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The Impact Of Intrauterine Experiences On Psychosomatic Reactions In Adulthood

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Abstract: The article presents an analysis of contemporary scientific approaches to understanding the impact of intrauterine experiences on the development of psychosomatic reactions in adulthood. The study is based on an interdisciplinary methodology that integrates neurobiological, epigenetic, and psychodynamic concepts of fetal programming. Particular attention is given to the analysis of structural and functional brain changes, epigenetic markers of stress reactivity (NR3C1, SLC6A4), and the role of metabolic disturbances identified in newborns exposed to prenatal distress. The mechanisms of unconscious somatic memory formation and its manifestation as persistent psychosomatic symptoms are examined. The paper systematizes neurophysiological and epigenetic effects with consideration of sex differences, the nature of maternal distress, and the quality of the postnatal environment. The limitations of universal fetal programming models are highlighted, particularly in relation to cultural-geographical, genetic, and environmental variables. The article substantiates the need to move from monofactorial to integrative models that combine insights from neuroscience, psychoanalysis, and social epidemiology. The theoretical analysis is accompanied by an original interpretation of symptoms as a form of unconscious reconstruction of prenatal affective bonds, reflected in the structure of somatic memory and cognitive-emotional regulation. This article will be of interest to professionals in psychosomatic medicine, perinatal psychology, epigenetics, and clinical neuroscience, as well as

practicing psychotherapists working with somatic symptoms of unclear origin.

Keywords: Intrauterine stress, psychosomatics, epigenetics, neurodevelopment, NR3C1, somatic memory, prenatal programming, hippocampus, psychoemotional development, psychodynamic model.

Introduction

Contemporary research in psychosomatics, epigenetics, and perinatal psychology provides increasingly compelling evidence that intrauterine experience exerts long-term effects on an individual's bodily and emotional regulation. Within the Developmental Origins of Health and Disease (DOHaD) paradigm, it has been established that prenatal exposures—particularly maternal emotional and stress states—can alter the development of key fetal brain structures and lay the groundwork for subsequent somatic and behavioral responses in adult life [1].

At the same time, prenatal psychology posits that a mother's intrauterine experiences are encoded in the fetus's neurophysiological parameters and embodied memory. A woman's psychoemotional states, her beliefs, bodily suffering, and characteristic stress reactions become imprinted in the child as unconscious patterns of somatic response. This process generates a hidden map of bodily blockages, muscular tensions, and emotional connections that may reemerge in adult life.

A number of neuroscientific studies—including imaging and morphometric analyses of newborn brains—indicate that elevated maternal anxiety and stress correlate with reduced volumes in regions such as the hippocampus and amygdala in the fetus, as well as disrupted myelination of white matter [8]. These alterations serve as neurodevelopmental markers and, longitudinal observations show, are associated with an increased propensity for anxiety, depressive, and somatoform disorders in adulthood [9].

Psychodynamic and body-oriented approaches extend the interpretation of these findings, asserting that a mother's intrauterine emotional states can be preserved in cognitive memory, the neurosensory matrix, and somatic response patterns—manifested in muscle tone, breathing, heart rate, and visceral sensitivity. In later life, such unconscious patterns may be spontaneously reactivated in response to stress, loss, anxiety, or bodily intimacy, thereby reenacting the intrauterine connection as a means of maintaining an internal affective bond with the maternal image [6].

In the context of global crises such as the COVID-19 pandemic, the level of psychological distress among pregnant women rose sharply, allowing large-scale studies to demonstrate how transient, situational stressors affect fetal neurodevelopment—even in the absence of direct infection [2]. Structural brain changes observed in the neonatal period may thus serve as early biomarkers of potential psychosomatic disturbances.

An adult individual may, without conscious awareness, reenact the mother's intrauterine experiences on both bodily and emotional levels, unconsciously maintaining an affective bond with her. Such reenactment can manifest as recurring patterns of suffering, disrupted self-regulation, and somatic symptoms of unclear origin. Against this backdrop, particular interest attaches to work with bodily memory aimed at the safe release of unconscious prenatal material.

