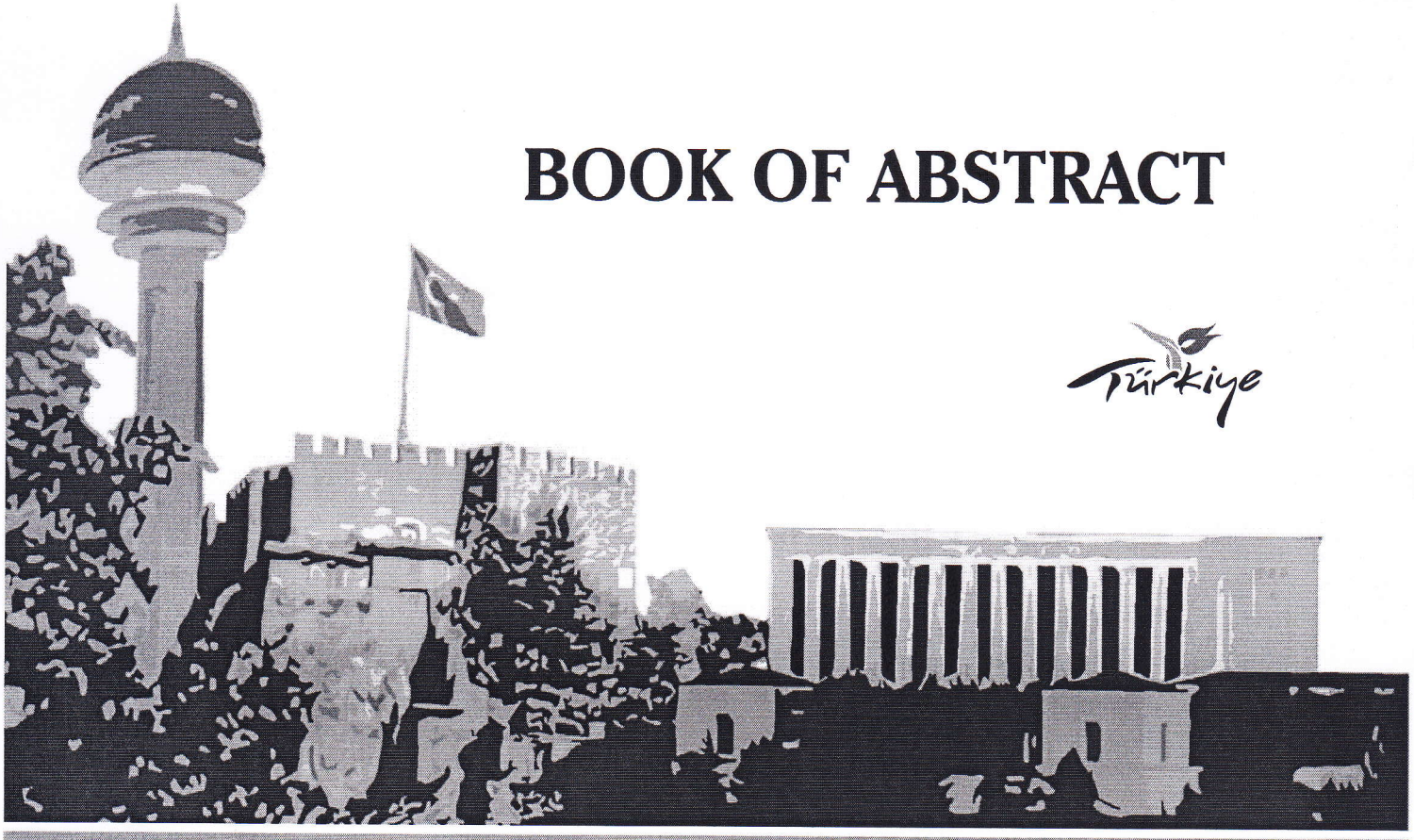
ANKARA UNIVERSITY
FACULTY OF PHARMACY

ISOPS 11th International SYMPOSIUM ON PHARMACEUTICAL SCIENCES

BOOK OF ABSTRACT



Türkiye

T.C.
KÜLTÜR VE TURİZM BAKANLIĞI

TUBİTAK

JUNE 09-12, 2015
ANKARA, TURKEY

Ankara University Faculty of Pharmacy Publication No: 112

ANKARA UNIVERSITY
FACULTY OF PHARMACY

ISOPS 11th International SYMPOSIUM ON PHARMACEUTICAL SCIENCES

Book of Abstracts

June 9-12, 2015
ANKARA, TURKEY

Book of Abstracts
Ankara University Faculty of Pharmacy Publication No: 112
June 2015 Ankara, Turkey

