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Psychopathological and Neuroendocrine Mechanisms of "Pregorexia": The Impact of Caloric Restriction and Gestational Dysmorphic Disorder on Fetoplacental Homeostasis and Long-Term Health Outcomes in Mother and Child

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Abstract

Introduction. "Pregorexia" is a popular term used to describe restrictive eating behaviors and excessive physical activity during pregnancy aimed at controlling body weight. Although it is not recognized as a formal psychiatric diagnosis, this phenomenon is associated with serious psychosocial and somatic consequences.

Aim of the study. This review aims to synthesize current evidence on the psychopathological and neuroendocrine mechanisms of "pregorexia," as well as its impact on fetoplacental homeostasis and the long-term health outcomes of both mother and child.

Materials and methods. A literature review was conducted using Google Scholar and PubMed databases to analyze publications concerning the effects of maternal stress, malnutrition, and eating disorders - including anorexia nervosa and pregorexia - on the placenta, the fetus, and the mother.

Review of the literature. Chronic caloric restriction leads to disruptions in neuroendocrine axes (such as the HPA and HPT axes) and induces oxidative stress and systemic inflammation. This impairs placental structure and function (including glucose and amino acid transport) and results in intrauterine growth restriction (IUGR). Epigenetic modifications and fetal programming increase the offspring's risk of metabolic disorders (obesity, insulin resistance) and neurodevelopmental conditions (ADHD, autism spectrum disorders) in adult life. In the mother, nutritional deficiencies and stress exacerbate anxiety, postpartum depression, and hinder the mother-infant bonding process.

Conclusions. "Pregorexia" represents a significant yet frequently overlooked clinical issue requiring early diagnosis and multidisciplinary management involving obstetric, psychological, psychiatric, and dietary care.

Key words: Pregorexia, Eating disorders in pregnancy, Caloric restriction, Fetoplacental homeostasis, Fetal programming, Maternal malnutrition, Gestational weight gain, Maternal stress, Anorexia nervosa.

1. Introduction

Eating disorders represent a significant public health concern, affecting individuals across diverse age groups, genders, and sociocultural backgrounds. While traditionally associated with adolescent females, eating disorders are increasingly recognized across a much broader range of populations. Increasing evidence highlights the impact of eating disorders among populations that have historically received less attention, including adult males, older adults, particularly perimenopausal women, and members of the LGBTQIA+ community (1).

This broader recognition also extends to pregnancy, a period characterized by substantial physical and psychological changes that may increase vulnerability to disordered eating behaviors (2). During pregnancy, a pattern of disordered eating commonly referred to as "pregorexia" may emerge. Although "pregorexia" is not recognized as a formal psychiatric diagnosis, the term, derived from the words "pregnancy" and "anorexia", has been used in popular media to describe disordered eating behaviors occurring during pregnancy. It generally refers to restrictive eating behaviors and excessive physical activity aimed at limiting or controlling gestational weight gain, often accompanied by body image disturbance and concerns about the physical changes associated with pregnancy (3). These behaviors contrast with the physiological weight gain expected during pregnancy and with the evidence-based recommendations for gestational weight gain (for detailed recommendations, see Table 1) (4).

Table 1. Recommended gestational weight gain by pre-pregnancy BMI category

Pre-pregnancy BMI category	Underweight (BMI < 18.5)	Normal BMI (18.5 - 24.9)	Overweight (BMI 25 - 29.9)	Obesity (BMI ≥ 30)
Optimal gestational weight gain	12.5 - 18 kg	11.5 - 16.0 kg	7.0 - 11.5 kg	5.0 - 9.0 kg

Source: own elaboration based on (4).

1.1. Psychopathological Background and Mechanisms of "Pregorexia"

Pregnancy-related changes in body weight and shape may heighten concerns about physical appearance and contribute to disturbances in eating behavior. Approximately 7.5% of pregnant women meet diagnostic criteria for eating disorders, while 23.4% report significant concerns regarding body weight and shape (5).

The etiology and course of eating disorders during pregnancy are influenced by multiple interacting factors, including hormonal and physiological changes, body image concerns, psychological distress, a history of eating disorders, and sociocultural pressures. Specifically, women with a history of anorexia nervosa, restrictive dieting, pregnancy-related body dissatisfaction, or greater susceptibility to media influences may be at increased risk of developing disordered eating behaviors during pregnancy (6,7). These factors may also be

associated with neuroendocrine alterations that can influence energy homeostasis and appetite regulation (6).

These interacting biological, psychological, and social factors may contribute to the development and maintenance of "pregorexia" and related disordered eating behaviors. Hormonal and neurotransmitter changes during pregnancy may influence eating behavior, while sociocultural pressures surrounding the "perfect pregnancy body" and minimal weight gain may increase body image distress. Combined with lifestyle changes, these influences may lead to maladaptive coping strategies, including restrictive eating, excessive exercise, emotional eating, or the use of laxatives and diuretics, thereby reinforcing disordered eating patterns and preoccupation with weight and shape (8).

From a classical diathesis–stress perspective, pregnancy may therefore be viewed as a period in which individual vulnerabilities interact with the physical and psychological changes, ultimately triggering or maintaining disordered eating behaviors (9).

