We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Impacts of Environmental Stressors on Autonomic Nervous System

Mayowa Adeniyi

Abstract

Stress can be described as the perception of discomforts physically, psychologically, or physico-psychologically. During stress, the perceived discomfort indicates there is a deviation from homeostasis. In stress, there is a nonspecific physiological response to stressors, a group of stress-inducing phenomena. Stress-inducing phenomena can be defined as environmental insults, such as perturbed levels of light, temperature, chemicals, ambient oxygen, and noise. Response to stress occurs via the chemical messenger-mediated sympathetic nervous system including the autonomic-adrenal axis. Furthermore, the chemical messenger-mediated sympathetic nervous system determines nonhormonal effects which are often devised as general stress markers. Examples of general stress markers include changes in heart rate, heart rate variability, blood pressure, body temperature, blood glucose, baroreflex sensitivity, among others.

Keywords: stress, autonomic nervous system, hormonal stress markers, nonhormonal stress markers

1. Introduction

The autonomic nervous system is a nervous control saddled with the regulation of vegetative functions, moderating homeostatic processes in the body. It mediates growth, reproduction, digestion, respiration, blood pressure, heart rate, thermoregulation, osmoregulation, penile tumescence, maintenance of glucose, and electrolyte balance in the body. It connects the peripheral organs in the body with the central nervous system. The autonomic nervous consists of the parasympathetic nervous system and the sympathetic nervous system.

The parasympathetic nervous system plays important role in the homeostasis and restoration of homeostasis. It is evoked during rest and relaxation. Parasympathetic signals are transmitted from one neural cell to another cell through cholinergic nerve endings. Examples of such nerve endings include preganglionic cholinergic nerve fibers, postganglionic cholinergic nerve fibers, and preganglionic sympathetic cholinergic nerve fibers and postganglionic sympathetic cholinergic nerve fibers. The acetylcholine released by parasympathetic nerve fibers binds with muscarinic receptors (m_1 - m_5) to elicit specific responses.

In some cases, parasympathetic activities are not directly mediated by acetylcholine but by other chemical messengers. For instance, the cholinergic nerve endings to

cavernosal arterioles increase the expression of nitric oxide synthase leading to the secretion of nitric oxide causing vasodilation and increased blood flow to penile erectile tissues. Vagal nerve endings in the stomach secrete nitric oxide leading to expansion of the stomach before the arrival of bolus (receptive gastric relaxation) and relaxation of the stomach in the presence of bolus (adaptive gastric relaxation).

The parasympathetic nervous system originates from the cranial nerves and sacral segments of the spinal cord (craniosacral outflow). Usually, the preganglionic parasympathetic cell bodies are situated in the cranial nerves and sacral segments of the spinal cord. However, the parasympathetic ganglia are located close to the effector structures. This makes the postganglionic nerve fibers shorter. Examples are otic ganglion, sphenopalatine ganglion, submaxillary ganglion, ciliary ganglion, among others. Effects of parasympathetic innervation may either be excitatory (involving the development of excitatory postsynaptic potential) or inhibitory. Examples of the inhibitory effects of parasympathetic stimulation include decreased heart rate, the force of cardiac contractility, and blood pressure. Other effects include miosis, increased gastrointestinal motility and secretion, peripheral vasodilation, increased cutaneous blood flow, penile, and clitoral tumescence among others.

The sympathetic nervous system evolved to enable organisms to cope with the emergency, specifically, increased metabolic demand associated with an emergency, flight, and fight. The sympathetic nervous function is executed by the thoracolumbar outflow of the spinal cord and lower brain areas. Unlike the parasympathetic nervous system, the sympathetic ganglia are situated close to the spinal cord and from where postganglionic fibers originate to innervate effectors. The effect of sympathetic stimulation is mediated through binding with adrenergic receptors (alpha and beta receptors). The effects of sympathetic stimulation are increased heart rate and cardiac contractility, increased blood pressure, mydriasis, decreases gastrointestinal motility and secretion, peripheral vasoconstriction among others. It is interesting to note that many phenomena can elicit sympathetic nervous activity. One of them is stress.

Stress is a perception of physical, psychological, or physico-psychological strains or discomforts. During stress, the discomfort perceived physically, psychologically or physical-psychologically represents deviations from what the human body has recognized as normal. Stress is characterized by a nonspecific physiological response to stressors, stress-inducing phenomena. Stress-inducing phenomena can be defined as environmental insults, such as perturbed levels of light, temperature, chemicals, ambient oxygen, and noise. In terms of rapidity of physiological response to stress, the chemical messenger-mediated sympathetic nervous system including the autonomic-adrenal axis plays a major role. Furthermore, the chemical messenger-mediated sympathetic nervous system is a determinant of nonhormonal effects which are often used as general stress markers, such as change in heart rate, heart rate variability, blood pressure, body temperature, blood glucose, baroreflex sensitivity, skin conductance, among others. The chapter looks at different forms of environmental stressors and their autonomic characterizations.

