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ABSTRACTS OF THE PROCEEDINGS OF THE FORTY-SECOND ANNUAL SCIENTIFIC CONFERENCE OF THE PHYSIOLOGICAL SOCIETY OF NIGERIA

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ALTERED COGNITIVE FUNCTION IN PREGNANCY AND ALUMINUM CHLORIDE-INDUCED ALZHEIMER'S DISEASED FEMALE WISTAR RATS.

Onyinye Cynthia Okeke O.C[1], Esther Iyobo-Oghosa Odiase[2], Becky Ogie[2], Paulina Ada Ojo[2], Omoyemwen Osariemen[2]

[1]*Department of Medical Physiology, University of Rwanda, Butare, Rwanda.*

[2]*Department of Physiology, Igbinedion University, Okada, Edo State, Nigeria.*

Email: okekecynthia12@gmail.com; Tel: +250796594747

Cognition has been observed to decline during pregnancy, a condition colloquially described as "baby/pregnancy brain." Like in pregnancy, a decline in cognition is also a common feature amongst people with Alzheimer's disease. This research studied the cognitive functions in pregnancy, comparing them with aluminium chloride-induced Alzheimer's models to explore links between pregnancy and early-onset dementia. Estrus cycles of the animals were monitored and they were mated to achieve pregnancy. Also, calculated doses of aluminium chloride were injected intraperitoneal to a different group to induce Alzheimer's disease. Neurobehavioral studies were conducted for all the groups, and blood samples were collected for assay of reproductive hormones and cognition-related neurotransmitters. Results are presented as Mean±SEM. Student t-test and ANOVA were used to compare the means obtained and p-values of less than 0.05 were considered as statistically significant. Results obtained indicate a similar decline in cognition in both pregnant and Alzheimer's disease groups compared with controls. In conclusion, this study projects the need to be thoroughly decisive concerning childbearing in females with a family history ofAlzheimer's disease, as pregnancy may be an early trigger for the cascade of reactions that culminate in early or late onset dementia.

Keywords: *Cognition, Pregnancy, Aluminium chloride, Alzheimer's disease*.

MODULATION OF KEY INFLAMMATORY GENE EXPRESSION IN THE UTERUS OF PREGNANT RATS FOLLOWING EDIBLE CLAY (CALABASH CHALK) INGESTION.

[1, 2]*Edeha, D.O., [2]Agoreyo, F.O. and [3]Agoreyo, B.O.

Department of Physiology, [1]Benson Idahosa University, and [2]University of Benin, Nigeria.

Email: daniel.edeha@yahoo.com; Tel: +2348073210782

Calabash chalk consumption is a common practice among individuals of African descent, particularly pregnant women and children, as it is culturally believed to satisfy cravings, support detoxification, and relieve morning sickness. This study investigated the gene expression of interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and cyclooxygenase-2 (COX-2) in uterine tissue following oral ingestion of edible clay during pregnancy. Seventy-five pregnant Wistar rats, separated into five groups and three subgroups (n = 5), representing gestational day (GD) 7, 14, and 20, were orally administered 2000 mg/kg (high dose), 1000 mg/kg (medium dose), and 500 mg/kg (low dose) of edible clay, distilled water (control) and lead acetate (15 mg/kg). Uterine tissues were harvested on GD7, 14 and 20, and gene expression of IL-6, TNF-α, and COX-2 was analyzed using real-time quantitative polymerase chain reaction (RT-PCR). Results revealed a significant decrease in the expression of IL-6, TNF-α and COX-2 genes across all doses of edible clay and lead acetate group for the IL-6 gene ($p<0.05$) at GD7. GD14 showed a significant increase in the expression of IL-6, TNF-α, and COX-2 genes of pregnant rats exposed to a medium dose of edible clay and lead acetate, but a significant decrease in the expression of the COX-2 gene of pregnant rats treated with high and low doses of edible clay ($p<0.05$). At GD20, there was a significant decrease in the expression of IL-6, COX-2, and TNF-α genes in the uterus of pregnant rats across all treatment groups compared with control ($p<0.05$). The observed downregulation indicates a weakened uterine immune signaling for implantation, pregnancy maintenance and parturition.

Keywords*: Pregnancy, Edible clay, Uterus, Inflammatory genes, immune signalling.*

ANTIPROLIFERATIVE EFFECT OF *MARGARITARIA DISCOIDEA* STEM BARK EXTRACT ON BENIGN PROSTATIC HYPERPLASIA IN RATS

Bernard Opeyemi Adedeji[1,3], Tolulope David Oluwole[2,3], Ayodeji Folorunsho Ajayi[2,3]

[1]*Department of Physiology, Federal University of Health Sciences, Ila-Orangun, Osun-State, Nigeria.*

[2]*Department of Physiology, Crescent University, Abeokuta, Ogun-State, Nigeria*

[3]*Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.*

(Email: bernard.adedeji@fuhsi.edu.ng; Tel.: 08133869196).

Age-associated benign prostatic hyperplasia (BPH) is a prevalent condition affecting a large percentage of men above the age of 50 years. Although BPH is a benign process, the fact that it often develops into malignant tumour (e.g., prostate carcinoma) makes it a condition that requires urgent attention. Presently, surgical, minimally invasive, and pharmacological interventions are the common available treatment options. Unfortunately, these available interventions have been associated with erectile dysfunction, male infertility and depression. Therefore, this study sought to explore the antiproliferative effect of aqueous extract of *M. discoidea* stem bark (AESBM) in a rat model of BPH. Twenty male rats were randomized into four groups (n=5) as follows: control, untreated BPH, finasteride-treated (5 mg/kg), and AESBM-treated (300 mg/kg) groups. BPH was

induced by subcutaneous injection of testosterone propionate (6 mg/kg) for 28 days, and AESBM was administered orally after BPH induction for 28 days. BPH induction was evident by the increased prostate weight, PSA, and testosterone levels. This was accompanied by a significant increase in IL-6, TNF-α, IL-1β, NO, and caspase 3 and a decrease in enzymatic antioxidant activities. These events were associated with increased glandular and stromal hyperplasia within the prostate gland. Interestingly, these observed oxido-inflammatory-mediated apoptotic responses were abrogated in animals treated with AESBM. In fact, AESBM's ameliorative effects were comparable to those observed in animals treated with a standard drug (finasteride). Therefore, findings from this study established the efficacy of AESBM as a potential phytotherapeutic agent for BPH management.

Keywords: *Antiproliferative, Benign Prostatic Hyperplasia, Inflammation, Oxidative stress, Phytotherapy*

ASSESSMENT OF THE EFFECTS OF OMEGA-3 FATTY ACIDS ON TESTICULAR INTEGRITY IN METHAMPHETAMINE-EXPOSED WISTAR RATS

Florence Bukola Adisa[1, 2], Ayodeji Folorunsho Ajayi[1, 2]

[1]*Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.*

[2]*Anchor Biomed Research Institute, Ogbomoso, Oyo State, Nigeria*.

Email: adisaflorence124@gmail.com; Tel: 09035874993

Methamphetamine (MA) abuse poses a growing global health concern, with detrimental effects on male reproductive health often underemphasized. Notably, MA impairs testicular function, disrupting spermatogenesis and hormonal balance. Omega-3 fatty acids (Ω-3FAs), known for their anti-inflammatory and antioxidative properties, may offer protective benefits against such reproductive toxicity. Therefore, this study explored the modulatory effects of Ω-3FAs on testicular integrity in MA-exposed Wistar rats by assessing sperm quality, hormonal and steroidogenic profiles, oxidative stress markers, inflammatory cytokines, apoptotic indices, and histological architecture. Twenty adult male Wistar rats were randomized into four groups (n = 5): control (vehicle), MA (10 mg/kg), Ω-3FAs (300 mg/kg), and combined MA + Ω-3FAs. Treatments were administered orally for 60 days. On day 61, rats were euthanized, and biological samples were collected for comprehensive biochemical and histological analyses. MA exposure significantly reduced testicular and body weights, sperm count, motility, and viability while increasing sperm deformities. It also disrupted hormone levels, elevated oxidative stress (MDA, H_2O_2), inflammation (TNF-α, IL-1β), and apoptosis (caspase-3, DNA fragmentation index), and led to adverse histological changes. Conversely, co-administration of Ω-3FAs significantly ameliorated these MA-induced alterations. Rats in the MA + Ω-3FAs group showed improved testicular and body weights, sperm parameters, hormone balance, and antioxidant enzyme levels. Furthermore, Ω-3FAs attenuated inflammatory and apoptotic markers and restored normal testicular histoarchitecture. In conclusion, Ω-3FAs exhibit significant protective effects against MA-induced testicular toxicity via antioxidant, anti-inflammatory, and anti-apoptotic pathways. Therefore, these findings underscore the therapeutic potential of Ω-3FAs in mitigating reproductive dysfunction associated with methamphetamine abuse.

Keywords: *Infertility, Testicular toxicity, Oxidative stress, Apoptosis, Inflammation, Spermatogenesis, Antioxidants, Drug abuse, Methamphetamine, Omega-3 fatty acids.*

CHLORPROMAZINE-INDUCED SEDATION: A STUDY OF THE DOPAMINERGIC MECHANISM UNDERLYING SLEEP AND LOCOMOTOR ACTIVITY IN WISTAR RAT.

Abuda Onobu and Alufohai Paul Imevbore

Department of Physiology, Benson Idahosa University, Benin City.

Tel: 08037368784; Email: webmasterhonour@gmail.com

The dopaminergic system plays a vital role in regulating motor control, mood, cognition, and sleep. Dopamine antagonists such as chlorpromazine hydrochloride influence these functions by modulating dopaminergic neurotransmission. Acute administration of chlorpromazine has been shown to affect locomotor activity and sleep in rodents; limited research exists on its chronic effects. This study aims to investigate the effects of prolonged administration of chlorpromazine hydrochloride on locomotor activity and thiopental-induced sleep in Wistar rats. The objectives of the study are; to determine the effect on the onset time of sleep in rats following chronic doses of chlorpromazine hydrochloride, examine the effect on the duration of sleep in rats following chronic doses of chlorpromazine hydrochloride and evaluate the locomotor activity following the administration of chronic doses of chlorpromazine hydrochloride. Sixty healthy adult male albino Wistar rats were randomly divided into control and treatment groups. Animals were acclimatized for two weeks with free access to food and water. A stock solution of chlorpromazine hydrochloride was prepared by dissolving a known quantity of the drug into a known volume of physiological saline. Chlorpromazine hydrochloride was administered intraperitoneally at doses of 1, 3, and 10 mg/kg, either with thiopental sodium (40 mg/kg) for sleep studies or alone for locomotor assessments. The control groups received thiopental alone or normal saline. Sleep parameters (sleep latency and sleep duration) were recorded every three days for 21 days. Locomotor activity was assessed in an open-field apparatus, with observations repeated across the 21-day study period. Data were expressed as mean ± SEM and analyzed using one-way ANOVA with Dunnett's multiple comparison test, with $p \leq 0.05$ considered statistically significant. The result showed that chronic administration of chlorpromazine hydrochloride significantly decreased sleep latency at 3 mg/kg (days 6 and 9) and 10 mg/kg (days 1, 3, 18, and 21). It also showed that sleep duration was significantly prolonged at 3 mg/kg (days 15, 18, 21) and 10 mg/kg (days 1, 3, 6, 21). These results demonstrate the potentiation of thiopental-induced sleep by chlorpromazine, consistent with its central nervous system-depressant effect. In locomotor studies, 1 mg/kg and 3 mg/kg doses showed no significant effects across the study period. However, 10 mg/kg caused a significant reduction in locomotor activity on days 2 and 3, followed by a significant increase on day 21. This late increase may suggest behavioral sensitization (reverse tolerance), likely due to receptor-level adaptations in dopaminergic signaling or may also have been an artifact. The findings of this study confirms that chlorpromazine hydrochloride enhances barbiturate-induced sleep in Wistar rats under chronic administration, supporting its sedative profile. The observed

reversal in locomotor suppression to increased activity after prolonged dosing indicates neuroadaptive changes, consistent with dopaminergic receptor sensitization. This aligns with previous evidence that dopamine D2 receptor antagonism initially reduces locomotor activity but may trigger compensatory excitatory responses with chronic exposure. In conclusion, chronic administration of chlorpromazine hydrochloride promotes thiopental-induced sleep and alters locomotor activity in Wistar rats. While low to moderate doses maintained sedative properties, prolonged high-dose administration induced behavioral sensitization, suggesting a potential dual central nervous system effect.

