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Shaping minds: Family environmental influences on early neurodevelopment

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ABSTRACT

Neurodevelopment arises from the complex interplay of genetic, epigenetic, neurobiological, environmental, and experiential factors that shape the maturation of brain and adaptive functions. Early experiences, particularly within sensitive developmental periods, can either support or disrupt these processes, influencing vulnerability to neurodevelopmental disorders. Among early environmental determinants, family represents the most proximal and dynamic context for the developing child, providing the primary sources of stimulation, emotional regulation, and caregiving. The family environment encompasses both structural (e.g., socioeconomic conditions) and relational (e.g., parent–child interactions, parental mental health) dimensions that jointly influence neurodevelopmental trajectories. This narrative review examines how key domains of the family environment serve as protective factors or sources of vulnerability through their mutual interplay in shaping child neurodevelopment, with the aim of synthesizing current evidence and highlighting directions for early preventive and therapeutic interventions.

Abbreviations

ADHD	Attention-Deficit/Hyperactivity Disorder
AIRE	Assessment of Infant RElationships
ASD	Autism Spectrum Disorder
CNS	Central Nervous System
DOHaD	Developmental Origins of Health and Disease
EE	Enriched Environment
GxE	Gene-Environment Interaction
HPA Axis	Hypothalamic-Pituitary-Adrenal Axis
SES	Socioeconomic Status

1. Introduction

The term *neurodevelopment* refers to the process through which the central nervous system (CNS) and adaptive functions (i.e., sensory, motor, cognitive, communicative, emotional–relational, and social) mature from conception, throughout the first 1000 days of life, and into childhood and young adulthood. This process unfolds through a continuous interplay among genetic, epigenetic, neurobiological, environmental, and experiential factors, each of which may act as either a risk or a protective factor.[1]

It is well established that early environmental factors exert a profound influence on brain development,[2] particularly when they occur within specific developmental windows, referred to as sensitive or critical periods.[3] Common risk factors, together with genetic, neurobiological, and environmental components, both age-specific and transdiagnostic, can interfere early with the development of the central nervous system, disrupting the reorganization of neural networks that support learning, emotion, relationships, and behavior.[4,5] Such alterations influence sensitivity to and responses toward environmental stimuli, which become progressively more complex with development, potentially leading to the onset of neurodevelopmental disorders, which represent the phenotypic expressions of these complex processes. The effects of these alterations may emerge from the earliest years of life, as in autism spectrum disorders, language and learning disorders, attention-deficit/hyperactivity disorder, or neurological conditions such as cerebral palsy and epilepsy,[1] or later in adolescence, with psychiatric disorders such as behavioral, mood, or eating disorders, or schizophrenia, all of which are now considered neurodevelopmental disorders.[6]

The infant's family environment represents one of the most

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influential and proximal contexts shaping early neurodevelopment. From the prenatal period onwards, the home and family setting provide the primary sources of stimulation, caregiving, and emotional regulation upon which neural and behavioral systems are scaffolded. Within this microenvironment, children are exposed to a complex combination of factors, including parental sensitivity and mental health, parenting practices, family cohesion, socioeconomic conditions, and household stress, that dynamically interact with biological predispositions to influence developmental trajectories.[2,7] The family environment is thus not a single construct but a multidimensional system encompassing both structural aspects (e.g., family composition, socioeconomic status, housing conditions) and relational processes (e.g., attachment relationships, emotional and communication patterns). These dimensions contribute to shaping the quality and timing of experiences that guide synaptic plasticity and the maturation of circuits involved in cognition, affect regulation, and social behavior.[6,8]

In this narrative review we examine the influence of the family environment on child neurodevelopment, with a particular focus on factors that may contribute to vulnerability to neurodevelopmental and psychiatric outcomes. Although broader environmental determinants such as air pollution, toxic exposures, and infections are also important contributors to neurodevelopment, they are not addressed in this review because the present article focuses specifically on the family environment as the most proximal and modifiable context shaping early developmental trajectories. Recognizing the breadth and complexity of the literature, this review focuses on four interconnected domains of the family environment that have been consistently associated with children's developmental trajectories[9]: parent–child interactions, parental mental health, family socioeconomic conditions and home environment and psychological functioning. These domains do not operate independently but dynamically interact across development. Relational processes within the caregiver–child dyad are shaped by parental psychological well-being, material and social resources, and the broader developmental opportunities available in the home context. Examining these domains together may therefore provide a more integrated understanding of how family environmental experiences contribute to early neurodevelopmental outcomes. The specific objectives of this narrative review are: (1) to synthesize evidence on how parent-infant interactions, parental mental health, socioeconomic conditions, and the home environment influence early neurodevelopment; (2) to describe the underlying biological and relational mechanisms; and (3) to identify translational implications for early preventive and therapeutic interventions. To better contextualize how these domains contribute to developmental trajectories, we first outline a conceptual framework of vulnerability and resilience that informs the interpretation of the evidence reviewed in the following sections.