2. Stress

Stress is a perception of physical, psychological, or physico-psychological strains or discomforts. Stress as a sensation of discomfort may be subjective, that is, qualitative. For instance, during a prolonged examination procedure, a student may report having muscle ache or nausea without any confirmatory screenings. In objective

cases, medical assessments are done to ratify the existence of stress [1]. Examples of these include determinations of catecholamine, cortisol, prolactin, heart rate, blood pressure, body temperature, brain rhythm, cardiac rhythm, blood glucose, respiratory rate, peripheral oxygen saturation, urine specific gravity among others. During stress, the discomfort perceived either physically or psychologically or both represents deviations from what the human body has recognized as normal. For example, sudden standing elicits perception of physical and physico-psychological strains because of the sudden diversion of a large amount of blood to structures below the heart including the lower extremities at the detriment of other structures including the brain. Events such as driving a car, teaching a crowd of students, having sexual intercourse, exposure to high altitude or low atmospheric oxygen tension, exposure to noise, or running a 100meter race and nighttime study [2, 3] will result in the perception of physical and physico-psychological discomfort in naïve individuals. It is important to add that a measure of stress is beneficial for physical fitness and health status. This is simply known as eustress. An example of eustress is exercise, any programmed and purposeful physical activity. This invariably means that it is not all physical, psychological, and psychophysical sensations of discomfort that can be imaged as harmful. For instance, in order to be able to run a marathon race, a high $\dot{V}O_2$ max is needed. Achieving this high $\dot{V}O_2$ max through training is not unassociated with sensations of discomfort. The term “distress” comfortably describes all sensations of discomfort either physically, psychologically or both that are not beneficial to human health and survival [4].

Furthermore, sudden standing, driving a car for the first time, acute exposure to high altitude or low atmospheric oxygen tension, exposure to unfamiliar noise, or executing new physical tasks like running a 100meter race, clearly indicate that stress is part of man. If stress is part of man, then, are there natural defensive, protective, and modulatory mechanisms that come into play during stress? The human body was designed to respond to stress nonspecifically through two mechanisms and they are hormonal and nonhormonal mechanisms. Hormonal mechanisms operate through the autonomic-adrenal axis, hypothalamo-hypophyseal-adrenal axis, tuberoinfundibular dopamine axis, and autocrine, paracrine, and neurohormonal pathways. The nonhormonal mechanisms are outcomes of hormonal mechanisms. Examples include changes in heart rhythms, blood pressure, skin conductance, blood glucose, body temperature, respiratory rate, peripheral oxygen saturated among others.

Any agents or conditions that orchestrate deviations from what the human body has recognized as normal or homeostatic are known as a stressor. They are stress-inducing situations and events [1]. These agents may be exogenous, chemical, and biological. Information made available by Study.com indicates hyperthermia results in discomfort. In diurnal mammals, secretions of melatonin predicates photic signals. Hence, disruption of photic signal and photic stress impairs melatonin secretion resulting in circadian desynchronization, impaired glucose homeostasis [2, 3], weight gain, diabetes mellitus, sexual disorder, and breast cancer [3]. Thermal changes, unfamiliar noise, job-related mental and physical exertions [4], job loss, increase in expected work output, overcrowding, marital problem, irregular lighting, bereavement, barotrauma, traveling, exposure to low atmospheric oxygen, economic hardship, heightened family needs and demands, drug and chemical use, and abuse are stressors. Hence, they demonstrate a huge tendency of triggering certain effects. The effects produced depend on the type of stressor. For instance, metabolic perturbation and cytokine production can be produced by chemical stressors. Intake of tobacco brings about an increase in metabolic rate causing tachycardia, palpitation, tachypnea

among others. Alcohol induces secretions of cytokines such as dopamine, endorphin, and serotonin secretion resulting in motivation and addiction. Direct and indirect trauma on the body tissues such as epithelium, muscles, connective tissues, and neural structures and pain-mediating cytokines such as substance P and glutamate are results of both physical and chemical stressors. Both physical and chemical stressors may activate hormonal mechanisms through the autonomic-adrenal axis, hypothalamic hypophyseal-adrenal axis, tuberoinfundibular dopamine axis, autocrine, paracrine, and neurocrine pathways resulting in the release of norepinephrine, dopamine, cortisol, prolactin, prostaglandins interleukin-6, leptin, dehydroepiandrosterone, salivary alpha-amylase, alpha tumor necrosis factor, ghrelin, and growth hormone. As widely believed, human existence has always revolved around not only the surroundings but also the conditions of the environment (Psychology second course) [5].

