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## Maternal Stress and the Future Child: Pathways Linking Prenatal Stress with Labor and Developmental Outcomes

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### Abstract

**Introduction:** Maternal stress during pregnancy is a key determinant of obstetric and neonatal outcomes, influencing both fetal development and maternal health. Psychosocial and biological stress pathways, particularly dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, and elevated cortisol are linked to adverse labor progression (including prolonged labor, obstetric complications) and neonatal outcomes (Apgar scores at 1 and 5 minutes, newborn anthropometrics). Understanding these mechanisms is essential for developing integrative maternal care strategies that mitigate stress-related risks. **Objective:** To assess the association between maternal stress during pregnancy, labor progression, and neonatal outcomes. **Methods:** A cross-sectional study was conducted among 398 pregnant women in Tbilisi, Georgia. A validated pregnancy-specific questionnaire was used to assess self-reported stress, and salivary cortisol was measured in a subgroup of 95 women

reporting high stress. Labor characteristics and neonatal outcomes (Apgar scores at 1 and 5 minutes, newborn anthropometrics) were analyzed. Statistical analyses were performed using SPSS v.23.0. **Results:** Two groups were formed: the Study Group (n=172; stressed, with a subgroup of 95 women, showing elevated cortisol levels) and the Control Group (n=226; non-stressed). Delivery complications occurred in 51.9% of stressed women versus 26.6% of controls (p=0.001), with more than twice the odds of complications (OR = 2.21; 95% CI = 1.13–4.33; p = 0.021). Newborns of stressed mothers were twice as likely to have low 1-minute Apgar scores (OR = 2.12; p = 0.028). No significant difference was found in 5-minute Apgar scores (OR = 1.13; p = 0.257). **Conclusions:** Elevated maternal stress and cortisol levels were significantly associated with higher risks of delivery complications and reduced neonatal vitality at birth. These findings position maternal stress as a clinically relevant and modifiable determinant of intrapartum risk. The markedly higher rates of delivery complications and early neonatal compromise in stressed women support the integration of routine stress screening and psychosocial assessment into standard antenatal care. From a policy and practice perspective, targeted stress-reduction interventions—such as psychological counseling, social support programs, and stress-management strategies—should be incorporated into prenatal services to improve obstetric and neonatal outcomes. Future research should focus on evaluating the effectiveness of such interventions, identifying critical gestational windows of vulnerability, and clarifying the biological pathways linking maternal stress, cortisol dysregulation, and perinatal outcomes.

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**Keywords:** Prenatal stress, HPA axis dysregulation, Maternal health, Cortisol levels, Intrapartum complications, Neonatal outcomes

## Introduction

Pregnancy is an intricate biopsychosocial process requiring physiological and emotional adaptation. Maternal psychological well-being during this period has profound implications for both the mother and her developing child. Chronic or intense stress exposure during gestation can activate neuroendocrine pathways that influence placental function, fetal growth, and the timing and course of labor (Dunkel Schetter & Tanner, 2012; Wadhwa et al., 2011). Epidemiological and experimental studies have increasingly demonstrated that prenatal stress exerts programming effects on the offspring's neurodevelopment and stress-response systems (Sandman & Davis, 2012; O'Donnell et al., 2020). Elevated maternal cortisol a hallmark of hypothalamic–pituitary–adrenal (HPA) axis activation, crosses the placental barrier and may alter fetal HPA calibration, affecting long-term cognitive and behavioral outcomes. In the obstetric context, maternal stress has been linked

with prolonged labor, dysfunctional uterine contractions, and a higher incidence of cesarean delivery (Glynn et al., 2018). Stress also correlates with reduced Apgar scores, lower birthweight, and impaired neonatal adaptation (Bussi eres et al., 2015). Beyond immediate perinatal consequences, these effects may predispose children to later emotional dysregulation and neurocognitive difficulties.

In Europe, data from the Euro-Peristat Network indicate that the median caesarean section (CS) rate was 26.0% (interquartile range, IQR: 20.7–32.1 %) for the period 2015-2019. The median instrumental vaginal delivery rate during the same period was reported at 6.1% (IQR: 3.5–9.8%). In the United States, data from the National Center for Health Statistics (NCHS) show the CS rate increased to 32.3% of all live births in 2023. Observational studies linking maternal prenatal psychological stress and delivery complications highlight a significant association. For example, women with a strong fear of childbirth were found to have higher odds of elective cesarean section (adjusted odds ratio, OR ~ 2.4; 95% CI = 1.2–4.9) in a Norwegian cohort. A systematic review found that antenatal depression and fear of childbirth are linked to increased CS risk, though findings vary. Thus, the convergence of high operative delivery rates and documented associations of stress/anxiety with intrapartum complications underscores the importance of considering prenatal psychological stress as a potential contributor to delivery-stage morbidity in both Europe and the U.S.