2. Methods

This brief narrative review followed the recognized principles of narrative reviews,[10,11] which seek to provide a qualitative, critical, and integrative synthesis of the literature on a specific topic while offering a coherent overview rather than adhering to rigid, pre-defined protocols. Accordingly, an interpretive rather than systematic approach was adopted to examine family-related environmental domains that have been consistently associated with developmental trajectories during the prenatal period, infancy, and early childhood. In line with the conceptual framework of the manuscript, the family environment was considered in both its structural dimensions (e.g., socioeconomic conditions and home characteristics) and relational dimensions (e.g., parent–infant interactions and parental psychological well-being). A targeted literature search was conducted between August and October 2025 using the electronic databases PubMed/MEDLINE, Scopus, and Google Scholar. These sources were selected to capture relevant literature from developmental neuroscience, child psychiatry, developmental psychology, pediatrics, and public health. In addition to database searching, backward citation tracking of relevant reviews and seminal articles was performed to identify additional studies of conceptual relevance.

The search strategy combined terms related to neurodevelopment with terms related to family environment and early-life contextual exposures. Keywords included neurodevelopment, early brain development, family environment, parent–infant interaction, parental sensitivity, dyadic synchrony, parental mental health, maternal depression, prenatal stress, socioeconomic status, home environment, environmental enrichment, early childhood development, and developmental outcomes. Search terms were combined using Boolean operators (AND/OR), with examples such as "neurodevelopment AND family environment," "parent–infant interaction AND cognitive development," "prenatal stress AND child neurodevelopment," and "socioeconomic status AND brain development."

The review primarily considered articles published in English, with particular attention to studies published from 2000 onward in order to reflect contemporary advances in developmental neuroscience and early developmental research. Earlier seminal contributions were also included when considered essential for conceptual framing. Priority was given to systematic reviews, meta-analyses, longitudinal cohort studies, prospective observational studies, and influential conceptual papers addressing mechanisms linking family-related environmental exposures to neurodevelopmental outcomes.

Studies were selected on the basis of their relevance to the central review question: how family environmental factors may act as protective factors or sources of vulnerability in shaping neurodevelopmental trajectories. The evidence was then organized narratively into four thematic domains that were consistently represented across the literature: (1) parent–infant interactions, including synchrony, contingency, sensitivity, and developmentally appropriate stimulation; (2) parental mental health, including prenatal and postnatal psychological well-being; (3) socioeconomic conditions, including parental education, household income, and related contextual adversity; and (4) home environment, including developmental stimulation and enriched environmental experiences.

As this was a brief narrative review, no formal systematic review procedures were applied. Consistent with the narrative nature of the review, no protocol registration, formal risk-of-bias assessment, or quantitative meta-analytic synthesis was performed. Rather than aiming for exhaustive coverage, the purpose was to integrate representative and conceptually informative evidence across selected domains in order to highlight converging developmental mechanisms and inform early preventive and therapeutic interventions.

3. Results

A robust understanding of neurodevelopment requires a clear definition of the constructs that shape individual susceptibility to adversity. Within this review, vulnerability is conceptualized as a multidimensional state comprising four distinct aspects: (a) an increased risk of the onset of neurodevelopmental or psychiatric disorders; (b) a predisposition toward greater symptom severity following onset; (c) the potential for a poorer response to standard therapeutic interventions; and (d) a fundamental reduction in adaptive resilience.[12,13] Although these indicators do not necessarily constitute the disorder itself, they represent conditions that may contribute to its phenotypic expression when combined with environmental triggers. In contrast, resilience refers to the capacity for positive adaptation despite significant adversity[14] and is often strengthened by high-quality relational support and stable environmental resources.[13,15]

The "three-hit" concept provides a unifying framework for understanding this susceptibility.[16] Hit 1 involves genetic predisposition, which provides the initial biological sensitivity. Hit 2 refers to the early-life environment, particularly during critical developmental periods when neural circuits are most plastic. Hit 3 encompasses later-life environmental exposures. Vulnerability is substantially increased when

the capacity to cope with adversity is compromised across these successive hits, leading to a cumulative maladaptation.[16]

A key concept underlying vulnerability and resilience is that of sensitive periods, namely developmental windows during which neural circuits are particularly plastic and responsive to environmental input.[17] Experience during these windows can produce more efficient and enduring neural reorganization than at other times, although plasticity does continue across the lifespan.[17] Sensitivity is generally highest during the prenatal period and declines progressively throughout childhood.[17]

The first six months of life represent a critical window for sensory system maturation and the development of early attachment.[18] Evidence from studies on institutional deprivation suggests that deprivation confined to the first 4–6 months of life may not lead to long-term cognitive deficits if the child is subsequently placed in a high-quality caregiving environment.[19] In contrast, deprivation extending beyond this period is associated with an increased risk of persistent neurodevelopmental impairments.[19,20]

Similarly, the first three years of life are essential for the establishment of functional brain organization.[21] Harsh parenting during this stage has been associated with altered fronto-amygdala connectivity and reduced global network efficiency, potentially compromising later emotional regulation.[22,23] Conversely, middle childhood (5–9 years) represents another sensitive developmental window during which warm and supportive parenting may enhance emotional regulation and reduce the risk of later anxiety and depression.[23]

Taken together, these models highlight how vulnerability and resilience emerge through the dynamic interaction between biological susceptibility and family environmental experiences across development. The following sections therefore examine four key domains of the family environment that shape neurodevelopmental trajectories.