3. Effect of photic stress on autonomic nervous system

Indiscriminate and prolonged exposure to light has a great deal of impact on the autonomic nervous system. Yasushi *et al.*, [6] examined the effect of exposure to bright light (5000 lux) on sympathetic nervous activity for a period of 20 minutes. The authors reported that exposure increased heart rate while no change was noticed in blood pressure. Muscle sympathetic nerve activity was found to increase after exposure. Sleep is an autonomic phenomenon, Katsanis *et al.*, [7] reported that exposure to color light stimulation shortened time for sleep onset and influence heart rate variability in healthy humans. Specifically, color light preference was found to lower frequency/high-frequency ratio. The study simply indicated that color light preference induces parasympathetic nervous stimulation leading to enhancement of sleep.

To demonstrate the role of dynamic lighting system after fatigue, Maietta *et al.*, [8] utilized a self-reported questionnaire and heart rate variability to measure the autonomic nervous system. There was a rise in parasympathetic nervous mechanisms in the dynamic lighting system within 25 minutes. Heart rate variability means interbeat time interval. Usually, heart rate variability is inversely related to heart rate. This implies the higher the heart rate variability, the smaller the heart rate. Schaefer and Kraft [9] studied the impacts of light stress courtesy of illumination with a colored fluorescent light on autonomic regulation and heart rate variability. He studies the exposure of 12 healthy individuals to red, blue, and green light (700 lux) for 10 minutes. With a green light, there was a reduction in high-frequency components; thus indicating that illumination with colored light can affect heart rate variability.

Glucose metabolism is influenced by the autonomic nervous system. The islet, the birthplace of insulin and glucagon, is innervated by autonomic nerve fibers. Target tissues, such as the liver, muscles, and adipose tissues, express receptors for these hormones. The expression of these receptors is necessary for the hormone to exert its effects. Hormone resistance or hormone tolerance occurs when there is a reduction in the levels of expressed hormone receptors. Sympathetic stimulation of islet is widely known to increase glucagon secretion. Usually, glucagon, when secreted, is in immature form (preproglucagon) which will become activated through intracellular mechanisms. On the other hand, sympathetic stimulation brings about the breakdown of hepatic glycogen and gluconeogenesis. Exposure to light at night was found to increase glucose intolerance in rats. Specifically, the green light was shown to exert more glucose intolerance. However, blue and red lights did influence glucose intolerance. At 50 and 150 lx, greater glucose responses were produced than 5–20 lx [10].

The study results clearly showed that the intensity of light exposure affects glucose homeostasis.

When C57Bl/6 J mice were placed on constant light in a bid to understand how constant length-induced circadian rhythm abnormality affects energy metabolism and insulin sensitivity, Coomans *et al.*, [11] showed a pronto decline in the amplitude of circadian rhythm. Energy expenditure fell by -13% and food intake rose by $+26\%$ culminating in pronto weight gain. Energy metabolisms, insulin sensitivity, and endocrine functions of the islet are autonomically controlled processes. Parasympathetic stimulation of the pancreatic islet through m4 receptors results in a CAMP-mediated increase in the secretion of preproinsulin, which will then be converted to insulin. In the quantification of insulin sensitivity, it is not only insulin secretion that receives consideration. Insulin sensitivity which examines the responsiveness of target tissues to insulin is essential. Insulin sensitivity involves both the reception of insulin by target tissue and associated transduction.

Similarly, a study by Abulmeaty *et al.*, [12] investigated the impact of prolonged continuous exposure on energy homeostasis as well as adropin expression, ROR α , and Rev-erb- α nuclear receptors in 32 rats over a period of 3 months. Continuous light exposure was found to raise total energy expenditure. Lowered respiratory quotient and Rev-erb- α hepatic and renal nuclear receptors coupled with elevation in ROR α and plasma levels of adropin and expressions. The study indicated the possible involvement of adropin, ROR α , and plasma levels of adropin in energy homeostasis. The parasympathetic nervous system is a component of the autonomic nervous system concerned with energy restoration and homeostasis.

Melatonin secretion is an autonomically dependent process. The pineal-innervating postganglionic fibers from the superior cervical ganglion are known to secrete norepinephrine. Elsaid and Fahim [13] showed that maternal exposure to excess artificial light using female rabbits orchestrated a decline in maternal melatonin. In addition, epidermal vacuolation, swollen mitochondria, and shrunken indented nuclei were gotten. As far as the fetus was concerned, reduced thickness of the epidermis, decreased hair follicle number, raised collagen surface area, suppressed proliferating cell nuclear antigen-positive cells were gotten. It is possible that excess light-induced suppression of melatonin is responsible for maternal and fetal histological changes in the study. Furthermore, light also influences body temperature and the reproductive cycle. Specifically, in rodents' study, exposure to light stress has been shown to lengthen the estrous cycle and estrous cycle ratio [2, 3, 14] and impairs body temperature rhythm [15]. Light exposure may also influence the activities of antioxidant enzymes. At least, a study has shown that ovarian glutathione peroxidase increased in light-deprived rats [16].