This review aims to synthesize current evidence on the psychopathological mechanisms, clinical consequences, diagnostic challenges, and multidisciplinary management of "pregorexia".

2. Review of the literature

2.1. Neuroendocrine and Metabolic Consequences of Caloric Restriction

Chronic caloric restriction and malnutrition, typical of eating disorders including pregorexia, induce profound disturbances within key neuroendocrine axes and alterations in the secretion of peptides regulating energy homeostasis. These deviations essentially constitute an evolutionary adaptive mechanism aimed at protecting the body against the drastic effects of weight loss and preserving its critical energy reserves (10).

Chronic starvation induces massive physiological stress, which acts as a stimulus shifting the hypothalamic-pituitary-adrenal (HPA) axis into a state of chronic overactivation, leading to hypercortisolemia (10, 11). Its etiology is multifactorial, with one of the primary causes being the hypersecretion of corticotropin-releasing hormone (CRH) in the paraventricular nucleus of the hypothalamus. The stimulation of CRH-ergic neurons is driven, among other factors, by an increase in ghrelin levels induced by chronic starvation. Importantly, CRH itself exerts a potent anorexigenic effect, which may exacerbate anorexia and body mass deficit (10, 11). Another cause of elevated cortisol levels is the increased pulsatile frequency of its secretion, as well as a significant reduction in its hepatic metabolic clearance, resulting in a prolonged half-life (10, 11, 12). Furthermore, the negative feedback loop inhibiting glucocorticoid secretion is

disrupted due to the blunted sensitivity of the hypothalamus and pituitary gland to high circulating glucocorticoid concentrations, while the diurnal rhythm of this hormone's secretion is simultaneously preserved (10, 11, 12). In contrast to the overproduction of glucocorticoids, adrenal androgen secretion is suppressed; patients exhibit significantly reduced concentrations of dehydroepiandrosterone sulfate (DHEA-S) and total testosterone (11, 12, 13). Furthermore, the pulsatile secretion of gonadotropins is inhibited, resulting in reduced estrogen concentrations (10, 11).

Another hormonal axis disrupted by nutritional deprivation is the hypothalamic-pituitary-thyroid (HPT) axis, where the observed alterations resemble the clinical picture of sick euthyroid syndrome. The peripheral conversion of thyroxine (T4) to active triiodothyronine (T3) is inhibited, resulting in decreased T3 levels in laboratory findings, accompanied by typically normal or low-normal free thyroxine (fT4) levels and a lack of a compensatory increase in thyrotropin (TSH) concentration (10, 14).

In response to starvation, the body attempts to minimize anabolic processes through alterations in the somatotropic axis. Growth hormone (GH) concentration is elevated due to the loss of negative feedback and increased ghrelin levels; however, there is receptor resistance to its action (15). The expression of hepatic GH receptors is down-regulated, consequently causing a drastic decline in the production of insulin-like growth factor 1 (IGF-1) (10, 15). This GH resistance is selective—only the IGF-1 synthesis pathway is inhibited, while the direct metabolic effects of GH are preserved. This enhances gluconeogenesis and lipolysis, protecting the organism against hypoglycemia and influencing adipose tissue redistribution (10, 15). The IGF-1 deficiency impairs the bone-forming capacity of osteoblasts, resulting in a decrease in bone mineral density (10, 15).

In addition to its impact on the previously discussed hormonal axes, the loss of adipose tissue triggers a cascade of alterations in peripheral signaling molecules, particularly those regulating hunger (orexigenic) and satiety (anorexigenic). There is a decrease in the concentration of appetite-suppressing leptin, which evolutionarily aims to drive caloric intake; however, it has been shown that hypoleptinemia can also induce increased physical activity in these patients (10, 15, 16). Conversely, the concentration of orexigenic ghrelin increases, which, as mentioned earlier, stimulates GH secretion and influences the HPA axis, helping to maintain euglycemia under conditions of starvation. Reports regarding peptide YY (PYY) are inconsistent; however, some studies have demonstrated paradoxically high concentrations of this hormone, even though it exerts an anorexigenic effect (10, 15, 16). The prevention of hypoglycemia is further

supported by a decrease in fasting insulin levels, which is accompanied by reduced amylin secretion. The concentrations of incretin hormones, specifically glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP), are also decreased (10, 15, 16, 17).

Complementing the neuroendocrine and metabolic consequences of anorexia nervosa is the development of chronic oxidative stress and systemic inflammation associated with prolonged tissue starvation (18, 19, 20). Studies demonstrate unphysiologically elevated levels of inflammatory mediators in patients, such as interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), alongside an increased CD4/CD8 ratio and enhanced spontaneous TNF production by monocytes (19, 20). This is accompanied by an overproduction of reactive oxygen species (ROS), which initiate lipid peroxidation processes, subsequently damaging cell membrane structures and disrupting intracellular signaling pathways (20). Furthermore, a pro-inflammatory environment within the central nervous system, coupled with oxidative stress, has been shown to directly impair neuroplasticity; this likely represents a crucial mechanism that modulates eating behaviors and drives the psychopathology of the disorder (19, 20).