Keywords: *Chlorpromazine hydrochloride, dopaminergic system, thiopental-induced sleep, locomotor activity, Wistar rats, behavioral sensitization*

CURCUMIN MITIGATES THE DELAY IN PUBERTAL DEVELOPMENT AND ONSET OF PUBERTY OF THE OFFSPRING OF FOOD-RESTRICTED LACTATING WISTAR RATS

Katchy N.A, Adeniyi D.B, Nwachukwu D.C, Adigwe C.R,Nwannadi V.I

Department of Physiology, College of Medicine, University of Nigeria, Enugu campus

Email: katchyruky@gmail.com; +2348138256395

Maternal food restriction (FR) affects puberty onset and contributes to the developmental programming of reproductive dysfunction in adulthood. Polyphenols such as Curcumin has been increasingly recognized in prevention and treatment of diseases. This study investigated the effects of Curcumin on pubertal development and puberty onset of the offspring of food-restricted lactating Wistar rats. Twenty five female Wistar rats were used for the study. At birth, the dams were then randomly assigned to five (5) groups of five (5) rats each. Group I (Normal control (100% Control diet), Group 2 (FR control (50% Control diet), Group 3 (25 mg/kg Curcumin +50% Control diet) Group 4 (50 mg/kg Curcumin +50% Control diet) and Group 5 (100 mg/kg Curcumin +50% Control diet). The administration commenced throughout lactation after which the offspring were weaned and placed on a standard diet until puberty onset. From PND 21, pups were monitored for balano-preputial separation and vaginal opening. At puberty onset, blood samples were collected from the offspring for the assay of leptin, ghrelin, reproductive hormones and oxidative stress markers. Offspring of Curcumin-treated groups showed a significant increase ($P<0.05$) in food intake, body weight, leptin, reproductive hormones and antioxidant markers (CAT,SOD,GSH) and decreased ($P<0.05$) ghrelin and MDA levels compared to FR control group. Curcumin administration significantly reduced ($P<0.05$) the delay in puberty onset when compared with FR control. It was concluded that maternal consumption of Curcumin during lactation can mitigate the negative effects of food restriction on offspring by preventing the delay in puberty onset and development.

Keywords: *Food restriction, puberty onset, Curcumin, lactation, offspring*

NEUROPROTECTIVE EFFECTS OF N-HEXANE EXTRACT OF *Anacardium occidentale* NUT ON ACETIC ACID-INDUCED COLITIS-MEDIATED ANXIETY AND DEPRESSIVE-LIKE BEHAVIOUR IN WISTAR RATS

Kalesanwo V. O., Ismail H. O.

Department of Physiology, University of Ibadan, Ibadan.

E-mail: kalesanwovictor@gmail.com

Ulcerative colitis is a chronic inflammatory condition of the colon associated with oxidative stress, impaired antioxidant defenses, and mood disturbances. *Anacardium occidentale* possesses antioxidant and anti-inflammatory properties. This study investigated its neuroprotective effects on acetic acid-induced colitis in rats. Thirty rats were divided into six groups of five. Group 1 received saline, Group 2 acetic acid only, and Groups 3–5 varying doses of n-hexane extract of A. occidentale (25, 50, 100 mg/kg) for seven days before colitis induction. Group 6 received prednisolone (2 mg/kg) post-induction. Treatments continued for seven days, and behavioral tests—Open Field, Elevated Plus Maze, and Forced Swimming—were conducted. Biochemical assays assessed antioxidant (glutathione, catalase, GST) and oxidative stress markers (nitrites, malonaldehyde). Group 4 (50 mg/kg) showed enhanced locomotion, grooming, and greater time in the open arms, suggesting anxiolytic activity. Group 5 (100 mg/kg) displayed reduced immobility in the Forced Swimming Test, indicating antidepressant effects. Both extract-treated and prednisolone groups exhibited increased antioxidant levels and reduced oxidative markers compared to untreated colitis rats. The n-hexane extract of Anacardium occidentale attenuates anxiety and depressive-like behavior in ulcerative colitis through antioxidant enhancement and ROS suppression.

Keywords: *Anacardium occidentale,* Colitis, n-hexane extract, Antidepressant

MATERNAL VITAMIN E SUPPLEMENTATION PREVENTED THE DEVELOPMENT OF CARDIOVASCULAR DYSFUNCTIONS IN FEMALE OFFSPRING OF SLEEP-DEPRIVED WISTAR RAT DAMS

Ojewale-Jimoh Aishat Oladoyin.

Federal University of Health Sciences Ila-Orangun. aishat.jimoh@fuhsi.edu.ng | +2347052231009

Maternal sleep deprivation is known to negatively impact the cardiovascular health of offspring, primarily due to increased gestational oxidative stress. This study investigated the protective role of maternal vitamin E supplementation—a potent antioxidant—on cardiac function in female offspring of sleep-deprived pregnant Wistar rats. Twenty pregnant rats (120–150 g) were assigned into four groups from Gestation Day 1 (GD1): Control, Sleep-Deprived (SD), Vitamin E (200 mg/kg), and SD + Vitamin E. Sleep deprivation was induced from GD14 to GD20 for 20 hours daily using the Modified Multiple Platform Method, alongside concurrent vitamin E administration. Cardiovascular assessments were performed at Post-Natal Week (PNW) 25, and at PNW 26, biochemical and histological evaluations were conducted.

The SD group showed significant increases in body weight, heart rate, malondialdehyde, erythrocyte sedimentation rate, corticosterone, C-reactive protein, blood pressure, and mean arterial pressure. Antioxidant enzyme activities (superoxide dismutase, catalase, and glutathione peroxidase) were markedly reduced in this group. Vitamin E supplementation improved these enzyme levels and significantly attenuated blood pressure and inflammatory markers. Histology revealed mild myocardial inflammatory infiltration in both

SD and SD + Vitamin E groups. This study suggests that maternal vitamin E administration during late gestation may offer partial protection against prenatal sleep deprivation-induced cardiovascular dysfunctions—especially oxidative stress and hypertension—in female offspring.

VITAMIN D_3 MITIGATES MONOSODIUM GLUTAMATE–INDUCED TESTICULAR DYSFUNCTION AND SPERM DAMAGE IN RATS THROUGH ITS ANTIOXIDANT, ANTI-INFLAMMATORY, AND CALCIUM MOBILIZATION EFFECTS

Oluwole David Tolulope[1,4*], Ebiwonjumi Oladipupo Samuel[1,3], Yakubu Aliyah Ochuwa[1], Lanase Balqees Sholape[1], Faleye Kaosarat Olajumoke[1], Suleiman Tawakalitu Oluwaseun[1], Oladimeji Abdullahi Abayomi[2], Haruna, Abdullahi[5] Akinleye Olamide Azeezat[1,4], Ajayi Ayodeji Folorunsho[4,5].

[1]Department of Physiology, College of Health Sciences, Crescent University, Abeokuta, Ogun State, Nigeria.
[2]Department of Anatomy, College of Health Sciences, Crescent University, Abeokuta, Ogun State, Nigeria.
[3]Department of Physiology, University of Medical Sciences, Ondo, Ondo State, Nigeria
[4]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[5]Department of Biochemistry, Kogi State University, Anyigba, Nigeria
[6]Anchor Biomed Research Institute, Ogbomoso, Oyo State, Nigeria
Email: david.oluwole-t@cuab.edu.ng;

Western dietary habits have increased male exposure to monosodium glutamate (MSG), a common food additive linked to testicular and sperm dysfunction. This study explored vitamin D3's protective role against MSG-induced reproductive damage in male Wistar rats. Twenty rats (180–200 g) were divided into four groups: Vehicle control, MSG (150 mg/kg), vitamin D3 (500 IU/kg), and MSG + vitamin D3. Treatments were administered orally for 30 days. After sacrifice under anesthesia, the hormonal levels, oxidative stress markers, inflammation, apoptosis indicators, and sperm quality were evaluated using histological and biochemical methods. Statistical analysis was done using one-way ANOVA with Tukey's post hoc test (GraphPad Prism 10.3.0, $p<0.05$). Vitamin D3 significantly improved reproductive hormones (LH, FSH, testosterone), antioxidant enzyme activity (SOD, catalase, GST), sperm motility, viability, and Ca^{2+} ATPase function. It also reduced estradiol, MDA, TNFα, MPO, and caspase-3, indicating lower oxidative stress and inflammation. Histological damage from MSG was visibly reduced with vitamin D3 co-treatment. Vitamin D3 alleviates MSG-induced reproductive harm by improving biochemical and histological parameters. Further molecular studies are suggested.

Keywords: Monosodium glutamate, Vitamin D_3, Testicular functions, Sperm functions.

ANTIDEPRESSANT-LIKE AND ANXIOLYTIC-LIKE EFFECTS OF SESAME OIL IN MICE SUBJECTED TO OPEN-SPACE FORCED SWIM TEST

C. Enesi, Y. Yusha'u, A. AbdulWahab and A.S. Isa
Department of Human Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria-Nigeria.
Email: enesi4comfort@gmail.com

Depression is a mood disorder that causes a persistent feeling of sadness and loss of interest. This study investigated the effect of sesame oil on open-space forced swim-induced depression in mice. Thirty (30) apparently healthy Swiss albino mice (18–26 g), were assigned to six groups (n=5) and treated for 21 days. Group 1 received normal saline (NS) 10 ml/kg; Group 2 was subjected to open-space forced swim test (OSFST) + NS; Groups 3, 4, 5, and 6 were exposed to OSFST + Sesame oil 100, 200, 400 mg/kg, and fluoxetine 20 mg/kg, respectively. All administration of drugs was via oral gavage. At the end of 21 days, Neuro-behavioural assessments were carried out using sucrose preference test, open field test, Y-Maze, and NORT. The animals were sacrificed, and brain samples were obtained for serotonin levels assessment. All results were analyzed using ANOVA. Sesame oil (200 and 400 mg/kg) significantly decreased immobility time ($p<0.05$) and increased sucrose preference ($p<0.05$). Notably, sesame oil (100, 200, and 400 mg/kg) significantly increased serotonin levels in the brain ($p<0.05$). In conclusion, the findings suggest that sesame oil may have antidepressant-like effects, with potential mechanisms involving modulation of serotonin levels and behavioural responses to stress.

ROLES OF PERIODIC ORTHOSTASIS ON THE ELECTROENCEPHALOGRAM IN YOUNG MALE INDIVIDUALS

Mayowa Jeremiah Adeniyi,[1]* Oyesanmi Fabunmi,[2] Awosika Ayoola,[2]
[1]Department of Physiology, Federal University Health Sciences Otukpo, Nigeria
[2]School of Laboratory Medicine and Medical Science, KwaZulu-Natal, Durban, South Africa
[3]College of Medicine, Illinois State University, USA
Email: 7jimade@gmail.com; Tel: +23408066796517

Monitoring brain waves through electroencephalography does not only help in prevention and treatment of diseases, but also provides information about physiological responses to physical changes. We investigated the effect of periodic orthostasis on the electroencephalogram. 30 of 40 male individuals recruited through respondent driven sampling who satisfied the inclusion criteria were selected for the study. The participants underwent orthostasis for 20 minutes at 10.00am, 2.00pm and 6.00pm respectively. Electroencephalogram was recorded using PowerLab 26T. On assumption of erect position, EEG recording was started and continued for 20 minutes during which the standing lasted. At the end of the 20[th] minute of standing, blood pressure and pulse rate were determined using standard procedures. Orthostasis in the morning caused significant increase and decrease in beta wave frequency and alpha/beta ratio when compared to afternoon respectively. Orthostatic baroreflex sensitivity was found to be significantly higher in the morning when compared to afternoon. However, there was no correlation between orthostatic alpha/beta ratio and orthostatic baroreflex sensitivity during morning, afternoon or evening. Morning orthostasis elicited increased cortical activity when compared to afternoon. Adequate attention should be given to the time of the day before the conduct of orthostatic tolerance test for the diagnosis of orthostatic hypotension, differentiation of the causes of syncope and evaluation of hypovolemia and autonomic dysfunctions.

Keywords: Orthostasis, Electroencephalogram, Baroreflex sensitivity, Alpha/beta ratio, Blood pressure

OC-D-9

INTEGRATING ARTIFICIAL INTELLIGENCE AND ENVIRONMENTAL PHYSIOLOGY TO PREDICT CLIMATE CHANGE IMPACT ON HEALTH IN NIGERIA

Ekenedilichukwu Goodness Maduagwuna
Department of Clinical Physiology, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.
Email: ekenedilichukwugoodness@gmail.com

Climate change presents significant health challenges, particularly in regions such as Nigeria where rising temperatures, extreme weather events, and environmental degradation are increasingly prevalent. Environmental physiology, which examines how variables such as temperature, humidity, and air quality affect human function, is critical for understanding these impacts. However, traditional research approaches often struggle to synthesise large, heterogeneous datasets, limiting their ability to generate timely, actionable insights.
This study applies artificial intelligence (AI), specifically machine learning and deep learning, to forecast climate change–related health risks. Multi-source datasets including meteorological records (temperature, humidity, rainfall, air quality), satellite observations, local health statistics (hospital admissions, disease incidence), and wearable physiological sensor data will be integrated. Rigorous data cleaning and ground-truth validation will ensure accuracy. Predictive models, such as neural networks and random forests, will be trained to identify complex relationships between environmental factors and physiological stress responses. Preliminary outcomes are expected to include spatial risk maps identifying health vulnerability hotspots and temporal forecasts for climate-sensitive disease outbreaks (e.g., malaria, heat-related illnesses). These tools will facilitate the development of early warning systems, guide targeted public health interventions, and inform policy measures, such as heat action plans and climate-resilient urban design. By integrating AI with environmental physiology, this work offers a scalable, data-driven approach to safeguard vulnerable populations, improve adaptive capacity, and support Nigeria's public health resilience in the face of climate change.

