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Chronic Daily Stress and Its Impact on Glycemic Elevation and Diabetes Mellitus: A Multicenter Study

ABSTRACT

Objective: Chronic psychosocial stress is increasingly recognized as a contributor to impaired glucose regulation and diabetes progression. This study examined the association between daily stress levels and glycemic control in adults with long-standing diabetes.

Methods: A multicenter cross-sectional study was conducted in five hospitals several public and private hospitals in West Sumatra from July 2023 to February 2025 involving 214 adults with type 2 diabetes (≥3 years). Stress was measured using the Perceived Stress Scale, and fasting plasma glucose (FPG), postprandial glucose (PPG), and glycated hemoglobin (HbA1c) were analyzed. Multivariable linear regression adjusted for age, sex, body mass index, diabetes duration, and medication use.

Results: Higher stress scores were significantly correlated with increased FPG ($r = 0.62$, $P < .001$), PPG ($r = 0.55$, $P < .001$), and HbA1c ($r = 0.48$, $P < .001$). Each 1-unit increase in stress score predicted rises of 2.8 mg/dL in FPG, 4.5 mg/dL in PPG, and 0.07% in HbA1c, independent of confounders.

Conclusion: Chronic daily stress is strongly associated with poor glycemic control. Integrating stress management strategies may improve metabolic outcomes among diabetic patients.

Keywords: Chronic stress, diabetes mellitus, glycemic control, insulin resistance, neuroendocrine mechanisms

Introduction

Type 2 diabetes mellitus (T2DM) is a major and growing global health burden, affecting more than 500 million adults worldwide and projected to become one of the leading causes of morbidity and mortality by 2045. It is characterized by insulin resistance, progressive β-cell dysfunction, and chronic hyperglycemia that together accelerate the development of macrovascular and microvascular complications such as cardiovascular disease, nephropathy, retinopathy, and neuropathy. In addition to well-established risk factors such as sedentary lifestyle, obesity, and genetic predisposition, accumulating evidence implicates chronic psychosocial stress as a critical but often overlooked determinant of both the onset and progression of glycemic dysregulation.[1-4]

Psychosocial stress, defined as a sustained perception of threat or pressure that exceeds an individual's adaptive capacity, elicits a coordinated physiological response involving the hypothalamic–pituitary–adrenal (HPA) axis and the sympathoadrenal system. These pathways promote the release of glucocorticoids (primarily cortisol) and catecholamines (epinephrine and norepinephrine), key hormones that modulate energy metabolism, immune activity, and cardiovascular function. When activated acutely, this neuroendocrine stress response is adaptive and ensures adequate glucose supply to vital organs. However, when stress becomes chronic, persistent elevations in these hormones lead to metabolic perturbations including enhanced hepatic gluconeogenesis, increased glycogenolysis, reduced insulin sensitivity, and dysregulated inflammatory signaling.[5-8]

At the biomolecular level, chronic stress is believed to induce insulin resistance through multiple interlinked mechanisms. These include the upregulation of hepatic gluconeogenic enzymes such as phosphoenolpyruvate carboxykinase and glucose-6-phosphatase, increased adipose tissue lipolysis and the consequent rise in circulating free fatty acids, as well as the activation of proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). These inflammatory mediators interfere with insulin receptor signaling by promoting serine phosphorylation of insulin receptor substrate proteins,

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leading to impaired phosphoinositide 3-kinase–protein kinase B (PI3K–AKT)signaling pathway activation and decreased glucose uptake in skeletal muscle and adipose tissues.[9-11] Furthermore, oxidative stress resulting from the overproduction of reactive oxygen species (ROS) and the diminished antioxidant defense system contributes to both systemic inflammation and endothelial dysfunction, worsening insulin resistance and promoting atherogenic processes.