3.1. Parent-infant interactions

Caregiver–child interactions represent one of the most direct and immediate influences on children, providing a crucial context for the development of cognitive, social, and emotional skills.[24] Depending on the nature of the caregiving relationship, parents can function as promotive, protective, or risk factors in child development.[25] Parental responsiveness has been defined as being accepting, nurturing, supportive, sensitive, and warm,[9] thereby fostering a secure and supportive environment for the child's development. In this context, research has identified several relational qualities that shape children's early socioemotional and cognitive development, including dyadic synchrony, contingency, developmentally appropriate stimulation, and parental sensitivity.

3.1.1. Dyadic synchrony and contingency

Dyadic synchrony, defined as the coordinated and reciprocal timing of behaviors between caregiver and infant, allows infants to experience contingent responses to their emotional and behavioral cues, supporting self-regulation, attachment, social engagement, and cognitive skills.[26,27] Synchrony emerges through the mutual exchange of verbal and nonverbal behaviors between infants and their caregivers. These interactions begin at birth and continue to shape the child's growth over time.[28] For example, a caregiver may gently rock the infant during feeding, and the infant may respond by calming and maintaining eye contact. The caregiver then acknowledges this response with a warm smile, reinforcing the connection. This process encompasses physiological, behavioral, and temporal components that together help explain how dyadic synchrony develops and evolves. Physiologically, mechanisms such as hormonal activity (e.g., oxytocin release), autonomic regulation (e.g., vagal tone), and activation of the brain's reward system serve as key foundations for observable interaction patterns.[29] Behaviorally, synchrony is expressed through caregiving actions such as eye contact, vocalizations, and physical touch, which vary in timing, duration, intensity, and frequency.[30] Contingency, defined as the timely and appropriate response of a caregiver to an infant's signals, is central to synchrony.[26,28,30] It reinforces predictability and trust and lays the groundwork for emotional and behavioral regulation.[27] Both synchrony and contingency appear to be linked to neural processes, with synchronized and contingent interactions associated with coordinated activity in brain regions such as the prefrontal cortex (supporting executive function and emotion regulation), the temporoparietal junction (involved in social cognition), and the anterior cingulate cortex (associated with attention and affective processing) .[31,32] Importantly, infants are active participants in these exchanges, contributing to the quality of synchrony through individual differences in temperament, attention, and physiological regulation.[33,34] Although the capacity for synchrony appears to be biologically grounded, its expression is shaped by cultural norms and caregiving practices. Cross-cultural research has highlighted variations in the use of gaze, touch, and vocalization.[35] High levels of synchrony and consistent contingent responses are associated with better stress regulation, more adaptive emotional expression, and stronger socioemotional competence, whereas inconsistent or poorly attuned interactions have been linked to heightened physiological stress and socioemotional difficulties.[28,34] These early relational experiences are especially influential during sensitive periods of brain development and often predict mental health, social functioning, and cognitive flexibility in later childhood and adolescence.[34] Accordingly, the study of dyadic synchrony has informed targeted interventions, particularly in high-risk populations such as preterm infants or dyads affected by maternal depression, emphasizing the role of caregiver responsiveness in promoting healthy developmental trajectories. Early interventions that prioritize parental empowerment and strengthen parent–infant relationships may yield greater benefits for later cognitive and neuromotor development than approaches focused solely on the infant's functional impairments.[36,37]

3.1.2. Developmentally appropriate stimulation

The quality of early interactions also depends on developmentally appropriate stimulation, whereby parental input is tailored to the infant's developmental stage and current state in order to maintain an optimal level of arousal for engagement.[38] Parents interpret the infant's cues and adjust their responses accordingly, while infants sustain the interaction through engagement and responsiveness. For instance, if the infant shows signs of fatigue, the parent may reduce the intensity of play or shift to a calmer activity, allowing the infant to regulate. Conversely, when parental behaviors are not attuned to the infant's needs, interactions may become stressful, with potential long-term effects on development.[38]

3.1.3. Caregiver sensitivity and attachment-related processes

Finally, caregiver sensitivity, that is the ability to accurately perceive and interpret infants' signals and to respond promptly, appropriately, and with warmth, engagement, and support,[39] plays a central role in these processes. Particularly in contexts that elicit stress or require emotion regulation, maternal sensitivity has been linked to children's socioemotional development and physiological regulation[38] and is therefore considered a strong predictor of infant attachment.[40]

