Dear Friends and Colleagues,

I'm pleased to welcome you to the 3rd conference of " ***Egyptian Society of Physiological Sciences".***

It's a good opportunity to share our thoughts regarding our future research which I hope it would be a valuable addition to the scientific field and a good presentation to all the new breakthroughs in our different departments.

We have to guide all our researched to the right track to pursuit the modern topics and techniques.

A gratitude to ***Physiology Department*** *in* ***Tanta University*** *for hosting this conference and everyone participated in organizing this conference.*

I would like to thank also all my colleagues who helped in re-formatting and developing the journal of the association to reach the standard we are looking for and willing to be.

Hope you will spend some quality time in this conference with our scientific researches through a friendly and familiar spirit.

Best Regards.

President of "ESPS"

Prof. Sobhy Elkafafy

Physiology Department

Faculty of Medicine

Alexandria University

Welcome Message

It is our honor in ***physiology department, Tanta University*** *to host you in our faculty for the 3rd annual conference of* ***Egyptian Society of Physiological Sciences*** *(ESPS) titled*

Physiology: A framework of integrated research

What a privilege to have such a prestigious attendance of physiology professors and staff from all over Egypt, it is always great for this unique family of hard workers , dedicated and truth seekers to meet and share ideas about "the science of life ".

With the activity of participants, we will make sure that this conference will be a memorable and highly educational event. We truly value your participation and support for this conference.

Thank You.

President of the Conference

Prof. Sahar Ahmed El Sawy

Head of Physiology Department

Tanta Faculty of Medicine

Main Topics

- **Aging and pancreas**
- **Metabolic sensing of glucose and fructose**
- **Molecular biology of diabetic complications**
- **Olive Oil in renal Ischemia / Reperfusion**
- **Umbilical cord blood CD34+ stem cell in Parkinsonism in mice**
- **Corticosterone & Growth Hormone on Hippocampal Neurons.**
- **Darbepoetin-alpha& Mesenchymal stem cells on chronic Nephropathy.**
- **Stress & brain oscillations during memory task in males.**
- **Tissue pathology & hsCRP,IL-6,TNF-a in diabetic treated with aspirin.**
- **The Effect of resveratrol in fructose- fed rats.**
- **The role of endocannabinoid system in the obesity .**
- **phospholipase A2 in patients with COPD and obstructed sleep apnea.**
- **Systemic ghrelin on Myocardial Infarction Induced by Isoproterenol**

Venue: Main Conference Hall, Second floor, Faculty of medicine, Tanta University

Conference Coordinator

Dr. Ahmed Abdalfattah

Dr. Ahmed Alm El-Din

Conference President

Prof. Sahar Elsawy

President of ESPS

Prof. Sobhy Kafafy

اللجنه المنظمة:

الرئيس الشرفى للجمعية	ا.د :إبتسام الباجوري	أستاذ الفسيولوجى بطب القاهرة
رئيس الجمعية	ا.د: صبحى الكفافى	أستاذ الفسيولوجى بطب الأسكندرية
رئيس المؤتمر	ا.د: سحر الصاوي	أستاذ و رئيس قسم الفسيولوجى بطب طنطا
مقرر المؤتمر	د: أحمد عبد الفتاح د: أحمد علم الدين	مدرس الفسيولوجى بطب طنطا مدرس مساعد الفسيولوجى بطب طنطا

Scientific Committee:

Prof Sobhy El Kafafy	Alexandria University
Prof Haiat El Sayad	Alexandria University
Prof Kawkab Elsabah Ragab	Alexandria University
Prof Hanaa El serougy	Mansoura University
Prof Soheir Mohamed helmi	Mansoura University
Prof Fatma Aly Lebda	Ain shams University
Prof Essam Ahmed El Shamy	Alexandria University
Prof Maha Mohamed Gamal Eldin	Cairo university
Prof Hemmat Aly Khloussy	October 6 University
Prof Alaa El Din Hassan	Alexandria University
Prof samir Eskaros	Alexandria University
Prof Maged Haroun	Cairo university
Prof Maha Hegasy	Alexandria University
Prof Bataa el Kafoury	Ain shams University