Parallel to these peripheral mechanisms, chronic stress may also exacerbate β-cell dysfunction in the pancreas. Sustained exposure to hyperglycemia and elevated glucocorticoid levels triggers glucotoxicity, mitochondrial oxidative damage, and endoplasmic reticulum stress in β-cells, thereby impairing insulin synthesis and secretion. Epigenetic modifications induced by chronic stress—such as DNA methylation and histone acetylation changes—can further alter the expression of genes regulating insulin production, apoptosis, and metabolic adaptation.[12-14] Collectively, these processes create a vicious cycle wherein stress-driven hormonal and molecular alterations reinforce hyperglycemia and β-cell failure.

Despite the growing mechanistic understanding of stress-induced metabolic dysfunction, robust clinical evidence linking real-world daily stress exposure to longitudinal glycemic deterioration remains scarce, particularly in regions with distinct cultural, occupational, and environmental stressors compared to Western populations. Therefore, this multicenter study conducted across several hospitals in West Sumatra sought to fill this knowledge gap by examining whether chronic daily stress, evaluated over several months, is significantly associated with elevated fasting blood glucose and glycated hemoglobin (HbA1c) levels among individuals with established T2DM. The present investigation integrates psychometric assessments of perceived stress with biochemical and biomolecular parameters to elucidate the stress–metabolic axis in an adult diabetic population in across several public and private hospitals in West Sumatra.

Material and Methods

Design and Setting

A multicenter, cross-sectional observational study was conducted across both public and private healthcare facilities to ensure heterogeneity and enhance external validity of findings. The study was performed at three state hospitals and two private hospitals in West Sumatera between July 2023 and February 2025. This timeframe captured seasonal and occupational variations in stress exposure and glycemic control.

Inclusion criteria were: (1) age ≥56 years, representing a population with higher physiological stress vulnerability and cumulative metabolic risk;[1,2] (2) confirmed diagnosis of T2DM for at least 3 years based on the American Diabetes Association (ADA) diagnostic criteria;[3] (3) employment status in either government or private institutions, reflecting different psychosocial stressors and workload characteristics;[4] and (4) both male and female participants were included to explore possible sex-based biomolecular differences in stress response and glucose regulation.[5,6]

Exclusion criteria comprised the presence of acute infection or inflammation (to avoid stress-related transient hyperglycemia),[7] hospitalization within the last 3 months, any known endocrine disorders such as Cushing's syndrome or hyperthyroidism, and ongoing systemic glucocorticoid therapy, which can confound cortisol-mediated glucose elevation.[8,9]

A total of 214 eligible participants provided written informed consent and underwent a comprehensive evaluation. Ethical approval was obtained in 20th of June 2023 (No. 254/KEPK/2023) from the Institutional Ethics Committee of Health Polytechnic of the Ministry of Health, West Sumatera in accordance with the Declaration of Helsinki.[10]

Stress Assessment

Psychosocial stress was assessed using the Perceived Stress Scale (PSS-10), a widely validated instrument adapted for the Indonesian population with established reliability (Cronbach's $\alpha = 0.85$).[11,12] The PSS-10 comprises 10 items, each scored from 0 (never) to 4 (very often), yielding a total score ranging from 0 to 40; higher scores indicate greater perceived stress intensity.

In addition to the PSS-10, participants completed a structured demographic and occupational stressor questionnaire that included working hours, job strain, sleep quality, social support, and income stability—all previously recognized as major determinants of stress affecting metabolic and hormonal pathways.[13-15]

The biomolecular rationale underpinning this component is that chronic activation of the HPA axis due to prolonged stress exposure results in elevated cortisol and catecholamine release, leading to increased hepatic gluconeogenesis, lipolysis, and inhibition of peripheral glucose uptake.[16-18] These mechanisms are known to contribute to insulin resistance and sustained hyperglycemia, forming the pathophysiological link under investigation in this study.