The impact of maternal stress on pregnancy outcomes is mediated through a complex network of biological and psychosocial mechanisms. The primary physiological pathway is the hypothalamic–pituitary–adrenal (HPA) axis, which coordinates the body’s stress response through the secretion of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol (Glover, 2015). During pregnancy, both maternal and placental CRH production increase substantially, resulting in a natural elevation of cortisol that supports fetal organ maturation. However, chronic psychological stress exaggerates this physiological rise, creating a hypercortisolemic environment that can disrupt placental homeostasis and uterine function (Sandman & Davis, 2012; O’Donnell & Meaney, 2017).

Excess cortisol readily crosses the placental barrier, although normally it is partially inactivated by the placental enzyme 11 $\beta$ -hydroxysteroid dehydrogenase type-2 (11 $\beta$ -HSD2). Sustained stress downregulates 11 $\beta$ -hydroxysteroid dehydrogenase type-2, allowing more active cortisol to reach the fetus (Seckl & Holmes, 2007). This exposure may prematurely mature fetal tissues, alter neural differentiation, and reset the fetal HPA axis to heightened reactivity—an example of fetal programming (Barker, 1998; Wadhwa et al., 2011). In obstetric terms, elevated cortisol interferes with oxytocin receptor sensitivity and myometrial contractility, which may prolong

the latent phase of labor or increase the risk of dystocia and operative delivery (Glynn et al., 2018). Increased catecholamine levels associated with acute stress further constrict uterine blood flow, predisposing to fetal hypoxia and low Apgar scores (Dunkel Schetter, 2011).

Beyond biological processes, psychosocial stressors—such as financial insecurity, occupational overload, family conflict, or limited social support—have been strongly associated with adverse obstetric outcomes (Lobel et al., 2008; Giurgescu et al., 2015). Psychosocial distress can exacerbate health-risk behaviors (e.g., poor sleep, inadequate nutrition) and reduce adherence to prenatal care, compounding physiological stress effects. The buffering role of social support and adaptive coping has been well documented: women reporting higher perceived support exhibit lower cortisol reactivity and fewer labor complications (Guardino & Dunkel Schetter, 2014). Consequently, both the psychological appraisal of stress and the availability of protective social and occupational environments are critical determinants of maternal and fetal well-being.

An expanding body of evidence underscores the influence of prenatal stress on obstetric and developmental outcomes. Classic longitudinal studies by Wadhwa, Sandman, and colleagues (1993–2011) first demonstrated that high maternal cortisol and anxiety during pregnancy predicted shorter gestation and lower birthweight.

A 2020 meta-analysis encompassing 29 cohort studies concluded that maternal stress and anxiety increase the risk of preterm delivery (OR = 1.56, 95 % CI = 1.25–1.94) and cesarean section (Staneva et al., 2020). Similarly, Glynn et al. (2018) reported that high stress levels in late pregnancy correlated with reduced oxytocin responsiveness and longer labor durations. Biological plausibility lies in stress-induced suppression of prostaglandin synthesis and uterine receptor sensitivity.

Prenatal stress has been associated with lower 1-minute Apgar scores, impaired neonatal adaptation, and smaller head circumference (Bussi eres et al., 2015; Van den Bergh et al., 2020). These neonatal markers often predict later cognitive and emotional trajectories.

Follow-up studies show that children of highly stressed mothers demonstrate elevated cortisol reactivity, greater behavioral inhibition, and increased risk of anxiety and attention-deficit and hyperactivity disorders (O'Donnell et al., 2020; Kim et al., 2021). Animal and human data converge on an epigenetic mechanism involving glucocorticoid-receptor gene methylation that mediates intergenerational stress effects (Meaney & Szyf, 2005; Palma-Gudiel et al., 2019).

While subjective questionnaires capture self-perceived stress, biomarkers such as salivary cortisol or hair cortisol provide objective validation. Combining both questionnaire and cortisol measurement enhances

predictive accuracy (Hodyl et al., 2016). The present study applies this dual approach in the Georgian context, where culturally specific stressors—economic transition, social expectations, and healthcare accessibility—may uniquely shape maternal stress experiences.