GLUCO-REGULATORY AND CHONDROPROTECTIVE EFFECTS OF MELATONIN IN DIABETES-OSTEOARTHRITIC MALE WISTAR RATS

[1]SALAM, B.S., [2]ASUKU, A.O., [1]AYINLA, M.T., [1]OLAYAKI, L.A
[1]Department of Physiology, Faculty of Basic Medical Sciences, College of Health Sciences, University of Ilorin, Nigeria.
[2]Bioresources Development Centre, National Biotechnology Research and Development Agency, Ogbomoso, Nigeria.
*Email: salambat50@gmail.com, Tel: +2348101130648

Type 2 diabetes mellitus (T2DM) is a chronic disorder with systemic metabolic abnormalities which influences the pathophysiology of osteoarthritis (OA) (He *et al.,* 2022). Melatonin (MEL), an endogenous hormone has anti-inflammatory, antioxidant, antiapoptotic and immunomodulatory effects. The present study is aimed to examine the effect of melatonin on diabetes-osteoarthritic (DOA) male Wistar rats. Thirty male Wistar rats weighing 175-200 g were divided into five groups at random: control, sham, DOA, MEL and metformin groups. Fasting blood glucose was done before the rats were anaesthetised; OA was induced by injecting 2 mg of monoiodoacetate intra-articularly, while diabetes was induced by high-fat diet and streptozotocin (35 mg/kg). All drugs were administered orally. After 21 days of treatment, animals were anaesthetised; blood, synovial fluid and joint cartilage were collected for analysis. Data were analysed by one-way ANOVA at the $p < 0.05$ significance level. There was a significant increase in fasting blood glucose, insulin resistance, lipid profile, interleukin-1β, tumor necrosis factor-α, caspase-3 levels and matrix metalloproteinase activity, and a significant decrease in catalase, superoxide dismutase and collagen type-2 in the DOA group compared to the control group. Also, immunohistochemical evaluation shows little expression of collagen type-2 in DOA group. However, administration of melatonin significantly reversed the trends. Melatonin improves gluco-regulatory and chondroprotective effects in the DOA Wistar rats via anti-inflammatory, antioxidant, and anti-apoptotic mechanisms. Therefore, melatonin could be used in the management of DOA.

Keywords: Melatonin; Weight; Lipid Profile; Fasting Blood Glucose; Apoptosis

EFFECT OF *ALLIUM SATIVUM* ON BLOOD GLUCOSE, TOTAL AND CONJUGATED BILIRUBIN, AND SERUM AMYLASE LEVELS IN WISTAR RATS

Onunu, G.C., Ezenwanne, E.B., *Onunu, A.N.
Department of Physiology, School of Basic Medical Sciences, College of Medicine, University of Benin, Benin City, Edo state, Nigeria.
*Department of Medicine, University of Benin Teaching Hospital, Benin City, Edo state, Nigeria.
Email: goodluckchukwudinazu@gmail.com.

Allium sativum (garlic) has been heavily employed in traditional medicine because of its hypoglycemic, hepatoprotective, and anti-inflammatory activities. Nevertheless, its specific actions on blood glucose levels, bilirubin metabolism, and pancreatic enzyme activity are not yet elucidated in detail. The objective of this work aimed at investigating the effects of ethanolic garlic extract on blood glucose level, total and conjugated bilirubin level, and serum amylase activity in Wistar rats. A total of 25 male Wistar rats after a 2 weeks acclimatization period were divided into five groups: a control and four treatment groups receiving 100 mg/kg, 250 mg/kg, 400 mg/kg, and 550 mg/kg BW of *Allium sativum* extract every two days for a four weeks administration period. Blood samples at the termination of the experiment were collected for biochemical analysis. The obtained data from all the groups were presented as Mean ± S.E.M (Standard Error of Mean), (n=5) in each group and analyzed for statistical significance by using one-way Analysis of Variance (ANOVA). Values were considered significant at ($P \leq 0.05$). Blood glucose levels decreased significantly (**$p \leq 0.01$) in 250 mg/kg and 400 mg/kg groups, indicating a hypoglycemic effect. However, no significant change was observed in the 100 mg/kg and 550 mg/kg groups ($p > 0.05$). There was no significant difference ($p > 0.05$) in total and conjugated bilirubin levels, and serum

amylase activities. Conclusively, this study on the effect of *Allium sativum* demonstrated garlic's hypoglycemic potential at moderate doses.

Keywords: *Allium Sativum*, Glucose, Bilirubin, Serum Amylase, Wistar Rats.

MODULATORY EFFECTS OF CANNABIDIOL (CBD) OIL ON CIRCADIAN RHYTHM DISRUPTION IN PREGNANT SPRAGUE-DAWLEY DAMS.

Okwute P.G.[1*], Adeleke F.F[1]., Mofolorunso A.M[1]., Oloyo A.K[2].

[1]Department of Physiology, Babcock University, Ilisan-Remo, Ogun State, Nigeria

[2]Department of Physiology, College of Medicine of the University of Lagos, Lagos State, Nigeria

Email: okwutep@babcock.edu.ng; Tel: +234708038082953

The suprachiasmatic nucleus (SCN) regulates circadian rhythms and responds to light cues. Disruption of circadian rhythm during pregnancy is linked to adverse maternal and fetal outcomes. While the endocannabinoid system modulates circadian rhythms, little is known about the effects of cannabidiol (CBD) oil during pregnancy. This study examined the modulatory role of CBD oil on light-induced circadian disruption in pregnant Sprague Dawley rats. Pregnant rats were randomized into five groups: (1) Control, (2) Constant Light (LL), (3) LL + CBD oil, (4) Constant Darkness (DD), and (5) DD + CBD oil, from gestational day 0 to 18. Parameters measured included daily food intake, weekly body weight, melatonin, Oxidative stress, and gene expression.Constant light increased gestational weight gain, indicating enhanced adiposity, while CBD reduced weight gain, particularly in the DD + CBD group. Lighting altered appetite, with DD increasing and LL reducing food intake. Melatonin was suppressed in LL and LL + CBD groups but elevated in DD and DD + CBD groups, suggesting CBD enhances melatonin signalling. LL + CBD group upregulated the expressions of CLOCK and BMAL1 genes. CBD oil was able to mitigate the adverse effects of circadian disruption in pregnancy by enhancing antioxidant defence, supporting melatonin secretion, and modulating circadian gene expression.

Keywords: *Cannabinoid (CBD) Oil, Circadian rhythm, Melatonin, Pregnancy*

SLEEP DEPRIVATION AGGRAVATE PLACENTAL OXIDATIVE STATUS AND ALTERS HAEMATOLOGICAL VARIABLES IN PREGNANT WISTAR RATS

Abraham, T.F[1,3]., Akindele, O.O. and Raji Y.

[1]Department of Physiology, Faculty of Basic Medical Sciences, College of Medicine, Federal University of Health Sciences, Ila-Orangun, Nigeria.

2Department of Physiology, Faculty of Basic Medical Sciences, College of Medicine, University of Ibadan, Ibadan, Nigeria.

3Department of Physiology and Biophysics, Institute of Biological Sciences, Federal University of Minas Gerais, Belo Horizonte, Brazil.

E-mail: tunde.abraham@fuhsi.edu.ng

Prenatal sleep deprivation is associated with increased risk for preterm delivery and complications, partly due to burst in oxidative stress from increased placental mitochondrial activity and establishment of intervillous circulation. Studies linking this relationship is limited, hence, we evaluated how maternal sleep deprivation affect placental stress and haematology in Wistar rats. Thirty pregnant Wistar rats (180g-200g) were assigned into six groups (n=5): control and sleep deprived (SD) subgroups for three Gestational periods/Days (GD1-7,GD8-14 and GD15-21). Sleep deprivation was by the multiple platform technique (20h/day for 7 days). The dams were euthanized on GD7,14 and 21 respectively, after which the placentas were excised for biochemical studies and blood collected for haematological analysis. Haemoglobin level decreased significantly at GD15-21 ($P<0.05$), while WBC increased ($P<0.0001$) at GD8-14 and GD15-2, with concomitant increase in platelets ($P<0.0001$) at GD15-21 in the SD group. Lymphocytes increased ($P<0.05$) at GD1-7, whereas monocytes exhibited trimester-specific variation in SD rats, decreasing ($P<0.05$) at GD1-7 and increasing ($P<0.05$) at GD15-21. Eosinophils decrease ($P<0.05$) at GD15-21 in the SD. Placental SOD activity decreased ($P<0.05$) at GD1-7 while MDA levels increased ($p<0.05$) at GD1-7 and GD15-21 in SD, with no significant difference in catalase concentrations. Placental histology shows significant haemorrhage, disrupted cytoarchitecture of the frondosum layer and infiltration of inflammatory cells in SD. Maternal sleep deprivation worsens oxidative stress, impairs haematological functions and placental cytoarchitecture. This may adversely affect the mother and fetal health, with the potential to epigenetically program the fetus for increased susceptibility to chronic diseases later in life.

Keywords: Sleep deprivation, Pregnancy, Placenta, Oxidative stress, Haematology.

TIME-DEPENDENT EFFECTS OF ASCORBIC ACID AND α-TOCOPHEROL ON TOTAL WHITE BLOOD CELL COUNTS AND PLATELET INDICES, FOLLOWING LEAD ACETATE INDUCED IMMUNE TOXICITY IN MALE WISTAR RATS.

Olatunde B. J, [1]*, Ojeka S. O, [1], Dapper D. V, [1].

[1]Department of Human Physiology, Faculty of Basic Medical Sciences, University of Port Harcourt, Choba, Rivers State, Nigeria.

E-mail: babatunde_olatunde@uniport.edu.ng. +2348054711938

The study was undertaken to establish the time-dependent effects of ascorbic acid and α-tocopherol on total white blood cell counts and platelet indices, following lead acetate induced immune toxicity in male wistar rats. 110 adult male wistar rats with an average body weight of 180 - 200g, were bought from the Animals house in Faculty of Basic Medical Sciences, University of Port Harcourt. The animals were acclimatized for two weeks and thereafter divided into 6 groups (groups 1 to 6). The study was carried out in 3 phases; acute (7 days), sub-chronic (30days) and chronic (60 days) phases. Group 1 represented the normal control and was given only distilled water and normal rat feed while the rest of the groups were orally given; group 2: 10 mg/kg body weight of lead acetate only, group 3: 200 mg/kg body weight of ascorbic acid only, group 4: 1000 iu/kg body weight of α-tocopherol only, group 5: 10 mg/kg of lead acetate + 200 mg/kg of ascorbic acid, group 6: 10 mg/kg of lead acetate + 1000 iu/kg of α-tocopherol respectively. At the expiration of the experimentation for each phase, five (5) rats were sacrificed and blood samples collected from each rat and examined for total white blood cell counts and differentials and platelet counts and indices. The results showed a significant decrease in the concentrations of the total white blood cell counts and differentials in the lead acetate group

with respect to the control indicating impaired immune functions, in all the phases. There was also a significant decrease in the concentrations of Plateletocrit (PCT) and Platelet count (PLT) and a significant increase in the concentrations of mean platelet volume (MPV), platelet distribution width (PDW) and Platelet large cell ratio (PLCR), all in the lead acetate group and in all the phases, with respect to the control group indicating their dysfunctions. However, there was significant increase in the concentrations of the total white blood cell counts in groups 5 and 6 with respect to lead acetate group indicating enhancement in the immune functions, across the phases. There was significant increase in the concentrations of (PCT) and (PLT) and a significant decrease in the concentrations of (MPV), (PDW) and (PLCR) in groups 5 to 6 with respect to lead acetate group indicating amelioration of the impaired functions across the phases. In conclusion, ascorbic acid and α-tocopherol demonstrated the ability to ameliorate the lead acetate induced immune toxicity on these parameters, in a time-dependant manner.

Keywords: *Lead acetate, Ameliorate, total white blood cell counts and differentials, Platelet indices.*

PERINATAL HIGH SALT EXPOSURE PROGRAMS HYPERTENSION AND OXIDATIVE STRESS IN ADULT OFFSPRING OF SPRAGUE-DAWLEY RATS

Ismail-Badmus K. B., Balogun Z. T., Adejare, A. A., Iranloye B., Oloyo A. K.
College of Medicine, University of Lagos, Physiology, Surulere, Lagos.

