehavioral pain responses in preterm infants (Cong et al., 2011), further supporting the idea that skin-to-skin contact may be one of the most effective, nonpharmacological pain reduction techniques. In sum, the evidence for a direct link between

pain reduction techniques in the NICU and later IDD outcomes is not robust yet, but given the promising initial findings, future research is highly encouraged.

NICU-Based Nutritional Interventions NICUs play a pivotal role in providing the necessary care for at-risk infants during the perinatal period and are ideal locations for implementing interventions that can be continued at home (Aucott et al., 2002; Soleimani et al., 2020). Nutritional interventions in the NICU and following discharge from the hospital aim to optimize the overall health of preterm infants by ensuring adequate nutrition through parenteral and enteral feeding. Although breastfeeding may lead to slower weight gain in preterm infants than when receiving formula, breastfeeding has been associated with reduced risk for poor neurodevelopmental outcomes and better odds for a larger head circumference at 6 months and 2 and 5 years of age (Rozé et al., 2012). Correlational research also suggests that breastfeeding may reduce the risk of certain developmental disorders, such as autism spectrum disorders (Xiang et al., 2023) and attention-deficit/hyperactivity disorder (Oddy et al., 2010). While these interventions are essential for neonatal survival and growth, more evidence is needed to measure the impact on reducing developmental and intellectual disability risk.

Medical Treatments as Prevention Medical prevention and interventions are commonly provided in NICUs for respiratory support and infection management to promote infant survival and long-term health (Ho et al., 2023). Effective respiratory support can reduce the risk of brain injury and improve neurodevelopmental outcomes in at-risk infants (Polin & Sahni, 2017). For instance, surfactant therapy is commonly used to treat respiratory distress syndrome in preterm infants, as it improves lung function and oxygenation while simultaneously reducing the risk of hypoxia-related brain injury and improving neurodevelopmental outcomes (Polin & Sahni, 2017). Other medical interventions focus on neuroprotection, by minimizing brain injury and improving long-term neurodevelopmental outcomes with medications, including caffeine citrate for apnea of prematurity (Schmidt & Davis, 2016). Such interventions have the potential to indirectly reduce risk for IDD, but their causal effects are yet to be fully understood.

Maternal-Fetal Dyadic Interventions

Family-based prevention and intervention programs prior to birth and in the NICU not only contribute to the overall well-being of the infant but also empower parents to become active participants in their children's care and development. These interventions can help reduce the risk of developmental disorders by creating a supportive, nurturing, and cognitively stimulating environment for infants. We discuss two examples below.

Kangaroo Care Kangaroo care (KC), also known as skin-to-skin care, involves placing the infant on their parents' chest in a diaper and a hat, maximizing skin-to-

skin contact. Randomized controlled trials (RCT) suggest that KC has causal benefits for infant neurocognitive health. For example, El-Farrash et al. (2020) found that KC causally improves infant attention and regulation, lowers scores on lethargy, and reduces nonoptimal reflexes. Moreover, a 20-year follow-up of an RCT conducted by Charpak et al. (2017) showed that KC significantly reduced the risk of neurodevelopmental disabilities among young adults who were born preterm. The study found sustained positive effects of KC on cognitive and motor outcomes compared to the traditional incubator care group. Correlational work also suggests that KC is associated with better emotional bonding between the parent and the infant, which may have an indirect positive impact on infants' cognitive development (Feldman et al., 2002), reduces infant stress hormone levels, lowers maternal postpartum depression (Cristóbal Cañadas et al., 2022), improves cognitive control, and enhances autonomic nervous system functioning (Feldman et al., 2014). Finally, a meta-analysis by Conde-Agudelo and colleagues (2011) found a positive impact of KC on developmental outcomes, and a recent systematic review by Boundy et al. (2016) suggested reduced incidence of sepsis, improved weight gain, and enhanced neurodevelopmental outcomes in preterm infants who received KC. The strength of evidence for KC's impact on reducing IDD risk is substantial, making it a cornerstone intervention in the NICU.

Attachment-Based Interventions One such early parenting program is the modified Attachment and Biobehavioral Catch-up (mABC), which was developed for low-income mothers who are dependent on opioids (Labella et al., 2021). Intervention sessions begin during the third trimester when parent coaches support mothers in preparing for anticipated challenges, such as caring for their infants who may be challenging to soothe while remaining sensitive and nurturing to the infant. mABC parenting program sessions continue throughout the first 6–12 months of the infant's life. Recent evidence from an RCT suggests that ABC causally enhances opioid-exposed infants' autonomic nervous system functioning (Tabachnick et al., 2022) and high-risk children's long-term executive functioning (Korom et al., 2021; Lind et al., 2017).