Scientific Program:

Thursday	20 /3 /2014
8.30 - 9.30 AM	Registration
9.30 - 10.00 AM	Opening Ceremony
10.00 – 11.00 AM	Plenary session 1
Chair persons:	
Prof. Mohamed Madi	**Prof. Sobhy Kafafy**
Prof. Mohamed Zamzam	
Speaker:	
Prof. Fouad Kandeel	Aging and pancreas.
11.00 am -12.0 0 pm	**Oral session 1**
Chair persons:	
Prof. Hayat El sayad	**Prof. Hanna Abdalmonem**
Prof. Soher Helmy	
Speakers	
Prof. Ansam Aly (Ain Shams) 11:00-11:15	**Olive oil surpasses Ginger in attenuating renal I/R injury**
Dr. Noha Abo-Grisha.(Suez Canal) 11:15-11:30	**Effects of intravenous human umbilical cord blood CD34+ stem cell therapy versus levodopa in experimentally induced Parkinsonism in mice**
Prof. Ghada Mahmoud (Assiut) 11:30-11:45	**Effect of co-application of Glucocorticoid and growth hormone on hippocampal neurons**

Prof. Abdalazez Hussen (Mansura) 11:45-12:00	**Effect of Darbepoeitin-alpha and mesenchymal stem cells and (BDNF) in a rat model of vascular dementia.**
12.00 -12.30 pm	**Coffee Break**
12.30 pm – 1. 45 pm	**Plenary session 2**

Chair persons:

Prof. Yasser El-Wazir	**Prof Kowkab ragab**
Prof. Faten Diab	**Prof. Soad Abdallah Seleem**

Speakers:

Prof. Andrew Thomas	**A tale of two sugars: metabolic sensing of glucose and fructose.**
Prof. Maessa Alnahas	**Molecular Biology of diabetic complications**
1.45 pm-2.30 pm	**LUNCH & POSTER SESSION**
2.30-3.30 pm	**Annual Meeting of ESPS**
3.30- 4.15 pm	**Oral session 2**

Chair persons:

Prof Hanaa El serougi	**Prof Aly Khalil**
Prof. Soher Saleh	**Prof Mohamed Hussein**

Speakers:

Prof. Amany Elbaz (Suez Canal) 3.30-3.45 pm	**The effect of examination stress on brain oscillations during memory task in males**
Prof. Yahya Naguib	**Correlation between tissue pathology and vitreal levels of hsCRP, IL-6 and TNF-a in**

Menoufia 3.45-4.00 pm	**diabetic rats treated with aspirin**
Dr. Ahmed Abdalfattah (Tanta) 4.00-4.15 pm	**The effect of resveratrol on insulin resistance, metabolic syndrome and hepatic oxidative stress in Fructose Fed Rats.**

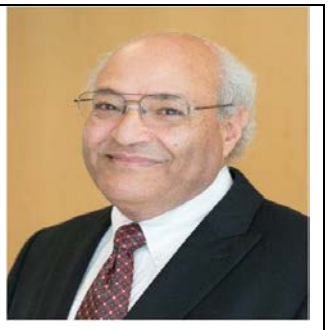
Poster Presentation

Prof. Sheren Bedeer (Zagazig)	**The role of endocannabinoid system in the obesity induced atherogenesis.**
Prof. Suzan Hazzaa (Menoufiya)	**Lipoprotein associated Phospholipase A2 levels as a predictor of Cardiovascular risks in patients with COPD and obstructive sleep apnea.**
Dr. Abeer Abo-zeid (Tanta)	**Effect of systemic grelin on experimental myocardial infarction**

Closing Ceremony

Aging of the pancreas

Prof. Fouad Kandeel, MD, PhD
Chair and Professor, Department of Clinical Diabetes, Endocrinology & Metabolism, City of Hope, Duarte, CA, USA.