Emerging evidence supports the concept that hypertension may have developmental origins, with perinatal environmental exposures such as maternal high salt intake implicated in the early programming of cardiovascular dysfunction. The heritability of salt sensitivity and the role of oxidative stress in vascular pathophysiology further strengthen this hypothesis. This study evaluated the effect of perinatal high salt exposure on blood pressure and oxidative stress markers in adult offspring. Pregnant Sprague-Dawley rats were assigned to either a normal salt (0.3% NaCl) or high salt (8% NaCl) diet throughout gestation and lactation. After weaning, the offspring were further maintained on either a normal or high salt diet until 14 weeks of age, forming four experimental groups. Systolic, diastolic, and mean arterial blood pressure were measured via femoral artery cannulation under IP anaesthesia with 25% urethane and 1% alpha-chloralose. Serum and urinary sodium and potassium concentrations were assessed, along with oxidative stress markers, including superoxide dismutase (SOD) activity and malondialdehyde (MDA) levels. Data were analyzed using one-way ANOVA with a significance threshold of $p < 0.05$. Offspring of high salt-fed dams exhibited significantly elevated mean arterial blood pressure even when maintained on a normal salt diet post-weaning. Although serum sodium levels were comparable across groups, urinary sodium and potassium excretion were higher in offspring exposed to post-weaning high salt diets. Offspring from high salt-fed dams also showed reduced SOD activity and elevated serum MDA concentrations, indicating heightened oxidative stress. These findings suggest that perinatal exposure to a maternal high salt diet predisposes offspring to adult-onset hypertension, potentially through mechanisms involving persistent oxidative stress. This supports the developmental programming hypothesis and highlights the critical influence of maternal nutrition on long-term cardiovascular health.

Keywords: *Perinatal programming, high salt diet, hypertension, oxidative stress, superoxide dismutase, malondialdehyde, developmental origins.*

HAEMATORESTORATIVE EFFECTS OF *CITRULLUS LANATUS* JUICE IN PHENYLHYDRAZINE-INDUCED ANAEMIA IN SPRAGUE DAWLEY RATS

***Aikpitanyi-Iduitua R.O[1], Aikpitanyi-Iduitua G.A[2].**
[1]Department of Physiology, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria
[2]Department of Medical Laboratory Science, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria.
*Email: rosemary.aikpitanyi-iduitua@uniben.edu; Tel: 08027448703

Anaemia is a public health problem with a remarkable global impact, and haemolytic anaemia caused by oxidative erythrocyte damage poses a major concern. *Citrullus lanatus* (watermelon), known for its antioxidant and L-citrulline content, may support red blood cell recovery, yet its efficacy in haemolytic anaemic conditions appears to be remotely investigated. This study evaluated the haematorestorative potential of *C. lanatus* juice in phenylhydrazine-induced anaemia in male Sprague Dawley rats. Fifty rats were acclimatized for two weeks; five served as non-anaemic controls. The remaining forty-five were induced for haemolytic anaemia using phenylhydrazine hydrochloride and anaemia was confirmed by a packed cell volume (PCV) ≤ 20%. These were divided into three groups: distilled water (negative control), *C. lanatus* juice (10 ml/kg/day), and vitamin B complex plus folic acid (positive control). Treatments were administered orally for three weeks. Weekly, five rats per group were euthanized for haematological analysis using automated analyzer to assess RBC indices: RBC, HCT, HGB, MCV, MCH, MCHC, RDW-SD, and RDW-CV. Data were analyzed using one-way ANOVA ($p < 0.05$). By week one, all the anaemic groups showed significant reductions in red cell parameters when compared with the non-anaemic control. By weeks two and three, the *C. lanatus* group showed full restoration of erythrocyte indices, comparable to the vitamin-treated group ($p < 0.05$). These findings highlight the potential of *C. lanatus* juice as a natural, functional intervention in anaemia management. Follow-up study to ascertain the specific mechanisms is recommended.

Keywords: *Citrullus lanatus*; haemolytic anaemia; haematology; antioxidants; Sprague Dawley rats

INSUFFICIENT SLEEP MODEL AND MALE REPRODUCTIVE FUNCTION; THE ROLE OF CONCOMITANT ZINC SUPPLEMENTATION

***[1]Muhammad, S., [1]Salisu, A. I., [2]Tankon Y. and [2]Mohammed, A.**
[1]Department of Human Physiology, Faculty of Basic Medical Sciences, Collage of Health Sciences, Bayero University, Kano, Nigeria.
[2]Department of Human Physiology, Faculty of Basic Medical Sciences, Collage of Health Sciences, Ahmadu Bello University, Zaria, Nigeria.
Email; shihabuddeenm@gmail.com, Tel: +234 8036890654

Insufficient sleep (IS) in today's society is gaining recognition as a public health concern, with increasing evidences that linked it to poorer health outcomes. Coincidentally, there is remarkable decline in fertility rates, in these industrialized societies which is believed to be due to lifestyle modifications. We therefore set up IS model of sleep deprivation (SD), to study its effects on male reproductive functions and the influence of concomitant Zinc supplementation on those effects. Twenty-four (24) male Wistar rats (aged 12- 14 weeks) were randomly grouped into three: Control, IS and ISZ models. IS and ISZ models were subjected to SD for 18 hours (07:00pm – 01:00pm next day) using Modified Multiple Platform Method (MMPM). The rats in ISZ model were given orally given Zinc sulphates (5mg/animal/day) while those in control and IS models were given distilled water (1ml/animal/day) daily for 56 days respectively. Sleep deprivation in IS model resulted in significant increase (p<0.05) in serum Corticosterone, testicular tissue MDA, serum FSH and significant decrease (p<0.05) in testicular tissue TAC, serum Testosterone, serum Estradiol, sperm count and percentage of sperm with active progressive motility compared to the control. On the other hand, concomitant Zinc supplementation (ISZ model) significantly (p<0.05) increases testicular tissue TAC, serum Estradiol and significantly (p<0.05) decreases testicular tissue MDA and serum FSH compared to the IS model. The IS model of SD deteriorates male reproductive functions, while concomitant Zinc supplementation ameliorates some of these functions.

Keywords: *Insufficient Sleep; Sleep Deprivation; Total Anti-oxidant Capacity;*

CASTRATION AND METFORMIN SYNERGISTICALLY REPRESS P16 SENESCENCE ASSOCIATED GENE THROUGH P16 DNA HYPERMETHYLATION IN MALE PIGS

EHIAGWINA, Isaiah Osayande[(1)], AGOREYO, Freddy Ohworode[(1)] and OMOROGIUWA, Arthur[(1)]

[(1)] Department of Physiology, School of Basic Medical Sciences, University of Benin, Edo State. Nigeria.
Email: ehiagwina.isaiah@yahoo.com, Tel: 08136919336

P16 is a senescent-associated gene whose expression increases with age to reflect the rate of ageing at a molecular level. This study's aim was to investigate the combined effects of metformin and castration on P16 gene expression and DNA methylation. Twenty clinically healthy, young male pigs (51 ± 11.4 days old) from two paternal and five maternal bloodlines were randomly assigned into four equal experimental groups (n=5): Group-A: Control (saline), Group-B: Metformin-only, Group-C: Unilateral castration + Metformin, Group-D: Bilateral castration + Metformin. Treatment was administered orally, daily for 120 days at 25 mg/kg BW. Following sacrifice 24 hours after the final dose, blood and thymus samples were harvested for gene expression and DNA methylation analyses. Data were evaluated using one-way ANOVA and Tukey's multiple comparison test. P<0.05 was considered statistically significant. Metformin alone significantly down-regulated P16 gene both in the blood and thymus (P<0.05). Metformin and castration synergically repressed P16 gene expression in the blood and thymus (P<0.05) with bilateral castration significantly more potent than unilateral castration particularly in the blood. Metformin alone resulted to significant P16 DNA hypermethylation (P<0.05) suggesting a mechanism for its down-regulation. Metformin combined with unilateral castration resulted to further P16 DNA hypermethylation though not significantly (P>0.05). Bilateral castration with metformin significantly resulted to P16 DNA hypermethylation aligning with its gene repression. This study demonstrated a synergy between metformin and castration in down-regulating P16 gene in blood and thymus likely through P16 DNA hypermethylation with implications of improving ageing in male pigs.

AMELIORATIVE EFFECT OF *Solenostemon monostachyus* (African Dead Nettle) ON LIVER FUNCTIONS IN STZ-INDUCED DIABETIC RATS

Eriata, B.O[1*]; Okonkwo, O.V[2] ; Adie, P.U[1]; Uquetan, S. U[1]; Ante, E.E[1]; Ibitham, I.I[1]; Obembe, A.O[1]

[1]Department of Physiology, Faculty of Basic Medical Science, University of Calabar, Calabar – Nigeria
[2]Department of Physiology, Faculty of Basic Medical Science, Arthur Jarvis University, Calabar - Nigeria
E-mail:eriataomon13@gmail.com; 08159459335,

Diabetes mellitus is a widespread metabolic disorder causing systemic complications. *Solenostemon monostachyus* may improve hyperglycemia associated with diabetes, but there is a paucity of information on its effect on diabetes related liver dysfunctions. It becomes expedient to access its ability to not only ameliorate diabetes but also diabetes induced disorders on liver functions with emphasis on Bile secretion, Bile electrolytes and Liver enzymes. A total of twenty-five (25) *Wistar* rats grouped into five groups of 5 (five) rats weighing 110 – 200g were used. The first group was the control group (3ml of normal saline daily), the second group was the diabetic untreated (50mg/kg), the third group was administered metformin (200mg/kg), while the fourth and fifth groups were administered low dose (200mg/kg) and high dose (400mg/kg) of *S. monostachyus* extract respectively after STZ induction. The treatment lasted for 21 days. The result showed a significant (P<0.05) increase in the extract treated group compared to the control group on bile secretion. The results on the electrolytes showed a significant (P<0.05) increase in the extract treated groups compared to the control group except in the case of chloride ion concentration which showed a decrease in the extract treated groups. The result showed a significant (P<0.05) decrease in enzymes concentration in the extract treated groups compared to the untreated group. There was amelioration of hepatocytes and cytoplasmic granularity in the extract treated group. The results show that *S. monostachyus* may not have a significant ameliorative effect on liver damage.

EFFECTS OF PAPIN AND PHYCOCYANIN ON SOME BIOCHEMICAL PARAMETERS AND LIVER HISTOLOGY IN WISTAR RATS.

Adie P.U[1]*, Udemeobong E.O[1], Atim I. O[1], Eriata B.O[1], Ekpe C. G[1], Uket, J.M[1]. Asuquo. N. G[2], Uquetan S. U[1]

[1]Department of Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, University of Calabar, Calabar.
[2]Department of Physiology, School of Basic Medical Sciences, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria.
Email: polycarp.physiol@unical.edu.ng; 08134205676

Papain (from *Carica papaya*) and phycocyanin (from *Spirulina platensis*) are widely consumed food additives with digestive and antioxidant roles. However, their systemic impacts remain poorly understood. This study assessed the individual and combined effects of papain and phycocyanin on serum electrolytes, protein profile, lipid parameters, and liver histology in Wistar rats. 20 Male Wistar rats were divided into four groups and administered potable water and rat feed (control), papain (100 mg/kg), phycocyanin (200 mg/kg), or both papain + phycocyanin, orally for 28 days. After which the animals were sacrificed and blood samples were collected via cardiac puncture and analyzed for serum electrolytes- Na^+, K^+, Cl^-, HCO_3^-, serum proteins- total protein, albumin, globulin, Lipid profile- total cholesterol, triglycerides, HDL-c, LDL-c, and VLDL-c while Liver tissues were examined histologically using H&E and Masson's Trichrome stains. Papain treatment led to hyperkalemia, hypoproteinemia, altered lipid profile, and centrilobular hepatic damage. Phycocyanin significantly improved electrolyte and lipid indices and preserved hepatic architecture. Co-treatment attenuated papain-induced disruptions, showing intermediary effects between monotherapies. Papain may cause systemic and hepatic stress, whereas phycocyanin confers hepatoprotection. Co-administration offers a protective balance and supports phycocyanin's potential as a nutraceutical adjunct.