ISBN: 978-605-136-221-2

Basım: Başak Matbaacılık ve Tanıtım Hiz. Ltd. Şti.
T.0312 397 1617 F.0312 397 0307

Honory President of the symposium

Prof. Dr. Erkan İBİŞ

President of Ankara University

ORGANIZING COMMITTEE

Maksut COŞKUN (Chair)
Gülbin ÖZÇELİKAY (Treasurer)
Sinan SÜZEN
Nilüfer YÜKSEL
Betül SEVER YILMAZ
Arzu ONAY BEŞİKCİ
Tansel ÇOMOĞLU
Sinem ASLAN ERDEM (Secretariat)
Meltem CEYLAN ÜNLÜSOY
Müjde YÜCE ERYILMAZ
Arzu Zeynep KARABAY
Burcu DOĞAN TOPAL
Işıl ÖZAKCA

SOCIAL COMMITTEE

Selen ALP
Ebru ARIOĞLU İNAN
İlker ATEŞ
Filiz BAKAR
Kayhan BOLELLİ
Derya ÇİÇEK POLAT
Merve DEMİRBÜGEN
Zehra Ceren ERTEKİN
Ayşe Mine G. ÖZKAN
Alper GÖKBULUT
Gizem GÜLPINAR
Mehmet GÜMÜŞTAŞ
Berna GÜVEN
M. Mesud HÜRKUL
Özge İNAL
Melek KARACAOĞLU
Banu KAŞKATEPE
Ecem KAYA
Gizem KAYKI MUTLU
Merve E. B. KIYMACI
Dilan KONYAR
Sevinç KURBANOĞLU
Aslıhan H. ALGAN
Umut Can ÖZ
Selman ŞABANOĞLU
Tangül ŞEN
Mehmet Barlas UZUN
Gülderen YILMAZ
Serap YILMAZ

SCIENTIFIC COMMITTEE

Nurten ALTANLAR (Turkey)
M. Levent ALTUN (Turkey)
Zeynep ATEŞ ALAGÖZ (Turkey)
Tunca Gül ALTUNTAŞ (Turkey)
Asuman BOZKIR (Turkey)
Zeliha BÜYÜKBİNGÖL (Turkey)
Benay CAN EKE (Turkey)
Nina CHANISHVILI (Georgia)
Tülay ÇOBAN (Turkey)
Tuncer DEĞİM (Turkey)
Bilgehan DOĞRU (Turkey)
Bülent GÜMÜŞEL (Turkey)
Christoph HIEMKE (Germany)
Ayşegül KARATAŞ (Turkey)
Ceyda S. KILIÇ (Turkey)
Marie-Aleth LACAILLE-DUBOIS (France)
Henk LINGEMAN (The Netherlands)
Arzu ONAY BEŞİKCİ (Turkey)
Feyyaz ONUR (Turkey)
A. Tanju ÖZÇELİKAY (Turkey)
Gülbin ÖZÇELİKAY (Turkey)
Ayşe Mine ÖZKAN (Turkey)
Keykavous PARANG (USA)
Gülçin SALTAN (Turkey)
Sevgi ŞAR (Turkey)
Istvan TOTH (Australia)
Bengi USLU (Turkey)
Sulhiye YILDIZ (Turkey)

serum albumin as standard. Statistical analysis was performed using SPSS 10.0 for windows software. The obtained data were presented as mean ± SE (standard error) unless otherwise specified. The differences were considered as statistically significant when $p<0.05$.

