Psycho-Neuro-Endocrine-Immunology, DOHaD and Mindfulness: Towards an Integrated Model of Prenatal Salutogenesis

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Abstract: *Over the past thirty years, the DOHaD (Developmental Origins of Health and Disease) paradigm has radically transformed our understanding of the aetiology of chronic diseases, shifting the focus from genetic determinism to the biological plasticity of early life. This perspective converges naturally with Psycho-Neuro-Endocrine-Immunology (PNEI), which interprets the organism not as a collection of isolated systems but as an interconnected network in which the nervous, endocrine, immune and metabolic systems engage in constant dialogue with the psychosocial environment.*

The intersection of these two approaches reveals pregnancy as the temporal window of maximum susceptibility and evolutionary opportunity. Within this scenario, the placenta emerges as a sophisticated interface organ, capable of translating maternal environmental stimuli — including stress signals and adaptive responses — into molecular signals that shape the neurodevelopment, hypothalamic-pituitary-adrenal (HPA) axis and metabolic setting of the offspring.

In this research landscape, mindfulness-based practices are emerging as promising non-pharmacological and transdiagnostic interventions. Although scientific evidence has already validated their efficacy in regulating the stress response, autonomic nervous system balance and low-grade chronic inflammation, the hypothesis that such modulation may directly influence fetal programming remains an unexplored frontier of considerable clinical potential.

The present review aims to bridge this gap by offering a critical synthesis that integrates contemplative neuroscience, polyvagal theory, epigenetics and perinatal medicine. Through the pro-

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posal of an original theoretical model, it explores how maternal neurovegetative regulation — analysed in terms of heart rate variability (HRV) and placental communication — may act as a systemic determinant of health. From this perspective, mindfulness is redefined not merely as a tool for distress reduction, but as an active modulator of salutogenic processes, capable of fostering, from the earliest stages of development, the biological foundations of human resilience.

Keywords: *Developmental Origins of Health and Disease (DOHaD), fetal programming; mindfulness, heart rate variability (HRV), salutogenesis, placenta.*

Introduction

The Developmental Origins of Health and Disease (DOHaD) paradigm has marked a decisive conceptual transition, overcoming genetic determinism in favour of a dynamic model in which adult health is shaped from the earliest stages of development (Barker, 1998; 2004; Gluckman *et al.*, 2008). Barker's epidemiological evidence demonstrated that low birth weight is predictive of chronic diseases in adulthood, identifying gestation as a critical window of biological plasticity (Barker, 1990; 1995; Hanson & Gluckman, 2011). Through predictive adaptive responses (PARs), the fetus modulates its phenotype in response to maternal signals; however, a potential developmental mismatch between the prenatal and postnatal environment may convert such adaptations into pathological risk factors (Gluckman & Hanson, 2005).

PNEI defines this programming as a systemic phenomenon mediated by bidirectional communication among the nervous, endocrine, immune and metabolic systems (McEwen, 1998; 2007; Ader, 2007; Bottaccioli & Bottaccioli, 2017). Pregnancy thus becomes a complex ecosystem in which maternal psychophysiological variables — from inflammation to stress regulation — shape fetal development (Van den Bergh *et al.*, 2005; 2015; Glover, 2014; O'Donnell & Meaney, 2017).

Adopting Antonovsky's salutogenic perspective, perinatal medicine must evolve toward strengthening maternal-fetal resilience mechanisms, moving beyond mere pathogenic prevention (Antonovsky, 1979; Hanson & Gluckman, 2011). In this context, contemplative neuroscience identifies mindfulness as a systemic modulator capable of influencing the HPA axis, heart rate variability, gene expression and the organisation of neural networks (Kabat-Zinn, 2003; Davidson & McEwen, 2012; Tang *et al.*, 2015). Exploring the impact of such practices on fetal programming represents an innovative clinical frontier (Crovetto *et al.*, 2023; Nakaki *et al.*, 2023). The present review proposes an integrated model of prenatal salutogenesis, synthesising PNEI evidence, polyvagal theory and clinical research on mindfulness-based interventions (Babbar *et al.*, 2021).

