

Evaluation of levels of stress hormones in patients with atherosclerosis

Ekhlas A. K. Kanani¹, Marwa T. Ahmed², Amenah Imad Fawzi^{3,*}, Hamza Fadhel Hamzah⁴ and Ahmed Abdullah Hussein⁵

¹Al-Manara College for Medical Sciences, Maysan, Iraq

²Department of Microbiology, College of Medicine, University of Tikrit, Iraq

³Department of Medical Laboratory Technology, College of Health and Medical Techniques, AL-Bayan University, Baghdad, Iraq

⁴Department of Medical Laboratories Technology, AL-Nisour University College, Baghdad, Iraq

⁵Department of Medical Laboratory Technology, College of Medical Technology, Al-Farahidi University, Baghdad, Iraq

Correspondance: (e-mail: amenah.i@albayan.edu.iq).

Received 30 November 2024

Revised 29 December 2024

Accepted 31 January 2025

Published 24 February 2025

ABSTRACT Chronic exposure to stress hormones, including adrenocorticotrophic hormone (ACTH) and cortisol, has complex effects on cardiovascular health. Cortisol has anti-inflammatory properties but contributes to AS by altering lipid profiles and vascular function. Chronic stress increases norepinephrine and catecholamine levels, which contribute to hypertension and vascular contraction through the sympathetic–adrenal–medullary system. The renin–angiotensin–aldosterone system also elevates angiotensin II levels, further influencing blood pressure and catecholamine activity. Imbalance of hormones brought about by chronic stress, mainly on the hypothalamic–pituitary–adrenal axis, intensifies the risk for cardiovascular diseases due to a disturbed vascular response and myocardial dysfunction. Socioeconomic development and individual physiological responses may mediate these effects. These mechanisms identify potential therapeutic targets, including stress hormone modulation, for preventing and managing CVD.

KEYWORDS atherosclerosis, adrenocorticotrophic hormone, cardiovascular disease, cortisol

1. INTRODUCTION

Atherosclerosis is an inflammatory chronic disease that is responsible for the growth of plaque in the arterial wall, which represents a leading cause of cardiovascular diseases and a public health problem of great magnitude. While traditional risk factors such as high cholesterol, hypertension, and diabetes have long been established, the role of psychosocial stress and associated hormonal changes in the pathogenesis of atherosclerosis has, in recent years, received greater attention [1]. Chronic stress has been established to cause damaging effects on the vascular health with increased stress hormones like adrenaline and cortisol levels in the blood being associated with atherosclerosis initiation and progression process [2]. Such hormonal imbalances due to chronic stress are linked to enhanced levels of blood pressure, lipids, and inflammation, all contributing to plaque development and instability [2]. Stress has also been linked to a higher incidence of atherosclerosis in women, potentially due to its interactions with other risk factors such as diet and smoking [3].

Extensive research has demonstrated that psychosocial stress, a known cardiovascular risk factor, can modulate inflammatory processes involved in the pathology of atherosclerosis [3]. Furthermore, environmental stressors such as noise pollution have been shown to elevate stress lev-

els, leading to increased atherosclerosis risk [2]. Profiling the levels of stress hormones in patients with atherosclerosis may provide valuable insights into the underlying mechanisms and contribute to the development of more targeted therapeutic approaches.

2. RISK FACTORS OF ATHEROSCLEROSIS

Certain risk factors and cardiovascular inflammation are connected throughout the pathophysiological processes leading to the development of atherosclerosis. Some classical risk factors are largely nonmodifiable. Being male, for example, is one such classical risk factor for CVD risk, and recent studies link at least part of its association with increased vascular inflammation in both animals and humans [4].

Aging is yet another independent risk factor for CVD; biological aging is associated with various inflammatory processes such as increased oxidative stress or more circulating inflammatory cytokines [5]. Clonal hematopoiesis, the process in which increasing numbers of white blood cells derive from one single clone due to specific somatic mutations in hematopoietic stem cells [6], has recently been identified as a pathophysiological mechanism that links aging with atherosclerosis [7]. In atherosclerotic mice, clonal hematopoiesis resulted in a stronger activation of inflammatory pathways and thus accelerated atherosclerosis progres-

sion [8].