Sleep is another autonomically controlled mechanism. Sympathetic nervous stimulation through activation of alpha or beta receptors results in increased cortical activities, thereby promoting alertness. On the other hand, the parasympathetic nervous system elicits a reduction in cortical activities, eliciting rest and sleep. Stenvers *et al.*, [17] reported a novel means of creating circadian desynchronization in rats. In their study, they exposed rats to 12-hour of light with the intensity of 150–200 lux and natural 12 hours of dark or 12-hour light (150–200 lux) and 12-hour dark (5 lux). The amplitude of REM and NREM sleep rhythms was attenuated with 12 hour-light (150–200 lux) and 12-hour dark (5 lux). Although 12-hour light (150–200 lux) and 12 hour-dark (5 lux) did not affect glucose tolerance and body weight, there was a reduction in suprachiasmatic expression of the clock gene and desynchronized locomotive activity.

Ishida *et al.*, [18] showed that exposure to light orchestrated the expression of the adrenal gland gene and the underlying mechanism involved suprachiasmatic nucleus-sympathetic nervous system connection. One of the most extensively documented suprachiasmatic nucleus-sympathetic nervous system connections is the retinohypothalamic-pineal gland pathway, through which ambient light passes through intrinsically photosensitive mesopic retinal ganglionic cells to the suprachiasmatic nucleus of the anterior hypothalamus and from there to the pineal gland via superior cervical ganglion. Before the advent of the electric bulb in 1860, human functions were governed by the natural light/dark cycle that consisted of 12 hours of daylight and 12 hours of dark. Cailotto *et al.*, [19] reported that exposure to nocturnal light exerted a rapid effect on peripheral clock gene expression with autonomic hepatic innervation being found to be essential for the photic signals from the suprachiasmatic nucleus.

Kalsbeek *et al.*, [20] identified gamma aminobutyric acid (GABA) as the endogenous mechanism involved in the transmission of photic signals from the suprachiasmatic nucleus to the pineal gland. In the study, the authors showed the role of the chemical messenger by administering GABA antagonist to some parts of the suprachiasmatic nucleus. The presence of GABAergic projection between ventrolateral and dorsolateral parts of the suprachiasmatic nucleus has also previously been reported.

4. Effect of thermal stress on autonomic nervous system

Exposure to thermal stress has a profound effect on the autonomic nervous system. Niimi *et al.*, [21], in a study, examined the effect of heat stress on the sympathetic nervous system in human subjects on the sympathetic nervous system. Nine subjects were deployed for the study and they were exposed to heat stress through the rise in environmental temperature gradually from 290°C to 400°C. Muscle sympathetic nerve activity, tympanic temperature, plasma arginine vasopressin, cutaneous blood flow, heart rate, blood pressure, cardiac output, and mean arterial blood pressure were monitored. The rise in muscle sympathetic nerve activity, tympanic temperature, cardiac output, mean arterial blood pressure, and cutaneous blood flow were recorded when heat stress was applied. Crandall *et al.*, [22] exposed seen subjects to whole-body heating through the infusion of 15 ml/kg of warm water. Skin blood flow, heart rate, arterial blood pressure, muscle sympathetic nerve activity, and sublingual temperature were measured. The authors observed that thermal exposure raised muscle sympathetic nerve activity but central venous blood and mean arterial blood pressure were unaffected. However, mean arterial pressure was found to increase when the effect of arterial baroreceptor loading on muscle sympathetic nerve activity during heat stress was conducted. Low *et al.*, [23] examined the possibility that a shift from moderate to severe thermal stress would raise muscle sympathetic nerve activity and sympathetic nerve activity continuously. Thirteen subjects were made to undergo passive thermal stress that raised body temperature by 1.3°C. There was a rise in mean cutaneous temperature. Mean body temperature, heart rate, and skin vascular conductance by thermal stress exposure. There was also a transient increase in muscle sympathetic nerve activity with thermal stress. Cui *et al.*, [24] investigated the role of thermal stress in the alleviation of muscle sympathetic nerve activity during gravitational stress. The authors found that there was a rise in sublingual temperature, sweat rate, cutaneous blood flow, heart rate, and muscle sympathetic nerve activity during whole-body heating. However, the rise in muscle sympathetic nerve activity which occurred in response to gravitational stress was not assuaged by thermal stress in

human subjects. Cutaneous heating depressed the high-frequency component of heart rate variability but raised the ratio of low-to-high frequency component and mayer waves. However, there was no increase in baroreflex sensitivity with cutaneous heating [25]. Kaho *et al.*, [26] investigated how thermal stress affects heart rate variability in freely moving ruminant animals. They measured autonomic nervous system markers such as heart rate variability, temperature-humidity index, among others. Heart rate variability was reported to decline with temperature-humidity index. Their study showed that high thermal stress may affect the autonomic equilibrium of ruminant organisms by inducing the sympathetic nervous system nonlinearly. Wilson and Ray, [27] reported that whole-body heating raised heart rate, internal temperature, and muscle sympathetic nerve activity but mean arterial blood pressure was unaffected.

