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

Stefania Carbonella*

Received 3 July, revised 27 July, accepted 30 July
Online first: 30 September 2026

* Psychologist, Psychotherapist, Specialist in Psychoanalytic Psychotherapy of Developmental Age, Turin. Coordinator, National Commission SIPNEI ELCA.
stefania.carbonella@gmail.com

Please cite: Carbonella S. (2026). Psycho-Neuro-Endocrine-Immunology, DOHaD and Mindfulness: Towards an Integrated Model of Prenatal Salutogenesis. *PNEI Review*. DOI: 10.3280/pnei2025oa23569

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-

PNEI review – ISSN 2532-2826 – DOI: 10.3280/pnei2025oa23569

Copyright © FrancoAngeli This work is released under Creative Commons Attribution – Non-Commercial – No Derivatives License. For terms and conditions of usage please see: <http://creativecommons.org>

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).

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 intra-uterine environment constitutes a dynamic biological ecosystem, constantly shaped by bidirectional communication between mother and fetus (O'Donnell & Meaney, 2017).

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 11 β -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).

Under conditions of chronic stress, reduced barrier efficacy of 11 β -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 pre-natal 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).

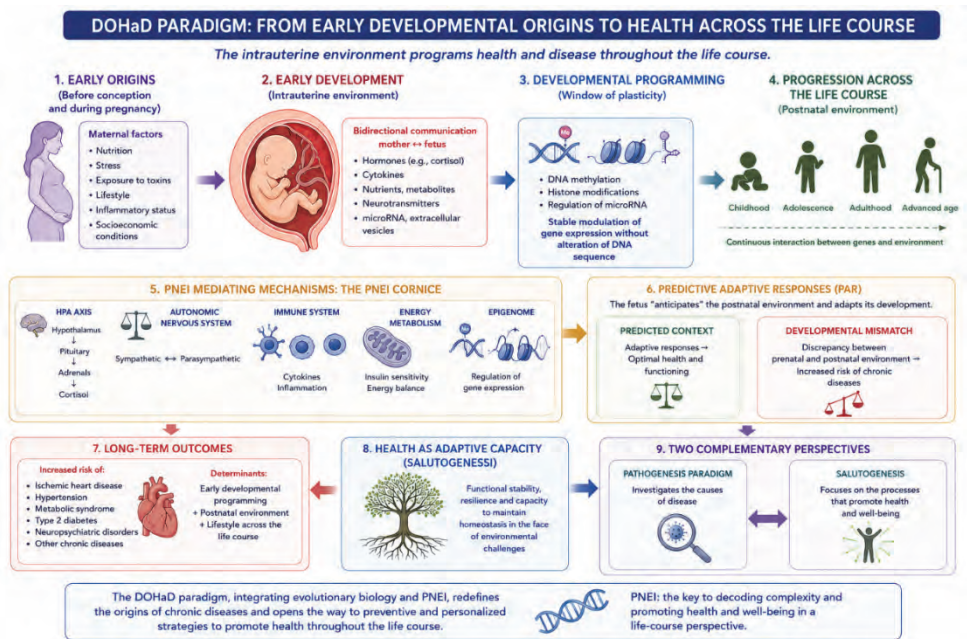


Fig. 1. DOHaD paradigm: from early developmental origins to health across the lifespan

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

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 re-modelling 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

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- κ B-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

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).