RESULTS AND DISCUSSION

SOD activities were found in Group I, Group II and Group III as 86.62±10.81 U/mg protein, 81.92±11.54 U/mg protein and 51.93±8.46 U/mg protein in liver tissue and 53.27±6.49 U/mg protein, 49.27±6.06 U/mg protein and 34.2±5.88 U/mg protein in kidney tissue, respectively. Catalase activities were found in Group I, Group II and Group III as 56.22±6.27 U/mg protein, 51.07±7.42 U/mg protein and 24.01±4.31 U/mg protein in liver tissue and 44.02±7.81 U/mg protein, 45.91±8.11 U/mg protein and 22.2±4.06 U/mg protein in kidney tissue, respectively. GSH levels were found in Group I, Group II and Group III as 0.81±0.06 µmol/mg protein, 0.84±0.06 µmol/mg protein and 0.53±0.04 µmol/mg protein in liver tissue and 0.67±0.08 µmol/mg protein, 0.66±0.09 µmol/mg protein and 0.41±0.06 µmol/mg protein in kidney tissue, respectively. MDA levels were found in Group I, Group II and Group III as 91.06±10.04 nmol/mg protein, 94.04±10.47 nmol/mg protein and 147.83±19.61 nmol/mg protein in liver tissue and 78.71±12.63 nmol/mg protein, 76.81±11.87 nmol/mg protein and 106.93±8.08 U/mg protein in kidney tissue, respectively. The electromagnetic field led to a significant increase in malondialdehyde (MDA) levels and significant decrease in SOD and CAT levels in the liver and kidneys tissue of rats ($p<0.05$). There was no significant difference in GSH levels in the same tissues ($p>0.05$).

CONCLUSIONS

In conclusion, electromagnetic field emitting from mobile phone might produce impairments in some oxidative stress parameters in the liver and renal tissue of albino rats.

REFERENCES

1. Li L, Xiong F, Liu JW, Li ZX, Zeng GC, Li HL. A cross-sectional study on oxidative stress in workers exposed to extremely low frequency electromagnetic fields. *Int J Radiat Biol* **2015**, 4, 1-23.

2. Ellman GL, Courtney KD, Andres V Jr, Feather-Stone RM. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochem Pharmacol* **1961**, 7, 88-95.

3. Yagi K. Lipid peroxides and related radicals in clinical medicine. *Adv Exp Med Biol* **1994**, 366, 1-15.

P-151: INFLUENCE OF SUBLETHAL CHLORPYRIFOS EXPOSURE ON OXIDATIVE STRESS AND ACETYLCHOLINESTERASE ACTIVITY IN CARP (*CYPRINUS CARPIO*)

M. Berkoz[1], S.G. Gunduz[2], F.Ö. Yilmaz[2], S. Yalin[3], A.O. Hunt[4], M. Yildirim[3]

Yuzuncu Yıl University, Faculty of Pharmacy, [1]Department of Pharmaceutical Biotechnology, Van, Turkey,
Mersin University, Faculty of Fisheries, [2]Department of Basic Sciences, [4]Department of Aquaculture, Faculty of Pharmacy, [3]Department of Biochemistry, Mersin, Turkey

INTRODUCTION

The commonly used pesticides in agriculture may react with macromolecules and may cause enzyme inactivation and DNA damage. Furthermore, they may also initiate peroxidation of poly-unsaturated fatty acids (PUFA) due to their deposition in fatty tissues by the generation of reactive oxygen species (ROS) as by-products. In the course of these events they can lead to oxidative stress. The objective of our study was to determine the oxidative and neurotoxic potential of sub-lethal concentrations (0.26 ppm and 0.52 ppm) of chlorpyrifos which is extensively used as a pesticide in Turkish agriculture in brain tissue at the 96th and 240th hours.

MATERIALS AND METHODS

In order to detect the levels of oxidative stress in brain tissue, glutathion levels were detected by using superoxide dismutase possessing antioxidant features. Moreover, malondialdehyde (MDA) levels and acetylcholine esterase (AChE) levels were examined for the determination of levels of lipid prexodiation and neurotoxic effect, respectively.Acetylcholine esterase activity in cerebral cortex was performed by utilizng the spectrophotometric method of described by Ellman, Courteney, Andres, and Featherstone. The levels of tissue lipid peroxidation products such as thiobarbituric acid (TBA)-malondialdehyde (MDA) adducts were measured spectrophotometrically by the method described by Yagi. Virtually, all of the nonprotein sulfhydryl compounds of tissue were existing in the form of GSH. 5,5′ Dithiobis (2-nitro benzoic acid) (DTND) is a disulfide compound readily which is reduced by sulfhydryl compounds that form a highly colored yellow anion by the method described by Beutler *et al.* The optical density of this yellow substance is measured at 412 nm. SOD activity was measured by the inhibition of nitroblue tetrazolium (NBT) reduction due to O_2 generated by the xanthine/xanthine oxidase system.