Glycemic and Biomarker Measurements

Blood samples were obtained after an overnight fast of at least 8 hours to determine fasting plasma glucose (FPG), followed by a standardized meal challenge to measure 2-hour postprandial glucose (PPG). Venous blood was drawn and analyzed using the enzymatic glucose oxidase–peroxidase method calibrated to ISO 15189 laboratory standards.[19]

Glycated hemoglobin levels were determined using high-performance liquid chromatography (Bio-Rad D-100 system) as per National Glycohemoglobin Standardization Program criteria.[20] Internal and external quality controls were performed monthly.

In a subsample (n = 120), serum cortisol levels were measured using a chemiluminescent immunoassay (Abbott Architect i2000SR) to serve as a molecular biomarker of stress activity.[21] Additional biochemical covariates included the fasting lipid profile (total cholesterol, low-density lipoprotein, high-density lipoprotein, triglycerides) and

MAIN POINTS

- Chronic daily stress is significantly associated with elevated fasting, postprandial glucose, and glycated hemoglobin levels.
- Each 1-unit increase in perceived stress score predicted a measurable increase in glycemic levels, independent of confounders.
- Psychosocial stress is a modifiable metabolic risk factor for insulin resistance.
- Stress management strategies may enhance long-term glycemic control in patients with diabetes.

C-reactive protein, an inflammatory marker that may modulate insulin sensitivity.[22]

Covariates and Confounding Control

Key clinical and lifestyle variables, including age, sex, body mass index (BMI), waist circumference, blood pressure, duration of diabetes, and use of anti-diabetic medications (metformin, insulin, sulfonylurea, dipeptidyl peptidase-4 inhibitors, or combination therapy), were recorded. These parameters are established confounders of glucose metabolism and stress reactivity.[23,24]

To ensure measurement accuracy, anthropometric assessments were performed using calibrated equipment by trained nurses. All data were double-entered and cross-verified for consistency.

Statistical Analysis

Descriptive statistics are presented as mean ± standard deviation (SD) for continuous variables and frequencies (%) for categorical variables. Normality was assessed using the Shapiro–Wilk test. Pearson's correlation coefficient (r) or Spearman's rank test was applied to assess univariable associations between perceived stress and glycemic indices (FPG, PPG, HbA1c, cortisol).

To determine independent predictors of hyperglycemia, multivariable linear regression models were constructed, adjusting for age, sex, BMI, diabetes duration, and medication use.[25-27] The regression coefficients (β) with 95% confidence intervals were reported.

Stratified analyses were performed by sex and by employment sector (government vs. private) to evaluate interaction effects on stress–glucose dynamics.[28] Model diagnostics included multicollinearity assessment (VIF < 5) and residual normality testing. All analyses were conducted using Statistical Package for the Social Sciences version 27.0 (IBM SPSS Corp.; Armonk, NY, USA), and 2-tailed $P < .05$ was considered statistically significant.[29,30]

Participant Characteristics

A total of 214 participants were included in the final analysis. The mean age was 59.8 ± 3.4 years, with 108 males (50.5%) and 106 females (49.5%). The mean duration of diabetes was 8.5 ± 2.1 years, indicating that most participants had long-standing type 2 diabetes with progressive insulin resistance. The average BMI was 26.3 ± 3.5 kg/m^2, classifying most participants as overweight, consistent with previous evidence linking adiposity to glucocorticoid-driven metabolic dysregulation.[1,2]

The mean PSS score was 21.4 ± 5.8 (range: 7-35), indicating a moderate-to-high level of daily psychological stress. Glycemic control was generally poor, with mean FPG = 165.2 ± 38.7 mg/dL, PPG = 228.5 ± 54.3 mg/dL, and HbA1c = 8.1 ± 1.4%, all exceeding ADA-recommended targets for optimal diabetic management.[3]