2.2. The Impact of Maternal Caloric Restriction and Stress on Fetoplacental Homeostasis

2.2.1. Introduction and Clinical Outcomes

While the specific term "pregorexia" is rarely used in scientific literature—with eating disorders during pregnancy primarily classified as Anorexia Nervosa (AN), Bulimia Nervosa (BN), or Binge Eating Disorder (BED) (23)—maternal energy and micronutrient deficits severely compromise fetoplacental homeostasis (21, 24). The most frequently reported complications of maternal undernutrition include intrauterine growth restriction (IUGR), small for gestational age (SGA), low birth weight, preterm birth, and microcephaly (21, 23, 24). Neonates born to mothers with severe eating disorders, particularly AN, often present with low APGAR scores, require resuscitation, and experience early environmental adaptation difficulties, including impaired thermoregulation and feeding disorders (22, 23). Furthermore, maternal malnutrition during critical gestational windows increases the risk of fetal death, miscarriage, and long-term neurocognitive delays (21, 24). However, the current literature evaluating these outcomes faces several methodological limitations, including small sample sizes, reliance on subjective questionnaires rather than clinical diagnoses, and a predominant focus on hospitalized patients (6).

2.2.2. Placental Morphological and Vascular Alterations

The placenta is highly sensitive to maternal nutritional status, with outcomes heavily dependent on the timing of the deficit. Starvation limited to the first trimester can paradoxically increase placental mass at term without affecting birth weight, whereas severe caloric restriction in the third trimester results in placental weight loss and subsequent IUGR. Maternal undernutrition (MUN) critically alters placental architecture by decreasing trophoblast cell density, increasing placental barrier thickness, and inducing excessive apoptosis in both the labyrinthine and junctional zones. What seems to be interesting is the significant difference among species; in some animal models, these structural damages reduce the placental villous exchange area by up to 70% (25).

These morphological changes are driven by disrupted angiogenesis. An energy-deficient diet reduces maternal arginine levels, which serves as the primary substrate for nitric oxide (NO) synthesis. Decreased NO impairs vascular endothelial growth factor (VEGF) and angiopoietin signaling, leading to increased vascular resistance, placental vascular dysfunction, and restricted maternal-fetal blood flow (25).

2.2.3. Impaired Nutrient Transport Mechanisms

The structural and vascular degradation of the placenta directly impairs the transport of crucial nutrients. MUN causes a profound decrease in maternal plasma glucose, minimizing the concentration gradient essential for facilitated diffusion, which is further exacerbated by the downregulation of the placental glucose transporter SLC2A3 (GLUT3). Similarly, maternal protein restriction reduces the activity of System A amino acid transporters. Notably, this reduction in amino acid transport occurs prior to the clinical manifestation of IUGR, suggesting it is a primary etiological factor rather than a secondary consequence of restricted growth. Lipid metabolism is also disrupted; altered lipid hydrolase activity in the microvillous membrane decreases placental membrane fluidity, thereby hindering the transfer of essential long-chain polyunsaturated fatty acids (e.g., DHA, arachidonic acid) to the developing fetus (25).

2.2.4. Maternal Stress, Glucocorticoid Exposure, and the HPA Axis

Caloric restriction, micronutrient deficiencies, and accompanying psychological stress trigger the activation of the maternal hypothalamic-pituitary-adrenal (HPA) axis (6, 26). Under physiological conditions, the placental enzyme 11-β-HSD2 protects the fetus from excessive stress hormones by converting active maternal cortisol into inactive cortisone. However, maternal stress downregulates 11-β-HSD2 expression, allowing up to 20% of maternal cortisol to cross the placental barrier, leading to a strong correlation between maternal and fetal blood

cortisol levels (27, 28). Paradoxically, the placenta responds to this cortisol by synthesizing corticotropin-releasing hormone (CRH), which creates a positive feedback loop that further stimulates the maternal HPA axis (27).

Placental adaptations to glucocorticoid exposure are sexually dimorphic. Placentas associated with male fetuses adopt a strategy of continued growth by developing glucocorticoid resistance, characterized by higher levels of inactive glucocorticoid receptor isoforms (GR-α, GR-A, GR-P). Conversely, placentas of female fetuses reduce growth to maximize survival, maintaining sensitivity to glucocorticoids via the active GR-α isoform (27). The excessive fetal exposure to cortisol and maternal CRH directly impacts neurodevelopment, leading to cortical thinning and structural alterations in the hippocampus and amygdala (28).

2.2.5. Epigenetic Modifications and Structural Fetal Programming

The combination of nutrient deprivation and oxidative stress alters fetal genetic programming through epigenetic mechanisms, such as DNA methylation and histone modifications, without changing the underlying DNA sequence (6, 25). Key epigenetic shifts include the hypomethylation of the placental Wnt2 promoter and the hypermethylation of genes regulating calcium homeostasis (CACNA1C, PDE1A), lipid metabolism (ABCA1, LEP), and inflammation (IL10, IL6) (6). A hallmark of early fetal malnutrition is the reduced methylation and subsequent silencing of the IGF2 gene, which severely restricts placental trophoblast expansion and fetal growth (6, 25, 26).