Aging is an important risk factor for metabolic disorders, including obesity, impaired glucose tolerance, and type 2 diabetes (T2D). It has been reported that the prevalence of T2D increases with age and peaks at 60–74 [1]. Almost one-third of the elderly have diabetes, and three-quarters have diabetes or prediabetes [2]. The higher incidence of diabetes is especially alarming, considering that diabetes in itself increases the risk for multiple other age-related diseases such as cardiovascular disease (CVD), atherosclerosis, stroke, Alzheimer disease (AD), Parkinson's disease, nonalcoholic fatty liver disease (NAFLD), and cancer [3]. In certain ethnic groups, the proportion is even higher: almost 1 in 3 older Hispanics and African Americans and 3 out of 4 Pima Indian elders have diabetes. Insulin, secreted from pancreatic beta cells, is the major hormone in regulating glucose homeostasis. Insulin secretion is a complex process involving the integration of multiple stimuli, such as nutrients, hormones, neurotransmitters, and drugs, but the primary stimulus for insulin secretion is circulating glucose. Aging is associated with a marked decline in glucose-stimulated insulin secretion (GSIS) in both humans and rodents, and the impairment of GSIS is one of the hallmarks of T2D. In rodents, a decrease in GSIS in vivo has been shown using the state-of-the-art hyperglycemic clamps [4]. In humans, disorderly insulin release, a decrease in insulin pulse amplitudes, and decreased response to glucose oscillations as well as alterations in insulin clearance have all been demonstrated [5]. When insulin secretory defects are superimposed over an increased need for insulin, as in old age, impaired glucose homeostasis, glucose intolerance, and diabetes can result. Many factors contribute to the decrease in insulin secretion in aging, including the age-associated loss of Sirt1-mediated GSIS [6], decreased beta-cell sensitivity to circulating incretins [5], age-associated decrease in mitochondrial function, and increased oxidative stress [7]. Aging of the pancreas is also associated with impaired islet cell turnover through changes in the rate of programmed beta cell death, and decreased beta cell proliferation. In addition to changes in insulin secretion, senescence also affects other islet functions and plasticity. Significant evidence suggests that

islets from individuals under the age of 20 years are healthier and proliferate more readily, versus those from individuals that are older (age 30 years and up), which are more susceptible to apoptosis and less likely to expand. Thus, islets from younger donors are greatly preferred for transplantation. New data are emerging that suggest the most successful islet transplantation outcomes may require age-matching of donors and recipients. This presentation will specifically address the various changes observed in the aging pancreas and how they affect islet physiology and function in the setting of diabetes pathophysiology in general, as well as in relation to islet transplantation for type 1 diabetes (T1D).

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A tale of two sugars: metabolic sensing of glucose and fructose.

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It is widely recognized that obesity is a major health problem, which has already reached epidemic levels in many western countries and is increasing at an alarming rate worldwide. Obesity is associated with metabolic syndrome, which is also characterized by other metabolic risk factors including high plasma triglycerides and LDL-cholesterol, elevated fasting blood glucose, insulin resistance and hypertension. Metabolic syndrome increases the risk for diabetes, coronary artery disease and stroke. For most individuals, the core cause of obesity is an imbalance between energy expenditure and food consumption, with the latter being exacerbated by the wide availability of inexpensive food items with high calorific content and often limited nutritional value. While fat consumption had been a focus in the past, the role of excessive consumption of carbohydrate has recently come to the fore as an equal, if not more insidious culprit. And although some studies have indicated that the key parameter is the excessive intake of calories *per se*, there is also evidence to suggest that fructose may be particularly deleterious as compared to other sugars. In recent years, consumption of free fructose has increased dramatically as a result of the growing use of high fructose corn syrup, particularly in soft drinks and processed foods. There are data correlating the growing use of fructose with the surging obesity rates.

In the liver the pathways, regulation and metabolic fates of glucose and fructose are distinct. One result of these differences is that fructose is much more lipogenic than glucose, and has been shown to cause fatty liver. Moreover, we have found that fructose can cause the generation of oxidative stress in animal models. Thus, through this dual hit mechanism fructose may lead to Nonalcoholic Steatohepatitis (NASH). Our studies with genetically-encoded targeted probes to measure reactive oxygen

species (ROS) have indicated that the primary site of ROS generation is the mitochondria in liver. The mechanism and consequences of these multiple effects of fructose on the liver will be discussed.
We have also examined the effect of fructose on hypothalamic nutrient sensing. These studies have revealed an unexpected action of elevated fructose levels to elicit signals normally generated in response to hypoglycemic levels of brain glucose. This aberrant central nervous system (CNS) nutrient sensing can lead to elevated peripheral blood glucose due to the inappropriate engagement of pancreatic glucagon release, and may also lead to dysregulation of CNS pathways responsible for controlling food intake.
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Molecular Cell Biology of Diabetic Complications

Prof. Dr. Maessa Alnahas

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Oxidative stress plays a pivotal role in the development of diabetes complications, both microvascular and cardiovascular.