In summary, prenatal stress acts through an integrated biopsychosocial model. Stress perception activates the HPA axis → increased CRH and cortisol → placental dysregulation and altered uterine activity → obstetric complications (e.g., prolonged labor) → neonatal distress (low Apgar, growth restriction) → potential long-term neurodevelopmental alterations. This conceptual pathway provides the foundation for interpreting the present study's findings.

In Georgia, despite a growing body of public health research on maternal and child outcomes, no systematic studies to date have investigated the relationship between maternal stress during pregnancy and subsequent physical or cognitive outcomes in children. This represents a significant gap in the national scientific literature, as stress is a highly relevant factor in a context characterized by socioeconomic transitions, cultural expectations surrounding pregnancy, and emerging structural health challenges. The absence of baseline data on prenatal stress and its biological and psychosocial correlates prevents the development of targeted maternal mental health interventions and undermines evidence-based policymaking.

Hence, no systematic studies have examined the relationship between maternal stress during pregnancy and child health and developmental outcomes in Georgia. This absence of evidence highlights a critical gap in both national and regional research, as neighboring CIS countries have only recently begun to investigate these associations, often in the context of war, displacement, or acute socioeconomic instability. By focusing on psycho-emotional stress during pregnancy and its biological correlates, the present study represents the first structured attempt to document these dynamics in the Georgian population. Its novelty lies not only in generating baseline data for Georgia but also in contributing to the broader CIS literature, where robust longitudinal studies remain scarce. Importantly, this research positions Georgia within the international discourse on fetal programming, underscoring the relevance of local findings to the global understanding of how maternal stress shapes early developmental trajectories.

Georgia, like many middle-income countries, faces growing recognition of perinatal mental-health challenges. While antenatal care increasingly integrates screening of somatic pathologies, stress assessment remains limited. Contextualizing stress-related obstetric and neonatal outcomes within Georgian populations is therefore critical for informing clinical and public-health responses aligned with WHO's Maternal Mental Health Framework (2022).

The present study examines associations between maternal stress, labor progression, and neonatal outcomes in a Georgian cohort. By combining self-reported stress and salivary-cortisol measures, it elucidates both psychosocial and biological pathways linking prenatal stress with obstetric and developmental outcomes of the newborns.

## **Study Methodology**

Cross-sectional research was conducted between April 2023 and September 2025 among pregnant women, from women's consultations and maternity homes in Tbilisi, Georgia. This study is part of a doctoral research project with the headline "Fetal Stress and its impact on physical and cognitive health of the children under 1 year. Association with socio-economic and cultural aspects". It is aimed to assess the relationship between maternal stress during pregnancy, labor progression, and neonatal outcomes. The design followed STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for cross-sectional research. The study was approved by the ethics committee of the University of Georgia (Research code: UGREC-03-23).

A total of 398 pregnant women at different stages of their pregnancy were enrolled after providing informed consent. Participants were recruited from the women's consultations and maternity homes of Tbilisi, Georgia.

Inclusion criteria were: (1) healthy pregnant woman, (2) women aged 18 and older (3) absence of chronic endocrine or psychiatric disorders, (4) no pharmacological corticosteroid therapy, (4) residency of Georgia (5) willingness to participate (6) signing of the informed consent.

Exclusion criteria included: (1) multiple gestations, (2) pre-existing hypertension, (3) gestational diabetes, (4) mental disorders, (5) psychotropic or teratogenic medications, (6) non-native language speakers without translation services, (7) known fetal genetic disorders or malformations, (8) cognitive or intellectual disabilities, (9) pregnant women who are currently enrolled in other research on pregnancy related with stress or interventions.

The eligibility criteria were designed to ensure a homogeneous and clinically comparable cohort, allowing the effects of prenatal stress to be examined with minimal confounding. Inclusion was limited to healthy adult pregnant women without chronic endocrine or psychiatric conditions and without corticosteroid therapy, as these factors directly influence cortisol regulation and stress physiology. Restricting participation to residents of Georgia ensured consistency in healthcare access and socio-cultural context, while informed consent guaranteed ethical compliance and voluntary participation.