In addition to ABC, a recent systematic review by McAndrew and colleagues (2022) reviewed 19 NICU-based studies, all involving family-focused interventions that actively engaged families in some aspect of patient care in intensive care units. Although no physiological differences were observed between control and intervention groups, those receiving family-based interventions showed higher weight, shorter NICU stay, and increased alertness and total wakefulness time compared to the control group. Parents in the intervention group also endorsed reduced stress and lower respiratory rates when interacting with their vulnerable infants than parents in the control group. Although family-based interventions are a promising avenue for mitigating the risk for IDD, additional investigation into the effectiveness of interventions that involve family participation is needed.

Postnatal Follow-Up Care

Interventions that are currently being implemented through early intervention in the United States encompass many developmental areas, such as language, motor, and cognition. While the origins and principles guiding these interventions are developmentally sound, it is important to remember the developmental considerations for each individual with IDD and other co-occurring conditions. The most common interventions and services provided are occupational (adaptive, sensory, fine motor), physical (gross motor, limbic), and speech-language therapy, as well as special instruction and applied behavioral analysis, which uses behavioral psychology to teach skills acquisition through natural reinforcement. The field has shifted to implement Naturalistic Developmental Behavioral Interventions (NDBI), which are implemented in the individual's known environment (often the home), "involve shared control between child and therapist, utilize natural contingencies, and use a variety of behavioral strategies to teach developmentally appropriate and prerequisite skills" (Schreibman et al., 2015). Other important strategies during early childhood include parenting interventions which have the potential to alleviate stress in the household, empower parents, and teach parents how to play with infants in cognitively stimulating ways. These strategies may alter the course of symptoms and improve the well-being of the family members caring for a child with an IDD (Vanegas et al., 2022).

Integrative Summary, Implications for Policy, and Future Directions

This section will provide an integrative summary of our review, with the goal of highlighting key directions for research, prevention, practice, and policy. First, as reviewed in part one of our chapter, IDD may result from a broad number of environmental, experiential, and nongenetic risk factors during the perinatal period of human brain development. Adverse exposures can occur antenatally, influencing fetal brain development, and separately or in combination with risk that are encountered perinatally, related to severe premature birth, anoxia or hypoxia, and be correlated with low APGAR scores and low birth weight or poor growth. Effects of perinatal exposures can be compounded by social and caregiving stressors experienced while infants are cared for in the NICU and throughout later infancy. For this reason, perinatal risk factors may be better understood as increasing initial risk or perturbation in fetal and infant brain development that may set the infant on a trajectory of risk that is exacerbated by additional challenges encountered following birth. Timing of exposures and severity of both risk and protective factors are therefore essential for understanding the likelihood of risk for IDD.

Part two of this chapter focused on defining the phenotype associated with IDD risk. IDD involves a constellation of delays across various cognitive, memory, and

learning domains and is often accompanied by additional syndromes and disorders, including autism, and ADHD. Much of the existing work has attempted to connect various perinatal exposures with late emerging latent or categorically defined IDD risk. As heterogeneity and comorbidity are the norm rather than the exception, progress in delineating etiological pathways may stem from more carefully considering developmental trajectories of specific neurocognitive processes that are measurable in newborns and infants (such as including development of processing speed, visual attention, working memory, attention, inhibition, cognitive flexibility, and social or emotional processing, attention to faces) and, over time, contribute to the development of more complex syndromes, such as IDD and related disorders later in life. In this sense, our field may need to place more focus on the developmental aspect of IDD in order to fully understand how to support individual pathways toward resiliency. As covered in our review, work must also consider the role of caregiving experiences, cultural factors, and additional environmental or systemic hazards will be essential for mitigating ongoing risk.

Considering the timing of exposures is also key for future work, given emerging evidence that the effects of some exposures are timing dependent. Advances in fetal and infant neuroimaging may offer new insight into how the impact of any given exposures may depend on the timing or extent to which it disrupts the dominant processes at that stage of central nervous system development (ranging from disruptions in neurogenesis to neural migration, to neuronal differentiation, to axonogenesis, to synaptogenesis, followed by normative apoptosis and pruning of excessive synapses through the first years of life; Linderkamp et al. (2009)). For example, certain exposures (brain insults) may have formative and severe influences if they occur early and disrupt the formation of the CNS, potentially leading to profound IDD risk. However, interruptions in synaptic remodeling at later stages of fetal and infant brain development may be more likely to impair neuronal networks that support ongoing memory, attention, and learning. With supportive caregiving over the subsequent periods of postnatal development, impacts of the perinatal exposures on risk for IDD may be more attenuated or mild.