The metabolic abnormalities of diabetes cause mitochondrial superoxide overproduction in endothelial cells of both large and small vessels, as well as in the myocardium.

This increased superoxide production causes the activation of 5 major pathways involved in the pathogenesis of complications: polyol pathway flux, increased formation of AGEs (advanced glycation end products), increased expression of the receptor for AGEs and its activating ligands, activation of protein kinase C isoforms, and overactivity of the hexosamine pathway.

It also directly inactivates 2 critical anti atherosclerotic enzymes, endothelial nitric oxide synthase and prostacyclin synthase. Through these pathways, increased intracellular reactive oxygen species (ROS) cause defective angiogenesis in response to ischemia, activate a number of pro inflammatory pathways, and cause long-lasting epigenetic changes that drive persistent expression of pro inflammatory genes after glycemia is normalized (“hyperglycemic memory”).

Atherosclerosis and cardiomyopathy in type 2 diabetes are caused in part by pathway-selective insulin resistance, which increases mitochondrial ROS production from free fatty acids and by inactivation of anti-atherosclerosis enzymes by ROS.

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Olive Oil Surpasses Ginger In Attenuating Renal Ischemic Reperfusion Injury

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Renal ischemia-reperfusion injury (IRI) is a common cause of acute kidney injury (AKI), occurring with hypotension, cardiovascular surgery and inevitably during kidney transplantation. Morbidity and mortality from AKI are high, and results from current available clinical therapeutic approaches are disappointing. Olive oil has been shown to have high antioxidant and anti-inflammatory properties. Ginger has been shown to ameliorate renal IRI. This study aimed to investigate the ability and the possible mechanisms of olive oil to protect kidneys against IR injury in comparison with ginger. Adult female Wistar rats were randomly divided into control, sham operated (sham), ischemic/reperfused non treated (I/R), ischemic-reperfused ginger treated (ginger) (200 mg/kg orally daily for 4 weeks) and ischemic-reperfused olive oil treated (olive oil) (1 ml/100 g B.W. orally daily for 4 weeks) groups. Rats in I/R, ginger and olive oil groups were subjected to renal ischemia for 60 min, followed by reperfusion. Markers of renal damage, oxidative stress and inflammation were evaluated. Renal morphology was examined. Olive oil supplemented rats had lower serum urea and creatinine levels, with lower malondialdehyde, nitric oxide, c-reactive protein, interleukin 1beta, tumor necrosis factor alpha, nuclear factor kappa B, and higher glutathione, superoxide dismutase, and interleukin 10 levels in renal homogenates compared to I/R and ginger supplemented rats. Histological examination showed marked improvement of renal tissue in the olive oil group compared to both I/R and ginger treated groups. In conclusion, olive oil has shown particular promise against experimental renal IRI, and provided superior protection compared to ginger mostly due to its higher antioxidative and anti-inflammatory properties.

Key words: ischemia- reperfusion, kidney, ginger, olive oil

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Effects of intravenous human umbilical cord blood CD34+ stem cell therapy versus levodopa in experimentally-induced Parkinsonism in mice

Noha Abo-Grisha, Soha Essawy, Dina M. Abo-Elmatty, Zenab Abdel-Hady

Introduction: Parkinson's disease (PD) is a progressive neurodegenerative disease with impaired motor function. The current research was directed to investigate the effect of CD34+ stem cells versus levodopa in 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-induced Parkinsonism in mice.