Exclusion criteria were applied to remove conditions independently associated with obstetric or neonatal risk (e.g., multiple gestations,

hypertension, gestational diabetes, fetal anomalies) and factors that could distort stress assessment or outcome interpretation (e.g., mental disorders, psychotropic or teratogenic medications, concurrent participation in stress-related studies). Non-native language speakers without translation support and individuals with cognitive impairments were excluded to ensure accurate comprehension of study procedures and reliable data collection. Collectively, these criteria enhanced internal validity, reduced bias, and improved the transparency and replicability of the study design.

Participants (N=398) were divided into two groups: the **study group** (n = 172) — women reporting high self-perceived stress levels, of whom 95 provided salivary cortisol samples; the **control group** (n = 226) — women reporting no stress symptoms.

Maternal stress was evaluated using a pregnancy-specific stress questionnaire in the Georgian language and adapted to the cultural context, created specifically for this study. The instrument assessed emotional, socio-economical, relationship, occupational, and financial stress domains. In a high-stress subgroup (N=172), the night salivary cortisol was collected using sterile saliva swabs (passive drool method). Saliva samples were randomly obtained from 95 pregnant women (out of N=172) who self-reported persistent stress symptoms. Sampling was performed in the afternoon to determine evening salivary cortisol levels, which better reflect chronic hypothalamic–pituitary–adrenal (HPA) axis activation. Participants refrained from eating, drinking, or performing oral hygiene for at least 30 minutes prior to collection. Samples were collected using sterile tubes (provided by the clinic Neolab LTD, where the laboratory analysis of cortisol was performed), stored at +4 °C, and transported under cold conditions to the laboratory. Cortisol concentrations were quantified using a competitive enzyme-linked immunosorbent assay (ELISA) (Enzo Biochem Inc., USA). The assay employs a monoclonal antibody to cortisol, with results read at 405 nm; color intensity is inversely proportional to cortisol concentration. Saliva samples were centrifuged and analyzed by enzyme-linked immunosorbent assay (ELISA) kit, provided by Enzo Biochem Inc., USA, following the salivary collecting protocols.

Labor characteristics collected included:

- Type of delivery (spontaneous vaginal, assisted, cesarean)
- Obstetric complications (e.g., weakness of labor activity, fetal distress, postpartum hemorrhage)

Neonatal outcomes included:

- Apgar scores at 1 and 5 minutes
- Birthweight, length, head circumference, chest circumference

Obstetric and neonatal data were extracted from standardized hospital records. Statistical analyses were performed using SPSS version 23.0 (IBM, USA). Descriptive statistics summarized participant characteristics. Chi-square tests and independent t-tests compared categorical and continuous variables between groups. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for key outcomes. Significance was set at  $p < 0.05$ . Confidence interval: 95%; margin of error: 5%.

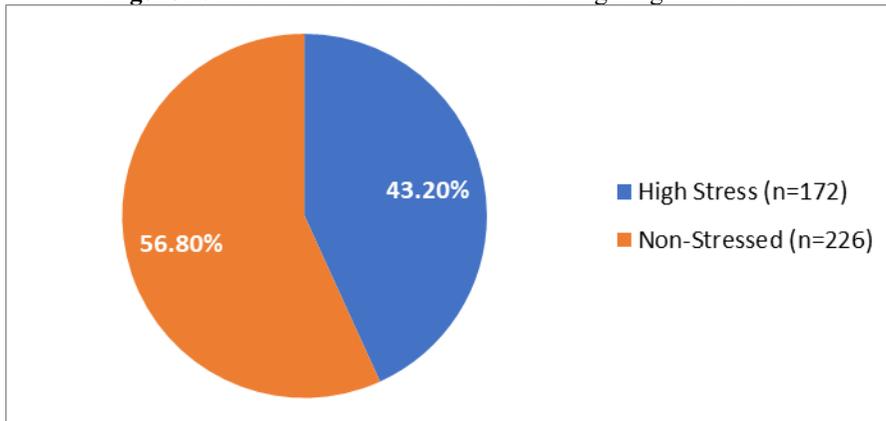
## Results

The mean maternal age was  $29.7 \pm 5.1$  years, with no significant difference between the study and control groups ( $p = .41$ ). Gestational age at delivery averaged  $38.6 \pm 1.5$  weeks. Socioeconomic and parity distributions were comparable, ensuring group homogeneity.

Among the 398 participants, 43.2 % ( $n = 172$ ) reported high stress levels.