In body-oriented approaches, practices are described that integrate and process early pain. One such method is standing on nails under therapist supervision—a technique that activates the bodily and emotional traces of prenatal experience. This method can be understood as a form of conscious living and transformation of intrauterine blocks. Additional theoretical support comes from the concept presented in the book The Matrix of Life, where bodily symptomatology is construed as a carrier of primary affective bonds.

Aim of the study – To analyze contemporary scientific approaches to understanding the influence of intrauterine experiences on the formation of psychosomatic reactions in adulthood, with an emphasis on the neuropsychological, epigenetic, and psychodynamic mechanisms of unconscious bodily memory.

Materials and Methods

This study is grounded in a content analysis of peer-reviewed scientific publications. Sources were selected based on their inclusion of empirically validated data and conceptual models describing how intrauterine stress and maternal emotional states influence the later development of psychosomatic reactions in adulthood. Priority was given to works detailing neuropsychological, epigenetic, and psychodynamic mechanisms underpinning the long-term impact of prenatal experiences.

The research methodology adheres to principles of substantive decomposition and interdisciplinary integration. During the literature review, we examined key concepts such as prenatal programming, psychosomatic memory, maternal–fetal signaling, and intergenerational transmission of emotional experience. These notions were explored within three principal scientific domains represented across the selected sources:

- **Biomedical;**
- **Neurocognitive;**
- **Psychoanalytic.**

Special attention was devoted to the biological pathways outlined in the literature—the hypothalamic–pituitary–adrenal (HPA) axis, inflammatory processes, and epigenetic mechanisms, including methylation of stress-related genes [3].

Among the pivotal contributions are Bush [1], who proposed an expanded model of prenatal programming, and Coussons-Read [2], who elucidated a dual (direct and indirect) pathway whereby prenatal stress shapes fetal development and subsequent psychosomatic outcomes. Lund's study [3] employed a triangulation design to assess causal links between intrauterine exposure and behavioral outcomes in childhood and adulthood. Musillo [4] offered a conceptual framework integrating neuroendocrine and metabolic pathways in the emergence of stable neurobehavioral risks.

Equally significant are the findings of Nolvi [5], which analyze the moderating role of the postnatal environment in buffering adverse prenatal influences; this work also addresses epigenetic modifications of glucocorticoid-receptor expression. Wall [7] highlights cultural and geographic variations in assessing psychological trauma during pregnancy and underscores the need for culturally adapted diagnostic tools.

Thus, the article's methodological strategy rests on a systemic, theoretical-analytical approach, enabling a robust interpretation of contemporary insights into prenatal programming and its effects on adult psychosomatic health, viewed through an interdisciplinary lens.

Results

Contemporary neuroimaging and longitudinal data provide strong evidence of enduring alterations in brain morphology and functional organization among children and adults exposed to prenatal stress. The literature converges on the view that intrauterine distress impacts the development of key regions such as the hippocampus, cerebellum, amygdala, and frontal cortex [9]. These alterations may remain latent at birth but emerge prominently during sensitive periods of cognitive and emotional maturation.

One of the most extensively documented effects is reduced hippocampal volume in children and adolescents who experienced prenatal stress. Wu et al. [9], using MRI, demonstrated that diminished hippocampal volume correlates with elevated anxiety levels, memory impairments, and learning difficulties in adolescence. Notably, Peterson et al. [6] observed more pronounced hippocampal changes in females, suggesting a sex-specific vulnerability in neuroplasticity. Functional shifts are equally significant: increased connectivity between the amygdala and prefrontal cortex appears as early as the first months of life and is interpreted as a marker of heightened emotional reactivity [2]. Wu et al. [9] further report that infants with high prenatal distress exhibit hyperconnectivity between the amygdala and ventromedial prefrontal cortex, potentially undermining the efficiency of neural circuits responsible for emotion regulation. These connectivity patterns persist into adulthood—especially among women—and predispose to affective disorders and impulse-control challenges.