6"-*O*-ACETYLGENISTIN-RICH FRACTION OF *TELFAIRIA OCCIDENTALIS* ACTIVATES GLP-1 AND IRS2/PI3K/AKT/PDX1 PATHWAYS IN HIGH-FAT DIET/STREPTOZOTOCIN-INDUCED DIABETIC RATS

[1,2,]*Feyitimi, A.A., [2]Salman, T.M. [3]Oguntoye, O.S. and [4]Alada, A.R.A.
[1]Department of Medical Physiology, University of Rwanda, Republic of Rwanda.
[2]Department of Physiology, University of Ilorin, Ilorin, Nigeria.
[3]Department of Chemistry, University of Ilorin, Ilorin, Nigeria.
[4]Department of Physiology, University of Ibadan, Nigeria.
Email:: aa.feyitimi@ur.ac.rw

Telfairia occidentalis (TO) is reported to have hypoglycaemic and antidiabetic potential, although the mechanisms remain unclear. This study evaluated the effect of 6"-*O*-Acetylgenistin-rich fraction of TO (AG) on insulin resistance, pancreatic β-cell function, and survival in type 2 diabetes (T2D). Thirty male Wistar rats (180 – 210 g) were randomly assigned to normal control (NC) and diabetic control (DC) groups, which received 1 mg/kg distilled water, while G5, AG100, AG200, and AG600 were high-fat diet/streptozotocin diabetic rats treated orally for 14 days with 5 mg/kg Glibenclamide, 100 mg/kg, 200 mg/kg, and 600 mg/kg AG, respectively. Serum, liver, and pancreas were evaluated for hormonal levels, histopathological changes, and protein expression. Results showed a significant ($p < 0.001$) reversal of elevated glucose and HOMA-IR in G5, AG100, and AG600, with a mitigating effect in AG200. Moreover, the reduced insulin sensitivity index (ISI) in untreated diabetic rats was markedly reversed in G5, AG100, and AG600, and mitigated in AG200. Similarly, the reduced HOMA-β was reversed in G5, AG100, and AG600. GLP-1 levels were elevated in rats treated with AG or Glibenclamide, while insulin levels increased in G5, AG100, and AG600. Reduced islet cellularity was fully reversed in G5 and AG100, increased significantly in AG200, and showed a slight increase in AG600. Insulin- and PDX1-positive cells increased in G5, AG100, and AG600. All treatments triggered an increase in p-PI3K expression. G5, AG100, and AG600 also exhibited increased expression of p-IRS2 and p-Akt. AG potentially attenuates insulin resistance and β-cell dysfunction via GLP-1 and IRS2/PI3K/Akt/PDX1 pathways.

Keywords: *Telfairia occidentalis*; 6"-*O*-Acetylgenistin; insulin; IRS2; PI3K; Akt; PDX1

EFFECT OF *PEPEROMIA PELLUCIDA* (PEPPER ELDER) ON ELECTROCARDIOGRAM AND CARDIAC INFLAMMATORY MARKERS IN *WISTAR* RATS FOLLOWING ADRENALINE-INDUCED MYOCARDIAL INFARCTION

Ante, E.E.*[1*]; Archibong, E.A[2]; Eriata, B.O[1]; Asuquo, J.O[3]; Nku C.[1]
[1]Department of Physiology, Faculty of Basic Medical Science, University of Calabar, Calabar – Nigeria
[2]Department of Physiology, Faculty of Basic Medical Science, David Umahi Federal University of Health Sciences, Uburu, Ebonyi State - Nigeria
[3]Department of Physiology, Faculty of Basic Medical Science, Arthur Jarvis University, Cross River State - Nigeria
Email: hoperantel55@gmail.com; 08033775184

Myocardial infarction (M.I), commonly called heart attack is a cardiovascular condition that occurs when the blood flow is decreased or terminated to a part of the heart, thereby resulting in damage to the heart muscle. A study shows that P. pellucida has an ameliorative effect on certain cardiovascular diseases. This present study was therefore targeted towards the Effect of *Peperomia pellucida* on the electrocardiogram and Cardiac inflammatory markers in albino Wistar rats following adrenaline-induced myocardial infarction. A total of twenty-five (25) *Wistar* rats grouped into five groups of 5 (five) rats weighing 110 – 200g were used. The first group was the control group, the second group was the adrenaline group (85mg/kg), the third group was administered the extract P. *pellucida*, while the fourth and fifth groups were administered adrenaline+extract and captopril (50mg/kg) respectively. The treatment lasted for 21 days. The result showed a significant ($P<0.05$) decrease in R-R, P-R, Q-T and QRS complex of extract treated groups compared to the control but no significant difference in the R-T interval. The ECG tracings showed amelioration upon administration of the extract. The cardiac inflammatory markers, Interleukin 6 and C - reactive protein showed a significant ($P<0.05$) decrease in all treated groups compared to the control. It is concluded that ethanolic extract of *P. Pellucida* has cardioprotective ability and is able to prevent myocardial injury induced by adrenaline toxicity, probably due to active phytochemicals present in the plant extract.

A SYSTEMATIC REVIEW EXPLORING THE ROLE OF KISSPEPTIN IN PROSTATE CANCER

Oyedokun P.A.[1,2], Oyedokun M.A.[2,3], Adegbola C.A.[1,2], Oladipo A.A.[1,2], Oladele T.A.[2,4], Oyeleke B.T.[2,5], Osikoya O.O.[2,6], Oyeniyi G.A.[7], Akhigbe TM[2,8], Akhigbe RE[1,2*]
[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Nigeria. [3]Department of Nursing, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

[4]Department of Pure and Applied Chemistry, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[5]Department of Medical Laboratory Science, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[6]Department of Medicine and Surgery, Babcock University, Ilishan Remo, Ogun State, Nigeria.
[7]Department of Surgery, Osun State University Teaching Hospital, Osogbo, Osun State, Nigeria.
[8]Breeding and Genetic Unit, Department of Agronomy, Osun State University, Osun State, Nigeria.
*Email: reakhigbe@gmail.com

Prostate cancer (CAP) is the second most commonly diagnosed cancer and the leading cause of cancer-related death in men globally. The growth and progression are dependent on androgens, which are regulated by the hypothalamic-pituitary-testicular (HPT) axis. Kisspeptin (Kiss1), the endogenous ligand for the G protein-coupled KISS1 receptor (KISS1R; also known as GPR54), is crucial in regulating the HPT axis and has emerged as a significant player in prostate cancer. Nonetheless, available data in the literature is scanty. The present study is a systematic review of human and animal studies evaluating the role of Kiss1 in CAP growth. This study was conducted in accordance with the PRISMA guidelines. After a pre-defined strategic protocol, 122 studies were screened, and 8 articles were identified as eligible for this study. The studies were observational (1), *in vitro* using human cell lines (2), randomized placebo-controlled (1), and animal studies (4). Kiss1 expression was downregulated in CAP cells. In addition, administration of Kiss1 analogue upregulated Kiss1 and KISS1R expression. Moreso, Kiss1 administration suppressed GnRH, LH, FSH, testosterone, and *FSH and LH* mRNA, demonstrating the suppressor effect of Kiss1 on the HPT axis. Also, Kiss1 administration reduced prostate tumor volume and prostate-specific antigen. Furthermore, Kiss1 administration downregulated *VEGF* mRNA and the migration and invasion of endothelial cells. This study reveals the role of Kiss1/Kiss1R signaling in incident CAP and its progression. Kiss1 suppresses prostate tumors by downregulating the HPT axis and *VEGF* mRNA.

Keywords: KISS1R; kisspeptin; malignancy; prostate cancer; testosterone; sex hormones

ACRIFLAVINE ATTENUATES METABOLIC SYNDROME AND ASSOCIATED CARDIO-RENAL DYSFUNCTION IN HIGH CARBOHYDRATE HIGH FAT DIET FED WISTAR RATS

Ademilusi E.O.[1,2], Alabi V.S.[2], Ajayi T.T.[2], Ademilusi O.M.[3], Omolaso B.O.[2]
[1]Department of Physiology, University of Ibadan, Nigeria.
[2]Department of Physiology, University of Medical Sciences, Ondo, Nigeria.
[3]Department of Health Policy and Management, University of Ibadan, Nigeria.
Email: ademilusiemma@yahoo.com; Tel: +2348055930578

There is presently no treatment for metabolic syndrome. Current remedy involves combination of different drugs which is demanding and reduces patient compliance. Acriflavine, has been reported to possess activity in the treatment of obesity, right ventriclar hypertrophy and right ventricle systolic pressure. Therefore, this study was designed to evaluate the potentials of acriflavine to act as remedy for metabolic syndrome and its associated cardio-renal dysfunction in rats. Twenty male Wistar rats were randomly divided into four groups(n=5): Control, Fructose (F) given high carbohydrate high fat diet (HCHFD) and fructose sweetened drink (FSD), Acriflavine (ACH) given feed, water and acriflavine (100 mg/kg/day), and F+ACH given HCHFD, FSD and acriflavine (100mg/kg/day) for 14 weeks. Fluid, feed, energy intake, urine output, blood pressure, anthropometric variables, fasting blood glucose, lipid profile, abdominal fat mass and morphometry, adipose tissue lactate and HIF-1α, serum insulin, IL-10, hs-crp, creatinine, blood urea nitrogen, bilirubin, albumin, leukocyte count, erythrocyte osmotic fragility, heart and kidney weight, heart failure markers, estimated glomerular filtration rate and HOMA-IR were determined. Heart and adipose tissue histopathological examination were done. Data were compared with ANOVA and $P<0.05$ was considered statistically significant. Mean blood pressure ($p<0.0001$), heart rate ($p=0.0004$), fluid ($p<0.0001$), and energy ($p<0.0001$) intake, weight gain ($p=0.002$), abdominal circumference ($p=0.0007$), abdominal fat ($p=0.004$), blood glucose ($p<0.0001$), insulin ($p=0.0002$), HOMA-IR ($p<0.0001$), hs-crp ($p=0.0021$), leukocytes ($p=0.0036$), triglyceride ($p=0.0011$), total cholesterol ($p=0.024$), adipose tissue diameter ($p<0.0001$), lactate ($p=0.0011$), and HIF-1α ($p=0.0205$), creatinine , blood urea nitrogen, bilirubin, albumin, erythrocyte osmotic fragility, heart weight ($p=0.0327$), heart failure markers, estimated glomerular filtration rate, increased significantly in F when compared with Control, while most of them reduced in ACH treated rats when compared with F.. The findings of this study showed that acriflavine ameliorated metabolic syndrome and its associated cardio-renal dysfunction by attenuating adipocyte dysfunction and insulin resistance in rats.

NARINGIN-RICH FRACTION OF TANGERINE PEEL EXTRACT IMPROVES MEMORY VIA THE YKL-40 AND BDNF PATHWAYS IN A NOVEL DIABETES-ALZHEIMER'S COMORBIDITY MODEL

Akintoye, O. O[1]., Aransi-ola, K. T[1]., Nkemakolam, E. O[1]., Okem, P[1]., Adejumo, M. O[1]., Ajibare, A. J[1]*
1*Neuro-Reproductive and Metabolism Unit, Department of Physiology, Faculty of Basic Medical and Clinical Sciences, College of Medicine, Lead City University, Ibadan, Oyo, State, Nigeria*
*Corresponding Author: Dr Ayodeji Johnson Ajibare. *Mobile:* +2348033273839
Email: ajibare.ayodeji@lcu.edu.ng.

The global prevalence of Diabetes-Alzheimer's comorbidity with its associated memory impairment has been steadily increasing over the past decades. However, available treatments are limited by single-pathology pathway approaches, hence the need for natural compounds with neuroprotective potentials for dual-pathology treatment. Naringin, a flavonoid found in tangerine peel has been shown to possess antioxidant and anti-inflammatory activities but its neuroprotective potentials on the Diabetes-Alzheimer's disease has not been yet explored. This study assessed the ameliorative effects of Naringin-rich fraction of tangerine peel extract on memory impairment in a Diabetes-Alzheimer's comorbidity.
Twenty-eight male Wistar rats (120-150 g) were fed high fat diet for 8 weeks. Obese rats (180-250g) were randomly assigned into four groups (n = 7). Control (distilled water p.o), DM (streptozotocin; 65 mg/kg i.p), DM + ALZ

(Streptozotocin + Scopolamine 1mg/kg i.p) to induce Diabetes-Alzheimer's comorbidity and DM + ALZ + NAR (150mg/kg p.o). Memory function was assessed by using the Y-maze and Novel Object Recognition Tests. Brain tissues were processed for biochemical assays and gene expression. Data were expressed as mean ± SEM and analyzed using ANOVA and Tukey's Multiple Comparison Test. Naringin-rich fraction of tangerine peel extract significantly enhanced recognition memory and spatial working memory ($p < 0.0001$). Neuroinflammatory biomarker expression (YKL-40) ($p < 0.0001$) and glutamate levels ($p = 0.001$) were significantly reduced while BDNF expression ($p < 0.0001$) and AChE levels ($p = 0.002$) were significantly increased. In conclusion, Naringin-rich fraction of tangerine peel extract exhibited ameliorative effects on memory impairment in Diabetes-Alzheimer's disease in Wistar rats.