Within this subset, the salivary cortisol subgroup ( $n = 95$ ) demonstrated mean cortisol concentrations significantly exceeding normal late-pregnancy values (mean  $14.2 \pm 4.8$   $\mu\text{g/dL}$  vs. expected  $9.1 \pm 3.2$   $\mu\text{g/dL}$ ;  $p < 0.001$ ). This proportion was calculated in order to define the burden of prenatal stress within the study population and to establish the size of the exposed group. (Figure 1.)

**Figure 1:** Distribution of Stress Levels Among Pregnant Women



*Distribution of stress levels among pregnant women (N=398). High stress group: Mean cortisol  $14.2 \pm 4.8$   $\mu\text{g/dL}$ . Vs. Normal  $9.1 \pm 3.2$   $\mu\text{g/dL}$ . ( $p < 0.001$ ).*

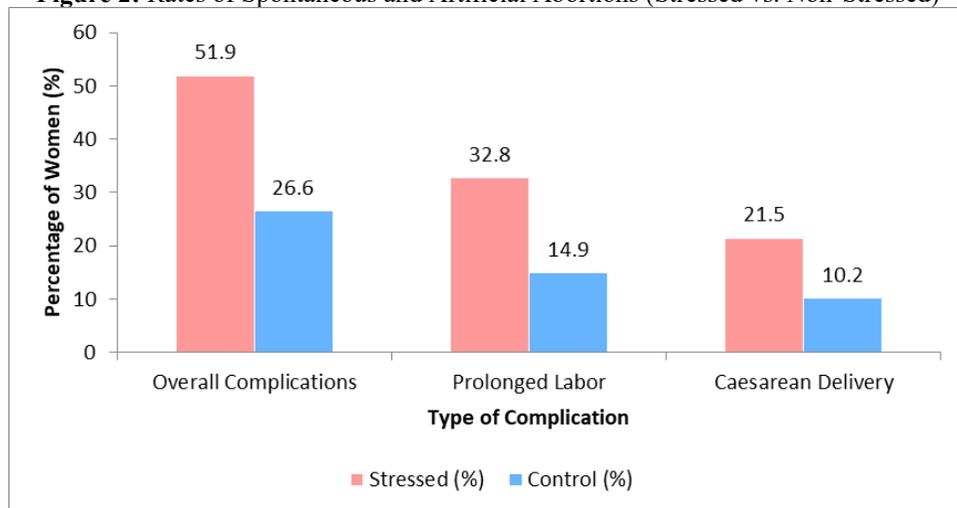
A statistically significant association was found between stress status and occupational activity. Employed women—either full-time or part-time—were more frequently represented in the stressed group than among controls (OR = 1.59;  $p = 0.02$ ; 95 % CI = 1.08–2.34). Within this subgroup, overtime work during pregnancy was strongly linked to stress (OR = 3.05;  $p = 0.001$ ; 95 % CI = 1.59–5.84). Likewise, continuing regular work duties throughout

pregnancy was more common among stressed participants (OR = 1.82;  $p = 0.01$ ; 95 % CI = 1.10–3.02). This analysis was performed to determine whether occupational activity represents a contextual risk factor for prenatal stress. The result indicates that employed women were significantly more likely to belong to the stressed group, suggesting that work-related demands contribute to psychological burden during pregnancy.

Among environmental and relational factors, pregnant women living with only one or two household members showed higher stress prevalence than those living in extended families (OR = 1.59;  $p = 0.038$ ; 95 % CI = 1.02–2.45). Comparing small households with extended-family settings quantifies the protective role of shared living and interpersonal support. The result indicates that limited household support is associated with a higher likelihood of stress during pregnancy. Similarly, the absence of a marital partner was associated with greater stress exposure ( $p = 0.01$ ), suggesting that reduced social support contributes to psychological vulnerability during pregnancy.

The rates of both spontaneous abortion (32.0 % vs. 17.7 %; OR = 2.19;  $p = 0.001$ ; 95 % CI = 1.37–3.49) and artificial abortion (21.5 % vs. 11.5 %; OR = 2.10;  $p = 0.007$ ; 95 % CI = 1.22–3.64) were significantly higher among women in the stressed group. This outcome provides evidence that prenatal stress is associated with increased biological vulnerability in early pregnancy. The significantly higher rate of spontaneous and artificial abortions among stressed women indicates that stress may act as an independent risk factor for adverse pregnancy continuation. (Figure 2.)