Hereditary factors also contribute to the risk of CVD, and a variety of genetic loci have been associated with CVD in large-cohort, genome-wide association studies [9]. Many of these loci appear to be associated with genes that are components of inflammatory pathways [10]. In addition to an intrinsic genetic component to atherosclerosis itself, some classical risk factors like high levels of cholesterol, arterial hypertension, and diabetes also have some degree of genetic predisposition [11], [12].

As outlined above, high cholesterol levels contribute to the initiation and progression of atherosclerosis by acting as an important proinflammatory stimulus. Of note, obesity and unhealthy diet, which are also considered classical risk factors for atherosclerosis [13]–[15], strongly promote high cholesterol levels [16]. Arterial hypertension is another classical risk factor and acts on the integrity of the endothelial cell layer [17]. Ongoing research is intensively investigating the link between atherosclerosis and diabetes, with the general proinflammatory milieu in diabetic patients most likely being a major player in the development of atherosclerosis [18], [19].

Smoking is yet another classical risk factor for cardiovascular inflammation and promotes atherosclerosis via both local and systemic proinflammatory effects, for example, by circulating toxic compounds and increasing hematopoietic stem cell proliferation [20]–[22]. Elevated hematopoiesis contributes to atherosclerosis progression by enhancing inflammatory leukocyte supply [23], [24].

It has become increasingly clear that apart from classical risk factors, additional lifestyle factors and other coexisting pathologies influence CVD. Many of these risk factors have only recently been identified and are thus referred to as nonclassical or nontraditional risk factors. Both viral and bacterial respiratory infections create a higher risk for MI not only during the acute infection but also in the postinfection phase [25], [26].

Acute increases in cardiovascular (CVD) events may result from elevated procoagulant activity, while long-term effects exacerbate vascular inflammation, promoting atherosclerosis progression. Sterile inflammatory conditions like prior myocardial infarction (MI), stroke, or rheumatoid arthritis, as well as nonsterile inflammation, increase cardiovascular risk by elevating proinflammatory leukocyte production in the bone marrow—a mechanism shared by risk factors such as sedentary lifestyles and sleep deprivation [27], [28].

Regular exercise reduces CVD risk through metabolic, antihypertensive, and anti-inflammatory effects. Studies show physical activity lowers leptin levels, induces bone marrow quiescence, and limits proinflammatory leukocyte supply, reducing cardiovascular inflammation and atherosclerotic plaque size [29], [30].

Poor sleep quality similarly raises CVD risk by increasing bone marrow leukocyte production [31]. Sleep fragmentation reduces hypocretin, a hormone that normally limits myeloid

cell production, thus promoting cardiovascular inflammation and atherosclerosis [32], [33].

Environmental factors such as air, light, and noise pollution also affect CVD health. Air pollution directly affects inflammatory pathways, whereas noise pollution indirectly increases risk by affecting sleep and stress [34]. Both promote cardiovascular inflammation through mechanisms such as cytokine production, endothelial dysfunction, and leukocyte infiltration [35], [36]. However, the exact pathways of these effects are still under investigation.

3. CHRONIC STRESS EFFECTS ON INFLAMMATION

Inflammation is a pathological process characterized by injury or destruction of tissues caused by a variety of cytological and chemical reactions. The typical signs of inflammation are pain, heat, redness, swelling and loss of function, and inflammation is related to chronic stress [37], [38]. Research shows that inflammation is strongly related to endothelial dysfunction, a preface to AS and thrombotic disease [39], [40]. Inflammatory reactions are generally considered the main causes of AS, and the influence of mononuclear cells, different subtypes of lymphocytes, neutrophils, and other immune and inflammatory cells on the pathological process of AS has been widely studied. However, in chronic stress, inflammation plays a critical role in the pathological process of AS. It is well-known that chronic stress reduces the activity of the hypothalamic–pituitary–adrenal axis and stimulates the sympathetic adrenal medulla, causing an increase in the production of inflammatory cytokines [41], [42]. Symes et al. hypothesized that chronic stress would result in higher alterations in the levels of proinflammatory factors and cell adhesion molecules; however, they observed that the interventions had only a moderate effect on vascular cell adhesion molecule-1 (VCAM-1) [43]. VCAM-1, a member of the immunoglobulin gene superfamily, is mainly expressed in vascular endothelial cells, and its ligands are $\alpha 4\beta 1$ (VLA-4) and $\alpha 4\beta 7$. Its interaction with VLA-4 is involved in the inflammation induced by leukocytes and improves the pathological process of AS [44], [45].