This intrauterine "fetal programming" induces permanent structural and functional tissue alterations—including reduced pancreatic islet vascularity, altered hepatic lobules, and premature maturation of the fetal HPA axis (22, 25, 26).

Furthermore, these early-life stressors may alter the microbiome, potentially contributing to the transgenerational transmission of eating disorders (21, 23), and can exert multigenerational effects, as seen in grandmaternal undernutrition increasing neonatal adiposity in the subsequent generation (26).

2.3. Long-Term Health Outcomes in the Child: Metabolic and Neurodevelopmental Fetal Programming

2.3.1. Long-Term Metabolic Programming and Epigenetic Regulation

The long-term metabolic consequences of prenatal malnutrition are tightly linked to these mechanisms of fetal programming, whereby limited availability of energy and nutrients forces permanent metabolic adaptations in the developing organism (29). Metabolically, low intrauterine fetal leptin levels stimulate orexigenic pathways (e.g., increased NPY mRNA)

while inhibiting satiety signals. Postnatally, this programs the infant for a rapid surge in leptin and subsequent fat accumulation, predisposing the individual to leptin resistance and obesity in adulthood (25).

Although direct clinical data regarding long-term metabolic effects specifically in offspring of women with "pregorexia" remain limited, the broader metabolic consequences of adverse prenatal developmental conditions are well documented (29). Intrauterine malnutrition significantly increases susceptibility to insulin resistance, type 2 diabetes, obesity, hypertension, cardiovascular disease, and metabolic syndrome later in life (22, 25, 26, 30, 31, 32).

Epigenetic mechanisms serve as the critical bridge sustaining these long-term alterations (29, 37). Beyond structural DNA methylation and histone modifications, chromatin remodeling alters the expression of key genes involved in appetite regulation and energy homeostasis—such as POMC, NPY, MC4R, LEPR, and INSR (37). These durable molecular shifts establish abnormal metabolic regulatory patterns that persist into adulthood (29, 37).

2.3.2. Central Nervous System Maturation and Neurostructural Vulnerabilities

The consequences of fetal programming extend profoundly into the development and long-term functional architecture of the central nervous system. Fetal brain development begins as early as 2–3 weeks post-fertilization, encompassing neuronal proliferation and migration, synapse formation, myelination, and programmed apoptosis. Due to the early and dynamic nature of these neurodevelopmental cascades, the developing brain is exceptionally vulnerable to adverse intrauterine environmental conditions and nutrient deficiencies (33).

Restricted energy intake during pregnancy alters the maturation of key hypothalamic systems controlling food intake and energy balance, primarily through an impaired response to leptin and insulin, which permanently recalibrates future perceptions of hunger and satiety (37). Concurrently, prenatal malnutrition adversely impacts other critical brain regions, particularly the hippocampus and amygdala, resulting in altered resting-state networks, lower IQ, and potential long-term impairments in memory, cognitive function, and emotional regulation (28, 34).

2.3.3. Clinical Profiles: Neurodevelopmental and Psychiatric Outcomes in Offspring

Clinical and epidemiological studies confirm that active maternal eating disorders during pregnancy carry severe long-term neurodevelopmental and psychiatric implications for the child. Maternal anorexia nervosa (AN) is associated with an increased risk of attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorders (ASD), and affective

disorders in offspring (28, 35). The persistence and timing of the disorder during pregnancy are decisive determinants of risk: offspring of women with active AN during pregnancy exhibit a substantially higher risk of ADHD and ASD compared to those whose mothers had a history of the disorder but were not actively affected at that time (35).

These clinical findings are corroborated by retrospective cohort studies examining offspring of women hospitalized for anorexia nervosa. Children born to these mothers demonstrate an elevated risk of developing mental and neurodevelopmental disorders, including severe anxiety and stress-related disorders. Notably, a particularly high risk of psychiatric hospitalization in offspring is observed among women who required hospitalization for AN specifically during pregnancy (36). Taken together, current evidence underscores that active or severe maternal anorexia nervosa during pregnancy exerts a profound, long-term impact on offspring neurodevelopment and mental health trajectories (35, 36).

2.4. Somatic and Psychological Complications in the Mother During the Perinatal Period

Peripartum eating pathology severely impairs maternal physiological stability and affective adaptation (22, 38). Severe energy restrictions trigger cachexia and muscle wasting, elevating the risk of critical electrolyte imbalances (8, 22). Cardiological integrity is concurrently compromised, predisposing patients to sinus bradycardia, QTc prolongation, and structural impairment (22). Furthermore, largely restricted intake disrupts prolactin and oxytocin axis dynamics, leading to delayed milk onset and insufficient lactation or premature glandular involution (22, 39).

Extended biological stress of calorie deprivation alters central neurotransmitter synthesis—particularly serotonin—while hyperactivating the hypothalamic-pituitary-adrenal axis (8, 38). This neurochemical shift amplifies maternal anxiety, intrusive food-related thoughts and proneness to postpartum depression (23, 38). In conjunction an obsessive cognitive focus on body shape and weight generates profound body dysmorphia which creates significant obstacles to „maternal adaptation", making women less likely to establish a strong early attachment bond with their newborn (8, 36). Excessive dietary monitoring and compensatory behaviors may lead to devour critical cognitive bandwidth needed to respond to infant cues, thereby impairing maternal sensitivity and emotional availability (8, 38).