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DOHaD and Developmental Plasticity: From Fetal Origins of Disease to the Medicine of Salutogenesis

The DOHaD paradigm represents an epistemological turning point in contemporary medicine, having relocated the aetiopathogenesis of chronic diseases from adult maturity phases to the early periods of human development (Barker, 2004). David Barker's epidemiological observations highlighted the correlation between low birth weight and increased incidence of chronic diseases — including ischaemic heart disease, hypertension and metabolic syndrome — in adulthood (Barker, 1990; 1995; 1998). These findings led to the formulation of the Fetal Origins Hypothesis, which postulates that the intrauterine environment exerts a lasting influence on the functional programming of organs and biological systems (Barker, 1995).

The evolution of the DOHaD model, integrated with evolutionary biology, introduced the concept of predictive adaptive responses (Gluckman *et al.*, 2008). Within this framework, the fetus is not a passive organism but a dynamic biological system capable of interpreting maternal environmental signals to adapt its development to anticipated postnatal conditions (Gluckman *et al.*, 2005). The resulting phenotype is the outcome of a continuous interaction between genetic heritage and early biological experience (Gluckman *et al.*, 2008). While such responses are adaptive in predictable contexts (Gluckman & Hanson, 2004), developmental mismatch between the prenatal and postnatal environment may convert these adaptations into factors of pathological susceptibility (Gluckman & Hanson, 2005).

These processes are mediated by epigenetic mechanisms — DNA methylation, histone modifications and microRNA regulation — which modulate gene expression without altering the nucleotide sequence (Meaney & Szyf, 2005; Moore *et al.*, 2013; Meaney, 2010). Within this framework, health is not understood as the mere absence of disease, but as an expression of the organism's adaptive capacity, in accordance with Antonovsky's salutogenic model: whereas the pathogenic paradigm seeks the causes of disease, salutogenesis focuses on the processes that sustain functional stability and resilience in the face of adverse conditions (Antonovsky, 1979).

The current extension of the DOHaD paradigm adopts a life-course perspective, recognising that developmental programming begins in the preconception phase and continues throughout the entire perinatal pathway. PNEI provides the essential theoretical framework for decoding this network of interactions (Bottaccioli & Bottaccioli, 2017): maternal stress is not a mere emotional experience, but a systemic event involving the HPA axis, autonomic nervous system, immune response, energy metabolism and epigenome (McEwen, 1998; 2007; Ader, 2007). This convergence confirms that the intrauterine environment constitutes a dynamic biological ecosystem, constantly shaped by bidirectional communication between mother and fetus (O'Donnell & Meaney, 2017).

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The Placenta: Neuroendocrine, Immune and Epigenetic Hub

The placenta is not a mere passive filter for nutritional exchange, but an organ of biological integration that translates maternal experience into developmental instructions for the fetus. As a neuroendocrine organ, it synthesises crucial mediators such as CRH (corticotropin-releasing hormone), capable of critically modulating the maternal-fetal HPA axis (Bronson & Bale, 2016; Kramer & Bale, 2023; Ragusa *et al.*, 2025). Placental immune function maintains a delicate balance at the maternal-fetal interface via cytokines and decidual cells; alterations in this profile are associated with fetal growth restriction, preeclampsia and neurodevelopmental dysfunctions (Burton & Fowden, 2015).

A fundamental protective mechanism is represented by the enzyme 11β-HSD2, which inactivates maternal cortisol, preventing chronic fetal exposure to glucocorticoids (Seckl & Holmes, 2007). Stress, inflammation and malnutrition may inhibit this enzymatic barrier, altering HPA axis programming and the neurobiological development of the offspring (Hodyl *et al.*, 2017). Concurrently, the placenta serves as an epigenetic interface, sensitive to variations in DNA methylation, microRNA regulation and histone modifications induced by maternal factors such as psychological stress, obesity or exposure to toxins (Kramer *et al.*, 2023).