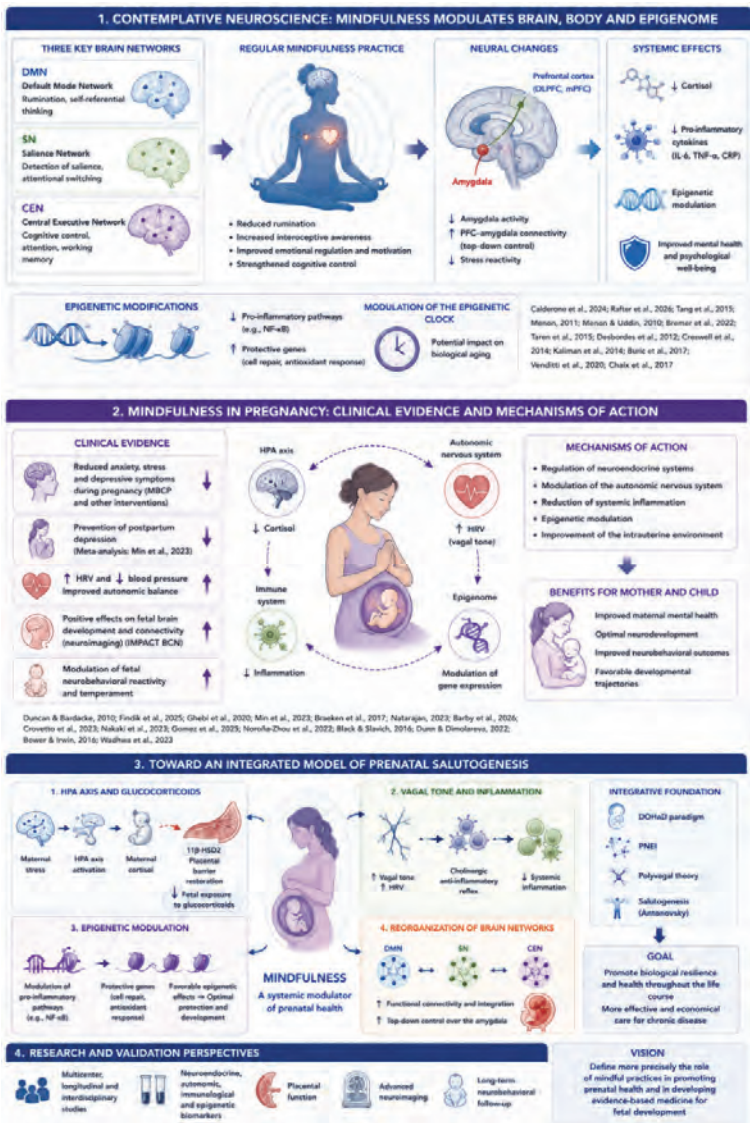


Fig. 2. The PNEI Integrated Model of Prenatal Salutogenesis and Contribution to the Salutogenesis of Meditative Practices

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 11 β -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.

Author Contributions: Conceptualization: S.C.; writing: original draft preparation, review and editing: S.C.
Funding: This research received no external funding. Conflicts of Interest: The authors declare no conflict of interest. All authors have read and agreed to the published version of the manuscript.

References

- Adler R. (2007).** *Psychoneuroimmunology*. London: Academic Press.
- Antonovsky A. (1979).** *Health, stress, and coping. New perspectives on mental and physical well-being*. California: Jossey-Bass.
- Babbar S., Oyarzabal A.J., & Oyarzabal, E.A. (2021).** Meditation and mindfulness in pregnancy and postpartum: a review of the evidence. *Clinical Obstetrics and Gynecology*, 64(3), 661-682. <https://doi.org/10.1097/GRF.0000000000000640>
- Barbry A., Gal E., Carton A., & Coquart J. (2026).** Effect of brief mindfulness meditation interventions
PNEI review – ISSN 2532-2826 – DOI: 10.3280/pnei2025oa23569

Copyright © FrancoAngeli This work is released under Creative Commons Attribution – Non-Commercial – No Derivatives License. For terms and conditions of usage please see: <http://creativecommons.org>