Alterations in cortical morphology have also been identified, including an elevated gyrification index. Although the functional implications of these structural variations remain under investigation, they have been linked to self-regulation deficits—including ADHD-like symptoms—by Musillo et al. [4]. Of particular note is the reduction in cerebellar volume: traditionally associated with motor coordination, the cerebellum also contributes to cognitive and emotional regulation. Peterson et al. [6] confirm that prenatal stress can induce diffuse cerebellar changes, leading to motor dysfunction, learning difficulties, and persistent attentional impairments.

Crucially, these neural effects are not uniform but vary according to sex, the timing of gestational exposure, and the quality of the postnatal environment. Wu et al. [9] found that females exhibit more marked neuroimaging responses to prenatal stress, whereas males more often manifest behavioral deviations in the absence of clear morphologic correlates. Table 1 systematizes the

principal neuromorphological and functional consequences of prenatal stress on the human brain, highlighting sex differences and long-term outcomes.

Table 1 – Key neuromorphological and functional changes in the brain following prenatal stress (Compiled by the author based on sources: [9], [6], [4])

Parameter	Effect	Sex Differences	Adult Consequences
Hippocampal volume	Decreased	Greater sensitivity in females	Memory impairments; anxiety
Amygdala connectivity	Increased	↑ in girls; ↓ in boys	Emotional hyperreactivity
Gyrification index	Increased	No significant difference	Self-regulation impairments
Cerebellar volume	Decreased	Both sexes affected	Motor disturbances; ADHD symptoms

This overview illustrates how even moderate prenatal distress can establish the groundwork for cognitive and emotional dysregulation that manifests in adulthood. These findings underscore the imperative to integrate neuropreventive strategies within obstetric and psychiatric practice.

Theoretical analysis of recent research confirms that the effects of prenatal distress emerge both as neuromorphological alterations and as durable epigenetic modifications that embody the organism's molecular "memory" of intrauterine experiences. These changes reshape the expression of genes governing stress-axis reactivity and the subsequent development of emotional and somatic responses in later life [8].

One pivotal marker is methylation of the NR3C1 gene, which encodes the glucocorticoid receptor. Bush [1] demonstrated that elevated prenatal stress reduces methylation in the NR3C1 promoter region, thereby heightening the body's sensitivity to cortisol. Such dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis can precipitate anxiety, sleep disturbances, and increased somatic reactivity.

Peterson [6] highlights another critical locus: the SLC6A4 gene, responsible for serotonin transport. Infants and adolescents exposed to prenatal stress exhibit increased methylation of SLC6A4—a change linked to serotonergic imbalance and a greater risk of depression and somatoform disorders. These conclusions echo earlier findings by Coussons-Read [2], who described a comprehensive fetal biochemical response to stress, characterized by altered neurotransmitter levels and hormonal regulators.

Wu's work [9] made a significant contribution to understanding metabolic alterations, demonstrating reduced levels of choline and creatine in fetuses exposed to prenatal distress. These metabolites play critical roles in neuroplasticity and energy metabolism, and their deficiency may increase the risk of cognitive and somatic impairments, including chronic fatigue, autonomic dysfunction, and emotional instability. Lund's multimodal study [3] links epigenetic markers to behavioral and physiological outcomes in preschool-aged children—such as adaptation difficulties and somatic complaints. Table 2 presents the principal epigenetic and biochemical markers described in the theoretical literature, along with their functional consequences and potential associations with adult psychosomatic manifestations.