5. Effect of chemical stress on autonomic nervous system

Chemical stressors such as exogenous chemicals have an influence on the autonomic nervous system. Gerald *et al.*, [28] reported increased chemosensitivity as well as baroreceptor reflex disruption, sympathetic hyper-excitation, increased heart rate, and high blood pressure. Shvachiy *et al.*, [29] investigated the impact of intermittent exposure to lead on autonomically controlled body functions such as cardiovascular and respiratory functions. High blood pressure, impaired baroreflex sensitivity and tachypnea were recorded following intermittent exposure to lead. In addition to the fact that chemical stressors suppressed parasympathetic actions in infancy and childhood, the effect may be scourged even during intrauterine life. Jurczyk *et al.*, [30] in their review showed that prenatal exposure to alcohol decreased heart rate variability. Prolonged alcohol exposure has been linked with an increased risk of heart disease [31]. Decreased heart rate variability implies a high heart rate. Yu *et al.*, [32] intended to examine the association between blood lead and autonomic nervous activity (heart rate variability and median nerve conduction velocity in 328 adult males aged 28 years. The authors found no association between blood level concentration and autonomic nervous activity. Liao *et al.*, [33] reported that there was an association between exposure to airborne heavy metals and autonomic dysfunction in 82 young adults. Other airborne chemical stressors which have been reported to impair autonomic nervous activity, raising systolic blood pressure and diastolic blood pressure and disturbing other autonomically controlled structures include dust [34], cadmium [35], aluminum [36], lead [37], and oil spillage [38].

6. Effect of hypoxic stress on autonomic nervous system

The environment is the primary source of oxygen in the body. Hypoxic stress represents the sensation of discomfort associated with exposure to the low partial pressure of oxygen. Exposure to high altitudes (3000–4000 m) has been reported to lead to depression of heart rate variability power spectra [39]. Exposure to hypoxia and hypercapnia has been reported to raise sympathetic burst frequency. However, hypoxia was found to produce a long-lasting effect on sympathetic activation in human subjects than exposure to hypercapnia [40]. Excessive hypoxia also causes disruption of peripheral chemoreceptors and hyperviscosity. Hyperviscosity leads to increased pulmonary blood pressure, right cardiac failure, inadequate brain perfusion, and death [41].

7. Effect of noise stress on autonomic nervous system

Unlike note, noise is produced when sound waves are emitted from multiple sources. Noise has been reported to impact autonomic nervous activity. Goyal *et al.*, [42] exposed 200 adult males to noise intensity of greater than 80 dB for about 6 months. Noise exposure increased heart rate and systolic blood pressure. Idrobo-Ávila *et al.*, [43] in their systematic review noted that noise exposure increased heart rate and blood pressure and caused alteration in both high frequency and low frequency/high-frequency ratio. Chen *et al.*, [44] examined the connection between occupational exposure to noise and high blood pressure using 1390 workers exposed to occupational noise. The authors reported that noise exposure caused an increase in systolic blood pressure and diastolic blood pressure. According to the study, the prevalence of high blood pressure was found as 17.8% in those who were exposed to noise and 9% in those who were not exposed to noise. Hey also claimed that there existed a stronger regression coefficient between diastolic blood pressure and noise exposure. Noise intensity, cumulative noise exposure, and years of exposure were related to high blood pressure risk. de Souza *et al.*, [45] claimed that exposure to noise was associated independently with high blood pressure at 75–85 dB. The study recruited 1729 subjects who were petrochemical workers. The authors also found that age, body mass index, and gender were associated with high blood pressure independently. A study by Brahem *et al.*, [46] was conducted on 120 electricity workers who were exposed to noise. Prevalence of high blood pressure was found in workers who were exposed to noise. Systolic blood pressure and diastolic blood pressure were higher in workers who were exposed to noise. Kuang *et al.*, [47] showed that increasing years of exposure to occupational noise increased systolic blood pressure and diastolic blood pressure.