Supporting this view, interventions that enhance sensitive caregiving, such as affective touch and skin-to-skin contact, improve physiological regulation, bonding, and neurodevelopment, particularly in preterm populations.[41] Recent evidence further highlights its importance for physical health, with higher maternal sensitivity across the first year of life being associated with fewer health complaints through adolescence.[42]

Although early attachment research has historically focused primarily on mothers, growing evidence highlights the independent and complementary contribution of fathers to infant neurodevelopment.[43] Recent evidence moves beyond maternal-centric models, showing that fathers' sensitivity, involvement, and characteristic forms of play have

specific links to children's social, emotional, and cognitive outcomes.[44] For instance, paternal sensitivity, defined as the ability to respond accurately and warmly to an infant's needs, is associated with positive cognitive and socio-emotional development.[45,46] Furthermore, evidence indicates that greater paternal involvement in childcare is linked to better outcomes for both mothers and infants, and can improve the quality of the perinatal environment.[43,45] Distinct from maternal styles, paternal interactions are frequently characterized by "play sensitivity" and high-intensity arousal, such as rough-and-tumble play, which effectively scaffold exploration, emotional resilience, and adaptive risk-taking in novel environments.[44] Neurobiologically, fathers undergo hormonal and functional adaptations during the transition to caregiving, including increased oxytocin and synchronized decreases in testosterone levels, which directly support nurturant behavior and parent-infant neural synchrony.[43]

3.1.4. Implications for early intervention

Taken together, these findings underscore that high-quality caregiver–child interactions, including synchrony, contingency, developmentally appropriate stimulation, and sensitivity, provide a developmental context that supports emotional, cognitive, social, and physical health outcomes. By fostering responsive and attuned caregiving from the earliest months of life, parents provide infants with the regulatory, relational, and cognitive scaffolding necessary for adaptive development across the lifespan.

3.2. Parental mental health

Parental mental health represents a critical determinant of offspring neurodevelopment, with both maternal and paternal well-being shaping children's cognitive, emotional, and social trajectories across the prenatal, perinatal, and postnatal periods.

3.2.1. Prenatal influences and fetal programming

Early environmental influences, beginning in utero, exert lasting effects on health and development across the lifespan.[47–49] The "fetal programming hypothesis",[48] the "developmental programming hypothesis" ,[50,51] and the "Developmental Origins of Health and Disease" (DOHaD) hypothesis[52] propose that disturbances during sensitive developmental windows, such as maternal stress, malnutrition, or glucocorticoid exposure, can recalibrate biological systems, with long-term consequences for brain development and mental health.

As noted above, maternal stress is among the most powerful prenatal influences, increasing offspring vulnerability to later somatic and psychiatric disorders. A recent comprehensive review by Van den Bergh et al. (2020) reported consistent associations between prenatal maternal stressors, including daily distress, major life events, and disaster exposure, and adverse offspring outcomes such as altered neonatal state regulation, poorer motor and cognitive development, and more difficult temperament.[53] These effects may persist into adulthood, where they are associated with elevated risks of anxiety, depression, attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), and schizophrenia.[53]

At the biological level, mechanistic accounts suggest that prenatal stress alters fetal programming through dysregulation of the hypothalamic–pituitary–adrenal axis, excessive glucocorticoid exposure, immune activation, microbiome alterations, and changes in telomere biology.[53,54] Supporting this view, neuroimaging studies have demonstrated structural and functional alterations in brain regions central to emotion regulation and cognitive control, including the amygdala, hippocampus, and prefrontal cortex, in infants exposed to poor maternal mental health during pregnancy.[53,55] In addition to fetal programming, genetic transmission must also be considered, as associations between prenatal maternal stress and offspring neurodevelopment may partly, or even entirely, reflect shared genetic liability.[56]

3.2.2. Postnatal parental psychopathology and the caregiving environment

Beyond prenatal maternal stress, a robust body of evidence demonstrates that maternal psychopathology, particularly depression, also adversely affects offspring neurodevelopment, increasing the risk of poorer cognitive outcomes, behavioral problems, and mental health disorders across the lifespan.[9, 57,58] Although both prenatal and postnatal exposure to maternal stress and psychopathology affect offspring neurodevelopment, the underlying mechanisms appear to differ. Prenatal maternal stress is thought to exert its effects primarily through biological pathways, including alterations in placental function, hypothalamic–pituitary–adrenal axis regulation, and inflammatory processes.[9,59,60] By contrast, the mechanisms through which postnatal maternal stress and psychopathology shape child neurodevelopment are largely behavioral.