**Figure 2:** Rates of Spontaneous and Artificial Abortions (Stressed vs. Non-Stressed)



*Rates of spontaneous and artificial abortions among stressed vs. non-stressed pregnant women ( $p = 0.001$  and  $p = 0.007$  respectively)*

No significant differences were observed regarding residence, education, material provision, income, family environment, or access to prenatal care ( $p > 0.05$ ).

In summary, the findings indicate that occupational overload, limited social and familial support, and adverse reproductive histories are associated with stress during pregnancy, underscoring the multidimensional nature of maternal psychosocial risk factors.

Delivery complications occurred in 51.9 % of stressed women compared with 26.6 % of controls ( $p = 0.001$ ). The most frequent complications were:

- Prolonged labors (32.8 % vs. 14.9 %)
- Caesarian delivery (21.5 % vs. 10.2 %)

A statistically significant association was observed between severe maternal stress and weakness of delivery activities. Among women experiencing severe stress, 64.5% demonstrated inefficient uterine contractions, compared to only 4.3% in those with moderate or mild stress ( $p < 0.001$ ). The calculated odds ratio (OR = 40.00, 95% CI = 4.73–338.23) indicates that severely stressed women were approximately forty times more likely to develop weak delivery activity. This exceptionally strong and significant association suggests that heightened maternal stress may exert a direct inhibitory effect on uterine contractility through cortisol- and catecholamine-mediated pathways (Table 1).

**Table 1:** Weakness of delivery activities (weak/inefficient uterine contractions during labor)

Weaknesses of Delivery Activities	Severe Stress (n = 31)	Moderate or Mild Stress (n = 23)
No	11 (35.5%)	22 (95.7%)
Yes	20 (64.5%)	1 (4.3%)
Total	31	23

Odds Ratio (OR) = 40.00

95% Confidence Interval (CI) = 4.73–338.23

$p < 0.001$

The odds of experiencing any complication were 2.21 times higher among stressed women (95 % CI = 1.13–4.33,  $p = 0.021$ ).

Newborns of stressed mothers were twice as likely to exhibit low 1-minute Apgar scores ( $<7$ ) (OR = 2.12,  $p = .028$ ). However, the difference in 5-minute Apgar scores was not statistically significant (OR = 1.13,  $p = .257$ ).

No significant differences were observed in birthweight or head circumference after adjusting for gestational age.

The key findings are demonstrated in Table 2.

**Table 2:** Summary Table of the key findings

Outcome	Stressed (n=172)	Non-Stressed (n=226)	Odds Ratio (95% CI)	p-value
Delivery complications	51.9 %	26.6 %	2.21 (1.13–4.33)	0.021
Low 1-min Apgar ( $<7$ )	17.4 %	8.6 %	2.12 (1.08–4.12)	0.028
Low 5-min Apgar ( $<7$ )	3.4 %	2.9 %	1.13 (0.59–2.16)	0.257
Mean maternal cortisol levels ( $\mu\text{g/dL}$ )	14.2 $\pm$ 4.8	9.1 $\pm$ 3.2	—	$<0.001$

## Discussion

The present study identifies significant associations between maternal stress during pregnancy and adverse intrapartum and early neonatal outcomes, namely an increased risk of delivery complications and reduced neonatal vitality as reflected by lower 1-minute Apgar scores. These findings are consistent with a substantial body of international evidence demonstrating that psychological and physiological stress responses during pregnancy are linked to impaired obstetric performance and compromised fetal adaptation (Glynn et al., 2018; Van den Bergh et al., 2020). In this respect, the current results corroborate established patterns observed across diverse populations and healthcare systems.

The more than twofold increase in delivery complications among stressed mothers is biologically plausible and aligns with experimentally and clinically supported mechanisms. Prior research has shown that hyperactivation of the hypothalamic–pituitary–adrenal (HPA) axis during pregnancy leads to elevated circulating cortisol and catecholamines, which suppress uterine contractility and interfere with oxytocin-mediated signaling pathways (Dunkel Schetter & Tanner, 2012). These neuroendocrine effects are known to impair myometrial efficiency and prolong labor. In addition, stress-induced vasoconstriction can reduce uteroplacental perfusion, increasing the likelihood of fetal distress and abnormal intrapartum heart-rate patterns—complications that were frequently observed in the present cohort. While the current study does not directly measure uterine dynamics or placental blood flow, the observed clinical patterns are consistent with these well-established physiological pathways.