8. Summary

Environmental stressors such as ambient threshold changes in light, heat, noise atmospheric oxygen, and chemical phenomena have a great impact on the autonomic nervous system causing impairment in general stress markers. Environmental stressors orchestrated an increase in systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and disrupted baroreflex sensitivity in both males and females. Derangement in body temperature and blood glucose and suppression of melatonin were also consequences of environmental stressors in both human beings and animals. Future studies will examine in detail the molecular mechanisms that underlie environmental stressor-induced changes in general stress markers.

IntechOpen

IntechOpen

Author details

Mayowa Adeniyi
Stress and Physical Exertion Unit, Department of Physiology, Edo State University
Uzairue, Iyamho, Edo State, Nigeria

*Address all correspondence to: 7jimade@gmail.com

IntechOpen

© 2022 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

References

- [1] Shawi A, Abdullateef AN, Khedher MA, Rejab MS, Khaleel RN. Assessing stress among medical students in Anbar governorate, Iraq: a cross-sectional study. *The Pan African Medical Journal*. 2018;**31**:96. DOI: 10.11604/pamj.2018.31.96.16737
- [2] Adeniyi MJ, Fabunmi O, Okojie AK, Olorunnisola OL, Odetola AO, Olaniyan OT, et al. Impact of night study frequency on sleep pattern, anthropometrical indices and peripheral oxygen saturation in age-matched nigerian female students prior to semester examination. *International Journal of Biomedical Sciences*. 2020a;**16**(3):37-42
- [3] Adeniyi MJ, Agoreyo FO, Olorunnisola OL, Olaniyan OT, Seriki SA, Ozolua PO, et al. Photo-pollution disrupts reproductive homeostasis in female rats: The duration-dependent role of selenium administrations. *The Chinese Journal of Physiology*. 2020b;**63**:235-243
- [4] Ulrich-Lai YM, Fulton S, Wilson M, Petrovich G, Rinaman L. Stress exposure, food intake and emotional state. *Stress*. 2015;**18**(4):381-399. DOI: 10.3109/10253890.2015.1062981. Epub 2015 Aug 13
- [5] Psychology Second Course 105-115. 222en23. Available from: <https://nios.ac.in/media/documents/secpsycour/English/Chapter-23.pdf> [Last accessed: October 22, 2021]
- [6] Saito Y, Shimizu T, Takahashi Y, Mishima K, Takahashi K-i, Ogawa Y, et al. Effect of bright light exposure on muscle sympathetic nerve activity in human. *Neuroscience Letters*. 1996;**219**(2):135-137
- [7] Lee S, Kim D. Effect of Color Light Stimulation Using LED on Sleep Induction Time. *Journal of Healthcare Engineering*. 2017;**2017**:6030268. DOI: 10.1155/2017/6030268. Epub 2017 May 11
- [8] Maietta S, Chen C-Y, Wu P-J, Hsiao Y-J, Tai Y-W. Autonomic nervous system under dynamic lighting environment during a short rest. *Changes in Humans*. 2021;**2021**:6697701. DOI: 10.1155/2021/6697701
- [9] Schaefer A, Kratky KW. The effect of colored illumination on heart rate variability. *Forschende Komplementärmedizin*. 2006;**13**:167-173
- [10] Opperhuizen AL, Stenvers DJ, Jansen RD, Foppen E, Fliers E, Kalsbeek A. Light at night acutely impairs glucose tolerance in a time-, intensity- and wavelength-dependent manner in rats. *Diabetologia*. 2017;**60**:1333-1343. DOI: 10.1007/s00125-017-4262-y
- [11] Coomans CP, van den Berg SA, Houben T, van Klinken JB, van den Berg R, Pronk AC, et al. Detrimental effects of constant light exposure and high-fat diet on circadian energy metabolism and insulin sensitivity. *The FASEB Journal*. 2013;**27**(4):1721-1732. DOI: 10.1096/fj.12-210898. Epub 2013 Jan 9
- [12] Abulmeaty MMA, Almajwal AM, Alnumair KS, Razak S, Hasan MM, Fawzy A, et al. Effect of long-term continuous light exposure and western diet on adropin expression, lipid metabolism, and energy homeostasis in rats. *Biology (Basel)*. 2021;**10**(5):413. DOI: 10.3390/biology10050413
- [13] Elsaid AG, Faheem NM. Impact of constant light exposure during pregnancy

on skin of neonatal New Zealand rabbits: structural and ultrastructural study. *Brazilian Journal of Medical and Biological Research*. 2021;54(6):e10722. DOI: 10.1590/1414-431X202010722

[14] Adeniyi MJ, Agoreyo FO. Estrous cycle ratio as a reproductive index in the rats. *American Journal of Biomedical Science and Research*. 2019;4(2):100-103

[15] Adeniyi MJ, Agoreyo FO. Duration-related modulation of body temperature rhythm and reproductive cycle in rats by photoperiodic perturbation. *DRJHP*. 2020;8(1):1-6