Keywords*:* Naringin, Memory Impairment, Diabetes-Alzheimer's disease, Streptozotocin, Neuroinflammation, Neuroplasticity

CAFFEINE CONSUMPTION REDUCES LIPID PROFILE BUT DISRUPTS LIVER CYTOLOGY AND REPRODUCTIVE FUNCTIONS OF PERI-PUBERTAL FEMALE WISTAR RATS

***Ogunwole E., Emojevwe V. O., Adesida A. O., Ojo J. B., Luwah P. E., Oloruntoba F. P., Odewale O. F., Enigbokan O. E. and Akinsefunmi O. A.**

Department of Physiology, University of Medical Sciences, Ondo City, Ondo, Nigeria

**Corresponding Author: eogunwole@unimed.edu.ng Phone: +2347059545657*

Consumption of over-the-counter medicines and caffeinated beverages is prevalent among adolescents. Caffeine has been associated with some health problems. This study investigates its consumption impacts on lipid profile, liver, and reproductive functions of peri-pubertal female Wistar rats. Five groups (n=5) of peri-pubertal Wistar rats received distilled water (control), II and III (120 and 180 mg/Kg/day) caffeine orally for 28 days and sacrificed, IV and V (120 and 180 mg/Kg/day) caffeine orally for 28 days but allowed to recover for the same period and then sacrificed. During the experiment, vaginal smears were obtained daily for estrous cycle study via microscopy. The levels of Malondialdehyde (MDA), Superoxide dismutase (SOD), and Catalase activities in the ovaries and uteri were determined by spectrophotometry. Histology of liver, ovaries, and uteri was assessed by microscopy. Some lipid profile was assessed by colorimetric test. Luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, tissue 8-hydroxyguanosine (8-OHdG), Tumor Necrosis Factor Alpha (TNF-α), and C-reactive protein levels were measured by ELISA. Data were analyzed using ANOVA at $p < 0.05$ significance. Caffeine reduced lipid profile, FSH, and LH, but increased estradiol levels and disrupted the metestrus and diestrus phases of the estrous cycle. It reduced tissue TNF-α and C-reactive protein levels but increased ovarian MDA, and altered ovarian histology reversibly. Caffeine increased uterine MDA and 8-OHdG levels but reduced SOD levels, and irreversibly distorted the endometrial and liver histology. Caffeine possesses beneficial effects via reduced lipid but is detrimental to liver and reproductive functions via the observed adverse alterations.

Keywords: Caffeine; Ovary; Uterus; Infertility; Peri-pubertal rats

L-ARGININE ATTENUATES CISPLATIN-INDUCED CARDIOTOXICITY BY ACTIVATING NRF2/HO-1 AND UPREGULATING GLUTATHIONE IN MALE WISTAR RAT

Saka W.A.[1], Adegbola C.A.[1,2], Akhigbe T.M.[2,3], Ayandele O.A.[1], Ashonibare V.J.[2,4], Akhigbe R.E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria.

[3]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria. [4]Cardiovascular Regenerative Medicine & Tissue Engineering 3D Lab, Department of Cardiovascular Surgery and Research Group for Experimental Surgery, Heinrich Heine University, Medical Faculty, Düsseldorf Germany.

*Corresponding author: reakhigbe@lautech.edu.ng

Cisplatin-induced cardiotoxicity is a major global health challenge among patients on platinum-based chemotherapy and it has limited the clinical use of this drug despite its effectiveness. It exerts its cardiotoxicity via the downregulation of Nrf2/HO-1 and glutathione system, while L-arginine, a semi-essential amino acid and an anti-oxidant, activates Nrf2/HO-1 and enhances glutathione antioxidants. However, data on the effect of L-arginine on cisplatin-induced cardiotoxicity is scarce. Therefore, this study examined the effect of L-arginine on cisplatin-induced cardiotoxicity. Also, the involvement of the Nrf2/HO-1 pathway and glutathione system was explored. Twenty-four male Wistar rats were randomly allotted into 4 groups after a 2-week acclimatization period; control, arginine-treated, cisplatin-treated, and cisplatin+arginine-treated. L-arginine blunted cisplatin-induced decrease in body and cardiac weight, and cardiac/tibial length ratio. Also, L-arginine attenuated the cisplatin-induced rise in cardiac lactate dehydrogenase and creatine kinase activities, as well as in lactate and troponin levels. Moreover, L-arginine attenuated the cisplatin-induced rise in circulating levels of glucose, total cholesterol, and triglycerides, as well as atherogenic indices and insulin resistance. Furthermore, L-arginine improved cisplatin-induced alteration in cardiac histoarchitecture. These findings were associated with L-arginine-led suppression of cisplatin-induced rise in cardiac MDA, IL-1β, TNF-α, and myeloperoxidase activity, and cisplatin-induced reductions in cardiac GPx, GST, SOD, catalase, and HO-1 activities, and Nrf2 expression. In conclusion, L-arginine attenuated cisplatin-induced cardiotoxicity and defective cardiac metabolic flexibility by activating Nrf2/HO-1 signaling and upregulating glutathione.

Keywords: Antioxidants; Amino acids; Arginine; Cardiotoxicity; Inflammation; Platinum-based chemotherapy

MICRONIZED PURIFIED FLAVONOID FRACTION IMPROVES CARDIAC STRUCTURAL AND FUNCTIONAL INTEGRITY BY MODULATING NLRP3/CASPASE-1/-3 SIGNALING IN CISPLATIN-TREATED MALE WISTAR RATS

Saka W.A.[1], Oladipo A.A.[1,2], Oyedokun P.A.[1,2], Adegbola C.A.[1,2], Akhigbe T.M.[2,3], Ashonibare P.J.[1,2], Kolawole O.R.[1,2], Akhigbe R.E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive

Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria.
*Correspondence: reakhigbe@gmail.com;
Although **c**isplatin is an effective chemotherapeutic agent, its use is restricted by its toxicity to non-target organs, such as cardiotoxicity that is mediated by NLRP3-driven oxido-inflammation. Conversely, micronized purified flavonoid fractions (MPFF) attenuate oxido-inflammation by downregulating NLRP3 inflammasome. However, there is a dearth of information on the effect of MPFF on cisplatin-induced cardiac injury. This study examined the possible protective effect of MPFF in cisplatin-induced cardiac injury, and the role of NLRP3 inflammasome and caspase-1/-3 signalling. Thirty-two adult male Wistar rats were randomly allotted to four equal groups (n=8 rats per group). The control received 0.5 mL of distilled water orally daily, the MPFF-treated rats received 100 mg/kg/day of MPFF orally for 14 days, the cisplatin-treated rats had 7 mg/kg of cisplatin via an intraperitoneal route on day 8, and the cisplatin+MPFF-treated rats received cisplatin and MPFF as those in the cisplatin- and MPFF-treated groups. Cisplatin significantly increased cardiac injury markers and plasma glucose levels and induced dyslipidemia and insulin resistance. Moreover, cisplatin altered cardiac histology, evidenced by vascular congestion, and increased myofibril thickness and interstitial space. These observations were accompanied by cisplatin-induced cardiac oxidative stress (increased malondialdehyde and a decline in reduced glutathione, superoxide dismutase, and catalase), inflammation (increased tumor necrosis factor-alpha, interleukin-1beta, and interleukin-6), apoptosis (increased caspase 1 and caspase 3), and an increase in NLPR3. These derangements were blunted by MPFF co-therapy. This study reveals that MPFF attenuated cisplatin-induced cardiac structural and functional damage via the downregulation of NLPR3 /caspase-1/-3 signaling.
Keywords: Antioxidant; Apoptosis; Chemotherapy; Flavonoid; Oxidative stress; Inflammation

COQ10 MITIGATES BPA-INDUCED CARDIOTOXICITY BY MAINTAINING CELLULAR ANTIOXIDANT LEVELS AND NA+/K+-ATPASE AND CA2+-ATPASE ACTIVITIES

Saka W.A.[1], Oladipo A.A.[1,2], Adegbola C.A.[1,2], Adelowo O.E.[1,2], Oyedokun P.A.[1,2], Kolawole O.R.[1,2], Owoeye M.T.[3], Akhigbe T.M.[2,4], Onaolapo A.Y.[3], Akhigbe R.E.[1,2*]
[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Department of Anatomy, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [4]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria.
*Corresponding author: reakhigbe@gmail.com;
The cardiotoxic effect of BPA involves multiple pathways and includes the depletion of cellular antioxidants and impairment of Na^+/K^+-ATPase and Ca^{2+}-ATPase activities. On the other hand, CoQ10 exerts cardioprotection through its antioxidant activities. However, little to no data have been reported on the cardioprotective effect of CoQ10 against BPA-induced cardiotoxicity. This study explored the impact of CoQ10 on BPA-induced cardiotoxicity and the involvement of cellular antioxidants, Na+/K+-ATPase, and Ca^{2+}-ATPase activities. Twenty-four adult male Wistar rats were randomized to four equal groups (n=6 rats/group). The control had 0.5 mL/day of corn oil, while the CoQ10-treated had 10 mg/kg/day of CoQ10, the BPA-treated had 50 mg/kg/day of BPA, and the BPA+CoQ-treated rats had 50 mg/kg/day of BPA and 10 mg/kg/day of CoQ10 for 4 weeks. BPA significantly increased cardiac injury markers (troponin, creatinine kinase, lactate, and lactate dehydrogenase) and altered cardiac histology as depicted by the presence of focal hemorrhage, inflammatory cell infiltration, reduced myofibril thickness, increased intercellular space, reduced nuclei width, and length, reduced total nuclei density, and increased pyknotic nuclei and amyloid accumulation. These findings were accompanied by oxidative stress (elevated malondialdehyde and reduced glutathione, catalase, and superoxide dismutase), inflammation (elevated myeloperoxidase activity), impaired cardiac Na^+/K^+-ATPase and Ca^{2+}-ATPase activities, and increased cardiac DNA fragmentation. These biochemical and histological perturbations induced by BPA exposure were attenuated by CoQ10 co-administration. In conclusion, the present study demonstrated that CoQ10 attenuated BPA-induced cardiotoxicity by maintaining cellular antioxidant levels and activities and improving Na^+/K^+-ATPase and Ca^{2+}-ATPase activities.
Key words: Antioxidant; Bisphenols; CoQ10; Environmental toxicants; Plasticizer; Oxidative stress

CARDIOPROTECTIVE EFFECT OF METFORMIN/DONEPEZIL COMBINATION INVOLVES ACETYLCHOLINESTERASE INHIBITION AND MODULATION OF BAX/BCL-2/CASPASE 3 PATHWAY IN HFD/STZ-INDUCED DIABETIC MALE WISTAR RATS

Saka W.A.[1], Kolawole O.R.[1,2], Olamoyegun O.M.[3], Ashonibare V.J.[2,4], Ashonibare P.J.[1,2], Oladipo A.A.[12], Adegbola C.A.[1,2], Adeyeni A.E.[1,2], Akinkunmi O.A.[1,2], Adebayo J.[1,2], Akhigbe T.M.[2,5], Akhigbe R.E.[1,2*]
[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Endocrinology Unit, Department of Medicine, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [4]Cardiovascular Regenerative Medicine & Tissue Engineering 3D Lab, Department of Cardiovascular Surgery and Research Group for Experimental Surgery, Heinrich Heine University, Medical Faculty, Düsseldorf, Germany. [5]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria.
*Corresponding author: reakhigbe@gmail.com;
Diabetes-associated cardiac damage is linked with cardiac acetylcholinesterase (AchE) increase and apoptotic injury. Although metformin is established in diabetes management and donepezil is a known acetylcholinesterase (AchE) antagonist with cardioprotective activity, the cardioprotective effect of combining metformin and donepezil in diabetes is yet to be reported. This study investigated the cardioprotective role of the metformin/donepezil in a Wistar rat model of high-fat diet/streptozotocin (HFD/STZ)-induced diabetes. Thirty-six

8-week-old male Wistar rats were acclimatized for a week and then randomly allotted into six equal groups (n=6 rats per group): control, diabetes (DM), DM+metformin, donepezil, DM+donepezil, and DM+metformin+donepezil groups. Diabetes was induced with HFD/STZ. In addition, DM+Metformin rats received 100 mg/kg of metformin *per os*, Donepezil received 10 mg/kg of donepezil *per os*, DM+Donepezil had 10 mg/kg of donepezil *per os*, and DM+Metformin+Donepezil had 100 mg/kg of metformin with 10 mg/kg of donepezil per os for six weeks. Metformin and donepezil independently improved fasting glucose, insulin, and insulin resistance, and lipid profile in diabetic rats. Also, metformin and donepezil reduced the damage caused by diabetes to the heart, including high levels of injury markers, amyloid buildup, and tissue damage. In addition, metformin and donepezil attenuated oxidative stress (MDA), inflammation (CRP, TNF-α, IL-1β, and IL-6), apoptosis (Bax/Bcl-2 and caspase-3), and AchE rise and antioxidant (GSH, SOD, and catalase) decline in the cardiac tissue of diabetic rats. Metformin/donepezil exerted a superior effect compared to either monotherapy. Metformin/donepezil combination exerts cardioprotective effects in diabetic rats through AchE inhibition and Bax/Bcl-2/caspase-3 modulation.