Table 2 – Epigenetic and Metabolic Consequences of Prenatal Stress (Compiled by the author based on sources: [1], [3], [5], [9])

Biomarker / Gene	Change	Consequence	Link to Psychosomatics
NR3C1 (glucocorticoid receptor)	Hypomethylation	Dysregulation of the HPA axis	Heightened anxiety; somatization
SLC6A4 (serotonin transporter)	Hypermethylation	Reduced serotonin expression	Emotional dysregulation; depressive symptoms
Choline & Creatine	Decreased levels	Impaired neuroplasticity	Cognitive deficits; autonomic instability

The epigenetic and biochemical alterations summarized in Table 2 position prenatal distress as an early molecular programming factor for psychosomatic vulnerability. These markers bear the biological imprint of intrauterine experience and form a foundation for future predictive–diagnostic models and early interventions. Importantly, their influence is not deterministic: several authors highlight the buffering potential of the postnatal environment—particularly the quality of maternal care and social support—to alter developmental trajectories even in the presence of epigenetic disruption.

Beyond neurophysiological and epigenetic indicators, bodily markers that preserve memory of intrauterine experience are gaining prominence. Clinically, persistent muscular patterns, restricted breathing, and autonomic dysfunctions—unaccounted for by current circumstances but correlating with maternal prenatal history—are increasingly observed. These manifestations may be regarded as somatic anchors of intrauterine experiences "imprinted" in the body, especially when pregnancy was accompanied by emotional instability, somatic illness, or stress.

Discussion

Contemporary concepts in psychosomatic medicine increasingly acknowledge the significance of intrauterine experience as the foundation for bodily memory and emotional patterns that persist into adulthood. One of the key theoretical frameworks in this context is the "funnel model" proposed by Musillo [4]. It posits that psychological or metabolic distress in the pregnant woman activates multiple neuroendocrine and immune transmission channels, which imprint on the fetal body as an unconscious "record." These imprints are far from neutral—they condense into the body as a kind of matrix capable of resurfacing as psychosomatic reactions later in life.

According to Musillo's interpretation, an adult's somatic symptoms may carry both biological and symbolic functions, acting as an unconscious continuation of the prenatal bond with the mother. Such reactions perform an "emotional reconstruction" of interrupted or distorted prenatal closeness. As demonstrated by Wu [9] and Peterson [6], children exposed to prenatal stress develop enduring alterations in limbic structures—most notably, amygdala hyperactivation and reduced hippocampal volume.

Review of the literature suggests that recurrent psychosomatic scenarios in adults with no overt organic pathology can be understood as somatized forms of an unconscious holding of the maternal connection established in utero. Specific maternal experiences—such as chronic anxiety, depression, or emotional ambivalence—leave distinct neurophysiological traces in the developing fetal brain, laying the groundwork for characteristic somatic responses in later life stages. Table 3 illustrates the relationship between the nature of prenatal emotional exposure, its neurobiological correlate, and the corresponding adult psychosomatic pattern.

Table 3 – Connection between intrauterine experience and recurring psychosomatic scenarios in adult life (Compiled by the author based on sources: [4], [9], [6])

Maternal experience type	Neurobiological footprint	Adult psychosomatic manifestation
Chronic anxiety	Amygdala–PFC hyperconnectivity	Panic attacks; spastic colitis
Depression and alienation	Reduced hippocampal volume	Sleep disturbances; feelings of dissociation
Maternal ambivalence	Functional imbalance in the DMN	Psychogenic pain; episodes of derealization

As Table 3 demonstrates, adult psychosomatic reactions may be interpreted both as responses to current stressors and as expressions of deep-seated unconscious processes rooted in the prenatal phase. This approach allows for a reappraisal of persistent somatic symptoms by integrating neurobiological data and psychoanalytic models into a unified explanatory framework. In particular, it underscores the body's capacity to "carry" relationships that have lost their tangible referent in the external world, continuing to enact them at a somatic level.

Despite the growing body of empirical evidence confirming the impact of intrauterine experiences on psychosomatic health, interpreting these links remains methodologically and conceptually challenging. One of the central problems highlighted in contemporary research is the difficulty of isolating the genuine effect of prenatal exposure from a host of competing or overlapping factors. For example, Lund et al. [3] emphasize the need for a triangulation approach—combining multiple methodological strategies (such as genetic controls, sibling-comparison designs, and instrumental-variable analyses)—to disentangle intrauterine influences from genetic and family-social variables. Their findings indicate that many observed associations between maternal stress during pregnancy and subsequent psychosomatic disturbances in the child can be partially or entirely attributed to shared genetic predispositions and stable environmental characteristics.

