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The Effect of Psychological Stress on Male Fertility Parameters: A Comparative Study Between Chronic Psychological Stress and Low-Stress

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ABSTRACT

Background: *Chronic psychological stress is increasingly recognized as a risk factor for male reproductive dysfunction. It may disrupt the GnRH–gonadotrophin axis and induce oxidative stress, negatively affecting spermatogenesis.* ***Purpose****: This study examined the relationship between chronic psychological stress and semen quality, including standard semen parameters and selected fertility indicators.* ***Methods****: Between 2023 and 2025, a cross-sectional study was done on 80 men selected from 500 participants. Forty participants were classified as low-stress and forty as high-stress using a validated stress questionnaire. Semen analysis was performed according to WHO guidelines, assessing sperm concentration, motility, morphology, and semen volume. Reproductive hormones and sperm DNA integrity were also evaluated when applicable. Statistical analyses were used to determine associations between stress and fertility parameters.* ***Results****: Higher levels of chronic psychological stress were significantly associated with lower sperm concentration, reduced progressive motility, and increased abnormal sperm morphology. Stress scores showed an inverse relationship with testosterone levels and sperm function, suggesting impaired male reproductive capacity.* ***Conclusion***: Chronic *psychological stress is strongly linked to reduced male fertility. These findings highlight the importance of psychological well-being in the evaluation and management of male infertility and suggest that stress-reduction strategies may improve reproductive outcomes.*

Keywords: Psychological stress, Male infertility, Semen parameters, DNA fragmentation.

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INTRODUCTION

Infertility is a universal health problem that affects about 10–15% of couples worldwide, with male factor alone being responsible for almost half of the cases (1). Even though there are new diagnostic methods, a high percentage of male infertility is still idiopathic and implies possible modifiable environmental, lifestyle or psychological factors (2). Among them, chronic psychological stress has been increasingly considered to be a possible cause that leads to poor male reproductive output (3). Psychological stress includes maintained emotional or psychological stress and has been reported to be related to changes in semen quality and reproductive outcome by clinical studies (4). Chronic stress induces activation of the hypothalamic–pituitary–adrenal (HPA) axis. This initiates sustained elevation of glucocorticoids, primarily cortisol. These can influence the role of the hypothalamic–pituitary–gonadal (HPG) axis and inhibit the secretion of luteinizing hormone (LH) and follicle-stimulating hormone physiology: gonadotropin inhibitory hormone. This can also

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inhibit testosterone production and result in low testosterone levels (5). The reduction of testosterone can cause damaging effects on spermatogenesis by inhibiting sperm production and maturation (6). Together with disturbances of the endocrine system, chronic psychological stress is also associated with enhanced oxidative and inflammatory responses (7).

Under oxidative stress, generated ROS can damage spermatozoa membranes and break molecular microenvironments such as mitochondria; it also causes the process of lipid peroxidation that is harmful to sperm motility and morphology (8). In addition, oxidative stress is one of the main factors contributing to sperm DNA fragmentation which has been correlated with decreased fertilization potential, altered embryo development, and poor reproductive outcomes (9). Clinical data indeed showed that men who suffer from significantly elevated levels of chronic stress or anxiety may present with ***lower*** sperm concentration, reduced progressive motility and increased percentages of abnormal forms than their less-stressed counterparts (10). Nevertheless, the strength and direction of these relationships in human populations are inconsistent; for example, some studies have indicated that stress may not consistently predict deleterious sperm quality (11).

Because of the amount of chronic stress in today's society and its potential influence on reproductive health, more information is needed to fully investigate the possible mechanisms which link stress with male fertility (12). Better appreciation of this association may allow stress to be validated as a modifiable risk factor and justify the incorporation of psychological evaluation and stress-reducing intervention into standard infertility care (13). Thus, in the present study we sought to examine the relationship between chronic psychological stress and semen quality, hormonal profile as well oxidative stress markers and sperm DNA integrity.

METHODS

Study Design and Participants

This cross-sectional comparative study was conducted between January 2023 and February 2025 at infertility clinics and teaching hospitals in Najaf Governorate, Iraq. Initially, 500 men attending infertility clinics were screened. Based on eligibility criteria and stress assessment results, 80 infertile men aged between 22 and 45 years were enrolled in the study. Participants were divided into two groups according to psychological stress level: low-stress group (n = 40) and high-stress group (n = 40).