Material and Methods: Female mice were divided into four groups. Group I: saline-injected group, group II: MPTP group, received four injections of MPTP (20 mg/kg, i.p.) at 2 h intervals, groups III, IV received MPTP and treated with levodopa/carbidopa (100/10 mg/kg/twice/ day for 28 days) or single intravenous injection of 10^6 CD34+ stem cells/mouse at day 7 and allowed to survive until the end of week 5.

Results: Levodopa and stem cell therapy improved motor deficits induced by MPTP; they abolished the difference in the stride length, decreased the percentage of foot slip errors and increased the ambulation, the activity factor and the mobility duration in parkinsonian mice. Further, they significantly increased striatal dopamine and ATP levels compared to MPTP group. Moreover, mitochondrial DNA from mice treated with levodopa or stem cells was in intact form and no appreciable fragmentation of nuclear DNA was found compared to MPTP group. Regarding tyrosine hydroxylase (TH) immunostaining, stem cell group showed a marked increase of TH-immunopositive neurons compared to both MPTP and levodopa groups

Conclusions: CD34+ stem cells ameliorated the motor, biochemical and histological deficits in MPTP-parkinsonian mice, these effects were superior to those produced by levodopa. CD34+ stem cells would be promising for the treatment of PD.

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Effects of Co-Application of Glucocorticoid and Growth Hormone on hippocampal neurons involve NMDA receptor upregulation ofNR2Bprotein expression and increasing NR2B/NR2A ratio

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Objectives In this study we investigated the role of growth hormone (GH) in protecting hippocampal function during episodes of acute stress.
Methods We tested the effects of co-application of GH and corticosterone (CORT) at different doses on field excitatory postsynaptic potential (fEPSPs) of hippocampal slices of rats at two different age groups. We examined the change of protein expression of N-mehyl-D-asparate receptor (NMDARs) subunits; NR1, NR2B, and NR2A in hippocampal brain slices treated with artificial cerebrospinal fluid (ACSF) or low dose of CORT alone or both CORT and GH for three hours.
Results We found an additive effect of co-application of CORT and GH on hippocampal synaptic transmission compared to either drug alone at all doses. Furthermore, we found that the combined use of low dose of GH and CORT have significantly higher effects on enhancement of fEPSPs in old rats compared to young ones. We showed that both GH and CORT enhanced protein expression NR2A subunit of NMDARs. Meanwhile, we demonstrated that the coexposure to low doses of GH and CORT significantly enhanced NR2B expression and increased the NR2B/NR2A while perfusion with CORT alone caused significant suppression in NR1 and NR2B protein expression and decrease in NR2B/NR2A.
Conclusion we suggest that NMDARs provide potential target for mediating GH potential protective effect against stress and age related memory and cognitive impairment

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Effects of Darbepoetin-alpha and Mesenchymal Stem Cells on Adriamycin-induced Chronic Nephropathy

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Objectives: To study and to compare the effects of bone marrow derived mesenchymal stem cells (BM-MSCs) and Darbepoetin-alpha (DPO-α) on adriamycin (ADR)-induced chronic nephropathy in rats.

Methods: 80 male Sprague Dawley rats divided into 4 groups (20 rats each); negative control group: normal rats received saline as a vehicle, positive control (ADR) group: rats received 2 iv injection of ADR *via* penile vein at 14 days interval without treatment; DPO group: as ADR group but rats received sc DPO once weekly for 12 weeks, and MSC group; as ADR group but rats received 2 iv injection of MSCs (5 days after each ADR injection). By the end of experiment hemoglobin (Hb) content, serum creatinine, BUN, albumin, triglycerides and cholesterol, urinary protein excretion and kidney injury molecule-1 (KIM-1) and GSH and catalase in kidney tissues. Also, immunohistochemical examination for caspase-3 and routine histopathological examination of kidney tissues were done.

Results: compared to ADR group, DPO group showed significant improvement in animal survival rate and body weight, Hb, serum BUN, triglycerides, cholesterol, and albumin and urinary protein excretion and KIM-1 in urine. Also, histopathological examination revealed improvement in caspase-3 expression in kidney tissues as well as in glomerular and tubulointerstitial damage in DPO group. On the other hand, MSCs failed to improve animal survival rate, body weight, Hb level, proteinuria, hypoalbuminemia in ADR-treated rats although; it caused mild improvement of BUN, hyperlipidemia and renal morphology.