The observed demographic and clinical characteristics reflect a typical high-risk metabolic profile found in aging diabetic populations, where persistent stress exposure, limited physical activity, and suboptimal treatment adherence contribute to glycemic instability.[4,5] From a biomolecular standpoint, chronic stress may induce overactivation of the HPA axis, resulting in excess cortisol and catecholamine secretion that enhances hepatic gluconeogenesis and inhibits peripheral glucose uptake.[6-8]

Correlation Analyses

As shown in Table 1, stress scores were strongly positively correlated with all measured glycemic parameters. The correlation coefficients were $r = 0.62$ ($P < .001$) for FPG, $r = 0.55$ ($P < .001$) for PPG, and $r = 0.48$ ($P < .001$) for HbA1c. These associations indicate that higher perceived stress levels were consistently associated with poorer glycemic control.

Table 1. Correlation Between Perceived Stress and Glycemic Parameters

Parameters	Correlation Coefficient (r)	P
PSS score vs. FPG	0.62	<.001
PSS score vs. PPG	0.55	<.001
PSS score vs. HbA1c	0.48	<.001

FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; PPG, postprandial glucose; PSS, Perceived Stress Scale.

The magnitude of these correlations aligns with findings from previous studies that link psychosocial stress and hyperglycemia via cortisol-mediated metabolic pathways.[9-11] Elevated cortisol levels promote hepatic glucose output and suppress insulin receptor signaling, leading to decreased GLUT4 translocation and reduced glucose uptake in skeletal muscle.[12-14] Such stress-induced insulin resistance explains the observed increase in HbA1c among participants with higher stress levels.

Multivariable Regression

After adjusting for age, sex, BMI, diabetes duration, and medication regimen, stress remained an independent and highly significant predictor of glycemic parameters (Table 2).

Each 1-point increase in stress score was associated with a 2.8 mg/dL increase in FPG, a 4.5 mg/dL increase in PPG, and a 0.07% increase in HbA1c. The adjusted R^2 values indicate that perceived stress explained approximately 28%-42% of the variance in glycemic outcomes, confirming that psychological factors exert a measurable metabolic burden independent of traditional risk factors.[15,16]

At the biomolecular level, stress hormones such as cortisol, epinephrine, and norepinephrine activate cAMP–protein kinase A signaling pathways, enhancing gluconeogenic gene expression (e.g., PEPCK and G6Pase) and impairing insulin-mediated suppression of glucose production.[17,18] Chronic overexposure to these hormones leads to β-cell exhaustion, further worsening glycemic regulation.[19,20]

Stratified Analyses

Subgroup analysis (Table 3) demonstrated that the stress–glycemia relationship was consistent across sex and occupational groups, though it was slightly more pronounced among females and government employees.

This gender-related difference aligns with evidence that female neuroendocrine responses to stress involve greater sensitivity to cortisol and corticotropin-releasing hormone, thereby amplifying glucose dysregulation under chronic stress exposure.[21,22] Government employees may experience prolonged occupational

Table 2. Multivariable Linear Regression: Effect of Stress Score on Glycemic Parameters

β coefficient per 1-unit PSS increase (95% CI)	Adjusted R^2	P
FPG (mg/dL)	2.8 (2.1-3.5)	<.001
PPG (mg/dL)	4.5 (3.2-5.8)	<.001
HbA1c (%)	0.07 (0.04-0.10)	<.001

FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; PPG, postprandial glucose; PSS, Perceived Stress Scale.

Table 3. Regression Slopes by Subgroup

Subgroup	β for FPG (mg/dL per PSS Unit)	β for HbA1c (%)	*P*
Males	2.5	0.06	<001
Females	3.1	0.08	<.001
Government employees	3.0	0.08	<.001
Private employees	2.6	0.06	<.001

FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; PSS, Perceived Stress Scale.

tension and administrative responsibility, further exacerbating sympathetic overdrive and neuroendocrine imbalance.[23,24]

Collectively, these findings confirm that daily psychological stress contributes significantly to interindividual variability in glycemic control among diabetic adults, providing empirical and biomolecular evidence of a stress–metabolism interaction.[25-30]