Infertility was defined as failure to achieve conception after at least one year of regular unprotected sexual intercourse. Exclusion criteria included smoking, alcohol consumption, chronic systemic diseases (such as diabetes mellitus, hypertension, renal disease, and endocrine disorders), history of reproductive tract infections, varicocele, hormonal therapy within the last six months, urogenital surgery, chemotherapy, radiotherapy, and known genetic causes of infertility.

Ethical Approval

The study was approved by institutional research ethics committee of Al-Furat AL-Awsat Technical university and Najaf Health Directorate. Written informed consent was obtained from all subjects prior to sampling.

Assessment of Psychological Stress

Psychological worry was measured using the validated Perceived worry Scale (PSS-10). The forms contain 10 questions to be scored on a 5-point Likert scale, ranging from 0 (not at all) to 4 (very often). Scores were on a scale of 0–40. Scores of 13 or less were low-stress; scores of 27 or more were high-stress.

Semen Sample Collection and Analysis

Drug used to collect sperm samples were obtained by masturbation after 3–7 days of sexual abstinence using clean wide-mouth containers. Samples were melted at 37°C for 30 min prior to analysis. Semen was analyzed

according to the World Health Organization (WHO, 2021) guidelines for analysis of sperm. Our examination covered the following issus: Semen Volume (mL), Sperm Concentration (×10⁶/mL), Sperm Motility (%), Sperm Total Motility (%) and Sperm Normal Morphology (%).

Makler counting box for analysing sperm count and motility with time observation under a microscope. We also used oil immersion microscopy to examine sperm morphology after Diff -Quik staining.

Hormonal Assay& Assessment of Oxidative Stress Markers

5 mL of blood from a vein was taken between 8 and 10 AM after an overnight fast. Serum was separated by spinning at 3000 rpm for 10 min and stored at -20C before analysis. The concentrations of cortisol and testosterone in the blood were measured according to manufacturer's protocols by using commercial ELISA kits .

To determine levels of malondialdehyde (MDA), an end-product of lipid peroxidation, which is detected as thiobarbituric acid reactive substances (TBARS). 8-OHdG (a marker of oxidative DNA damage) was measured by ELISA kits.

Sperm DNA Fragmentation Assay

Sperm DNA fragmentation was assessed using the Terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assay following the manufacturer's protocol. At least 500 sperm cells per sample were evaluated under fluorescence microscopy.

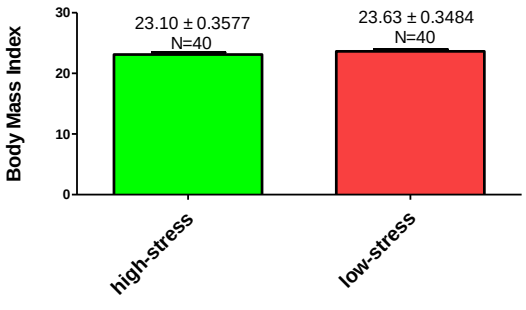
Statistical Analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) software version 26.0. Quantitative variables were expressed as mean ± standard error (Mean ± SE). Comparisons between groups were performed using independent sample t-test. Pearson correlation coefficient was used to evaluate associations between stress scores and fertility parameters. A p-value <0.05 was considered statistically significant.