Systemic integration is completed by the gut-brain-placenta axis, wherein maternal microbiota metabolites modulate trophoblastic function and the gestational immune profile (Cryan *et al.*, 2019). This bidirectional communication positions the placenta at the centre of the systemic pregnancy response (Dinan & Cryan, 2017; Hsiao *et al.*, 2013), defining it as a genuine "sensory organ" capable of transducing endocrine, metabolic and autonomic signals into biological input for the fetus. This view transcends functional reductionism, enshrining the central role of the placenta in mediating fetal plasticity and long-term health trajectories.

Prenatal Stress, HPA Axis and Neurobiological Programming

Maternal stress represents a critical environmental factor for fetal developmental programming, configuring itself, according to the PNEI paradigm, as a systemic event involving the HPA axis, autonomic nervous system and immune response (McEwen, 2007). Although physiological activation of the HPA axis is necessary for fetal pulmonary and metabolic maturation (Van den Bergh *et al.*, 2005), the chronic hyperactivation associated with Early Life Stress (ELS) results in prolonged exposure to glucocorticoids, potentially maladaptive for neurobiological development (Monk *et al.*, 2019; Sandman *et al.*, 2018).

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Under conditions of chronic stress, reduced barrier efficacy of 11β-HSD2 increases transplacental cortisol passage, altering neurogenesis, synaptogenesis and neuronal differentiation (Hodyl *et al.*, 2017). The role of systemic inflammation is additional: the increase in pro-inflammatory cytokines (IL-6, TNF-α) may compromise fetal microglial maturation, conditioning brain developmental trajectories (Slavich & Irwin, 2014). Neuroimaging studies confirm that elevated levels of prenatal stress are associated with structural and functional alterations in the amygdala, hippocampus, prefrontal cortex, cingulate cortex and insula (O'Donnell & Meaney, 2017; Buss *et al.*, 2012; Sandman *et al.*, 2015; Lautarescu *et al.*, 2021; Van den Bergh *et al.*, 2020; Thomason & Hendrix, 2024).

These alterations should not be interpreted as irreversible damage, but rather as modifications of maturational trajectories, intrinsically amenable to remodelling through positive postnatal environmental influences (Glover, 2014; Van den Bergh *et al.*, 2020). Fetal programming indeed creates a probabilistic vulnerability, not an immutable biological fate, confirming the permanent plasticity of the nervous system and validating the opportunity for interventions aimed at promoting biological resilience (Van den Bergh *et al.*, 2020).