[16] Agoreyo FO, Adeniyi MJ. Pattern of estrous cycle and ovarian antiperoxidative activity in light deprived sprague-dawley rats treated with sodium. *Journal of Medicinal Research and Biological Studies*. 2018;1(1):103

[17] Stenvers DJ, van Dorp R, Foppen E, Mendoza J, Opperhuizen AL, Fliers E, et al. Dim light at night disturbs the daily sleep-wake cycle in the rat. *Scientific Reports*. 2016;20(6):35662. DOI: 10.1038/srep35662

[18] Ishida A, Mutoh T, Ueyama T, Bando H, Masubuchi S, Nakahara D, et al. Light activates the adrenal gland: timing of gene expression and glucocorticoid release. *Cell Metabolism*. 2005;2(5):297-307. DOI: 10.1016/j.cmet.2005.09.009

[19] Cailotto C, Lei J, van der Vliet J, van Heijningen C, van Eden CG, Kalsbeek A, et al. Effects of nocturnal light on (clock) gene expression in peripheral organs: a role for the autonomic innervation of the liver. *PLoS One*. 2009;4(5):e5650. DOI: 10.1371/journal.pone.0005650

[20] Kalsbeek A, Cutrera RA, Van Heerikhuize JJ, Van Der Vliet J, Buijs RM. GABA release from suprachiasmatic

nucleus terminals is necessary for the light-induced inhibition of nocturnal melatonin release in the rat. *Neuroscience*. 1999;91(2):453-461. DOI: 10.1016/s0306-4522(98)00635-6

[21] Niimi Y, Matsukawa T, Sugiyama Y, Shamsuzzaman AS, Ito H, Sobue G, et al. Effect of heat stress on muscle sympathetic nerve activity in humans. *Journal of the Autonomic Nervous System*. 1997;63(1-2):61-67. DOI: 10.1016/s0165-1838(96)00134-8

[22] Crandall CG, Etzel RA, Farr DB. Cardiopulmonary baroreceptor control of muscle sympathetic nerve activity in heat-stressed humans. *The American Journal of Physiology*. 1999;277(6 Pt 2):H2348-H2352. DOI: 10.1152/ajpheart.1999.277.6.h2348

[23] Low DA, Keller DM, Wingo JE, Brothers RM, Crandall CG. Sympathetic nerve activity and whole body heat stress in humans. *Journal of Applied Physiology*. 1985;111(5):1329-1334. DOI: 10.1152/japplphysiol.00498.2011 Epub 2011 Aug 25

[24] Cui J, Wilson TE, Crandall CG. Muscle sympathetic nerve activity during lower body negative pressure is accentuated in heat-stressed humans. *Journal of Applied Physiology*. 1985;96(6):2103-2108. DOI: 10.1152/japplphysiol.00717.2003

[25] Gerald V, Carvalho M, Goncalves-Rosa N, Tavares C, Laranjo S, Rocha I. Lead toxicity promotes autonomic dysfunction with increased chemoreceptor sensitivity. *Neurotoxicology*. 2016;54:170-177. DOI: 10.1016/j.neuro.2016.04.016 Epub 2016 Apr 28

[26] Kitajima K, Oishi K, Masafumi M, Hirok A, Akira S, Yudai Y, et al. Effects of heat stress on heart rate variability in

free-moving sheep and goats assessed with correction for physical activity of muscle sympathetic nerve activity in heat-stressed humans. *The American Journal of Physiology*. 1999;277(6 Pt 2):H2348-H2352. DOI: 10.1152/ajpheart.1999.277.6.h2348

[27] Wilson TE, Ray CA. Effect of thermal stress on the vestibulosympathetic reflexes in humans. *Journal of Applied Physiology*. 2004;97(4):1367-1370

[28] Geraldes V, Carvalho M, Goncalves Rosa N, Tavares C, Laranjo S, Rocha I. Lead toxicity promotes autonomic dysfunction with increased chemoreceptor sensitivity. *Neurotoxicology* 2016;54:170-177. DOI: 10.1016/j.neuro.2016.04.016. Epub 2016 Apr 28

[29] Shvachiy L, Geraldes V, Amaro Leal Â, Rocha I. Intermittent low-level lead exposure provokes anxiety, hypertension, autonomic dysfunction and neuroinflammation. *Neurotoxicology*. 2018;69:307-319. DOI: 10.1016/j.neuro.2018.08.001 Epub 2018 Aug 8

[30] Jurczyk M, Dyląg KA, Skowron K, Gil K. Prenatal alcohol exposure and autonomic nervous system dysfunction: A review article. *Folia Medica Cracoviensia*. 2019;59(3):15-21. DOI: 10.24425/fmc.2019.131132