Conclusion: administration of DPO-α during induction of ADR-nephropathy might have renoprotective action against ADR-nephrosis. This might be due to improvement of Hb content, hyperlipidemia, enhancement of endogenous antioxidants, reduction of apoptosis and tubulointerstitial injury and maintaining the integrity of glomerular membrane. On the other hand, MSCs provide only partial protection that did not modify outcome of ADR-nephrosis.

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The Effect of examination stress on brain oscillations during memory task in males

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Background: Researches revealed that increased levels of stress hormones affects memory. Recently, it became apparent that the brain oscillations reflect cognitive aspects and information processing in the brain. Quantitative electroencephalogram (qEEG) provides an objective assessment of the electrical activity of the brain via many techniques such as power spectral analysis.

Aim: Assessment of the effect examination stress on quantitative electroencephalographic oscillations (qEEG) during delayed memory retrieval.

Methods: A prospective study was applied on **17** healthy, male undergraduate students. EEG was recorded during word recognition memory task (14 days after the encoding phase) during non-examination and examination (within 48 hour before periodic exams) periods. Quantitative analysis of the EEG was done using the relative power (RP). Serum cortisol (SC) was analyzed by enzyme linked immune-sorbent assay (ELISA) as a measure for stress.

Results:A bivariate correlational analysis showed positive significant correlation between the mean scores of SC and the mean scores of RP of δ and Θ bands (r: 0.53 and 0.55; p-value: 0.03 and 0.02 for δ and Θ bands respectively) during the memory task. There was a significant negative correlation between the mean scores of SC and the mean scores of RP of ß band (r: -0.51; p- value: 0.04). Insignificant correlation was found between SC and α band.

Conclusion: There is an obvious correlation betweenacute stress and brain oscillation in males.

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Correlation between tissue pathology and vitreal levels of hsCRP, IL-6 and TNF-α in diabetic rats treated with aspirin

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Diabetic retinopathy is a progressive neurological complication of diabetes. Microvascular changes accompany diabetic retinopathy and may result in blindness. Inflammation has been shown to underlie the pathogenesis of diabetic retinopathy. We investigated the correlation between inflammatory markers and tissue pathology in diabetic rat eye. We also tested the role of anti-inflammatory drugs (aspirin) in the prevention of diabetic retinopathy. Ninety male Wistar albino rats were used in the present study. Rats were equally divided into 3 groups; control group, diabetic (streptozotocin, 50 mg/kg i.p), and aspirin-treated (ASPIRIN PROTECT, 2mg/kg/d) diabetic group. All animals were scarified 6 months after the induction of diabetes. Histology and immunohistochemistry were conducted on the tissues of the rats' eyes, while vitreous samples were collected for the measurement of hsCRP, IL-6 and TNF-α. The levels of the measured inflammatory markers were significantly higher in diabetic rats, and notably correlated to the histo-pathological changes. Treatment with aspirin lowered the elevated levels of hsCRP, IL-6 and TNF-α with a considerable protective effect on the affected tissues. Our data suggest the possibility that hsCRP, IL-6 and TNF-α may be a cause of diabetic retinopathy progression and not necessarily a result. We also concluded that anti-inflammatory drugs which target hsCRP, IL-6 and TNF-α may play a crucial role in the prevention of diabetic microvascular complications .

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The Effect of Resveratrol on Insulin Resistance, Metabolic Syndrome and Hepatic Oxidative Stress in Fructose- Fed Rats

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Background/Aim: Metabolic syndrome and oxidative stress are common complications of type 2 diabetes mellitus. The aim of this work is to study the effect of resveratrol, a widely used nutritional supplement on insulin sensitivity, some metabolic parameters and hepatic oxidative stress in high fructose- fed rats.

Material & methods: Male Wister rats (180-200g) were divided into 3 groups 10 per each. 1-Control group was fed 65% corn starch diet.2- Fructose-fed insulin resistant group (HFD) was fed 65% fructose diet. 3- (HFD + Resveratrol) was fed with 65% fructose along with a single dose of 10 mg/kg/day of resveratrol orally through intra gastric tube for a period of 8 weeks.