It has long been appreciated that perinatal risk factors are not randomly distributed, but instead are stratified across the population, with the greatest burden of risk for more marginalized or impoverished families. Large-scale neuroimaging studies have drawn connections between perinatal risk and longer-term brain and cognitive delays and have revealed that effects may be exacerbated by lower socioeconomic status (Alnaes et al., 2020), pointing to the need for more progress in improved screening, detection, and prevention for marginalized populations. For example, infants with multiple significant pre- and perinatal risk factors can be offered additional follow-up or services even if they do not display overt clinical symptoms. Education to parents on early warning signs of IDD or other neurodevelopmental disorders can be offered prior to hospital discharge and at ongoing infant wellness checks. Preventative home visits that support cognitive stimulation and caregiver responsiveness can be offered to support families and reduce risk for compounding adversities that enhance IDD risk in toddlerhood and preschool years. As we

reviewed in part 3, there are a multitude of existing intervention and prevention programs that aim to reduce risk for IDD. Training hospital professionals to integrate ongoing educational and prevention strategies onto existing medical or preventative treatments may maximally support feasibility and implementation.

It is important to note that social-environmental factors are complex and multidimensional and include education, occupation, household income, and material resources but also factors such as parental psychosocial stress, parenting style, and a cognitively stimulating home environment which may all impact child development. To develop effective interventions and public policies that more equitably reduce risk for IDD, further studies are needed to understand the specific mechanisms through which social-environmental factors impact neurodevelopment. The effects of specific social adversities and early life stress on brain development and neurocognition can be studied in animal models; these are difficult to control in human studies given the complex intersections between these factors (Hackman et al., 2010). For example, postnatally, chronic stress during pregnancy in rodents may result in altered mother-infant interactions including a decrease in the frequency of licking and grooming behaviors of rat pups. These rat pups who are exposed to fewer licking and grooming behaviors have decreased NMDA receptor levels and expression of growth factors in the hippocampus which results in decreased synaptic formation and cognitive performance (Liu et al., 2000). This may provide some insight into the neural basis of parenting interventions, which have been shown to be effective in modifying neurodevelopmental outcomes in children including those with brain injury and IDD. In rodent models, environmental enrichment (Rosenzweig et al., 1978) results in increased neurogenesis, gliogenesis, and synapse formation in the hippocampus and cortex as well as improved memory and learning (Kempermann et al., 1997; Rampon et al., 2000; Van Praag et al., 2000). These findings complement those of human studies observing the importance of a cognitively stimulating environment on neurodevelopmental outcomes, providing a mechanism through which interventional programs reduce IDD risk and enhance long-term educational success and employability. Human clinical trials of social and parenting interventions involving randomized trials are the gold standard approach for establishing causation in humans. Results from such trials should be leveraged to guide policy changes, with the strongest effects for reducing IDD and comorbidities in at-risk families. Interestingly, a recent randomized control trial is testing the impact of improved financial support to impoverished families (based on the provision of monthly lower or higher levels of unconditional cash transfers) can improve early life brain function and psychosocial outcomes (Noble et al., 2021). They found that infants in the high-cash group showed more power in high-frequency bands on EEG, which has been previously associated with higher language and cognitive scores in other studies (Benasich et al., 2008; Williams et al., 2012). Longitudinal follow-up of this cohort is ongoing and will provide further insight into whether these alterations in the brain persist over time (Noble et al., 2021; Troller-Renfree et al., 2022).

Closing Summary

IDD can arise from genetic or nongenetic causes and encompass a range of neurodevelopmental disorders. The relatively high prevalence of IDD motivates the search for potential mechanisms and exposures during the perinatal period that can cause an IDD. Perinatal exposures known to increase risk for IDD in later childhood include medical exposures (perinatal brain injury, infection, and prematurity), environmental exposures (chemical exposure, environmental hazards known to undermine brain development, such as lead exposure, cigarette smoke, polluted air, and pesticides), and social adversities (maternal-infant separation in the NICU, and maternal mental health affecting bonding and responsiveness). As these adversities often co-occur, disentangling their individual versus interactive developmental effects is difficult. Postnatal neural plasticity and environmental supports have the potential to buffer relations between environmental insults and offspring brain development, potentially reducing later risk for IDD.

The extant literature reviewed here provides an overall understanding of the causes of IDD. Yet, more research is needed to understand its mechanisms and improve treatment or intervention options, including for co-occurring conditions. Progress on this front will inform expanded continuity of care and help close the gap on discontinuity, by implementing interdisciplinary approaches across all medical and clinical disciplines to support both the parent and child. Early intervention and services are critical to deliver positive outcomes in later child development. Yet, we need to consider the development trajectory of the child and the potential need for lifelong services, which currently arguably lack in resources given the structural barriers. Thus, future research should not only investigate the neural pathways and systems implicated in IDD but also critically examine ways to maximize the systematic implementation of relevant intervention programs in a variety of settings, thereby informing policy and improving access.

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