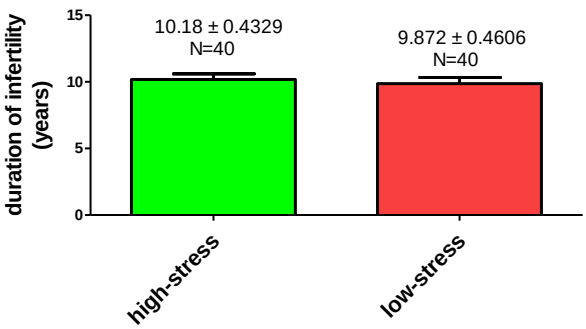
RESULTS

Participant Characteristics

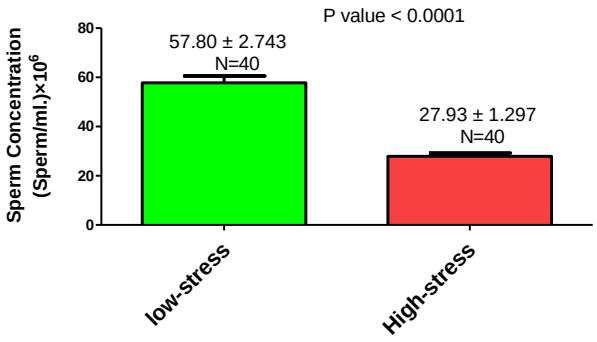
Total of 80 men; 40 high-stress, 40 low-stress. No significant differences in age, BMI, or duration of infertility ($p > 0.05$). Semen Parameters: High-stress group had lower sperm concentration, motility, progressive motility, and higher abnormal morphology ($p < 0.05$). Hormonal Profile: High-stress men had elevated cortisol and lower testosterone. Oxidative Stress and DNA Fragmentation: High-stress group showed increased sperm DNA fragmentation. Stress scores negatively correlated with sperm concentration and motility and positively with DNA fragmentation.



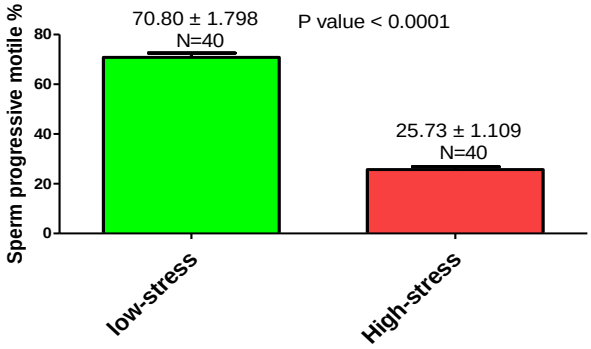
F. (1): Show body mass index.



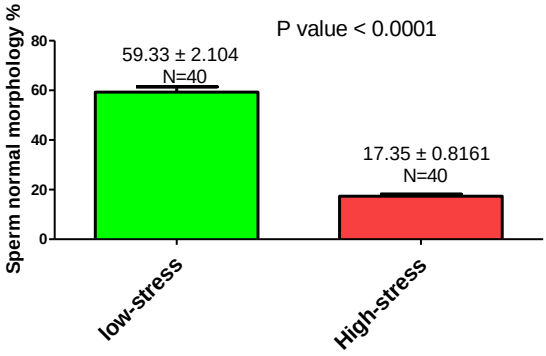
F. (2): Show duration of infertility.



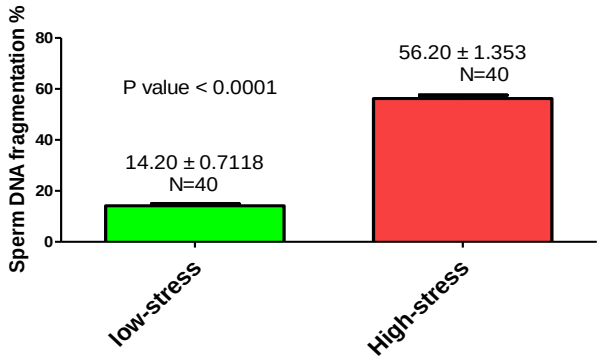
F. (3): Show Sperm Concentration.



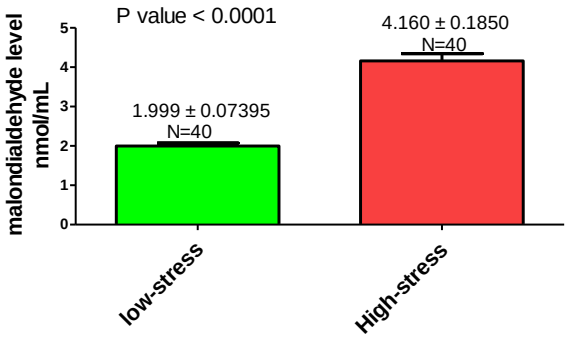
F. (4): Show Sperm progressive motile.



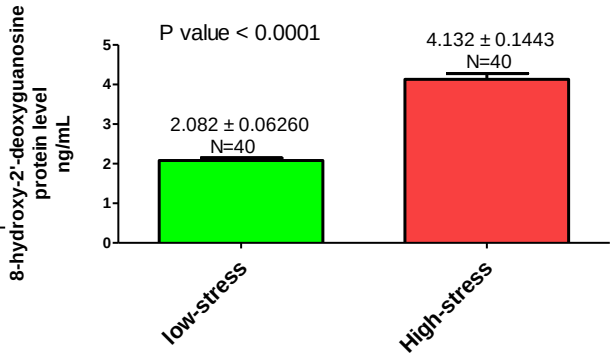
F. (5): Show Sperm normal morphology.