Research shows that the intercellular adhesion molecule-1, acute phase reactant C-reactive protein and proinflammatory cytokine interleukin-6 were significantly elevated in chronic stress-treated apolipoprotein E in knockout mice than in untreated animals [46]. Other inflammatory signals are found in the plasma cluster of differentiation, interleukin-8, 5-nucleotide ecto, programmed death ligand 1 and plasminogen activator inhibitor PAI-1 [47], [48]. In addition, chronic stress alters the sympathetic and vagal nervous systems homeostasis. Attenuation of the vagal tone contributes to a proinflammatory status, which can help to promote neurotransmitter regulation, particularly the spread of serotonin activation. For example, stress enhances the levels of plasma dipeptidyl peptidase-4 activity and weakens the concentration of plasma glucagon-like peptide-1 and both plasma and adipose adiponectin [49], [50].

4. THE ROLE OF CHRONIC EXPOSURE TO STRESS HORMONES IN CVD

A hormone is defined as a chemical substance that has a specific regulatory effect on the activity of a certain organ or organs. Chronic stress may affect quality of life [51]; however, the role of the stress-related adrenocorticotrophic hormone (ACTH) and cortisol in AS remains to be clarified. Some researches suggested that ACTH and cortisol influence the development of atherogenesis by regulating vascular endothelial action such as driving circulating monocytes to the vascular wall and causing them to disintegrate into macrophages or by controlling the production of inflammatory interleukins [52]. Whereas corticosterone is an anti-inflammatory hormone, it could promote AS in arteries, with increased dyslipidemia [53]. Moreover, one study demonstrated that the level of norepinephrine was increased in a chronic stress experimental group [54]. Thus, these hormones may become a new direction for the treatment and prevention of cardiovascular and cerebrovascular diseases.

Support for this suggestion is provided by the fact that rosiglitazone is connected with the content of cyclic corticosterone; however, experimental data indicate that rosiglitazone does not prevent the occurrence of chronic cardiac angiopathy [55]. In addition, one study showed that the concentrations of cortisol and catecholamines were correlated with socioeconomic development levels, although there was no correlation with hormone levels and education and psychological factors [56]. Levels of cortisol and catecholamine increase as socioeconomic development levels rise and people's lives speed up [56].

The sympathetic–adrenal–medullary system is another important factor in the pathogenesis of hypertension [57]. Under chronic stress, plasma epinephrine and norepinephrine increase rapidly. Past research has shown that the activity of the sympathetic nervous system is enhanced in hypertension; this sympathetic stimulation can result in the constriction of small arteries and veins, thereby elevating the diastolic/systolic blood pressure [58]. Catecholamines are key hormonal mediators of the sympathetic–adrenal–medullary system and play a role in the constriction of peripheral vessels to elevate diastolic blood pressure. The renin–angiotensin–aldosterone system also may play a critical role in chronic stress by increasing levels of angiotensin II, which regulates catecholamine secretion and blood pressure [59], [60]. It is well-known that sympathetic nerve excitement can promote the secretion of renin by stimulating the juxtaglomerular cell and β receptors of local tissues, thus increasing angiotensin II production.

In relation to the role of hormones in the hypothalamic–pituitary–adrenal cortical axis [61], [62], chronic psychological stress triggers the hypothalamus to discharge corticotrophin-releasing hormone and vasopressin that can cause an increase in ACTH secretion. Glucocorticoid is necessary to maintain the normal response of the circulatory system to catecholamine [63]. If the levels of glucocorticoid are low, then the response obviously decreases, the myocardial contraction force weakens, drops, and blood pressure

decreases [64], [65]. Some research suggests that there are racial and ethnic differences in the effects of chronic stress on blood pressure [66].