- on heart rate variability in adults: a systematic review. *Applied Psychophysiology and Biofeedback*, 51(1), 3-18. <https://doi.org/10.1007/s10484-025-09724-y>
- Barker D.J.P. (1990).** The fetal and infant origins of adult disease. *BMJ: British Medical Journal*, 301(6761), 1111. <https://doi.org/10.1136/bmj.301.6761.1111>
- Barker D.J.P. (1995).** The Wellcome Foundation Lecture, 1994. The fetal origins of adult disease. *Proceedings of the Royal Society of London*, 262(1363), 37-43. <https://doi.org/10.1098/rspb.1995.0173>
- Barker D.J.P. (1998).** *Mothers, babies, and health in later life*. Edinburgh: Churchill Livingstone.
- Barker D.J.P. (2004).** The developmental origins of adult disease. *Journal of the American College of Nutrition*, 23(sup6), 588S-595S. <https://doi.org/10.1080/07315724.2004.10719428>
- Beissner F., Meissner K., Bär K.J., & Napadow V. (2013).** The autonomic brain: an activation likelihood estimation meta-analysis for central processing of autonomic function. *Journal of Neuroscience*, 33(25), 10503-10511. <https://doi.org/10.1523/JNEUROSCI.1103-13.2013>
- Benarroch E.E. (1993).** The central autonomic network: functional organization, dysfunction, and perspective. *Mayo Clinic Proceedings*, 68(10), 988-1001. [https://doi.org/10.1016/S0025-6196\(12\)62272-1](https://doi.org/10.1016/S0025-6196(12)62272-1)
- Biaggi A., & Pariante C. M. (2020).** Risk factors for depression and anxiety during the perinatal period. In: Rosa Quatraro M. & Grussu P. (Eds.), *Handbook of Perinatal Clinical Psychology* (pp. 239-265). UK: Routledge.
- Biaggi A., Zonca V., Anacker C., Begni V., Benedetti F., Bramante A., Braniecka A., Brenna V., Bulgheroni M., Buss C., Cavaliere L., Cecil C.A.M., Couch A.C., de Barra D., El Marroun H., Entringer S., Grassi-Oliveira R., Jackowska M., Korosi A., Kwant P. J. C., Lahti J., Lekadir K., Mansuy I., Manuella F., Marizzoni M., Meyer U., Monk C., Radoš S. N., Pariante C. M., Pollux B.J.A., Priestley K., Räikkönen K., Richetto J., Riva M.A., Rothmann L.M., Simonetti V., Vai B., Vernon A.C., Žutić M., Cattaneo A., & HappyMums Consortium (2025).** Understanding, predicting, and treating depression in pregnancy to improve mothers' and offspring's mental health outcomes: The Happy Mums study. *Brain, Behavior, & Immunity-Health*, 44, 100961. <https://doi.org/10.1016/j.bbih.2025.100961>
- Black D.S., & Slavich G.M. (2016).** Mindfulness meditation and the immune system: a systematic review of randomized controlled trials. *Annals of the New York Academy of Sciences*, 1373(1), 13-24. <https://doi.org/10.1111/nyas.12998>
- Bottaccioli F., & Bottaccioli A.G. (2017).** *Psiconeuroendocrinoimmunologia e scienza della cura integrata*. Milano: Edra.
- Bower J.E., & Irwin M.R. (2016).** Mind-body therapies and control of inflammatory biology: A descriptive review. *Brain, Behavior, and Immunity*, 51, 1-11. <https://doi.org/10.1016/j.bbi.2015.06.012>
- Braeken M.A., Jones A., Otte R.A., Nyklíček I., & Van den Bergh B.R. (2017).** Potential benefits of mindfulness during pregnancy on maternal autonomic nervous system function and infant development. *Psychophysiology*, 54(2), 279-288. <https://doi.org/10.1111/psyp.12782>
- Bremer B., Wu Q., Mora Álvarez M. G., Hölzel B. K., Wilhelm M., Hell E., Tavacioglu E.E., Torske A., Koch K. (2022).** Mindfulness meditation increases default mode, salience, and central executive network connectivity. *Scientific Reports*, 12(1), 13219. <https://doi.org/10.1038/s41598-022-17325-6>
- Bronson S.L., & Bale T.L. (2016).** The placenta as a mediator of stress effects on neurodevelopmental reprogramming. *Neuropsychopharmacology*, 41(1), 207-218. <https://doi.org/10.1038/npp.2015.231>
- Buric I., Farias M., Jong J., Mee C., & Brazil I.A. (2017).** What is the molecular signature of mind-body interventions? A systematic review of gene expression changes induced by meditation and related practices. *Frontiers in Immunology*, 8, 259671. <https://doi.org/10.3389/fimmu.2017.00670>
- Burton G.J., & Fowden A.L. (2015).** The placenta: a multifaceted, transient organ. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370(1663), 20140066. <https://doi.org/10.1098/rstb.2014.0066>
- Buss C., Davis E.P., Muftuler L.T., Head K., & Sandman C.A. (2010).** High pregnancy anxiety during mid-gestation is associated with decreased gray matter density in 6–9-year-old children. *Psychoneuroendocrinology*, 35(1), 141-153. <https://doi.org/10.1016/j.psyneuen.2009.07.010>
- Calderone A., Latella D., Impellizzeri F., De Pasquale P., Famà F., Quartarone A., & Calabrò R.S.**