Results: At the end of the feeding schedule, HFD group had significant hyperglycemia, hyper insulinemia and insulin resistance as evident by HOMA IR. Significant increase in triglyceride and nitric oxide levels. Significant increase in hepatic Thio Babituric Acid Reactive Substance (TBARS) and significant decrease in vitamin C in HFD group compared with control group. Administration of resveratrol normalized altered metabolic parameters. Resveratrol also attenuate hepatic oxidative stress parameters and significantly reduced inflammatory markers (Monocyte Chemoattractant Protein -1(MCP-1), Tumor Necrosis Factorα (TNFα) and Regulated on Activation Normal T cells Expressed and Secreted (RANTES).

Conclusion: The results of the present work strongly suggest that resveratrol supplementation may be used as a therapeutic tools that target the hazards of metabolic syndrome.

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Effect of Systemic ghrelin administration on Experimental Myocardial Infarction Induced by Isoproterenol in Rats

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[2]Faculty of Medicine, Tanta University - Egypt

Background: Isoproterenol (ISO), is a synthetic non-selective β-adrenoceptor agonist, has been reported to be used in large doses to produce Myocardial infarction (MI) in rats. Ghrelin is an orexigenic peptide hormone secreted into the systemic circulation predominantly by the X/A-like cells in the mucosa of the stomach. Ghrelin is broadly expressed in myocardial tissues although its effect on myocardial infarction is not fully investigated

Objective: To investigate the possible effects of ghrelin administration on ISO-induced MI in Wistar rats.

Material and Methods: This study was carried out on 30 adult male Wistar rats divided into 3 groups, each consisted of 10 rats

Group I: control group were given 0.9 % NaCl Group II; isoproterenol treated group (ISO) injected subcutaneously to rats (100 mg/kg) at an interval of 24 h for 2 days to induce experimental myocardial infarction.. Group III; rats were pretreated with ghrelin (150 µg/kg, s.c.) for 15 days and at the 14 and 15 days received isoproterenol as in group2 . Animals were sacrificed 24 h after the second dose of ISO. plasma and serum were collected to measure The activities of creatine kinase (CK) Creatine kinase-MB fraction (CK-MB) and serum lactate dehydrogenase (LDH), TNF-α and IL-6 also Lipid profile including; total cholesterol , triglycerides , LDL-cholesterol, VLDL (, and HDL-cholesterol ,. Specimens from heart were obtained .The levels of TNF-α and IL-6 in the myocardial tissue homogenates were measured, tissue catalase (CAT) enzymes activities reduced glutathione (GSH) level , malondialdehyde(MDA), Superoxide dismutase (SOD) and paraoxonase(PON1) enzyme activity where determined in myocardial tissues.

Results: The ISO- treated rats showed significant increase in the activities of CK, CK-MB, and LDH in the serum, Also there is significant increase in the levels of

plasma and cardiac tissues TNF-α and IL-6,as well as in heart tissues level of MDA and GSH similarly a significant increase in the levels of serum cholesterol, triglycerides, LDL-cholesterol, VLDL where observed .On the other hand there significant decrease in Tissues SOD and PON1 enzyme activity and serum HDL-cholesterol when these results are compared to control rats. Pretreatment with ghrelin , result in significant decrease in the activities of CK, CK-MB, and LDH in the serum, the levels of plasma and cardiac tissues and TNF-α IL-6, as well as in heart tissues level of MDA and GSH .Similarly a significant decrease in the levels of serum cholesterol, triglycerides], LDL-cholesterol, VLDL. On the other hand there is significant increase in Tissues SOD and PON1 enzyme activity and serum HDL-cholesterol level when these results are compared to ISO-induced myocardial infracted rats

Conclusions: The present study demonstrated that ghrelin may be considered as promising new therapies for MI

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The role of endocannabinoid system in the obesity induced atherogenesis. What are the possible mechanism/s involved?

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*Physiology and** Clinical pharmacology Departments, Faculty of Medicine, Zagazig University

Background: The involvement of adipocytes in the induction of atherosclerosis has been extensively explored. The endocannabinoid system is a participant in regulation of lipogenesis and adipokines production. It is postulated that adipokines could be the link between endocannabinoid system and atherosclerosis.