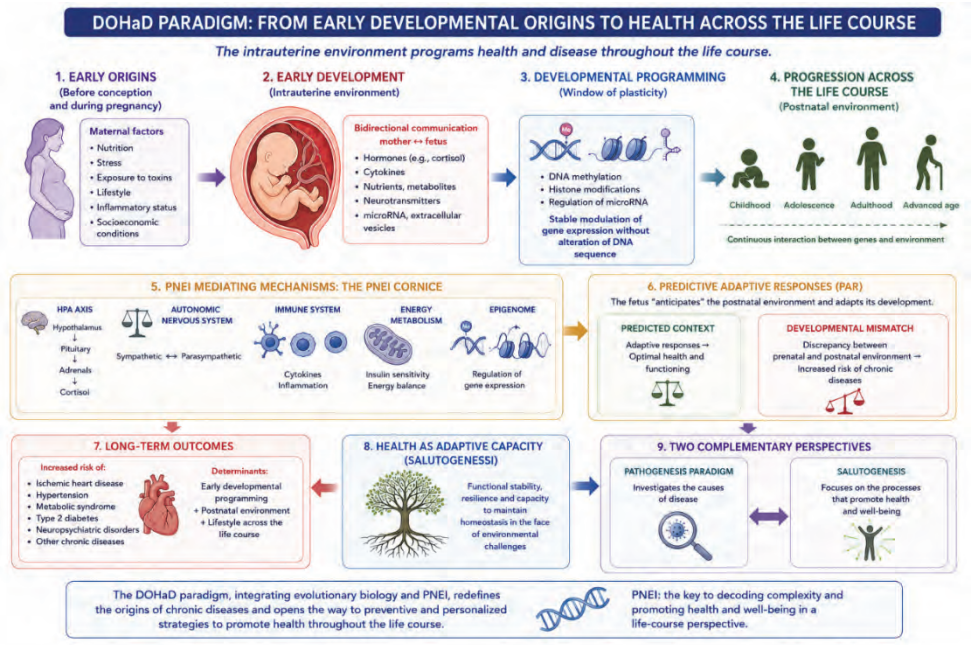


Fig. 1. DOHaD paradigm: from early developmental origins to health across the lifespan

Note. *The images in Figures 1 and 2 were created by the author with the help of AI.*

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The Autonomic Nervous System (ANS) as Mediator of Maternal-Fetal Communication

The Autonomic Nervous System (ANS), far from being a mere visceral effector system, is a complex regulatory network that coordinates cardiovascular, endocrine, immune and metabolic activity (Benarroch, 1993; Thayer & Lane, 2009; Critchley & Harrison, 2013). This function is mediated by the Central Autonomic Network (CAN), a network that integrates cognitive, emotional and homeostatic processes through structures such as the prefrontal cortex, insula and amygdala (Benarroch, 1993; Beissner *et al.*, 2013; Valenza *et al.*, 2019; Thayer *et al.*, 2012; 2025). The CAN modulates the dynamic balance between sympathetic and parasympathetic activation, ensuring the resilience and adaptive capacity of the organism (Craig, 2002; Thayer & Lane, 2009; Shaffer & Ginsberg, 2017).

During pregnancy, this balance is fundamental for maternal cardiovascular remodelling and the maintenance of metabolic homeostasis required for fetal development (Braeken *et al.*, 2017; Sharifiheris *et al.*, 2022). Although direct clinical evidence requires further confirmation, a plausible pathophysiological basis exists for the view that maternal autonomic regulation influences uteroplacental function and the fetal environment (Cerritelli *et al.*, 2021; Van den Bergh *et al.*, 2020). Porges' polyvagal theory (2022) underscores how vagal tone reflects the capacity to maintain biological safety and emotional regulation, with elevated parasympathetic activity associated with reduced systemic inflammation.

Heart rate variability (HRV) constitutes the primary non-invasive biomarker of such neurovegetative flexibility (Pinto *et al.*, 2025; Widatalla *et al.*, 2025). Through the cholinergic anti-inflammatory reflex, the vagus nerve modulates inflammation by inhibiting cytokine production (Tracey, 2002). Interventions such as mindfulness and slow breathing, by increasing vagal tone and HRV, offer indirect protective mechanisms for the fetal environment (Lehrer & Gevirtz, 2014; Kramar, 2025; Thayer & Lane, 2009; Braeken *et al.*, 2017; Sharifiheris *et al.*, 2022). Observational studies on maternal-fetal physiological synchrony suggest the existence of co-regulation processes (DiPietro *et al.*, 2006). Although direct causality on fetal programming remains under investigation, the convergence of PNEI data renders it biologically plausible that ANS modulation represents a pillar of prenatal salutogenesis (Van den Bergh *et al.*, 2020; Babbar *et al.*, 2021; Cerritelli *et al.*, 2021).