- (2024). Neurobiological changes induced by mindfulness and meditation: A systematic review. *Biomedicines*, 12(11), 2613. <https://doi.org/10.3390/biomedicines12112613>
- Cascio C.J., Moore D., & McGlone F. (2019). Social touch and human development. *Developmental Cognitive Neuroscience*, 35, 5-11. <https://doi.org/10.1016/j.dcn.2018.04.009>
- Cerritelli F., Frasch M.G., Antonelli M.C., Viglione C., Vecchi S., Chiera M., & Manzotti A. (2021). A review on the vagus nerve and autonomic nervous system during fetal development: searching for critical windows. *Frontiers in Neuroscience*, 15, 721605. <https://doi.org/10.3389/fnins.2021.721605>
- Chaix R., Alvarez-López M.J., Fagny M., Lemee L., Regnault B., Davidson R.J., Lutz A., Kaliman P. (2017). Epigenetic clock analysis in long-term meditators. *Psychoneuroendocrinology*, 85, 210-214. <https://doi.org/10.1016/j.psyneuen.2017.08.016>
- Craig A.D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews Neuroscience*, 3(8), 655-666. <https://doi.org/10.1038/nrn894>
- Creswell J.D., Pacilio L.E., Lindsay E.K., & Brown K.W. (2014). Brief mindfulness meditation training alters psychological and neuroendocrine responses to social evaluative stress. *Psychoneuroendocrinology*, 44, 1-12. <https://doi.org/10.1016/j.psyneuen.2014.02.007>
- Critchley, H.D., & Harrison, N.A. (2013). Visceral influences on brain and behavior. *Neuron*, 77(4), 624-638. <https://doi.org/10.1016/j.neuron.2013.02.008>
- Crovetto F., Nakaki A., Arranz A., Borrás R., Vellvé K., Paules C., & Gratacós E. (2023). Effect of a Mediterranean diet or mindfulness-based stress reduction during pregnancy on child neurodevelopment: a prespecified analysis of the IMPACT BCN randomized clinical trial. *JAMA Network Open*, 6(8), 2330255. <https://doi.org/10.1001/jamanetworkopen.2023.30255>
- Cryan J.F., O'Riordan K.J., Cowan C.S., Sandhu K.V., Bastiaanssen T.F., Boehme M., & Dinan T.G. (2019). The microbiota-gut-brain axis. *Physiological Reviews*, 99(4), 1877-2013. <https://doi.org/10.1152/physrev.00018.2018>
- Davidson R.J., & McEwen B.S. (2012). Social influences on neuroplasticity: stress and interventions to promote well-being. *Nature Neuroscience*, 15(5), 689-695. <https://doi.org/10.1038/nn.3093>
- Desbordes G., Negi L.T., Pace T.W., Wallace B.A., Raison C.L., & Schwartz E.L. (2012). Effects of mindful-attention and compassion meditation training on amygdala response to emotional stimuli in an ordinary, non-meditative state. *Frontiers in Human Neuroscience*, 6, 292. <https://doi.org/10.3389/fnhum.2012.00292>
- Dinan T.G., & Cryan J.F. (2017). The microbiome-gut-brain axis in health and disease. *Gastroenterology Clinics*, 46(1), 77-89. <https://doi.org/10.1016/j.gtc.2016.09.007>
- DiPietro J.A., Caulfield L.E., Irizarry R.A., Chen P., Merialdi M., & Zavaleta N. (2006). Prenatal development of intrafetal and maternal-fetal synchrony. *Behavioral Neuroscience*, 120(3), 687-701. <https://doi.org/10.1037/0735-7044.120.3.687>
- Duncan L.G., & Bardacke N. (2010). Mindfulness-based childbirth and parenting education: promoting family mindfulness during the perinatal period. *Journal of Child and Family Studies*, 19(2), 190-202. <https://doi.org/10.1007/s10826-009-9313-7>
- Dunn T.J., & Dimolareva M. (2022). The effect of mindfulness-based interventions on immunity-related biomarkers: a comprehensive meta-analysis of randomised controlled trials. *Clinical Psychology Review*, 92, 102124. <https://doi.org/10.1016/j.cpr.2022.102124>
- Findik E., Sezer N.Y., Aker M.N., & Badur D. (2025). The effects of the Mindfulness-Based Childbirth and Parenting Program (MBCP) on prenatal attachment, depression, stress, and anxiety in pregnant women: A randomized controlled trial. *Journal of Affective Disorders*, 376, 341-346. <https://doi.org/10.1016/j.jad.2025.02.030>
- Gheibi Z., Abbaspour Z., Haghighizadeh M.H., & Javadifar N. (2020). Effects of a mindfulness-based childbirth and parenting program on maternal-fetal attachment: a randomized controlled trial among Iranian pregnant women. *Complementary Therapies in Clinical Practice*, 41, 101226. <https://doi.org/10.1016/j.ctcp.2020.101226>