Maternal nutrient deficiencies are leading to compromise breast milk composition by reduction in vital micronutrients and essential fatty acids (e.g., DHA) which are directly causing disruption in optimal infant neurodevelopment (23, 39). Comorbid maternal affective blunting and feeding-related body distress unables skin-to-skin contact, severely disrupting early

mother–child bonding (36, 40). Consequently, these early interactive disruptions heighten the long-term risk of insecure attachment and neurodevelopmental morbidity in offspring (36, 40), while deepening maternal dissatisfaction and regret (41).

2.5. Diagnostic Challenges and Multidisciplinary Therapeutic Strategies

The diagnosis of "pregorexia" remains challenging because the term lacks universally accepted diagnostic criteria and is not recognized as a distinct psychiatric disorder in either the ICD-10 or DSM-5 (7, 21). Furthermore, its clinical presentation may overlap with normal changes in appetite and eating behaviors during pregnancy, potentially hindering early identification and appropriate management (42). Despite the well-documented risks, the identification and management of eating disorders in perinatal care remain suboptimal. Time constraints, limited healthcare provider training, and persistent social stigma may contribute to their underrecognition. Although depression and anxiety are routinely screened for during pregnancy, eating disorders may be overlooked (43).

The diagnostic process is further complicated by the difficulty of distinguishing pregnancy-related physical changes from symptoms associated with an eating disorder (42). In this context, commonly used screening instruments, including the SCOFF questionnaire and the Eating Attitudes Test (EAT-26), may have limited utility in pregnant populations because they were not specifically developed or validated for obstetric settings and may produce false-positive results. To address this gap, several promising tools have been developed to assess eating behaviors during pregnancy, including brief screening measures (PEBS and DEAPS) and a pregnancy-specific adaptation of an existing instrument (reduced EDE-PV), although further validation is warranted (44).

Once disordered eating is identified, "pregorexia" should be managed by a multidisciplinary team including an obstetrician, psychologist, psychiatrist, midwife, and dietitian. Treatment should include education on healthy eating and appropriate gestational weight gain, as well as a review of medication use and dietary habits (3). This process may require several months of regular care. Psychotherapy is an important part of treatment and may focus on body image and disordered eating behaviors, including food restriction, compensatory behaviors, and excessive exercise. Cognitive-behavioral therapy (CBT) is commonly recommended, while other approaches, such as acceptance and commitment therapy (ACT), dialectical behavior therapy (DBT), and art therapy, may also be considered (38). Support from family members or a partner also plays a key role in successful recovery. Antidepressants may be used in some cases, but

none are FDA-approved for “pregorexia” and they should not be the only form of treatment (21).

3. Conclusions

In conclusion, "pregorexia" represents a critical intersection of psychopathological and neuroendocrine disruptions during pregnancy, where severe caloric restriction and body dysmorphia severely compromise fetoplacental homeostasis. Maternal malnutrition and stress trigger chronic overactivation of the hypothalamic-pituitary-adrenal axis, altered nutrient transport, and structural placental degradation, which collectively restrict fetal growth and induce lasting epigenetic modifications. These intrauterine alterations result in adverse perinatal outcomes and drive long-term metabolic and neurodevelopmental programming in the child, increasing the lifelong risk of cardiometabolic diseases, ADHD, autism spectrum disorders, and psychiatric conditions. Furthermore, perinatal eating pathology imposes a profound physical and psychological burden on the mother, hindering affective adaptation and impairing early mother-child bonding. Given the diagnostic challenges and subtle presentation of disordered eating during gestation, routine screening with validated pregnancy-specific tools is essential. Effective management requires an early, multidisciplinary therapeutic approach involving obstetricians, psychiatrists, psychologists, dietitians, and midwives to mitigate short- and long-term risks, thereby safeguarding the health and well-being of both mother and child.

This review was undertaken to bridge a gap in the review literature by synthesizing the multifaceted psychopathological, neuroendocrine, and epigenetic mechanisms through which "pregorexia" compromises maternal-fetal health. As comprehensively outlined in the summary table of physiological impacts, gestational caloric restriction and body dysmorphia trigger a cascade of systemic disruption in mother organism as well as in offspring. Despite these profound implications, scientific progress in this field remains severely hindered by the lack of a standardized clinical definition and an overreliance on subjective questionnaires rather than objective physiological metrics. To overcome these limitations, future research must establish rigorous, standardized methodologies anchored in objective clinical data, thereby preventing the proliferation of low-quality studies and redundant review articles. Furthermore, untangling this condition is uniquely challenging due to the heavy semantic overlap with a multitude of existing medical terms covering similar behavioral and perinatal phenotypes. Ultimately, establishing a unified nomenclature and standardized diagnostic criteria is an urgent prerequisite to advance high-quality scientific literature, improve early clinical detection, and

optimize multidisciplinary therapeutic strategies for mothers and children affected by gestational eating disorders.

Table 2. All of the effects & mechanisms associated with pathophysiology of pregorexia.