Contemplative Neuroscience and Neurobiological Regulation

Over the past two decades, contemplative neuroscience has provided important

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evidence on the neurobiological correlates of mindfulness, demonstrating how regular practice is associated with functional modifications in brain networks involved in attention, interoceptive awareness, emotional regulation and cognitive control (Calderone *et al.*, 2024; Rafter *et al.*, 2026; Tang *et al.*, 2015). Neuroimaging studies have identified three principal connectivity systems — the Default Mode Network (DMN), the Salience Network (SN) and the Central Executive Network (CEN) — whose modulation is associated with reduced rumination, enhanced interoceptive awareness and strengthened attentional and emotional regulation (Menon, 2011; Menon & Uddin, 2010; Menon *et al.*, 2023; Bremer *et al.*, 2022; Tang *et al.*, 2015).

Of particular relevance is the down-regulation of the amygdala response, with consequent reduction in stress reactivity and enhanced connectivity with the prefrontal cortex (Taren *et al.*, 2015; Desbordes *et al.*, 2012). These neurofunctional modifications, interpretable as a strengthening of top-down control over limbic circuits, are accompanied by a reduction in cortisol and pro-inflammatory cytokine production, confirming the systemic action of mindfulness (Creswell *et al.*, 2014).

The neurobiological effects of mindfulness also extend to epigenetic modulation. Studies on expert practitioners have documented gene expression modifications, with down-regulation of NF-κB-mediated pro-inflammatory pathways and up-regulation of genes associated with cellular repair and antioxidant response (Kaliman *et al.*, 2014; Buric *et al.*, 2017; Venditti *et al.*, 2020; Venditti, 2025). Such effects have also been observed in the modulation of the epigenetic clock, suggesting a potential impact on biological ageing (Chaix *et al.*, 2017).

Mindfulness in Pregnancy: Clinical Evidence and Mechanisms of Action

The application of mindfulness-based interventions in the perinatal setting has registered significant growth. Randomised clinical trials have demonstrated that programmes such as Mindfulness-Based Childbirth and Parenting (MBCP) significantly reduce anxiety, stress and depression levels in pregnant women (Duncan & Bardacke, 2010; Findik *et al.*, 2025; Gheibi *et al.*, 2020). A meta-analysis by Min *et al.* (2023) confirmed the efficacy of such interventions also in preventing postpartum depression.

At the psychophysiological level, mindfulness during pregnancy is associated with increased HRV and reduced blood pressure, indicative of improved autonomic balance (Braeken *et al.*, 2017; Natarajan, 2023; Barbry *et al.*, 2026). The impact also extends to infant development: the IMPACT BCN study documented favourable effects of mindfulness-based stress reduction (MBSR) on fetal and neonatal brain development, assessed through neuroimaging (Crovetto *et al.*, 2023; Nakaki *et al.*, 2023; Gomez *et al.*, 2025).

Emerging evidence further suggests that maternal mindfulness may modulate

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neonatal autonomic and behavioural reactivity (Noroña-Zhou *et al.*, 2022). These data, albeit preliminary, support the hypothesis that mindfulness represents an epigenetic and neurophysiological modulator capable of influencing developmental trajectories through the regulation of maternal stress response systems (Black & Slavich, 2016; Dunn & Dimolareva, 2022; Bower & Irwin, 2016; Walton *et al.*, 2023).