Discussion

This multicenter study involving 214 adults with diabetes demonstrated a strong association between chronic daily stress and poor glycemic control, even after adjustment for key covariates. The findings support that psychosocial stress is a modifiable factor influencing glucose regulation through biological mechanisms rather than merely reflecting poor disease management.[15]

Consistent with prior studies, individuals with high perceived stress exhibited higher fasting glucose and HbA1c, confirming that chronic stress has both physiological and behavioral effects.[16,17] While acute stress causes transient cortisol spikes, chronic exposure leads to cumulative neuroendocrine and metabolic disruption.[11] The results extend previous work by focusing on older patients from Health Polytechnic of the Ministry of Health, West Sumatera and employing robust multivariable adjustments, emphasizing the clinical importance of daily stress in long-term glycemic regulation.[18]

From a mechanistic perspective, several interconnected pathways explain this relationship. Chronic activation of the HPA axis elevates cortisol, promoting hepatic glucose production and impairing insulin-mediated uptake.[2,9,23] Sympathetic overactivity increases catecholamine release, thereby enhancing glycogenolysis and inhibiting insulin secretion, which, over time, desensitizes β-cells.[24] Concurrent inflammation and oxidative stress elevate levels of cytokines (TNF-α, IL-6) and ROS, thereby disrupting insulin receptor signaling and reducing glucose uptake.[26] Lipotoxicity from excess free fatty acids exacerbates hepatic and muscular insulin resistance,[29] while epigenetic modifications—such as altered DNA methylation and microRNA expression—further suppress the expression of genes essential for insulin production and glucose metabolism.[30]

Collectively, these mechanisms create a vicious cycle of hormonal and metabolic imbalances that sustain hyperglycemia and accelerate β-cell exhaustion.[10] The findings highlight that stress is not only psychological but also a biological disruptor that perpetuates metabolic dysfunction.[27]

Clinically, this underscores the need for a biopsychosocial approach in diabetes care, combining stress management interventions—such as mindfulness, relaxation, and cognitive-behavioral therapy—with standard pharmacological treatment.[16-18] These strategies may attenuate HPA hyperactivity, lower inflammation, and enhance glycemic resilience.[20]

Strengths of this study include its multicenter design, standardized assays, and statistical adjustment for confounders. Limitations include its cross-sectional design, the absence of direct physiological stress biomarkers, and unmeasured lifestyle factors such as diet and sleep. Longitudinal and interventional studies are warranted to establish causality.

Routine stress assessment and psychosocial interventions should be incorporated into diabetes management. Addressing inflammation and oxidative stress may offer additional therapeutic targets for highly stressed individuals.

This study demonstrates that chronic psychosocial stress significantly contributes to poor glycemic control among individuals with diabetes mellitus. The findings confirm that stress acts not only as an emotional state but also as a persistent biological factor that disrupts neuroendocrine, inflammatory, and metabolic systems.

Chronic activation of the HPA axis and sympathetic nervous system increases hepatic glucose output, reduces peripheral glucose uptake, and impairs β-cell function, while inflammatory and oxidative processes further aggravate insulin resistance. These mechanisms collectively sustain hyperglycemia and accelerate metabolic dysfunction.

Clinically, psychosocial stress should be recognized as a modifiable determinant of diabetes management. Interventions such as mindfulness, relaxation therapy, behavioral counseling, physical activity, and social support can enhance glycemic stability and prevent complications. Integrating stress evaluation into routine diabetes care enables early identification of high-risk patients and supports personalized, holistic management that addresses both physiological and psychological well-being.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no Artificial Intelligence Tool was used in the preparation of the manuscript

Ethics Committee Approval: This study was reviewed and approved by the Research Ethics Committee Health Polytechnic of the Ministry of Health West Sumatra (Approval No. 254/KEPK/2023, Date: 20.06.2023).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

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