Mechanism / Type of Interaction	Maternal Effects	Placental Effects	Fetal & Offspring Effects
Neuroendocrine & Hormonal Axes	HPA axis overactivation (hypercortisolemia, ↑ CRH) Suppressed HPT axis (sick euthyroid syndrome, ↓ T3) Somatotropic axis disruption (↑ GH with receptor resistance, ↓ IGF-1) Suppressed sex hormones (↓ DHEA-S, testosterone, estrogen)	↓ 11-β-HSD2 enzyme activity (fails to inactivate maternal cortisol) Paradoxical ↑ synthesis of placental CRH Sexually dimorphic adaptation to glucocorticoids (resistance in males, sensitivity in females)	Premature maturation of the HPA axis Excessive exposure to cortisol Altered neurodevelopment due to hormonal imbalance
Metabolic & Nutrient Transport	Disrupted hunger/satiety signaling (↓ leptin, ↑ ghrelin) ↓ Fasting insulin, amylin, and incretin hormones (GLP-1, GIP) Enhanced gluconeogenesis and lipolysis	Downregulation of glucose transporters (GLUT3) Reduced activity of System A amino acid transporters Altered lipid hydrolase activity (hindered transfer of polyunsaturated fatty acids)	Low intrauterine leptin stimulating orexigenic pathways (↑ NPY) Long-term metabolic programming (risk of leptin/insulin resistance, obesity, type 2 diabetes, cardiovascular disease)
Morphological & Vascular	Decreased arginine levels (leading to ↓ nitric oxide synthesis) Decreased bone mineral density	Altered mass depending on starvation timing Disrupted angiogenesis (impaired VEGF) and increased vascular resistance ↓ Trophoblast cell density, ↑ barrier thickness, excessive apoptosis	Intrauterine growth restriction (IUGR) and small for gestational age (SGA) Microcephaly and low birth weight Reduced pancreatic islet vascularity and altered hepatic lobules
Epigenetic & Genetic Programming		Hypomethylation (e.g., Wnt2 promoter) Hypermethylation of genes regulating calcium, lipids, and inflammation Reduced methylation and silencing of the IGF2 gene	Chromatin remodeling of appetite-regulating genes (POMC, NPY, MC4R, LEPR, INSR) Permanent structural/functional tissue alterations Multigenerational transmission of adiposity
Immunological & Oxidative Stress	Systemic inflammation (↑ IL-1β, IL-6, TNF-α) ↑ CD4/CD8 ratio and reactive oxygen species (ROS) overproduction Lipid peroxidation and impaired neuroplasticity	Hypermethylation of inflammation-regulating genes (IL10, IL6) Oxidative stress contributing to altered genetic programming	Potential microbiome alterations Vulnerability to early environmental adaptation difficulties (thermoregulation, feeding disorders)
Neurodevelopmental & Psychiatric	Increased psychological stress triggering HPA axis Neuroplasticity impairment modulating eating disorder psychopathology		Cortical thinning, structural alterations in hippocampus and amygdala Altered resting-state networks and lower IQ Long-term psychiatric risks: ADHD, Autism Spectrum Disorders (ASD), affective disorders, and severe anxiety

Disclosure

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References

1. Hay P, Aouad P, Le A, Marks P, Maloney D, National Eating Disorder Research Consortium, Touyz S, Maguire S. Epidemiology of eating disorders: Population, prevalence, disease burden and quality of life informing public policy in Australia—A rapid review. Journal of Eating Disorders. 2023;11(1):23. https://doi.org/10.1186/s40337-023-00738-7

2. Martínez-Olcina M, Rubio-Arias JA, Reche-García C, Leyva-Vela B, Hernández-García M, Hernández-Morante JJ, Martínez-Rodríguez A. Eating disorders in pregnant and breastfeeding women: A systematic review. Medicina. 2020;56(7):352. https://doi.org/10.3390/medicina56070352
3. Harasim-Piszczatowska E, Krajewska-Kułak E. Pregorexia – anorexia of pregnant women. Pediatria i Medycyna Rodzinna. 2017;13(3):363–367. https://doi.org/10.15557/PiMR.2017.0038
4. Dobrowolska A, Baturo A, Marciniak M. Żywienie w okresie ciąży i połogu. W: Bręborowicz GH, redaktor. Położnictwo i ginekologia. Tom 1. Wyd. 3. Warszawa: PZWL Wydawnictwo Lekarskie; 2020. s. 69.
5. Easter A, Bye A, Taborelli E, Corfield F, Schmidt U, Treasure J, Micali N. Recognising the symptoms: How common are eating disorders in pregnancy? Eur Eat Disord Rev. 2013;21(5):340-344. https://doi.org/10.1002/erv.2229
6. Sebastiani G, Andreu-Fernández V, Herranz Barbero A, Aldecoa-Bilbao V, Miracle X, Meler Barrabes E, et al. Eating disorders during gestation: implications for mother's health, fetal outcomes, and epigenetic changes. Front Pediatr. 2020;8:587. https://doi.org/10.3389/fped.2020.00587
7. Mróz MW, Korek E. Review of non-specific eating disorders. Neuropsychiatria i Neuropsychologia / Neuropsychiatry and Neuropsychology. 2020;15(1):42-50. https://doi.org/10.5114/nan.2020.97400
8. Vasiliu O. The complex interplay between psychosocial and biological factors in pregorexia nervosa—a rapid review. Front Psychol. 2023;14:1168696. https://doi.org/10.3389/fpsyg.2023.1168696
9. Tarchi L, Merola GP, Selvi G, Caprara E, Pecoraro V, Cassioli E, et al. Pregorexia: a systematic review and meta-analysis on the constructs of body image dissatisfaction and eating disturbances by gestational age in the peripartum. Eat Weight Disord. 2023;28(1):64. https://doi.org/10.1007/s40519-023-01595-8
10. Misra M, Klibanski A. Neuroendocrine consequences of anorexia nervosa in adolescents. Endocr Dev. 2010;17:197-214. https://doi.org/10.1159/000262540
11. Calcaterra V, Magenes VC, Fratangeli N, Nigro G, Fabiano V, Mendolicchio L, et al. Hypothalamic-pituitary-adrenal axis in anorexia nervosa; an underestimated endocrine dysfunction among adolescents. Front Pediatr. 2024;12:1415061. https://doi.org/10.3389/fped.2024.1415061