5. CONCLUSION

Chronic stress significantly contributes to inflammation, endothelial dysfunction, and cardiovascular diseases (CVDs), particularly atherosclerosis (AS). It exacerbates AS through immune activation, proinflammatory cytokine production, and dysregulation of the hypothalamic–pituitary–adrenal axis and sympathetic nervous system, leading to hormonal imbalances and vascular dysfunction. Targeting stress-related pathways, such as reducing inflammation, regulating hormones, and managing sympathetic activity, offers promising strategies for CVD prevention. Lifestyle interventions that lower stress and improve autonomic balance may further enhance cardiovascular health. Continued research is essential to refine these approaches and address individual variations, including socioeconomic and ethnic factors.

ACKNOWLEDGMENT

We extend our sincere gratitude to Al-Bayan University for its valuable contribution in providing a conducive environment through its library and laboratories, which significantly facilitated the completion of our scientific research. Your unwavering support for researchers reflects the university's commitment to fostering knowledge and innovation.

REFERENCES

- [1] Hinterdobler, Julia, et al. "Impact of acute and chronic psychosocial stress on vascular inflammation." *Antioxidants & Redox Signaling* 35.18 (2021): 1531-1550.
- [2] Cai, Lina, et al. "Genome-wide association analysis of type 2 diabetes in the EPIC-InterAct study." *Scientific Data* 7.1 (2020): 393.
- [3] Carson, Jo Ann S., et al. "Dietary cholesterol and cardiovascular risk: a science advisory from the American Heart Association." *Circulation* 141.3 (2020): e39-e53.
- [4] Chen, Jie, et al. "Long-term exposure to fine particle elemental components and natural and cause-specific mortality—a pooled analysis of eight European cohorts within the ELAPSE project." *Environmental Health Perspectives* 129.4 (2021): 047009.
- [5] Ahmad, Hanan Shihab, Araf Sabah Abdulwahed, and Marwan Abdulrazzaq Kamil. "Evaluation of serum levels of irisin, tumor necrosis factor and some biochemical variables in males with prostate cancer in Baghdad City." *Cellular and Molecular Biology* 70.12 (2024): 152-156.
- [6] Ahmad, Hanan Shihab, and Saja Jamal Noman. "Correlation study of hemoglobin and hematocrit levels with BMI, age, and gender and determination of the risk of anaemia in adult residents of Iraq." *Applied Nanoscience* 13.8 (2023): 5357-5364.
- [7] Noman, Saja Jamal, and Hanan Shihab Ahmad. "Effects Of Some Fungal Secondary Metabolite Against Some Cancer Cell Line." *Bangladesh Journal of Medical Science* 22 (2023): p133.
- [8] Zangana, Ashraf Jamal Mahmoud, Hanan Shihab Ahmad, and Isra'a A. Ismail Al-Taii. "A therapeutic attempt by water extract of mentha piperita for amoebic dysentery in vivo and its effect on blood image." *AIP Conference Proceedings*. Vol. 2394. No. 1. AIP Publishing, 2022: 020037.
- [9] Alhaqmuhamad, Asmaa Abd, Mostafa Ali Abdulrahman, and Hanan Shihab Ahmad. "Effect of nanoparticles on liver functions and antioxidant in female rabbits treated with Domperidone." *Indian Journal of Forensic Medicine & Toxicology* 13.4 (2019): 553-557.
- [10] Ahmad, Hanan Shihab, et al. "Study of some hematological and biochemical among for employees of al-dour technical institute." *Biochemical & Cellular Archives* 19.1 (2019): pp. 63-67.