Objectives: The present work aims at studying the role of endocannabinoid system in the obesity associated atherogenesis and trying to clarify its possible mechanism/s of action. Also, we will investigate the role of adiponectin as a possible mediator of the cannabinoid action.

Design: Thirty adult male wistar albino rats were utilized in the present experiment. They were divided into three equal groups (each = 10 rats) as the following:**Group (1):** Lean control group, whichwere fed normal laboratory chow diet and gavaged with a single daily dose of the vehicle (0.6ml/kg /day of dimethyl sulfoxide)for 10 weeks. **Group (2):** Atherogenic diet group which were fed high fat diet and gavaged with a single daily dose of the vehicleas described for 10 weeks. **Group (3):** Atherogenic diet treated group which were treated by NIDA-41020(a selective cannabinoid receptor 1 blocker). They were fed high fat diet and gavaged with a single daily dose of NIDA-41020 of10mg/ kg /day for 10 weeks. Then body mass index, bleeding time, and total clotting time were assessed. After that, the animals were sacrificed andlipid profile, atherogenic index, bleeding time, platelet aggregation percentage, clot retractions, clotting time, prothrombin time, activated partial thromboplastin time, total &differential leukocytic counts and serum adiponectin levels were assessed in all groups. The aorta wasobtained from each animal dissected and stained by haematoxylin/eosin and oil Red O staining for histological examination and detection of aortic thickness and foam cells deposition.

Results: In the present study, the laboratory investigations and histological examination conducted on different groups revealed, significant increases in BMI,

lipid profile, atherogenic index, platelet aggregation%, peripheral monocytic count, and aortic thickness in the high fat diet received group versus lean controls which were otherwiseassociated with significant decreases in total clotting time,PT, PTT, serum HDL&adiponectinlevels. These previous changes were significantly and profoundly inhibited by the administration of the cannabinoid receptor antagonist.

Conclusion: The present study concluded that the endocannabinoid system is involved in the atherogenic changes associated with obesity. These effects were attributed to interference with serum adiponectin level, dyslipidemia, hypercoagulability, increased platelet activation &peripheral as well as endothelial recruitment of the monocytes .these endocannabinoid induced effects were found to be via activation of cannabinoid 1 receptor .These findings could offer a new potential therapeutic option for this condition.

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Lipoprotein-associated Phospholipase A2 Levels as a Predictor of Cardiovascular Risks in Patients with COPD and obstructive sleep apnea

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Background: Lipoprotein-associated phospholipase A_2 (Lp-PLA_2) is an inflammatory mediator used as a novel marker for diagnosis of cardiovascular diseases and atherosclerosis in human. Both chronic obstructive pulmonary disease (COPD) and obstructive sleep apnea (OSA) are associated with increased activation of inflammatory cells and molecular mechanisms associated with atherosclerosis.

Aim: to study the relation between Lp-PLA_2 level and arousal index in patients with OSA, COPD and overlap syndrome (COPD+OSA). **Methods:** Sixty patients were recruited, divided into 4 groups (15 in each group) based on their polysomnographic and spirometric data ; group I (normal), group II (OSA), group III (COPD) and group IV (overlap syndrome). Fasting serum samples were used to estimate lipid profile and Lp-PLA_2 concentrations.

Results: apnea-hypopnea, arousal, desaturation indices, lipid profile and Lp-PLA2 levels were significantly increased in all patients groups compared to control. The level of Lp-PLA_2 was significantly increased in overlap syndrome more than OSA and COPD patients and was positively correlated and it was independent predictor of arousal index in all patients groups.

Conclusion: Patients with overlap syndrome are more liable to cardiovascular disorders than either OSA or COPD alone and Lp-PLA_2 may be used as an independent predictor of cardiovascular disorders in patients with OSA, COPD and overlap syndrome.

Key words: Lipoprotein-associated phospholipase A_2, obstructive sleep apnea (OSA), chronic obstructive pulmonary diseases (COPD), overlap syndrome, arousal.

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