- Glover V. (2014).** Maternal depression, anxiety and stress during pregnancy and child outcome; what needs to be done. *Best Practice & Research Clinical Obstetrics & Gynaecology*, 28(1), 25–35. <https://doi.org/10.1016/j.bpobgyn.2013.08.017>
- Gluckman P.D., & Hanson M. (2005).** *The fetal matrix: evolution, development, and disease*. Cambridge, United Kingdom.
- Gluckman P.D., Hanson M.A., Cooper C., & Thornburg K.L. (2008).** Effect of in utero and early-life conditions on adult health and disease. *New England Journal of Medicine*, 359(1), 61–73. <https://doi.org/10.1056/NEJMra0708473>
- Gluckman P.D., Hanson M.A., & Low F.M. (2011).** The role of developmental plasticity and epigenetics in human health. *Birth Defects Research Part C: Embryo Today: Reviews*, 93(1), 12–18. <https://doi.org/10.1002/bdrc.20198>
- Gomez Y., Nakaki A., & Conti A. (2025).** Mindfulness-based stress reduction intervention during pregnancy changes maternal brain. *Scientific Reports*, 15, 21929. <https://doi.org/10.1038/s41598-025-07787-9>
- Hall H.G., Beattie J., Lau R., East C., & Biro M.A. (2016).** Mindfulness and perinatal mental health: a systematic review. *Women and Birth*, 29(1), 62–71. <https://doi.org/10.1016/j.wombi.2015.08.006>
- Hodyl N.A., Aboustate N., Bianco-Miotto T., Roberts C.T., Clifton V.L., & Stark M.J. (2017).** Child neurodevelopmental outcomes following preterm and term birth: What can the placenta tell us? *Placenta*, 57, 79–86. <https://doi.org/10.1016/j.placenta.2017.06.009>
- Hsiao E.Y., McBride S.W., Hsien S., Sharon G., Hyde E.R., McCue T., & Mazmanian S.K. (2013).** Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. *Cell*, 155(7), 1451–1463. <https://doi.org/10.1016/j.cell.2013.11.024>
- Kabat-Zinn J. (2003).** Mindfulness-based interventions in context: Past, present, and future. *Clinical Psychology: Science and Practice*, 10(2), 144–156. <https://doi.org/10.1093/clipsy.bpg016>
- Kaliman P., Alvarez-Lopez M.J., Cosín-Tomás M., Rosenkranz M.A., Lutz A., & Davidson R.J. (2014).** Rapid changes in histone deacetylases and inflammatory gene expression in expert meditators. *Psychoneuroendocrinology*, 40, 96–107. <https://doi.org/10.1016/j.psychneuen.2013.11.004>
- Khan S., Shehri F.A., Thalib H.I., Abbas O.S., Khan A., Mahmood A.M., & Hassan F.E. (2025).** Effect of maternal stress on the risk of development of autoimmune Guillain-Barré syndrome in children: a narrative review. *Pediatric Medicine*, 8. <https://doi.org/10.21037/pm-25-18>
- Kramar M. (2025).** The applications of the mindfulness-based cognitive therapy and HRV biofeedback in modern psychotherapy. *NeuroRegulation*, 12(2), 98–98. <https://doi.org/10.15540/nr.12.2.98>
- Kramer A.C., Jansson T., Bale T.L., & Powell T.L. (2023).** Maternal-fetal cross-talk via the placenta: influence on offspring development and metabolism. *Development (Cambridge, England)*, 150(20), 202088. <https://doi.org/10.1242/dev.202088>
- Lautarescu A., Hadaya L., Craig M.C., Makropoulos A., Batalle D., Nosarti C., & Victor S. (2021).** Exploring the relationship between maternal prenatal stress and brain structure in premature neonates. *PLoS One*, 16(4), 0250413. <https://doi.org/10.1371/journal.pone.0250413>
- Lehrer P.M., & Gevirtz R. (2014).** Heart rate variability biofeedback: how and why does it work? *Frontiers in Psychology*, 5, 756. <https://doi.org/10.3389/fpsyg.2014.00756>
- Lester B.M., & Marsit C.J. (2018).** Epigenetic mechanisms in the placenta related to infant neurodevelopment. *Epigenomics*, 10(3), 321–333. <https://doi.org/10.2217/epi-2016-0171>
- Marlin B.J., & Froemke R.C. (2017).** Oxytocin modulation of neural circuits for social behavior. *Developmental Neurobiology*, 77(2), 169–189. <https://doi.org/10.1002/dneu.22452>
- McEwen B.S. (1998).** Protective and damaging effects of stress mediators. *New England Journal of Medicine*. <https://doi.org/10.1056/NEJM199801153380307>
- McEwen B.S. (2007).** Physiology and neurobiology of stress and adaptation. *Physiological Reviews*. <https://doi.org/10.1152/physrev.00041.2006>
- McGlone F., Wessberg J., & Olsson H. (2014).** Discriminative and affective touch: sensing and feeling. *Neuron*, 82(4), 737–755. <https://doi.org/10.1016/j.neuron.2014.05.001>