Keywords: Acetylcholinesterase; Amyloidosis; Apoptosis; Biguanide; Cardiotoxicity; Diabetes

SILYMARIN AMELIORATES POST-WEANING BISPHENOL A EXPOSURE-INDUCED CARDIOTOXICITY VIA SUPPRESSION OF TLR4/VCAM SIGNALING IN MALE WISTAR RATS

Saka W.A.[1], Adeyeni A.E[1,2], Adegbola C.A.[1,2], Ashonibare V.J.[2,3], Aderoju R.A.[1,2], Lawal A.T.[1,2], Kajola P.O.[1,2], Akhigbe T.M.[2,4], Ogunkola B.D.[2], Akintola A.O.[5], Akhigbe R.E[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Cardiovascular Regenerative Medicine & Tissue Engineering 3D Lab, Department of Cardiovascular Surgery and Research Group for Experimental Surgery, Heinrich Heine University, Medical Faculty, Düsseldorf, Germany. [4]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria. [5]Department of Science Laboratory Technology (Biochemistry Unit), Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

*Corresponding author: reakhigbe@gmail.com

Bisphenol A (BPA) is globally used in the manufacture of a variety of wares, including children's toys and feeding bottles. Despite its usefulness, it exerts cardiotoxicity via the induction of oxidative stress. However, there is a dearth of data on the impact of post-weaning BPA induction of cardiotoxicity. Also, the effect of silymarin, a known antioxidant, on the possible impact of BPA post-weaning exposure is not documented to date. This study assessed the likely cardiotoxic effect of post-weaning BPA exposure. In addition, the potential ameliorative outcome of silymarin on BPA-induced cardiotoxicity was evaluated. Twenty-four male Wistar rats were randomized into four groups the vehicle-treated control group, the silymarin-treated group, which received 100 mg/kg/day of silymarin, the bisphenol A (BPA)-treated rats, which received 50 mg/kg/day of BPA, and the BPA+silymarin-treated rats, which received both BPA and silymarin as those in the BPA and silymarin groups, respectively. Silymarin attenuated the BPA-induced rise in cardiac injury markers, malondialdehyde, and iron contents, and BPA-induced downregulation of reduced glutathione and glutathione peroxidase 4. More so, silymarin suppressed BPA-induced upregulation of TNF-α, IL-1β, MPO, VCAM-1, NF-kB, TLR4, caspase 3, cytochrome c, and DFI. These were accompanied by improvement of cardiac histology and suppression of amyloid accumulation in rats co-treated with silymarin and BPA. In conclusion, silymarin attenuated post-weaning BPA-induced cardiotoxicity by suppressing amyloidosis, ferroptosis, and apoptosis through a TLR4/VCAM-mediated mechanism.

Keywords: Amyloidosis; Bisphenol A; Ferroptosis; Oxidative stress; Plasticizers; Toxicants

DONEPEZIL ENHANCES TESTICULAR PROTECTION OF METFORMIN IN HFD/STZ-INDUCED DIABETIC WISTAR RAT VIA MODULATION OF XANTHINE OXIDASE/URIC ACID AND BAX/BCL-2/CASPASE-3 SIGNALING

Akhigbe R.E.[1,2], Oyesode S.O.[1,2], Odeyemi A.A.[1,2], Chimezie J.[2,3], Adesogan F.[1,2], Oladipo A.A.[1,2], Ashonibare P.J.[1,2], Akhigbe T.M.[2,4], Ashamu E.A.[5], Olamoyegun M.A.[6]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Department of Physiology, Federal University of Technology-Akure (FUTA), Akure, Ondo State, Nigeria. [4]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria. [5]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [6]Endocrinology Unit, Department of Medicine, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

*Corresponding author: reakhigbe@lautech.edu.ng

Xanthine oxidase (XO)/uric acid (UA)/caspase-3-driven oxidative stress and apoptosis have been implicated in type 2 diabetes mellitus (T2DM)-induced testicular damage. Although metformin is effective in managing diabetes, combined therapy is more effective. Though Donepezil has been reported to protect against neuronal and retinal damage in diabetic rats, its role in diabetes-induced testicular damage has not yet been reported. This study investigated the effect of metformin/donepezil combined treatment on testicular dysfunction in a high-fat diet/streptozotocin (HFD/STZ)-induced T2DM rat model. Forty-eight 8-week-old male Wistar rats were randomly assigned into six equal groups: control, diabetes (DM), DM+metformin, donepezil, DM+donepezil, and DM+metformin+donepezil groups. T2DM was shown to cause testicular inflammation, oxidative stress, and apoptosis, evidenced by elevated proinflammatory cytokines (IL-1β, IL-6, TNF-α), activation of NF-κB signaling, increased oxidative markers (MDA, XO, and UA), and apoptosis markers (Bax, caspase-3), alongside reduced antioxidant enzyme activities and Bcl-2. T2DM also caused reduced serum testosterone, FSH, and LH, germ cell loss, and Leydig cell degeneration. Treatment with metformin and donepezil, singly or in combination, attenuated these alterations, with the combined therapy demonstrating superior effect. These results imply that the

metformin/donepezil combination provides a potentially effective treatment strategy for attenuating testicular damage caused by diabetes through modulation of oxidative, inflammatory, and apoptotic pathways.

Key words: Acetylcholinesterase; Apoptosis; Biguanide; Oxidative stress, inflammation

OC-C-13

DONEPEZIL/METFORMIN COMBINATION ATTENUATES NEUROTOXICITY AND COGNITIVE DEFICIT BY DOWNREGULATING CYTOCHROME-C/CAPASE-3 SIGNALING IN HFD/STZ-INDUCED DIABETES IN MALE WISTAR RAT

Akhigbe R.E.[1,2*], Odeyemi A.A.[1,2], Adejare M.A.[1,2], Olakunle A.B. [1,2], Kamorudeen Z.A[1,2], Amodu O.M[1,2], Olaniran DI[1,2], Julius J.B.[1,2], Thomas C.O.[3], Akhigbe T.M.[2,4], Adeleye O.O.[5], Ogundipe O.O.[1], Olamoyegun M.A.[6]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria.

[3]Department of Anatomy, University of Ibadan, Ibadan, Oyo State, Nigeria.

[4]Department of Agronomy, Osun State University, Ejigbo campus, Ejigbo, Osun State, Nigeria.

[5]Department of Anatomy, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

[6]Endocrinology Unit, Department of Medicine, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

*Corresponding author: reakhigbe@lautech.edu.ng

Diabetes is associated with several complications, such as neurocognitive deficit, and it is associated with oxidative stress-mediated mechanisms. Although metformin is effective in managing diabetes, it does not effectively attenuate diabetic neuropathy. On the other hand, Donepezil mitigates neuronal damage, but its role in diabetes-induced neurocognitive deficit has not been fully explored. This study investigated the effect of metformin/donepezil combination on diabetes-induced neurocognitive deficit in a high-fat diet/streptozotocin Wistar rat model. Forty-eight 8-week-old male Wistar rats were randomly assigned into six equal groups: control, diabetes (DM), DM+metformin, donepezil, DM+donepezil, and DM+metformin+donepezil groups. Metformin and donepezil, when administered singly or in combination, improved diabetes-induced neurocognitive deficit (increased total and primary latencies in Barne's test, reduced novel arm entry, time spent in exploring novel arm, and alternation in Y maze, reduced time spent exploring novel and familial objects and discrimination index in novel object recognition test, and reduced rearing and time spent in the centre of the field in the open field test). Also, Metformin/Donepezil alleviated the diabetes-induced decline in acetylcholinesterase and BDNF, and the diabetes-induced neurodegeneration and amyloid accumulation in the hippocampus, cerebrum, and cerebellum. In addition, Metformin/Donepezil attenuated diabetes-induced elevations in MDA, TNF-α, IL-1β, MPO, cytochrome c, and caspase-3, and the diabetes-induced decline in GSH, SOD, catalase, and total antioxidant capacity in the selected brain regions. The combined therapy exerted a superior effect than the monotherapies. These findings reveal that the metformin/donepezil combination is a promising, effective treatment strategy for attenuating diabetes-induced neurotoxicity and cognitive deficit.

Keywords: Apoptosis; Donepezil; Inflammation; Metformin; Neurocognition; Oxidative stress

OC-C-14

BETA-GLUCAN AND MYCELIA OBTAINED FROM MAGNESIUM-SUPPLEMENTED *PLEUROTUS OSTREATUS* ATTENUATES NEURODEGENERATION AND COGNITIVE DEFICIT IN METHOTREXATE-TREATED MALE WISTAR RAT

Akhigbe R.E.[1,2*], Thomas C.O.[2,3], Lasisi-Sholola A.S.[1,2], Oloke J.K.[4]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria.

[3]Department of Anatomy, University of Ibadan, Ibadan, Oyo State, Nigeria. [4]Department of Pure and Applied Biology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

*Corresponding author: reakhigbe@gmail.com

Despite the effectiveness of methotrexate (MTX) in the management of several cancers, rheumatoid arthritis, and psoriasis, it induces chemobrain via oxidative stress-mediated mechanisms, and it is associated with Alzheimer's disease and Parkinson's disease. *Pleurotus ostreatus* (PO), an edible mushroom, possesses antioxidant activity. Nonetheless, the effect of PO on MTX-induced neurocognitive deficit is yet to be reported. This study investigated the neuroprotective effects of a novel beta-glucan and mycelia obtained from magnesium-supplemented PO against MTX-induced Alzheimer-like symptoms in a Wistar rat model. Forty male Wistar rats were randomly allotted into five groups (n=8 rats/group): control, MTX-treated, MTX+ 100 mg/kg of PO-treated, MTX+ 250 mg/kg of PO-treated, and MTX+ 500 mg/kg of PO-treated. After completing the treatment regimen, novel object recognition, Barnes, Y-maze, and open-field tests were performed to evaluate cognition and anxiety. Subsequently, the cerebrum, hippocampus, and cerebellum were excised for biochemical assays and histopathological examinations. PO therapy attenuated cognitive deficit and anxiety in MTX-treated rats. Additionally, PO ameliorated the rise in MDA and enhanced the levels of GSH, SOD, and catalase, as well as dopamine, acetylcholinesterase, and BDNF in MTX-treated rats. More so, PO treatment suppressed MTX-induced rise in NF-kB, IL-1β, and TNF-α, and histological alterations in the cerebrum, hippocampus, and cerebellum. The effect of PO was not dose-dependent. This study demonstrates the protective effect of PO against MTX-induced chemobrain and Alzheimer's- and Parkinson-like symptoms, thus providing experimental evidence for a neuroprotective role for PO in neurodegeneration and cognitive impairment.

Keywords: Alzheimer's disease; Mushroom; Neurocognition; Oxidative stress, Parkinson's disease; *Pleurotus ostreatus*

OC-C-15

OESTROGEN REPLACEMENT THERAPY REVERSES MEMORY LOSS AND PYRAMIDAL CELL NEURODEGENERATION IN THE

HIPPOCAMPUS AND CORTEX OF LEAD-EXPOSED OVARIECTOMIZED WISTAR RATS

Lasisi-Sholola A.S.[1,2], Hammed S.O.[1], Ajike R.A.[1], Akhigbe R.E.[1,2*], Afolabi O.A.[1*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria.

*Corresponding author: reakhigbe@gmail.com;

Although menopause is a component of chronological aging, it may be induced by exposure to heavy metals like lead. Just like the postmenopausal state, lead exposure is associated with neurocognitive deficit; however, the impact of hormone replacement therapy (HRT) on menopause and lead-induced neurocognitive deficit is yet to be reported. This study explored the effect and associated mechanism of HRT on ovariectomized-led menopausal state and lead exposure-induced neurocognitive deficit. Thirty adult female Wistar rats were randomized into 6 groups (n = 5 rats/group): the sham-operated vehicle-treated, ovariectomized (OVX), OVX + HRT, lead-exposed, OVX+lead, and OVX+lead+HRT groups. Treatment was daily via gavage and lasted for 28 days. Ovariectomy and lead exposure impaired spatial memory, evidenced by a significant reduction in novel arm entry, time spent in the novel arm, alternation, time exploring novel and familiar objects, and discrimination index. These findings were accompanied by a marked distortion in the hippocampal and cortical histology, and a decline in pyramidal neurons and dopamine levels. In addition, ovariectomy and lead exposure elevated lactate, lactate dehydrogenase, and creatinine kinase activities, and induced oxidative stress (increased MDA and reduced GSH, SOD, and catalase), inflammation (elevated myeloperoxidase activity, and TNF-α and IL-1β), and apoptosis (increased caspase 3) in the hippocampus and cortex. The observed biochemical and histological perturbations were attenuated by HRT. This study revealed that HRT attenuated ovariectomy and lead-exposure-induced memory deficit and pyramidal neurodegeneration by suppressing oxidative stress, inflammation, and apoptosis of the hippocampus and cortex.