In this regard, postnatal parental mental health difficulties may reduce parental sensitivity and disrupt synchrony in parent–infant interactions, thereby compromising the quality of the caregiving environment. These disruptions may, in turn, have downstream consequences for infants' socioemotional regulation, attachment security, and neurodevelopment.[9, 61]

Finally, a recent meta-analysis suggests that paternal prenatal mood and anxiety disorders are associated with an elevated risk of offspring neurodevelopmental disorders, such as ADHD and ASD.[62]

3.2.3. Developmental implications and prevention

Taken together, these findings support a multi-layered framework in which parental mental health influences child neurodevelopment through both biological pathways and modifications of the caregiving environment. This body of evidence highlights the critical need for the early identification of, and intervention for, parental psychological difficulties, not only to enhance parental well-being, but also as a public health strategy aimed at promoting healthy neurodevelopment in the next generation.

3.3. Socioeconomic conditions

Parental socioeconomic status (SES) is a multidimensional construct encompassing income, education, occupation, and associated neighborhood characteristics.[63] It not only determines a family's position within society but also represents an important determinant of health across the lifespan, particularly during childhood.[64] SES influences neurodevelopment from its earliest stages, including the prenatal period.[65] While SES-related risks in high-income countries are often tied to relative poverty and reduced access to cognitively enriching environments, in low- and middle-income settings they frequently manifest as absolute deprivation, food insecurity, and limited healthcare infrastructure. These overlapping structural adversities can profoundly compound the biological embedding of stress and environmental risk during early neurodevelopment

3.3.1. Developmental pathways linking SES and neurodevelopment

Both family- and neighborhood-level SES are essential to shape key determinants of child development, including housing quality, nutrition, healthcare access, and educational opportunities, which together contribute to neurodevelopmental outcomes.[66] Household income and parental education are the most commonly used indicators of SES: the former is typically used to define poverty thresholds, while the latter reflects years of schooling or the highest degree attained.[66]

Material deprivation associated with low household income reflects limited access to cognitively enriching resources, such as books, educational toys, and high-quality early learning experiences. SES influences neurodevelopment through multiple pathways not only related to income, education, and occupation. These include prenatal health, quality of caregiving, cognitive stimulation, nutrition, exposure to stress and environmental toxins, and substance use.[67] All these determinants impact the neurodevelopmental trajectories at a molecular, functional,

Table 1
Overview of standardized assessment tools and evaluated developmental domains.

Scale / tool	Evaluated domains	Target age & administration	References
Griffiths Developmental Scales - III Ed.	*Learning foundations, language/communication, eye-hand coordination, social-emotional development*	*Birth to 5 years and 11 months Standardized assessment*	*Green E, Stroud L, Bloomfield S, Cronje J, Foxcroft C, Hurter K (2017) Griffiths III. Griffiths scales of child development, Third Edn. Edizione Italiana a cura S. Lanfranchi, M. Rea, R. Vianello, and R. Ferri (Firenze: Hogrefe).*
Bayley Scales of Infant and Toddler Development - III Ed.	*Global infant development: cognitive, language, motor, social-emotional, and adaptive functions.*	*1 to 42 months Standardized evaluation tool*	*Albers C.A., Grieve A.J. Test review: Bayley, N. Bayley scales of infant and toddler development–third edition. San Antonio, TX: Harcourt assessment. J. Psychoeduc. Assess. 2007;25:180–190.*
SINDA Scale	*Early neurodevelopmental screening: neurological, developmental, and socio-emotional domains.*	*6 weeks to 12 months corrected age Standardized screening*	*Hadders-Algra M, Tacke U, Pietz J, Rupp A, Philippi H. Reliability and predictive validity of the Standardized Infant NeuroDevelopmental Assessment neurological scale. Developmental Medicine & Child Neurology. 2019;61(6):654–660.*
Vineland Adaptive Behavior Scales (VABS)	*Adaptive behavior: communication, daily living skills, socialization, and motor skills.*	*Birth to adulthood Parent/caregiver questionnaire or semi-structured interview*	*Sparrow SS, Cicchetti DV, Balla DA (2016) Vineland Adaptive Behavior Scales-II—2nd ed. Survey Interview Firenze: Giunti Psychometrics*
HOME Scale	*Parental responsivity, physical environment, learning materials, active stimulation, emotional climate, parental involvement*	*Birth to adolescence Interview and observation*	*Bradley R.H., Caldwell B.M., Corwyn R.F. The Child Care HOME Inventories: Assessing the quality of family child care homes. Early Child. Res. Q. 2003;18:294–309.*
AIRE Scale	*Relational quality, communicative dyadic interaction, social/gaze interaction, play patterns.*	*Infants and toddlers Observation-based*	*Capotorti L, Luchino F, Siglienti L, Battaini A, Di Tullio F, Rosenholtz E, et al. La valutazione dell'ambiente familiare negli studi longitudinali di sviluppo infantile. Medico e bambino. 1987;9: 16–23.*

cognitive, and behavioral level.