- McGlone F., & Rankin L. (2025).** A reappraisal of attachment theory: The role of affective touch. *Infant Behavior and Development*, 79, 102053. <https://doi.org/10.1016/j.infbeh.2025.102053>
- Meaney M.J., & Szyf M. (2005).** Environmental programming of stress responses through DNA methylation: life at the interface between a dynamic environment and a fixed genome. *Dialogues in Clinical Neuroscience*, 7(2), 103–123. <https://doi.org/10.31887/DCNS.2005.7.2/mmeaney>
- Meaney M.J. (2010).** Epigenetics and the biological definition of gene × environment interactions. *Child Development*, 81(1), 41–79. <https://doi.org/10.1111/j.1467-8624.2009.01381.x>
- Menon V., & Uddin L.Q. (2010).** Saliency, switching, attention and control: a network model of insula function. *Brain Structure and Function*, 214(5), 655–667. <https://doi.org/10.1007/s00429-010-0262-0>
- Menon V. (2011).** Large-scale brain networks and psychopathology: a unifying triple network model. *Trends in Cognitive Sciences*, 15(10), 483–506. <https://doi.org/10.1016/j.tics.2011.08.003>
- Menon V., Palaniyappan L., & Supekar K. (2023).** Integrative brain network and salience models of psychopathology and cognitive dysfunction in schizophrenia. *Biological Psychiatry*, 94(2), 108–120. <https://doi.org/10.1016/j.biopsych.2022.09.029>
- Min W., Jiang C., Li Z., & Wang Z. (2023).** The effect of mindfulness-based interventions during pregnancy on postpartum mental health: a meta-analysis. *Journal of Affective Disorders*, 331, 452–460. <https://doi.org/10.1016/j.jad.2023.03.053>
- Monk C., Lugo-Candelas C., & Trumppff C. (2019).** Prenatal developmental origins of future psychopathology: mechanisms and pathways. *Annual Review of Clinical Psychology*, 15(1), 317–344. <https://doi.org/10.1146/annurev-clinpsy-050718-095539>
- Moore L.D., Le T., & Fan G. (2013).** DNA methylation and its basic function. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, 38(1), 23–38. <https://doi.org/10.1038/npp.2012.112>
- Nakaki A., Crovetto F., Urru A., Piella G., Borrás R., Comte V., & Gratacós E. (2023).** Effects of Mediterranean diet or mindfulness-based stress reduction on fetal and neonatal brain development: a secondary analysis of a randomized clinical trial. *American Journal of Obstetrics & Gynecology MFM*, 5(12), 101188. <https://doi.org/10.1016/j.ajogmf.2023.101188>
- Natarajan A. (2023).** Heart rate variability during mindful breathing meditation. *Frontiers in Physiology*, 13, 1017350. <https://doi.org/10.3389/fphys.2022.1017350>
- Noroña-Zhou A.N., Coccia M., Epel E., Vieten C., Adler N.E., Laraia B., & Bush N.R. (2022).** The effects of a prenatal mindfulness intervention on infant autonomic and behavioral reactivity and regulation. *Biopsychosocial Science and Medicine*, 84(5), 525–535. <https://doi.org/10.1097/psy.0000000000001066>
- Notaras M., & Buuse M. (2020).** Neurobiology of BDNF in fear memory, sensitivity to stress, and stress-related disorders. *Molecular Psychiatry*, 25(10), 2251–2274. <https://doi.org/10.1038/s41380-019-0639-2>
- O'Connor T.G., Monk C., & Fitelson E.M. (2014).** Practitioner review: maternal mood in pregnancy and child development – implications for child psychology and psychiatry. *Journal of Child Psychology and Psychiatry*, 55(2), 99–111. <https://doi.org/10.1111/jcpp.12153>
- O'Donnell K.J., & Meaney M.J. (2017).** Fetal origins of mental health: the developmental origins of health and disease hypothesis. *American Journal of Psychiatry*, 174(4), 319–328. <https://doi.org/10.1176/appi.ajp.2016.16020138>
- Pavlov V.A., & Tracey K.J. (2005).** The cholinergic anti-inflammatory pathway. *Brain, Behavior, and Immunity*, 19(6), 493–499. <https://doi.org/10.1016/j.bbi.2005.03.015>
- Pinto T.M., Nogueira-Silva C., & Figueiredo B. (2025).** Fetal heart rate variability and infant self-regulation: The impact of mother's prenatal depressive symptoms. *Journal of Reproductive and Infant Psychology*, 43(3), 588–601. <https://doi.org/10.1080/02646838.2023.2257730>
- Porges S.W. (2022).** Polyvagal theory: A science of safety. *Frontiers in Integrative Neuroscience*, 16, 871227. <https://doi.org/10.3389/fnint.2022.871227>
- Rafter C., McCarthy E., Stilp C., & Brumitt J. (2026).** Mindfulness practice and increases in gray matter density, gray matter volume, and cortical thickness: A scoping review. *Brain Sciences*, 16(5), 483.