[31] Ige SF, Adeniyi MJ, Adeniyi AM, Ibrahim SD. Hematological and immunological characterizations of experimental ulcerative colitis rats: The impact of alcohol consumption pattern. *International Journal of Biomedical Science*. 2021;17(2):6-14

[32] Yu CG, Wei FF, Yang WY, Zhang ZY, Mujaj B, Thijs L, et al. Heart rate variability and peripheral nerve conduction velocity in relation to

blood lead in newly hired lead workers. *Occupational and Environmental Medicine*. 2019;76(6):382-388. DOI: 10.1136/oemed-2018-105379 Epub 2019 Mar 30

[33] Liao YH, Chen WL, Wang CC, Lai CH. Associations between personal exposure to metals in fine particulate matter and autonomic nervous system dysfunction among healthy adults. *Aerosol and Air Quality Research*. 2020;20:1842-1849. DOI: 10.4209/aaqr.2020.04.0156

[34] Oni TJ, Adeniyi MJ. Postural difference in expiratory rate among female sanitary workers and its relationship with blood pressure and anthropometric indices. *Biomedical Journal of Scientific & Technical Research*. 2017;1(2):311-315

[35] Ige SF, Adeniyi MJ, Ademilua OB, Fatola AO, Adeyemi IA. Allium cepa remediates oxidative stress-mediated hepatic DNA damage in cadmium-exposed rats through enhanced p53 expression and inhibition of bcl2. *International Journal of Biomedical Sciences*. 2020;16(2):11-17

[36] Ige SF, Adeniyi MJ, Iyalla GO. Allium cepa mitigates aluminum chloride induced hepatotoxicity in male wistar rats. *Journal of Biomedical Science*. 2017;6(4):27

[37] Ige SF, Adeniyi MJ, Joanna AO, Oluwaseyi DA. Allium Cepa juice prevented oxidative stress-mediated metabolic disorder following chronic lead acetate exposure in male rats. *Albanian Journal of Medical and Health Science*. 2019;1(40):11

[38] Adeniyi MJ, Oni TJ. Water contamination in Nigeria and body defense issues. *Research Chronicler*. 2016;3(3):11-21

- [39] Zhang D, She J, Zhang Z, et al. Effects of acute hypoxia on heart rate variability, sample entropy and cardiorespiratory phase synchronization. *Biomedical Engineering Online*. 2014;**13**:73. DOI: 10.1186/1475-925X-13-73
- [40] Xie A, Skatrud JB, Puleo DS, Morgan BJ. Exposure to hypoxia produces long-lasting sympathetic activation in humans. *Journal of Applied Physiology*. 2001. DOI: 10.1152/jappl.2001.91.4.1555
- [41] Hainsworth R, Drinkhill MJ, Rivera-Chira M. The autonomic nervous system at high altitude. *Clinical Autonomic Research*. 2007;**17**:13-19
- [42] Goyal S, Gupta V, Walia L. Effect of noise stress on autonomic function tests. *Noise & Health*. 2010;**12**(48):182-186. DOI: 10.4103/1463-1741.64976
- [43] Idrobo-Ávila EH, Loaiza-Correa H, van Noorden L, Muñoz-Bolaños FG, Vargas-Cañas R. Different types of sounds and their relationship with the electrocardiographic signals and the cardiovascular system - review. *Frontiers in Physiology*. 2018;**22**(9):525. DOI: 10.3389/fphys.2018.00525
- [44] Chen S, Ni Y, Zhang L, Kong L, Lu L, Yang Z, et al. Noise exposure in occupational setting associated with elevated blood pressure in China. *BMC Public Health*. 2017;**17**(1):107. DOI: 10.1186/s12889-017-4050-0
- [45] de Souza TC, Périssé AR, Moura M. Noise exposure and hypertension: investigation of a silent relationship. *BMC Public Health*. 2015;**3**(15):328. DOI: 10.1186/s12889-015-1671-z
- [46] Brahem A, Riahi S, Chouchane A, Kacem I, Maalel OE, Maoua M, et al. Impact du bruit professionnel sur le développement de l'hypertension artérielle : enquête réalisée au sein d'une centrale de production d'électricité et de gaz en Tunisie [Impact of occupational noise in the development of arterial hypertension: A survey carried out in a company of electricity production]. *Annales de Cardiologie et d'Angéiologie (Paris)*. 2019;**68**(3):168-174. French. DOI: 10.1016/j.ancard. 2018.10.008
- [47] Kuang D, Yu YY, Tu C. Bilateral high frequency hearing loss is associated with elevated blood pressure and increased hypertension risk in occupational noise exposed workers. *PLoS One*. 2019;**14**(9):e0222135. DOI: 10.1371/journal.pone.0222135