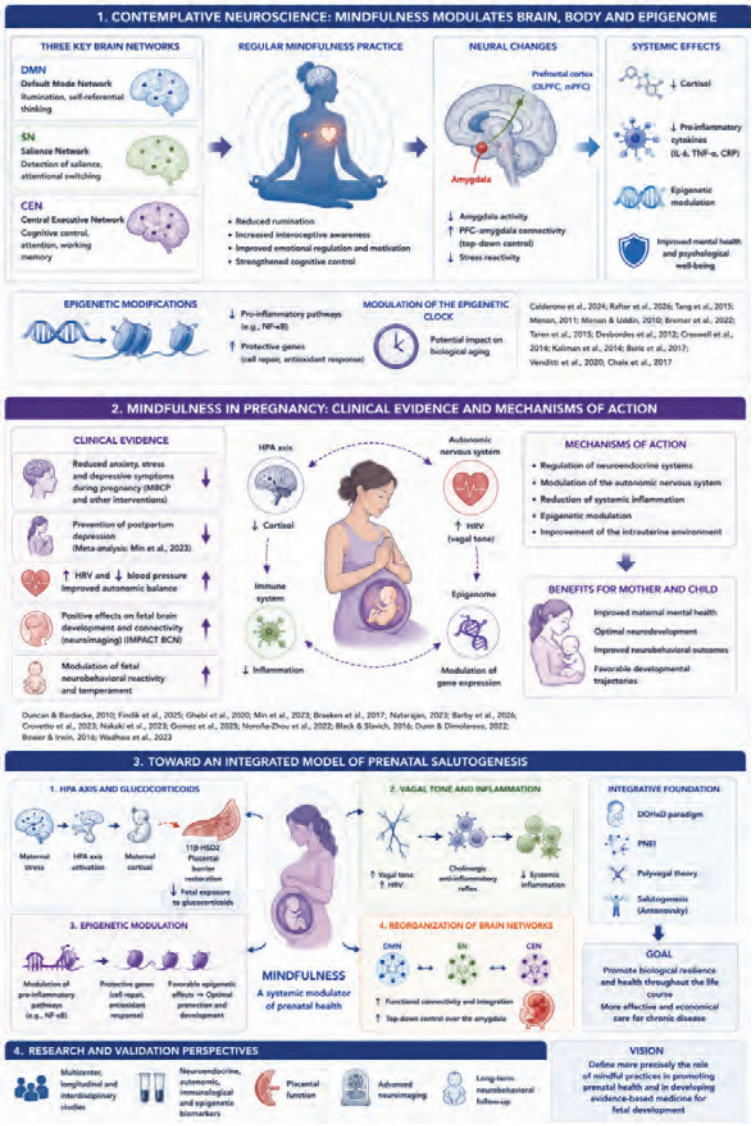


Fig. 2. The PNEI Integrated Model of Prenatal Salutogenesis and Contribution to the Salutogenesis of Meditative Practices

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Towards an Integrated Model of Prenatal Salutogenesis

The evidence reviewed thus far converges in delineating an integrated model in which maternal neurovegetative regulation acts as a systemic determinant of fetal health. Within this framework, mindfulness is configured not merely as an intervention for reducing psychological distress, but as an active modulator of salutogenic processes, capable of influencing the intrauterine environment through neuroendocrine, autonomic, immune and epigenetic mechanisms.

The proposed model rests on four interconnected axes: (1) regulation of the HPA axis and reduction of fetal glucocorticoid exposure, mediated by the restoration of the placental 11β-HSD2 enzymatic barrier; (2) enhancement of vagal tone and HRV, with consequent modulation of the cholinergic anti-inflammatory reflex and reduction of maternal systemic inflammation; (3) epigenetic modulation, with down-regulation of pro-inflammatory pathways and up-regulation of protective genes; (4) functional reorganisation of brain networks involved in emotional and interoceptive regulation, with particular reference to DMN-SN-CEN connectivity and top-down control over the amygdala.

This model finds its foundation in the convergence of the DOHaD paradigm, PNEI and polyvagal theory, and aligns with Antonovsky's salutogenic perspective, according to which promoting biological resilience is more effective and cost-efficient than treating overt pathology.

The clinical validation of this model will require multicentre, longitudinal and interdisciplinary studies integrating neuroendocrine, autonomic, immune and epigenetic biomarkers, measures of placental function, advanced neuroimaging techniques and follow-up of offspring neurobehavioural development. Only through this systemic approach will it be possible to define more precisely the role of integrative medicine interventions in promoting prenatal health and developing a genuinely evidence-based developmental medicine.

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