- [11] Majeed, Moazaz Abd Alrida, et al. "Effect of Costus Speciosus Extract on Some Types of Pathogenic Bacteria." *Journal of Global Pharma Technology* 11 (2019): 481-485.
- [12] Laylani, L. S., et al. "The effect of carotenoids of *Rhodotorula glutinis* and probiotic of *Lactobacillus acidophilus* on physiological and histological variables of the kidney in male rats exposed to ultraviolet radiation." *Journal of Animal Health and Production* 12.s1 (2024): 326-331.
- [13] Ahmad, Hanan Shihab, Araf Sabah Abdulwahed, and Marwan Abdulrazzaq Kamil. "Evaluation of serum levels of irisin, tumor necrosis factor and some biochemical variables in males with prostate cancer in Baghdad City." *Cellular and Molecular Biology* 70.12 (2024): 152-156.
- [14] Al-Aubaidi, Israa Kasim, Marwa Ali Al-Oqaily, and Sadia Shahab Hamad. "Role of Interleukin 33 During Infection with Toxoplasmosis in Rheumatoid Arthritis Patients." *Indian Journal of Forensic Medicine & Toxicology* 14.1 (2020): 526-531.
- [15] Hamad, Sadiashahab, Batool AH Al-Haidary, and Zaman Abdul-Sahib Abed. "Effects of Two Genotypes of *Toxoplasma gondii* Strains on DNA Sequence of Females' Oocytes with Polycystic Ovarian Syndrome." *Annals of Tropical Medicine & Public Health* 23.13B (2020): SP231362.
- [16] Hamad, Sadia Shahab, Halala Mohammed Abdulla, and Israa Kasim Al-Aubaidi. "Epidemiological Study of Toxoplasmosis in Patients with Renal Failure from Kirkuk City/Iraq." *Journal of Global Pharma Technology* 11.02 (2019): 578-584.
- [17] Mustafa, M. A., et al. "Virulence factors of proteus mirabilis isolated from urinary tract infection patients." *Latin American Journal of Pharmacy* 42 (2023): 418-421.
- [18] Mustafa, Harith Ahmed, Husham Najji Hameed, and Thabet Mudheher Khalaf. "Prevalence of Ticks and Lice parasites on a certain number of buffaloes in the district of Samarra/Iraq." *Indian Journal of Forensic Medicine & Toxicology* 13.4 (2019): p637.
- [19] Mustafa, Harith A., et al. "Study on some physiological, biochemical and hormonal parameters of seminal fluid of infertile men." *Biochemical and Cellular Archives* 19.Supplement 1 (2019): 1943-1947.
- [20] Skeoch, Sarah, and Ian N. Bruce. "Atherosclerosis in rheumatoid arthritis: is it all about inflammation?." *Nature Reviews Rheumatology* 11.7 (2015): 390-400.
- [21] Smith, Caleb J., et al. "Leukocytosis and tobacco use: an observational study of asymptomatic leukocytosis." *The American Journal of Medicine* 134.1 (2021): e31-e35.
- [22] Tobaldini, Eleonora, et al. "Short sleep duration and cardiometabolic risk: from pathophysiology to clinical evidence." *Nature Reviews Cardiology* 16.4 (2019): 213-224.
- [23] Vermeulen, Roel, et al. "The exposome and health: Where chemistry meets biology." *Science* 367.6476 (2020): 392-396.
- [24] Vineis, Paolo, et al. "The exposome in practice: design of the EXPO-SOMICS project." *International Journal of Hygiene and Environmental Health* 220.2 (2017): 142-151.
- [25] Wang, Julie C., and Martin Bennett. "Aging and atherosclerosis: mechanisms, functional consequences, and potential therapeutics for cellular senescence." *Circulation Research* 111.2 (2012): 245-259.
- [26] Wang, Zeneng, et al. "Impact of chronic dietary red meat, white meat, or non-meat protein on trimethylamine N-oxide metabolism and renal excretion in healthy men and women." *European Heart Journal* 40.7 (2019): 583-594.
- [27] Liu, Yun-Zi, Yun-Xia Wang, and Chun-Lei Jiang. "Inflammation: the common pathway of stress-related diseases." *Frontiers in Human Neuroscience* 11 (2017): 273283.
- [28] Smith, Jennifer A., et al. "Neighborhood characteristics influence DNA methylation of genes involved in stress response and inflammation: The Multi-Ethnic Study of Atherosclerosis." *Epigenetics* 12.8 (2017): 662-673.
- [29] Halaris, Angelos. "Inflammation-associated co-morbidity between depression and cardiovascular disease." *Inflammation-Associated Depression: Evidence, Mechanisms and Implications* (2016): 45-70.
- [30] Li, Chuan, et al. "Macrophage polarization and meta-inflammation." *Translational Research* 191 (2018): 29-44.
- [31] Doğan, Halef Okan, et al. "Evaluation of the associations between endothelial dysfunction, inflammation and coagulation in Crimean-Congo hemorrhagic fever patients." *Archives of Virology* 163 (2018): 609-616.
- [32] Ferreira, Bárbara Isabel Roque Cunha, et al. "Psoriasis and associated psychiatric disorders: a systematic review on etiopathogenesis and clinical correlation." *The Journal of Clinical and Aesthetic Dermatology* 9.6 (2016): 36.