The finding that low Apgar scores were more prevalent at 1 minute but not at 5 minutes suggests that neonatal compromise was predominantly transient. Established neonatal literature interprets such patterns as indicative of delayed adaptation rather than irreversible injury. This interpretation is supported by experimental and epidemiological data demonstrating that acute intrapartum stressors can temporarily depress neonatal tone and respiration without leading to sustained hypoxic damage. Nevertheless, longitudinal studies indicate that when early neonatal instability co-occurs with prenatal stress exposure, it may contribute to less favorable developmental trajectories (Bussi eres et al., 2015; O’Donnell et al., 2020). The present data therefore, align with existing evidence on early vulnerability, although direct developmental outcomes cannot be inferred from this study.

The associations observed here are consistent with large-scale syntheses of prior research. Meta-analyses by Staneva et al. (2020) and Lebel et al. (2022) demonstrate that antenatal anxiety significantly increases the likelihood of cesarean and assisted deliveries, reinforcing the concept that psychological states exert measurable effects on parturition. Similarly, Glynn

et al. (2018) report stress-related downregulation of oxytocin receptors as a mechanistic contributor to dysfunctional labor. The current findings extend these well-documented relationships to a Georgian population, in which such associations had not previously been empirically examined.

Within Georgia, no prior studies have systematically evaluated prenatal stress in relation to obstetric or early neonatal outcomes. The present investigation therefore, provides the first population-based evidence that global stress–outcome pathways are also operative in this context. This does not in itself establish causality, but it demonstrates that Georgian pregnant women exhibit stress-related risk patterns comparable to those observed internationally. From an interpretative standpoint, this suggests that culturally adapted mental-health interventions integrated into perinatal care may be both relevant and necessary in this setting.

Beyond immediate perinatal outcomes, extensive experimental and longitudinal research supports the concept of fetal programming by maternal stress. Excess prenatal cortisol exposure has been shown to modify glucocorticoid receptor expression (NR3C1) and alter amygdala–prefrontal circuitry, increasing susceptibility to affective and attentional disorders later in life (Palma-Gudiel et al., 2019; Kim et al., 2021). Cohort studies further demonstrate that children of highly stressed mothers display heightened cortisol reactivity, cognitive delays, and impaired emotional regulation across childhood and adolescence (O’Donnell & Meaney, 2017; Van den Bergh et al., 2020). While the present study does not include postnatal follow-up, these established pathways provide a biologically grounded framework within which the observed intrapartum effects acquire broader developmental significance. On this basis, it is reasonable to interpret prenatal stress as a modifiable upstream determinant of longer-term vulnerability, even though direct developmental outcomes remain beyond the scope of the current data. The psychosocial correlates identified in this cohort—particularly occupational overload, financial insecurity, and limited social support—mirror patterns reported in other middle-income contexts characterized by constrained social safety nets and limited maternity protections (Giurgescu et al., 2015; Guardino & Dunkel Schetter, 2014). Evidence from randomized and quasi-experimental studies indicates that stress-reduction programs emphasizing peer support, partner involvement, and mindfulness-based interventions can lower maternal cortisol levels and improve birth outcomes (Beddoe et al., 2009; Lebel et al., 2022). The present findings do not directly test such interventions; however, they identify a population in which these evidence-based strategies may be particularly relevant.

Several limitations warrant consideration. The cross-sectional design precludes causal inference and limits assessment of temporal dynamics. Cortisol was measured once and only in a subgroup, which does not capture

diurnal variation or longitudinal endocrine patterns. Although participants were clinically healthy, unmeasured confounders—such as nutritional status or subclinical inflammation—may have contributed to the observed associations. Nonetheless, the large sample size, standardized stress assessment, and integration of subjective and biological markers strengthen the robustness of the findings.

This investigation is among the first in Georgia to integrate psychosocial and biochemical indices of maternal stress with detailed obstetric outcomes. By situating local data within an established international evidence base, it provides a foundation for future longitudinal research and for the development of context-sensitive preventive strategies in Georgian perinatal care.

### **Public Health and Policy Implications**

The findings of this study carry direct and urgent implications for clinical practice and public-health policy in Georgia. Maternal stress emerges not as a peripheral emotional state but as a measurable biopsychosocial determinant of perinatal health—one that shapes obstetric performance, early neonatal vitality, and potentially the child’s long-term developmental trajectory. In this context, addressing prenatal stress is no longer optional; it is a necessary component of safe and effective maternity care.