12. Thavaraputta S, Ungprasert P, Witchel SF, Fazeli PK. Anorexia nervosa and adrenal hormones: a systematic review and meta-analysis. Eur J Endocrinol. 2023;189(3):S64-S73. https://doi.org/10.1093/ejendo/lvad123
13. Lawson EA, Misra M, Meenaghan E, Rosenblum L, Donoho DA, Herzog D, et al. Adrenal glucocorticoid and androgen precursor dissociation in anorexia nervosa. J Clin Endocrinol Metab. 2009;94(4):1367-1371. https://doi.org/10.1210/jc.2008-2558
14. Sato T, Igarashi N, Miyagawa K, Nakajima T, Katayama K. Catecholamine and thyroid hormone metabolism in a case of anorexia nervosa. Endocrinol Jpn. 1988;35(2):295-301. https://doi.org/10.1507/endocrj1954.35.295
15. Misra M, Klibanski A. Endocrine consequences of anorexia nervosa. Lancet Diabetes Endocrinol. 2014;2(7):581-592. https://doi.org/10.1016/S2213-8587(13)70180-3
16. Schorr M, Miller KK. The endocrine manifestations of anorexia nervosa: mechanisms and management. Nat Rev Endocrinol. 2017;13(3):174-186. https://doi.org/10.1038/nrendo.2016.175
17. Tomasik PJ, Sztefko K, Malek A. GLP-1 as a satiety factor in children with eating disorders. Horm Metab Res. 2002;34(2):77-80. https://doi.org/10.1055/s-2002-20519
18. Wagner-Skacel J, Haidacher F, Wiener M, Pahsini K, Marinschek S, Lahousen T, et al. Oxidative Status in Adult Anorexia Nervosa Patients and Healthy Controls—Results from a Cross-Sectional Pilot Study. Antioxidants. 2022;11(5):842. https://doi.org/10.3390/antiox11050842
19. Spero V, Scherma M, D'Amelio S, Collu R, Dedoni S, Camoglio C, et al. Activity-based anorexia (ABA) model: Effects on brain neuroinflammation, redox balance and neuroplasticity during the acute phase. Neurochem Int. 2024;180:105842. https://doi.org/10.1016/j.neuint.2024.105842
20. Gibson D, Mehler PS. Anorexia Nervosa and the Immune System-A Narrative Review. J Clin Med. 2019;8(11):1915. https://doi.org/10.3390/jcm8111915
21. Grajek MK, Grot M, Kujawińska M, Nigowski M, Kryska S. Pregorexia and its significance for physiological processes in the fetus - a review of current knowledge. Psychiatria Polska. 2023;57(6):1181–94. https://doi.org/10.12740/PP/OnlineFirst/150421
22. Sowińska-Przepiera E, Krzyścin M, Syrenicz I, Bumbuliene Z, Wajs-Syrenicz A, Przepiera A, et al. Dilemmas concerning the course of pregnancy in patients with anorexia nervosa considering hormonal and somatic parameters. Endokrynol Pol. 2024;75(3):279-290. https://doi.org/10.5603/ep.99255