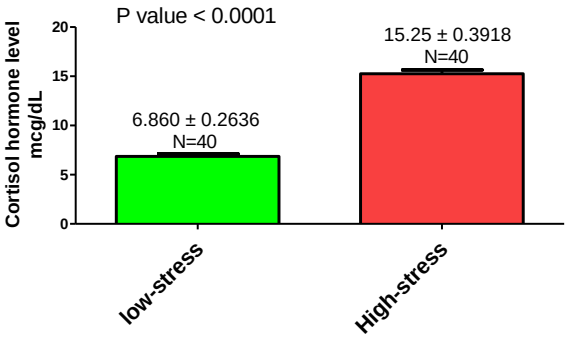
F. (6): Show Sperm DNA fragmentation.



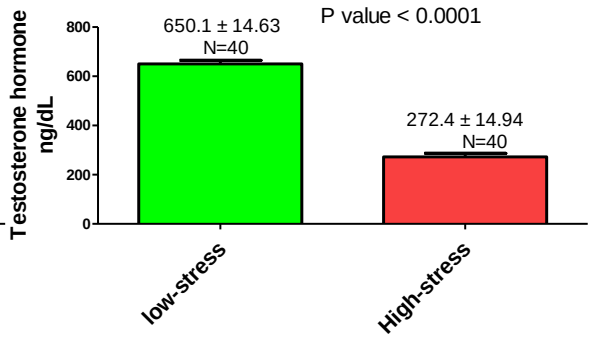
F. (7): Show malondialdehyde level.



F. (8): Show 8-hydroxy-2'-deoxyguanosine protein level.



F. (9): Show Cortisol hormone.



F. (10): Show Testosterone hormone.

DISCUSSION

The results in the present study showed that there were notable changes in the analyzed biochemical and molecular parameters between experimental and control groups, which suggest oxidative imbalance and its related physiological disorder. These findings are in concordance with the large literature documenting oxidative stress as a fundamental contributor to cellular dysfunction and disease evolution of diverse pathologies (14,15).

Oxidative stress occurs when ROS exceed the antioxidant capacity of organisms. Excessive ROS induces lipid, protein and nucleic acid damage in the process of destroying cell structure and function. Such a model is consistent with the extensive biomolecular studies that have identified specific markers of oxidative damage and radical infliction in clinical and experimental systems. For instance, malondialdehyde (MDA) and 8-hydroxydeoxyguanosine (8-OHdG) are widely used measures for lipid peroxidation and DNA oxidation, respectively, which have been demonstrated as a robust marker of oxidative damage in human studies (16,17).

Cortisol levels are usually much higher in infertile men with high psychological stress, when compared to the low-stress group. The elevation shown here is indicative of continuing HPA axis activation and correlates with findings that chronic stress also causes a prolonged cortisol secretion. High levels of cortisol have been reported to suppress the activity of the hypothalamic–pituitary–gonadal (HPG) axis by inhibiting gonadotropin-releasing hormone (GnRH) secretion, resulting in reduced luteinizing hormone (LH) and follicle-stimulating hormone (FSH) production. This results in deficient testosterone production by Leydig cells, which has detrimental effects on spermatogenesis and semen quality (18,19).

In contrast, low-stress infertile men have normal or near-normal cortisol levels within the physiological range. Infertility is observed in this group, but the normal cortisol level provides evidence that psychological stress may not be so significant compared to some aspects of the oxido-reductive equilibrium or metabolic perturbation, genetic abnormalities, and infections as etiological factors in infertility. The lack of long-term exposure to excess cortisol in these derangements may account for the less severe disruption of reproductive homeostasis and sperm quality (20,21,22).

In addition, high cortisol in association with stress was observed to cause infertile male individuals, which might indirectly lead to infertility via increased oxidative stress. Cortisol surplus has also been correlated with overproduction of reactive oxygen species (ROS) and decrease in antioxidant capacity, resulting in lipid peroxidation, sperm DNA fragmentation and sperm motility dysfunction. These pathways can also selectively potentiate the injurious effect of stress on male fertility (23,24).

CONCLUSION

Chronic psychological stress is significantly associated with impaired semen parameters, hormonal imbalance, increased oxidative stress, and elevated sperm DNA fragmentation. Stress should be considered a modifiable risk factor in male infertility, and integrating stress management into clinical practice may improve reproductive outcomes.

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