<https://doi.org/10.3390/brainsci16050483>

- Ragusa A., Re A.L., Segata M., Rinaldo D., Motta G., Vito M., & Svelato A. (2025).** The placenta and the development of human health. In: *Epigenetics and rare diseases* (pp. 57–75). Cham: Springer Nature Switzerland.
- Reynolds R.M. (2013).** Programming effects of glucocorticoids. *Clinical Obstetrics and Gynecology*, 56(3), 602–609. <https://doi.org/10.1097/GRF.0b013e31829939f7>
- Sandman C.A., Buss C., Head K., & Davis E.P. (2015).** Fetal exposure to maternal depressive symptoms is associated with cortical thickness in late childhood. *Biological Psychiatry*, 77(4), 324–334. <https://doi.org/10.1016/j.biopsych.2014.06.025>
- Sandman C.A., Curran M.M., Davis E.P., Glynn L.M., Head K., & Baram T.Z. (2018).** Cortical thinning and neuropsychiatric outcomes in children exposed to prenatal adversity: a role for placental CRH? *American Journal of Psychiatry*, 175(5), 471–479. <https://doi.org/10.1176/appi.ajp.2017.16121433>
- Seckl J.R., & Holmes M.C. (2007).** Mechanisms of disease: glucocorticoids, their placental metabolism and fetal “programming” of adult pathophysiology. *Nature Clinical Practice Endocrinology & Metabolism*, 3(6), 479–488. <https://doi.org/10.1038/ncpendmet0515>
- Shaffer F., & Ginsberg J.P. (2017).** An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, 5, 290215. <https://doi.org/10.3389/fpubh.2017.00258>
- Sharifiheris Z., Rahmani A., Onwuka J., & Bender M. (2022).** The utilization of heart rate variability for autonomic nervous system assessment in healthy pregnant women: systematic review. *JMIR Bioinformatics and Biotechnology*, 3(1), 36791. <https://doi.org/10.2196/36791>
- Slavich G.M., & Irwin M.R. (2014).** From stress to inflammation and major depressive disorder: a social signal transduction theory of depression. *Psychological Bulletin*, 140(3), 774. <https://doi.org/10.1037/a0035302>
- Tang Y.Y., Hölzel B.K., & Posner M.I. (2015).** The neuroscience of mindfulness meditation. *Nature Reviews Neuroscience*, 16(4), 213–225. <https://doi.org/10.1038/nrn3916>
- Taren A.A., Gianaros P.J., Greco C.M., Lindsay E.K., Fairgrieve A., Brown K.W., & Creswell J.D. (2015).** Mindfulness meditation training alters stress-related amygdala resting state functional connectivity: a randomized controlled trial. *Social Cognitive and Affective Neuroscience*, 10(12), 1758–1768. <https://doi.org/10.1093/scan/nsv066>
- Thakur M., Patil Y., Philip S.T., Hamdule T., Thimmapuram J., Vyas N., & Thakur K. (2023).** Impact of Heartfulness meditation practice on anxiety, perceived stress, well-being, and telomere length. *Frontiers in Psychology*, 14, 1158760. <https://doi.org/10.3389/fpsyg.2023.1158760>
- Thayer J.F., & Lane R.D. (2009).** Claude Bernard and the heart–brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience & Biobehavioral Reviews*, 33(2), 81–88. <https://doi.org/10.1016/j.neubiorev.2008.08.004>
- Thayer J.F., Åhs F., Fredrikson M., Sollers J.J., & Wager T.D. (2012).** A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews*, 36(2), 747–756. <https://doi.org/10.1016/j.neubiorev.2011.11.009>
- Thayer J.F. (2020).** Heart rate variability. In: *Encyclopedia of behavioral medicine* (pp. 1048–1049). Cham: Springer International Publishing.
- Thayer J. (2025).** Bidirectional communication between the heart and the brain: importance of vagal afferents. *Psychophysiology*, 62.
- Thomason M.E., & Hendrix C.L. (2024).** Prenatal stress and maternal role in neurodevelopment. *Annual Review of Developmental Psychology*, 6(1), 87–107. <https://doi.org/10.1146/annurev-devpsych-120321-011905>
- Townshend K., Caltabiano N.J., Powrie R., & O’Grady H. (2018).** A preliminary study investigating the effectiveness of the Caring for Body and Mind in Pregnancy (CBMP) in reducing perinatal depression, anxiety and stress. *Journal of Child and Family Studies*, 27(5), 1556–1566. <https://doi.org/10.1007/s10826-017-0978-z>
- Tracey K.J. (2002).** The inflammatory reflex. *Nature*, 420, 853–859. <https://doi.org/10.1038/nature01321>
- Trapani S., Caglioni M., Villa G., Manara D.F., & Caruso R. (2024).** Mindfulness-based interventions