23. Janas-Kozik M, Żmijowska A, Zasada I, Jelonek I, Cichoń L, Siwiec A, et al. Systematic review of literature on eating disorders during pregnancy—risk and consequences for mother and child. Front Psychiatry. 2021;12:777529. https://doi.org/10.3389/fpsyt.2021.777529
24. Esra Tunçer, Aylin Bayındır Gümüş, Alev Keser. The Importance of Pregorexia Awareness. Clinical and Experimental Health Sciences. 2020 Sep. 1;10(3):186-90. doi:10.33808/clinexphealthsci.673306
25. Belkacemi L, Nelson DM, Desai M, Ross MG. Maternal undernutrition influences placental-fetal development. Biol Reprod. 2010;83(3):325-331. https://doi.org/10.1095/biolreprod.110.084517
26. Roseboom TJ, Painter RC, van Abeelen AF, Veenendaal MV, de Rooij SR. Hungry in the womb: what are the consequences? Lessons from the Dutch famine. Maturitas. 2011;70(2):141-145. https://doi.org/10.1016/j.maturitas.2011.06.017
27. St-Pierre J, Laurent L, King S, Vaitys M. Effects of prenatal maternal stress on serotonin and fetal development. Placenta. 2016;48(Suppl 1):S66-S71. https://doi.org/10.1016/j.placenta.2015.11.003
28. Lautarescu A, Craig MC, Glover V. Prenatal stress: Effects on fetal and child brain development. Int Rev Neurobiol. 2020;150:17-40. https://doi.org/10.1016/bs.irn.2019.11.002
29. Kwon EJ, Kim YJ. What is fetal programming?: a lifetime health is under the control of in utero health. Obstet Gynecol Sci. 2017;60(6):506-519. https://doi.org/10.5468/ogs.2017.60.6.506
30. Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, et al. Maternal and child undernutrition: consequences for adult health and human capital. Lancet. 2008;371(9609):340-357. Erratum in: Lancet. 2008;371(9609):302. https://doi.org/10.1016/S0140-6736(07)61692-4
31. Lumey LH, Ekamper P, Bijwaard G, Conti G, van Poppel F. Overweight and obesity at age 19 after pre-natal famine exposure. Int J Obes (Lond). 2021;45(8):1668-1676. https://doi.org/10.1038/s41366-021-00824-3
32. Meng R, Lv J, Yu C, Guo Y, Bian Z, Yang L, et al. Prenatal famine exposure, adulthood obesity patterns and risk of type 2 diabetes. Int J Epidemiol. 2018;47(2):399-408. https://doi.org/10.1093/ije/dyx228

33. Li M, Francis E, Hinkle SN, Ajjarapu AS, Zhang C. Preconception and Prenatal Nutrition and Neurodevelopmental Disorders: A Systematic Review and Meta-Analysis. Nutrients. 2019;11(7):1628. https://doi.org/10.3390/nu11071628
34. Cortés-Albornoz MC, García-Guáqueta DP, Velez-van-Meerbeke A, Talero-Gutiérrez C. Maternal Nutrition and Neurodevelopment: A Scoping Review. Nutrients. 2021;13(10):3530. https://doi.org/10.3390/nu13103530
35. Mantel Ä, Örtqvist AK, Hirschberg AL, Stephansson O. Analysis of Neurodevelopmental Disorders in Offspring of Mothers With Eating Disorders in Sweden. JAMA Netw Open. 2022;5(1):e2143947. https://doi.org/10.1001/jamanetworkopen.2021.43947
36. Amar S, Côté-Corriveau G, Israël M, Steiger H, Low N, Chadi N, et al. Maternal anorexia nervosa and risk of mental and neurodevelopmental morbidity in offspring. Child Adolesc Ment Health. 2026;31(2):125-133. https://doi.org/10.1111/camh.70051
37. Mall S, Oladun B, Kim MH. Maternal Nutrition and Hypothalamic Programming of Offspring Metabolic Health. J Nutr. 2026;156(6):101515. https://doi.org/10.1016/j.tjnut.2026.101515
38. Fomicheva NS. Pregorexia: a psychotherapy strategy for eating disorders in pregnant women. Consortium psychiatricum. 2023;4(2):111–4. https://doi.org/10.17816/CP6642
39. Gerontidis A, Grammatikopoulou MG, Tzimos C, Gkiouras K, Taousani E, Athanasiadis L, Goulis DG. Effectors of Pregorexia and Emesis among Pregnant Women: A Pilot Study. Nutrients. 2022;14(24):5275. https://doi.org/10.3390/nu14245275
40. Watson HJ, Zerwas S, Torgersen L, Gustavson K, Diemer EW, Knudsen GP, Reichborn-Kjennerud T, Bulik CM. Maternal eating disorders and perinatal outcomes: a three-generation study in the Norwegian Mother and Child Cohort Study. J Abnorm Psychol. 2017;126(5):552-564. https://doi.org/10.1037/abn0000241
41. Baumgardt S, Asselmann E. Parenthood regret: an under-recognised aspect of parental wellbeing. J Reprod Infant Psychol. 2026;44(5):1143-1145. https://doi.org/10.1080/02646838.2026.2679863
42. Bannatyne AJ, Hughes R, Stapleton P, Watt B, MacKenzie-Shalders K. Signs and symptoms of disordered eating in pregnancy: a Delphi consensus study. BMC Pregnancy Childbirth. 2018;18(1):262. https://doi.org/10.1186/s12884-018-1849-3
43. Policy Center for Maternal Mental Health, National Eating Disorders Association. Eating Disorders and Maternal Mental Health [Issue Brief]. 2026 Feb. https://doi.org/10.69764/EDMH2026

44. Stephens J, Ellis A, Roberts S, Gillespie K, Bannatyne A, Branjerdporn G. Disordered eating instruments in the pregnancy cohort: a systematic review update. Eat Disord. 2025;33(4):512-536. https://doi.org/10.1080/10640266.2024.2386469