An additional limitation to the universality of prenatal-programming models is their sensitivity to social context. As Bush [1] notes, intrauterine exposure does not occur in isolation; it is modulated by a multitude of social, behavioral, and cultural factors, including chronic stress, poverty, lack of social support, and high levels of stigma. Cultural norms around the perception of trauma and symptom reporting also play a crucial role. In her systematic review, Wall [7] demonstrates that the types of prenatal trauma assessed, the diagnostic methods employed, and even the very definition of what constitutes a psychologically significant exposure vary widely by geographic and cultural setting.

In light of these constraints, it is advisable to develop integrative explanatory models rather than mono-factorial ones—models that combine insights from neuroscience, psychoanalysis, and epigenetics. A theoretical perspective grounded in such interdisciplinary linkage allows for a more realistic reflection of the complex nature of psychosomatic symptom formation, wherein prenatal, genetic, and postnatal influences function as interconnected and mutually reinforcing components. This broader analytical framework does not diminish the importance of intrauterine factors but rather refines the boundaries of their influence and mitigates the risk of overly deterministic interpretations.

Given the described patterns of bodily encoding of prenatal experience, methods that work directly with the body as a repository of unconscious memory

become especially pertinent. One such approach is the practice of standing on nails under therapeutic supervision, which can activate deep layers of prenatal material and provoke spontaneous affective and somatic releases. This process enables decompression of internal tensions, followed by conscious recognition and release of unconscious maternal scenarios. In this context, the framework presented in the book The Matrix of Life, which conceptualizes bodily symptoms as archetypal traces of early relational patterns and survival mechanisms, offers a compelling theoretical foundation. Such an interpretation opens new avenues for somato-emotional reprocessing and therapeutic integration of intrauterine experience.

Conclusion

The present study revealed that intrauterine experience—most notably the mother's psychoemotional state during pregnancy—shapes the development of brain structures and functions and establishes neuropsychological and somatic patterns that persist into adulthood. This influence unfolds not as a straightforward causal chain but as a complex network of reciprocal modifications spanning molecular, neurosensory, and affective-symbolic levels.

It was demonstrated that an adult's psychosomatic symptomatology can represent more than a mere aftermath of prenatal distress: it may constitute its unconscious re-enactment, whereby the body maintains an emotional tether to the maternal image. In this way, the bodily symptom transcends a purely "functional" role and becomes a vessel for biographical—and in some instances, transgenerational—information.

Conceptual analysis exposed the shortcomings of reductionist models that confine psychosomatic phenomena to discrete biomarkers or morphofunctional measures. Instead, it underscores the value of integrative explanatory frameworks capable of capturing the interplay among genetic predispositions, intrauterine signaling mechanisms, postnatal caregiving quality, and sociocultural modes of symptom interpretation.

Particular attention was drawn to observed sex differences in neuro-vulnerability and to how maternal emotional states translate across levels into the child's cognitive and somatic response strategies. These insights argue for a differentiated prevention and intervention approach that attends both to biological sex and to the cultural context of pregnancy.

Accordingly, an adult's psychosomatic vulnerability emerges at the nexus of multiple temporal and modal axes—from prenatal programming and epigenetic markings to the psychodynamic dimensions of symptom expression. This perspective reframes psychosomatic symptoms not as isolated reactions to stress, but as the product of a multilayered neuro-biographical assembly in which intrauterine experiences underpin bodily self-expression, identity, and memory. Future research should aim to develop multilevel diagnostic and therapeutic models that integrate findings from neuroscience, epigenetics, and psychoanalysis into a cohesive clinical-preventive paradigm.

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