Children from socioeconomically disadvantaged backgrounds are at increased risk of reduced psychological well-being and poorer emotional and cognitive development.[66] In particular, parental education, often used as a proxy indicator of SES, has emerged as a strong predictor of neurodevelopment. Low maternal education has been consistently associated with poorer cognitive and motor outcomes, particularly in preterm populations.[68,69] Evidence further suggests that maternal education may buffer the effects of early-life adversity, whereas its absence may constitute a risk factor for suboptimal neurodevelopment.

Similar associations between low parental education and adverse developmental outcomes have been reported in children with hypoxic–ischemic encephalopathy, in whom parental educational level was identified as an independent predictor of cognitive developmental outcomes, even after adjustment for clinically relevant parameters.[70] Comparable protective effects have also been reported in children with epilepsy and congenital heart disease.[71] These findings suggest that socioeconomic conditions may influence neurodevelopment not only through environmental pathways but also through biological mechanisms operating at the molecular level.

3.3.2. Neurobiological correlates of socioeconomic disadvantage

Recent neuroimaging studies have identified neural correlates of socioeconomic disadvantage, providing mechanistic insight into how SES shapes brain development. Although effects have been observed across distributed neural networks, the most consistent findings involve regions supporting executive function, language, and emotion regulation. Specifically, lower SES has been associated with structural alterations in the prefrontal cortex, hippocampus, and amygdala, regions that play central roles in self-regulation, reasoning, learning, memory, and emotional processing.[72,73]

Lower SES is also associated with reduced breastfeeding rates,[74] which may limit benefits for offspring cognitive development and maternal well-being. In addition, low SES has been linked to a higher prevalence of externalizing disorders during childhood and adolescence, including attention-deficit/hyperactivity disorder (ADHD) and disruptive behavior disorders, and, to a lesser extent, to internalizing symptoms such as anxiety and depression.[75,76]

Psychosocial stressors that frequently characterize low-SES contexts, including interpersonal violence, overcrowding, and unstable caregiver–child relationships, may further amplify developmental vulnerability and increase the risk of mental health disorders in infants and children exposed to these experiences. Greater investment in early childhood development programs and public policies is therefore needed to reduce the risk that children from socioeconomically disadvantaged backgrounds across different countries fail to reach their developmental potential because of adverse home environmental conditions.[77]

3.4. Home environment and developmental opportunities

The family environment, with the varied opportunities, challenges, and experiences it provides, plays a critical role in shaping neurodevelopmental outcomes in infants and children.[9] In defining the home environment, it is crucial to consider multiple aspects that contribute to its impact on a child's neurodevelopment. These are not confined to the home itself but also include neighborhood characteristics, family- and neighborhood-level SES, and the roles of individuals within the home, such as parents, siblings, extended family, and peers,[78] all of whom are important determinants of neurodevelopmental outcomes.[79]

Favorable home environments are consistently associated with better neurodevelopmental performance in early childhood. Studies conducted in diverse settings, from rural China to Vietnam and urban Italy, show that higher scores on home environment measures (such as the HOME scale and AIRE) are associated with enhanced cognitive, language, motor, and intelligence quotient outcomes.[66,80,81] The literature further suggests that enriched home environments mediate the effects of socioeconomic status and maternal cognitive factors on child development.[66] For a concise overview of the specific neurodevelopmental and environmental domains assessed by the standardized scales, please refer to Table 1.

3.4.1. Environmental enrichment and neural plasticity

Within this context, a key aspect concerns the early promotion of an enriched environment. Early exposure to enriched experiences, combined with active social interaction, has been studied through the paradigm of the enriched environment (EE), which has been shown to profoundly affect the central nervous system at functional, anatomical, and molecular levels.[82] The mechanisms involve enhanced neural plasticity, with environmental experiences modifying brain structure through epigenetic changes and neurotrophic factor expression.[83] Critically, these effects are most pronounced during early developmental sensitive periods, suggesting interventions targeting infancy and early childhood could have long-lasting neurological benefits,[84,85] although