International frameworks already provide a clear mandate. The World Health Organization (WHO, 2022) and the European Regional Office of the United Nations Population Fund (UNFPA, 2023) identify maternal mental health as a cornerstone of safe motherhood. Yet in Georgia, antenatal services remain largely biomedical in focus, prioritizing blood pressure, glucose levels, and infection screening, while psychological distress, occupational burden, and social strain are rarely assessed in a systematic manner. This gap represents a missed opportunity for early risk detection and prevention.

Integrating routine stress and anxiety screening—using validated tools such as the Pregnancy-Specific Stress Questionnaire (PSSQ)—into standard antenatal visits would immediately strengthen Georgia’s perinatal care framework. Early identification of vulnerable women would enable timely counseling, referral to mental-health services, and individualized care planning, thereby reducing preventable obstetric and neonatal complications.

The demonstrated link between stress and labor outcomes also calls for a structural reorientation of prenatal care toward interdisciplinary models. Antenatal teams that combine obstetric, psychological, and social expertise are no longer a luxury but a necessity. Evidence from Scandinavian and Western European systems shows that collaborative care reduces adverse outcomes and enhances maternal satisfaction (Lebel et al., 2022). Georgia can adapt these models by equipping midwives and general practitioners with core

competencies in psychological first aid and stress-management strategies. Embedding mental-health training within obstetrics curricula at women's consultations and maternity homes in Tbilisi would operationalize this shift.

The psychosocial correlates identified in this study—particularly occupational and financial strain—reflect broader structural determinants of health. In Georgia, many women continue full professional duties into late gestation without formal accommodations. This reality positions workplace stress as a critical yet underrecognized contributor to prenatal vulnerability. Policy reform should therefore extend beyond healthcare institutions and into labor regulation, including the enforcement of maternity-friendly workplace standards, flexible schedules, and protection from occupational hazards. Such measures have been shown to reduce stress and improve neonatal outcomes (Giurgescu et al., 2015; Guardino & Dunkel Schetter, 2014).

Public-health initiatives must also challenge cultural norms that minimize emotional distress during pregnancy. National campaigns, prenatal education programs, and digital health platforms can normalize discussions of mental well-being and empower women to seek support. Actively involving partners and family members in stress-reduction strategies further strengthens protective social networks.

These results provide concrete evidence base for Georgia's alignment with *WHO's 2030 goals for maternal, infant, and child health and the Comprehensive Mental Health Action Plan (2013–2030)*. Policymakers are encouraged to:

- Establish national guidelines for prenatal stress screening and management.
- Integrate psychosocial and, where indicated, cortisol monitoring into high-risk antenatal care;
- Allocate funding for professional training in perinatal mental health.
- Support longitudinal research on the intergenerational effects of prenatal stress.

By recognizing maternal stress as both a clinical variable and a public-health priority, Georgia has the opportunity to assume regional leadership in holistic maternal care. Bridging biological and social dimensions of pregnancy will ensure that “safe motherhood” encompasses not only physical survival, but psychological resilience and developmental equity for future generations.

## Conclusions

This study demonstrates that maternal stress during pregnancy constitutes a significant multidimensional determinant of obstetric and neonatal outcomes. In a cohort of 398 pregnant women in Tbilisi, elevated psychological stress and increased salivary cortisol levels were associated with a higher incidence of delivery complications and reduced early neonatal

vitality. These findings elucidate the biological and psychosocial pathways—particularly hypothalamic–pituitary–adrenal (HPA) axis activation—through which prenatal stress may disrupt labor dynamics and immediate neonatal adaptation.

The implications extend beyond childbirth. Sustained prenatal stress has the potential to shape long-term neurodevelopmental vulnerability, positioning the maternal–fetal stress continuum as both a clinical and public health priority. Recognizing prenatal stress as a modifiable risk factor supports the integration of routine psychosocial screening, targeted stress-management interventions, and biomarker monitoring into standard antenatal care. In the Georgian context, embedding psychological and social support services within maternity care pathways could address existing gaps and meaningfully improve maternal and neonatal outcomes.

At the policy level, incorporating maternal mental health into national reproductive health strategies is essential for advancing global objectives in maternal and child well-being. Emotional health should be regarded as an integral component of comprehensive antenatal care rather than a peripheral concern.

Ultimately, safeguarding the emotional stability of expectant mothers represents an investment in future generations. By addressing prenatal stress through evidence-based, compassionate, and culturally responsive approaches, healthcare systems can transform a trajectory of risk into one of resilience and healthier beginnings.

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