- [33] Vegas, Oscar, et al. "Chronic social stress Ameliorates psoriasiform dermatitis through upregulation of the Hypothalamic-Pituitary-Adrenal axis." *Brain, Behavior, and Immunity* 68 (2018): 238-247.
- [34] Symes, Lene, et al. "Exploring violence against women and adverse health outcomes in middle age to promote women's health." *Critical Care Nursing Quarterly* 33.3 (2010): 233-243.
- [35] Isingrini, Elsa, et al. "Early and late-onset effect of chronic stress on vascular function in mice: a possible model of the impact of depression on vascular disease in aging." *The American Journal of Geriatric Psychiatry* 19.4 (2011): 335-346.
- [36] Abey, Sarah K., et al. "Lysozyme association with circulating RNA, extracellular vesicles, and chronic stress." *BBA Clinical* 7 (2017): 23-35.
- [37] Castro, Elena, Vladimir Oviedo-Rodríguez, and Luis I. Angel-Chávez. "WRN polymorphisms affect expression levels of plasminogen activator inhibitor type 1 in cultured fibroblasts." *BMC Cardiovascular Disorders* 8 (2008): 1-10.
- [38] Halaris, Angelos. "Co-morbidity between cardiovascular pathology and depression: role of inflammation." *Inflammation in Psychiatry* 28 (2013): 144-161.
- [39] Lei, Yanna, et al. "Increased dipeptidyl peptidase-4 accelerates diet-related vascular aging and atherosclerosis in ApoE-deficient mice under chronic stress." *International Journal of Cardiology* 243 (2017): 413-420.
- [40] Ulmer-Yaniv, A., et al. "Maternal immune and affiliative biomarkers and sensitive parenting mediate the effects of chronic early trauma on child anxiety." *Psychological Medicine* 48.6 (2018): 1020-1033.
- [41] Fantidis, Panayotis. "The role of the stress-related anti-inflammatory hormones ACTH and cortisol in atherosclerosis." *Current Vascular Pharmacology* 8.4 (2010): 517-525.
- [42] Okutsu, Mitsuharu, et al. "Corticosterone accelerates atherosclerosis in the apolipoprotein E-deficient mouse." *Atherosclerosis* 232.2 (2014): 414-419.
- [43] Noller, Crystal M., et al. "The influence of social environment on endocrine, cardiovascular and tissue responses in the rabbit." *International Journal of Psychophysiology* 88.3 (2013): 282-288.
- [44] Goodson, M. L., et al. "Chronic stress and Rosiglitazone increase indices of vascular stiffness in male rats." *Physiology & Behavior* 172 (2017): 16-23.
- [45] Castro-Diehl, Cecilia, et al. "Associations of socioeconomic and psychosocial factors with urinary measures of cortisol and catecholamines in the Multi-Ethnic Study of Atherosclerosis (MESA)." *Psychoneuroendocrinology* 41 (2014): 132-141.
- [46] Imperatore, Roberta, Letizia Palomba, and Luigia Cristino. "Role of orexin-A in hypertension and obesity." *Current Hypertension Reports* 19 (2017): 1-13.
- [47] Li, Tiejun, et al. "Elevated oxidative stress and inflammation in hypothalamic paraventricular nucleus are associated with sympathetic excitation and hypertension in rats exposed to chronic intermittent hypoxia." *Frontiers in Physiology* 9 (2018): 840.
- [48] Lezama-Martínez, Diego, et al. "Combination of β adrenergic receptor block and renin-angiotensin system inhibition diminished the angiotensin II-induced vasoconstriction and increased bradykinin-induced vasodilation in hypertension." *Dose-Response* 15.4 (2017): 1559325817737932.
- [49] Ishigaki, Sayaka, et al. "Melatonin ameliorates intrarenal renin-angiotensin system in a 5/6 nephrectomy rat model." *Clinical and Experimental Nephrology* 22 (2018): 539-549.
- [50] Tang, Andrew R., et al. "Prolonged hypothalamic-pituitary-adrenal axis activation after acute coronary syndrome in the GENESIS-PRAXY cohort." *European Journal of Preventive Cardiology* 25.1 (2018): 65-72.
- [51] O'Connor, Daryl B., et al. "Effects of childhood trauma on cortisol levels in suicide attempters and ideators." *Psychoneuroendocrinology* 88 (2018): 9-16.
- [52] Ewart, Craig K., et al. "The role of agonistic striving in the association between cortisol and high blood pressure." *Psychosomatic Medicine* 79.4 (2017): 416-425.
- [53] Kometani, Mitsuhiro, et al. "Cortisol overproduction results from DNA methylation of CYP11B1 in hypercortisolemia." *Scientific Reports* 7.1 (2017): 11205.
- [54] de Vries, Laura V., et al. "Twenty-four hour urinary cortisol excretion and the metabolic syndrome in prednisolone-treated renal transplant recipients." *Steroids* 127 (2017): 31-39.
- [55] Mujahid, Mahasin S., et al. "Neighborhood stressors and race/ethnic differences in hypertension prevalence (the Multi-Ethnic Study of Atherosclerosis)." *American Journal of Hypertension* 24.2 (2011): 187-193.
- [56] Jawad, Zainab Nizar. "Molecular detection of caspase 3, 8, 9 genes and ADIPOR1 (rs2275738) polymorphism in colorectal cancer." *Applied Nanoscience* 13.8 (2023): 5365-5368.