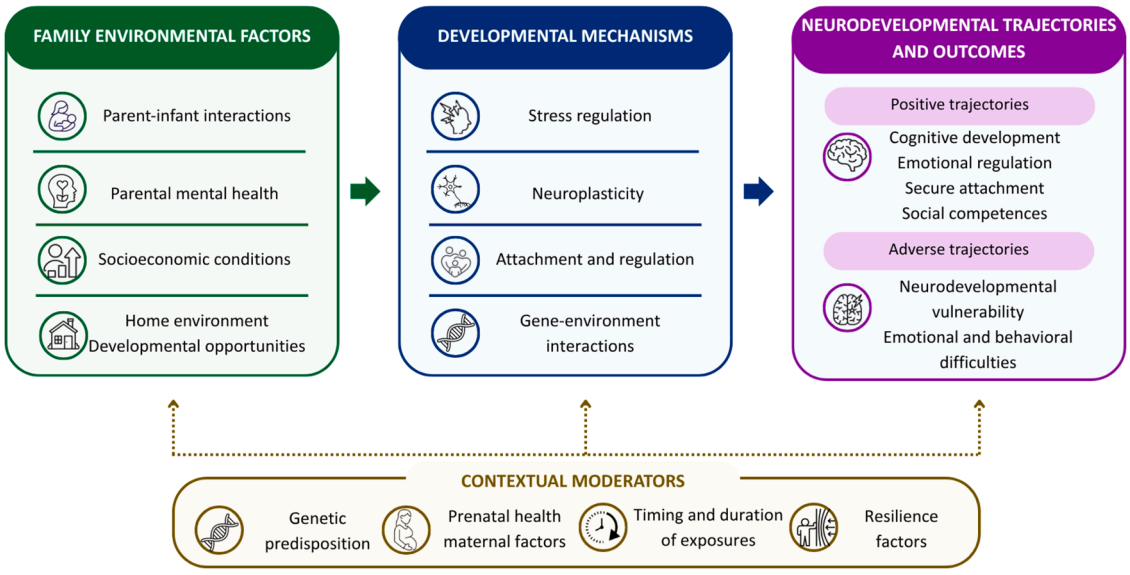


Fig. 1. Family environmental influences on neurodevelopmental trajectories.
Note: Family environmental domains influence neurodevelopmental trajectories through biological and psychosocial mechanisms. These pathways shape vulnerability or resilience to a range of neurodevelopmental and psychiatric outcomes.

evidence for the effectiveness of such approaches in human infancy remains limited.

Exposure to enriched environments has also been associated with improvements in visual acuity development, potentially reflecting accelerated maturation of factors involved in V1 cortical development in animal models.[86] Recently, EE principles have been incorporated into intervention programs aimed at promoting early visual function in infants with visual impairment.

The environment "gets under the skin" through biological embedding, whereby external experiences are translated into internal biological changes. This occurs through two primary, yet distinct, pathways: epigenetic modifications and gene–environment interactions (G × E).

3.4.2. *Epigenetic mechanisms of neurodevelopmental plasticity*

Epigenetic modifications, primarily DNA methylation and histone remodeling, act as a biological bridge between early environmental exposures, such as parental mental health and SES, and long-term neurodevelopmental outcomes.[87] The developing brain is particularly sensitive to these processes during intrauterine life and early infancy.[88] For example, prenatal maternal stress and depression have been associated with increased methylation of the glucocorticoid receptor gene, disrupting the regulation of the hypothalamic–pituitary–adrenal (HPA) axis in offspring.[89] Similarly, deficiencies in methyl donor nutrients such as folate during pregnancy can alter the methylation status of neurotrophic factors, impairing neural connectivity and cognitive development.[87,88] Furthermore, chronic stressors associated with low SES have been associated with differential DNA methylation in genes regulating inflammatory responses and, in some studies, stress- and plasticity-related genes, suggesting a molecular pathway for the biological embedding of social disadvantage.[90]

3.4.3. *Gene–environment interactions and risk amplification*

Gene–environment interactions (G × E) occur when the effect of an environmental exposure on a trait or disorder depends on an individual's genotype.[91] In many contemporary models, this implies that genetic liability may reflect differences in sensitivity to environmental conditions rather than a fixed, deterministic risk.

The "diathesis–stress" model proposes that environmental adversity, such as low SES or parental psychopathology, increases the likelihood of negative outcomes in genetically vulnerable individuals.[92] For instance, children carrying specific alleles in dopamine- and serotonin-related genes (e.g., *DRD4, 5-HTTLPR*) may show higher levels of ADHD symptoms or externalizing problems in the presence of harsh or conflictual family environments, whereas the same genotypes are less impairing under more supportive conditions.[92,93] Conversely, the "differential susceptibility" hypothesis suggests that some individuals carry "plasticity genes" that increase sensitivity to both negative and positive environmental influences.[94] In this framework, genetically "sensitive" children may have worse outcomes under high adversity but also show the greatest gains when exposed to responsive parenting or positive peer/educational contexts.[95]

Recent research emphasizes that these G × E dynamics are often mediated by the epigenetic mechanisms discussed above, where genetic background influences the degree to which environmental stress "embeds" biologically.[87]

4. Conclusions and translational implications

A child's development is founded upon various related domains, all of which are influenced by biological, social, and environmental factors that are vulnerable to protective or adverse situations[96] (Fig. 1, Tables 2 and 3). Because the family environment represents a modifiable set of risk factors, early child development programmes aimed at mitigating the impact of adverse home environments on infants' and children's developmental potential are urgently needed. Effective prevention and intervention strategies must be multidimensional (including components related to health, nutrition, caregiving quality, early learning, and social protection within an integrated framework), and initiated as early as possible, ideally during the prenatal period or the first 1000 days of life. Within this framework, interventions should prioritize parental empowerment and ensure that caregiver–child interactions are central to these programmes, as they have been shown to yield greater long-term benefits for cognitive and neuromotor development than approaches focusing exclusively on the infant's functional impairments. To be effective, such programmes should ensure frequent contact with healthcare professionals and be of sufficient duration to allow caregivers to practice and consolidate adaptive caregiving behaviours. Specific relational interventions—such as skin-to-skin contact (Kangaroo care) for preterm infants, book-sharing programmes, and play-based curricula—improve physiological regulation, bonding, and cognitive

Table 2
Family environmental domains, contrasting pathways, and neurodevelopmental outcomes.