- during pregnancy and long-term effects on postpartum depression and maternal mental health: a systematic review and meta-analysis of randomized controlled trials. *Journal of Integrative and Complementary Medicine*, 30(2), 107–120. <https://doi.org/10.1089/jicm.2023.0114>
- Turecki G., & Meaney M.J. (2016).** Effects of the social environment and stress on glucocorticoid receptor gene methylation: a systematic review. *Biological Psychiatry*, 79(2), 87–96. <https://doi.org/10.1016/j.biopsych.2014.11.022>
- Valenza G., Sclocco R., Duggento A., Passamonti L., Napadow V., Barbieri R., & Toschi N. (2019).** The central autonomic network at rest: Uncovering functional MRI correlates of time-varying autonomic outflow. *Neuroimage*, 197, 383–390. <https://doi.org/10.1016/j.neuroimage.2019.04.075>
- Van den Bergh B.R., Mulder E.J., Mennes M., & Glover V. (2005).** Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: links and possible mechanisms. A review. *Neuroscience & Biobehavioral Reviews*, 29(2), 237–258. <https://doi.org/10.1016/j.neubiorev.2004.10.007>
- Van den Bergh B.R. (2020).** Prenatal developmental origins of early brain and behavior development, of self-regulation in adolescence, and of cognition and central and autonomic nervous system function in adulthood. In: *Handbook of prenatal and perinatal psychology: Integrating research and practice* (pp. 83–113). Cham: Springer International Publishing.
- Van den Heuvel M.I., Johannes M.A., Henrichs J., & Bergh B.R.H. (2015).** Maternal mindfulness during pregnancy and infant socio-emotional development and temperament: The mediating role of maternal anxiety. *Early Human Development*, 91(2), 103–108. <https://doi.org/10.1016/j.earlhumdev.2014.12.003>
- Venditti S. (2025).** Remodeling the epigenome through meditation: Effects on brain, body, and well-being. In: *Neuroepigenetics mechanisms in health and disease* (pp. 231–260). Cham: Springer Nature
- Venditti S., Verdone L., Reale A., Vetriani V., Caserta M., & Zampieri M. (2020).** Molecules of silence: Effects of meditation on gene expression and epigenetics. *Frontiers in Psychology*, 11, 544346. <https://doi.org/10.3389/fpsyg.2020.01767>
- Walker S.C., Trotter P.D., Swaney W.T., Marshall A., & McGlone F.P. (2017).** C-tactile afferents: Cutaneous mediators of oxytocin release during affiliative tactile interactions? *Neuropeptides*, 64, 27–38. <https://doi.org/10.1016/j.npep.2017.01.001>
- Walton K.G., Wenuganen S., & Cole S.W. (2023).** Transcendental meditation practitioners show reduced expression of the conserved transcriptional response to adversity. *Brain, Behavior, & Immunity-Health*, 32, 100672. <https://doi.org/10.1016/j.bbih.2023.100672>
- Widatalla N., Alkhodari M., Koide K., Yoshida C., Kasahara Y., Saito M., & Khandoker A. (2025).** *Pattern-based assessment of the association of fetal heart variability with fetal development and maternal heart rate variability*. IEEE Access.
- Woody C.A., Ferrari A.J., Siskind D.J., Whiteford H.A., & Harris M.G. (2017).** A systematic review and meta-regression of the prevalence and incidence of perinatal depression. *Journal of Affective Disorders*, 219, 86–92. <https://doi.org/10.1016/j.jad.2017.05.003>