- [57] Jawad, Zainab Nizar. "Association between APC gene SNPs and the risk of occurrence of colorectal cancer." *AIP Conference Proceedings*. Vol. 2414. No. 1. AIP Publishing, 2023: 020021.
- [58] Jawad, Zainab Nizar, and Weaam Awad. "The effect of montelukast on CD14 gene expression in asthmatic patient in Kerbala." *AIP Conference Proceedings*. Vol. 2290. No. 1. AIP Publishing, 2020: 020053.
- [59] Jawad, Zainab Nizar, and Weaam Awad. "Association of urokinase and Vitamin D receptor genes SNPs and urolithiasis in an Iraqi population." *Meta Gene* 24 (2020): 100679.
- [60] Al-Abodi, Hiba Riyadh, et al. "Novel gold nanobiosensor platforms for rapid and inexpensive detection of Vibrio cholerae." *Reviews and Research in Medical Microbiology* 31.2 (2020): 70-74.
- [61] Jawad, Zainab Nizar, and Kamal AR. "Association of Additive Risk of Pioglitazone Use with the Presence of CYP1A1 Polymorphisms in the Occurrence of Bladder Cancer." *Systematic Reviews in Pharmacy* 11.5 (2020): p375.
- [62] Jawad, Zainab Nizar, Kamal Abdul Rasool, and Weaam Awad. "The relationship between smoking and Urokinase gene 3'-UTR T/C expression on occurrence of bladder cancer." *Indian Journal of Forensic Medicine & Toxicology* 14.1 (2020): 1169-1171.
- [63] Sangishetti Vijay Prasad, B Ghongane, B Nayak. Role of Oxidative Stress and Vitamin C, E on Male Fertility: Mini Review. *Int J Biomed Allied Heal Res* 2017;1(1):13-21
- [64] Jawad, Zainab Nizar. "The association of urokinase gene 3'-UTR T/C polymorphism with urinary bladder cancer." *Biochemical & Cellular Archives* 18.1 (2018): p657.
- [65] Jawad, Zainab Nizar, Zuhair Mohammed Ali Jeddooa, and Riadh Rasheed Al-Toama. "Association of MDM2 (T309G) gene polymorphism with interstitial urinary bladder cancer." *Biochemical & Cellular Archives* 18.2 (2018): p2505.
- [66] Jalal, Bnar J., and Mustafa RM Alqaisi. "Improving the production and quality of white button mushroom (*Agaricus bisporus*) by adding biochar and ash to the casing layer." *Tikrit Journal for Agricultural Sciences* 24.1 (2024): 22-33.