Keywords: Cognition; Heavy metals; Hormone replacement therapy; Lead; Memory; Oestrogen

CURCUMIN AMELIORATES DICHLORVOS-INDUCED CARDIAC INJURY BY SUPPRESSING OXIDATIVE STRESS AND DOWNREGULATING PRO-INFLAMMATORY CYTOKINES IN MALE WISTAR RATS

Saka W.A.[1], Kolawole O.R.[1,2], Oladipo A.A.[1,2], Oladepo S.[1], Igbayilola Y.D.[2,3], Akhigbe R. E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Department of Human Physiology, College of Medicine and Health Sciences, Baze University, Abuja, Nigeria.

*Corresponding author: reakhigbe@gmail.com

Dichlorvos (DDVP) induces toxicity in non-target cells via the initiation of inflammation and oxidative stress. Meanwhile, curcumin exerts antioxidant and anti-inflammatory activities. Nevertheless, there is an inadequate understanding of the beneficial potential of curcumin in dichlorvos-induced cardiotoxicity. This study investigated the cardio-protective effects of curcumin in dichlorvos-induced cardiotoxicity. Forty male Wistar rats were randomly allotted into four groups: the control (1 mL of olive oil), curcumin-treated (100 mg/kg), DDVP-treated (98.54 g/m 3 α of dichlorvos by inhalation), and DDVP + Curcumin-treated. It was observed that dichlorvos exposure led to cardiac histopathological damage such as focal vascular congestion, widened interstitial space between the cardiac myofibrils, and reduced thickness of the myofibrils. The results of the cardiac function test revealed a decrease in lactate dehydrogenase and a rise in creatinine kinase and troponin-I. These alterations were associated with significantly elevated systemic blood pressure, and reduced electrocardiographic P wave, PR interval, and baroreceptor sensitivity. Also, DDVP elevated levels of plasma cholesterol, triglycerides, and glycated hemoglobin, cardiac malondialdehyde (MDA), TNF, IL-1β, and C-reactive protein (CRP), and reduced superoxide dismutase activity. Meanwhile, curcumin co-treatment in dichlorvos-exposed rats restored cardiac histoarchitecture and electrophysiology and lipid profile. Curcumin co-treatment also attenuated DDVP-induced rise in cardiac MDA, TNF α, IL-1β, and CRP, and DDVP-induced decline in cardiac superoxide dismutase activity. Summarily, this study confirmed the cardiotoxicity of dichlorvos and further demonstrated the protective effect of curcumin against dichlorvos-induced cardiotoxicity by suppressing pro-inflammatory cytokines and oxidative stress.

Keywords: Oxidative stress, Curcumin, Dichlorvos, Male infertility, Organophosphate, Sexual dysfunction

DAFLON ATTENUATES SLEEP DEPRIVATION-INDUCED IMPAIRED REPRODUCTIVE FUNCTION IN MALE WISTAR RATS BY TARGETING OXIDATIVE STRESS AND INFLAMMATION

Oladapo D.D. [1,2], Ashonibare P.J.[1,2], Akhigbe T.M.[2,3], Adegbola C.A.[1,2], Thomas C.O.[2,4], Oladipo A.A.[1,2], Kolawole O.R.[1,2], Akhigbe R.E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria. [3]Department of Agronomy, Osun State University, Ejigbo, Osun State, Nigeria. [4]Department of Anatomy, Ahmadu Bello University, Zaria, Kano State.

*Corresponding author: reakhigbe@lautech.edu.ng

Sleep deprivation (SD) poses a global challenge to human health, including male reproductive health. It impairs male reproductive function via the induction of oxidative-inflammatory injury. On the other hand, daflon, containing diosmin and hesperidin, has been reported to confer cellular protection by suppressing inflammation and upregulating the expression and activities of antioxidants. However, whether or not daflon will attenuate SD-induced reproductive dysfunction is yet to be explored. This study investigated the effect of daflon on SD-induced male reproductive impairment. Thirty-two adult male Wistar rats were randomly assigned to four groups (n = 8): the vehicle-treated control (0.5 mL of water), daflon-treated (100 mg/kg), SD, and SD + daflon. SD was induced by exposing the animals to continuous SD for 96 h using the modified multiple-platform technique, followed by a 72 h recovery period for 8 cycles (56 days). Daflon improved SD-induced prolonged mount, intromission, and ejaculation latencies. Also, daflon

attenuated SD-induced reductions in mount, intromission, and ejaculation frequencies as well as daily and total spermatid production, sperm concentration, motility, viability, normal sperm morphology, and serum and testicular testosterone levels. Moreover, daflon alleviated the SD-led rise in testicular and epididymal xanthine oxidase, uric acid, MDA, TNF-α, IL-1β, TLR4, and NF-κB, and the SD-induced decline in GSH, SOD, and catalase. Furthermore, daflon improved SD-induced alterations in testicular and epididymal histoarchitecture. These findings suggest that daflon attenuated SD-induced impairment of reproductive function in male Wistar rats by targeting oxidative stress and inflammation.

Keywords: Antioxidant; Epididymides; Spermatozoa; Testis; Oxidative stress; Sleep deprivation

A SYSTEMATIC REVIEW AND META-ANALYSIS OF THE IMPACT OF TRICLOSAN EXPOSURE ON HUMAN SEMEN QUALITY

Adegbola C.A.[1,2], Akhigbe T.M.[2,3], Adeogun A.E.[1,2], Tvrdá E.[4], Pizent A.[5], Akhigbe R.E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Nigeria. [3]Breeding and Genetic Unit, Department of Agronomy, Osun State University, Osun State, Nigeria. [4]The Institute of Biotechnology, Faculty of Biotechnology and Food Sciences, Slovak University of Agriculture in Nitra, Trieda A. Hlinku 2, 949 76 Nitra, Slovakia. [5]Institute for Medical Research and Occupational Health, Ksaverska cesta 2, HR-10000 Zagreb, Croatia.

*Corresponding email: reakhigbe@lautech.edu.ng; reakhigbe@gmail.com

Triclosan has been reported to pose a threat to male reproductive health. It has been reported that Triclosan exposure caused a decline in semen quality and circulating levels of testosterone. Nevertheless, available data in the literature is scanty and inconsistent. The present study is a systematic review and meta-analysis of the impacts of Triclosan exposure on semen quality. This study was registered with PROSPERO (CRD42024524192) and was conducted according to PRISMA guidelines. Following a pre-defined strategic protocol, 166 studies were screened, and 5 articles were identified as eligible for this study. Included studies were from Belgium (1), China (3), and Poland (1). Two of these were case-control studies, while the others were cross-sectional studies. A total of 1312 male subjects were included in the meta-analysis. Triclosan exposure significantly reduced sperm concentration and sperm total motility. In addition, triclosan exposure marginally reduced sperm progressive motility. However, triclosan exposure did not significantly alter sperm count and sperm morphology. The present study provides the first comprehensive meta-analysis on the impact of Triclosan exposure on human semen quality. Data presented in this study demonstrate that triclosan exposure may impair male fertility by reducing sperm concentration and sperm motility. Our report augments existing data in the literature on the negative impact of triclosan exposure on male reproductive health.

Keywords: Endocrine disruptor; environmental toxicant; male infertility; oxidative stress; semen; sex hormones

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CURCUMIN ATTENUATES DICHLORVOS-INDUCED MALE REPRODUCTIVE TOXICITY BY UPREGULATING TESTOSTERONE, AND AMELIORATING INFLAMMATION, SPERM QUALITY, AND OXIDATIVE STRESS IN MALE WISTAR RATS

Saka W. A.[1], Oladipo A. A.[1,2], Kolawole O. R.[1,2], Olayioye A.[3], Oladepo S.[1], Akhigbe R. E.[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Osun State, Nigeria.

[3]Department of Crop and Environmental Protection, Faculty of Agricultural Sciences, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.

*Corresponding author: reakhigbe@gmail.com

Dichlorvos (DDVP) is an organophosphate pesticide that is commonly used for agricultural and domestic control of pests and insects. Despite its usefulness, it exerts reproductive toxicity and induces male sexual dysfunction. On the other hand, curcumin has been reported to attenuate reproductive toxicity by upregulating testosterone and ameliorating inflammation, sperm quality, and oxidative stress. However, to date, no study has reported the impact of curcumin on dichlorvos-induced male reproductive toxicity. This study investigated the effect and associated mechanism of curcumin on dichlorvos-induced male reproductive toxicity. Thirty-two male Wistar rats were randomized into four groups: the control (1 mL of olive oil), curcumin-treated (100 mg/kg), DDVP-treated (98.54 g/m3 of dichlorvos by inhalation), and DDVP + Curcumin-treated. Dichlorvos induced sexual incompetence as depicted by reduced motivation to mate, prolonged latencies, and reduced frequencies of mount, intromission, and ejaculation. Dichlorvos also significantly lowered sperm parameters (sperm concentration, motility, viability, and normal morphology) and male reproductive hormones (follicle-stimulating hormone, luteinizing hormone, and testosterone). Moreover, dichlorvos altered testicular histology, evidenced by reduced Leydig cells and scanty sperm cells in the seminiferous tubules. These observations were accompanied by dichlorvos-induced testicular oxidative stress (increased malondialdehyde and decreased reduced glutathione, superoxide dismutase, and catalase), inflammation (increased tumor necrosis factor-alpha, interleukin-1beta, and nitric oxide). These derangements were attenuated by curcumin co-therapy. This study reveals that curcumin administration improves male reproductive health by reducing inflammation and oxidative stress in DDVP-exposed male Wistar rats.

Keywords: Oxidative stress, Curcumin, Dichlorvos, Male infertility, Organophosphate, Sexual dysfunction

A SYSTEMATIC REVIEW EXPLORING THE ROLE OF KISSPEPTIN IN PROSTATE CANCER

Oyedokun P.A.[1,2], Oyedokun M.A.[2,3], Adegbola C.A.[1,2], Oladipo A.A.[1,2], Oladele T.A.[2,4], Oyeleke B.T.[2,5], Osikoya O.O.[2,6], Oyeniyi G.A.[7], Akhigbe TM[2,8], Akhigbe RE[1,2*]

[1]Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. [2]Reproductive Biology and Toxicology Research Laboratory, Oasis of Grace Hospital, Osogbo, Nigeria. [3]Department of Nursing, Ladoke Akintola University of Technology, Ogbomoso,

Oyo State, Nigeria. [4]Department of Pure and Applied Chemistry, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[5]Department of Medical Laboratory Science, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[6]Department of Medicine and Surgery, Babcock University, Ilishan Remo, Ogun State, Nigeria.
[7]Department of Surgery, Osun State University Teaching Hospital, Osogbo, Osun State, Nigeria.
[8]Breeding and Genetic Unit, Department of Agronomy, Osun State University, Osun State, Nigeria.
*Corresponding author: reakhigbe@gmail.com

Prostate cancer (CAP) is the second most commonly diagnosed cancer and the leading cause of cancer-related death in men globally. The growth and progression are dependent on androgens, which are regulated by the hypothalamic-pituitary-testicular (HPT) axis. Kisspeptin (Kiss1), the endogenous ligand for the G protein-coupled KISS1 receptor (KISS1R; also known as GPR54), is crucial in regulating the HPT axis and has emerged as a significant player in prostate cancer. Nonetheless, available data in the literature is scanty. The present study is a systematic review of human and animal studies evaluating the role of Kiss1 in CAP growth. This study was conducted in accordance with PRISMA guidelines. After a pre-defined strategic protocol, 122 studies were screened, and 8 articles were identified as eligible for this study. The studies were observational (1), *in vitro* using human cell lines (2), randomized placebo-controlled (1), and animal studies (4). Kiss1 expression was downregulated in CAP cells. In addition, administration of Kiss1 analogue upregulated Kiss1 and KISS1R expression. Moreso, Kiss1 administration suppressed GnRH, LH, FSH, testosterone, and *Fsh* and *Lh* mRNA, demonstrating the suppressor effect of Kiss1 on the HPT axis. Also, Kiss1 administration reduced prostate tumor volume and prostate-specific antigen. Furthermore, Kiss1 administration downregulated *VEGF* mRNA and the migration and invasion of endothelial cells. This study reveals the role of Kiss1/Kiss1R signaling in incident CAP and its progression. Kiss1 suppresses prostate tumors by downregulating the HPT axis and *VEGF* mRNA.

Keywords: KISS1R; kisspeptin; malignancy; prostate cancer; testosterone; sex hormones