Domain	Protective contexts and adaptive outcomes	Vulnerability context & adverse outcomes
Parent–infant interactions	***Protective Factors & Dimensions:*** *Synchrony* *Contingency* *Sensitivity* *Appropriate stimulation* *Paternal involvement*	***Vulnerability Factors:*** *Inconsistent, poorly attuned interactions* *Harsh parenting*
	Adaptive Outcomes: *Secure attachment* *Emotional regulation* *Cognitive and socioemotional competence*	***Adverse outcomes:*** *Physiological stress and socioemotional difficulties* *Stressful interactions with potential long-term effects on development* *Altered fronto-amygdala connectivity and reduced global network efficiency* *Compromised later emotional regulation*
Parental mental health	***Protective Factors & Dimensions:*** *Caregivers psychological well-being* *Early screening, support networks, timely treatment* *Warm, supportive parenting styles*	***Vulnerability Factors:*** *Prenatal stress* *Maternal depression* *Paternal psychopathology*
	Adaptive Outcomes: *Healthy, optimized offspring neurodevelopmental trajectories*	***Adverse outcomes:*** *Cognitive vulnerability* *Altered emotional regulation* *Behavioral problems* *Increased risk for neurodevelopmental disorders*
Socioeconomic conditions	***Protective Factors & Dimensions:*** *Higher parental educational attainment* *Stable household income* *Supportive neighborhood resources*	***Vulnerability Factors:*** *Low SES* *Low household income* *Material/resource deprivation* *Psychosocial stress* *Nutritional and healthcare access* *Toxins and reduced breastfeeding*
	Adaptive Outcomes: *Mitigation of early-life environmental adversity*	***Adverse outcomes:*** *Cognitive and language problems* *Compromised executive function* *Externalizing/internalizing symptoms*
Home environment	***Protective Factors & Dimensions:*** *Enriched environment (EE)* *Developmental stimulation* *Family and peer network roles*	***Vulnerability Factors:*** *Adverse home environments* *Severe early-life adversity (such as deprivation)* *Parental psychological difficulties*
	Adaptive Outcomes: *Enhanced cognitive, motor, language, and adaptive developmental outcomes.*	***Adverse outcomes:*** *Long-term neurodevelopmental impairments* *Failure of infants to reach their developmental potential*

development trajectories. Where feasible, these programmes should incorporate high-quality home visiting services (such as the Nurse-Family Partnership), providing structured curricula and appropriate supervision.[97]

Finally, translational strategies must address both structural and clinical challenges. For families facing material and economic deprivation, cash transfer programmes combined with complementary educational interventions can reduce parental stress, improve caregiving quality,[98] and provide resources necessary for an enriched home environment. Concurrently, early screening and strong care for parental mental health difficulties are essential, particularly in contexts where parental psychological difficulties may negatively affect children's neurodevelopment.[99] Protecting parental well-being is therefore a crucial public health strategy aimed at promoting healthy neurodevelopment in the next generation (Table 4).

Table 3
Mapping of family environmental factors onto neurodevelopmental disorders.

Family factor	Target disorder	Evidence consistency	References
Maternal perinatal mood and anxiety disorders (PMADs)	*ADHD*	*Increased risk, high consistency.*	Kępińska et al., 2024
Maternal perinatal mood and anxiety disorders (PMADs)	*ASD*	*Increased risk, high consistency.*	Kępińska et al., 2024
Paternal perinatal mood and anxiety disorders (PMADs)	*ADHD/ASD*	*Increased risk, emerging evidence.*	Kępińska et al., 2024
Low SES	*ADHD*	*Consistent evidence.*	Russell et al., 2016
Low SES	*Internalizing/ externalizing symptoms*	*Consistent evidence.*	Anderson et al., 2022
Harsh Parenting	*Externalizing problems*	*Consistent evidence.*	Koyama et al., 2024

Table 4
Implications for early prevention and intervention.

Target domain	Potential intervention strategies
Parent–infant interactions	*Parenting support programs, home visiting, responsive caregiving coaching, skin-to-skin interventions*
Parental mental health	*Early screening during pregnancy and postpartum, access to mental health care, dyadic interventions*
Socioeconomic adversity	*Early childhood development programs, social protection measures, integrated health and educational support*
Home environment	*Enrichment-based developmental programs, book-sharing, play-based interventions, caregiver-guided stimulation*

5. Declaration of generative AI in scientific writing

During the preparation of this work, the authors used Chat GPT in order to improve language and readability. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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