The contents of tissue protein were measured in accordance with the method developed by Lowry et al. by using bovine serum albumin as standard.

Statistical analysis was performed using SPSS 10.0 for windows software. The obtained data were

presented as mean ± SE (standard error) unless otherwise specified. ANOVA and Tukey multiple range tests were used to analyze differences between the groups. The differences were considered as statistically significant when $p<0.05$.

RESULTS AND DISCUSSION

AChE, SOD and GST activities and GSH and MDA levels were shown in the table.

	Control 96th hour	0.26 ppm 96th hour	0.52 ppm 96th hour	Control 240th hour	0.26 ppm 240th hour	0.52 ppm 240th hour
AChE (U/mg prt.)	0.14±0.02	0.08±0,01	0.06±0,01	0.13±0,02	0.07±0.01	0.05±0.01
SOD (U/mg prt.)	41.17±5.22	38.11±6.23	21.61±5.01	40.23±6.17	35.96±5.12	22.3±4.74
GST (U/mg prt.)	38.24±5.67	34.52±5.74	31.49±6.42	35.04±5.01	32.96±6.41	17.04±8.43
GSH (µmol/mg prt.)	0.08±0.01	0.07±0.01	0.04±0.01	0.07±0.01	0.07±0.01	0.03±0.01
MDA (nmol/mg prt.)	15.71±4.32	24.45±7.21	29.1±4.04	16.81±4.54	18.26±3.51	19.54±3.07

AChE esterase activity was reduced in both concentrations over the time ($p<0.5$). MDA levels were increased at the 96th hour for both concentrations ($p<0.05$). The levels of SOD and GSH were elevated at the 240th hour for both concentrations ($p<0.05$). GST activity reduced at 240th hour for only the concentration of 0.52 ppm ($p<0.05$).

CONCLUSIONS

We were able to observe an induced oxidative stress and significant inhibition of AChE in the brain tissues of *Cyprinus carpio* exposed to chlorpyrifos.

These findings manifest that sub-lethal concentration of chloropyrifos leads to significant systemic toxicity in the brain tissues of *Cyprinus carpio*.

REFERENCES

1. Kavitha P1, Rao JV. Toxic effects of chlorpyrifos on antioxidant enzymes and target enzyme acetylcholinesterase interaction in mosquito fish, Gambusia affinis. *Environ Toxicol Pharmacol* **2008**, 26 (2), 192-8.

2. Jin Y, Liu Z, Peng T, Fu Z. The toxicity of chlorpyrifos on the early life stage of zebrafish: A survey on the endpoints at development, locomotor behavior, oxidative stress and immunotoxicity. *Fish Shellfish Immunol* **2015**, 43 (2), 405-14.

P-152: IRON STATUS IN ADOLESCENT GIRLS-RELATION TO OBESITY AND INFLAMMATION

N. Kavaric [1], A. Klisic [1], T. Injac [2], D. Bozovic [3], N. Gligorovic-Barhanovic [3]

[1] Primary Health Care Center, Podgorica, Montenegro

[2] Primary Health Care Center, Niksic, Montenegro

[3] Center of Clinical-Laboratory Diagnostics, Clinical Center of Montenegro, Podgorica, Montenegro

INTRODUCTION

Obesity is associated with chronic low-grade inflammation [1]. Moreover, recent studies reported iron deficiency in overweight/obese adolescents [2, 3]. On the contrary, some other reports suggested that adiposity was sufficient to cause chronic inflammation but not to impair iron status [4]. Therefore, we aimed to determine some iron status biomarkers and to examine their potential association with anthropometric and inflammation parameters in normal weight and overweight/obese adolescent girls.

MATERIALS AND METHODS

A cross-sectional study was performed in a primary care setting. A total of 22 overweight/obese adolescent girls (mean age 17.50±1.34 years) and 16 age-matched normal weight controls were included. Biochemical and haematological parameters of iron status: serum iron, soluble transferrin receptor concentration (sTfR), transferrin, ferritin, red blood cell count (RBC), haemoglobin, haematocrit (Hct), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were measured. Inflammation was determined by high sensitivity C-reactive protein (CRP). Body weight and body height were obtained. Body mass index (BMI) z-score was calculated [5]. All the participants completed a questionnaire including dietary habits, somatic illnesses, medications use, and lifestyle habits. Adolescent girls younger than 16 years, and older than 19 years, as well as participants who had diabetes mellitus, renal, hepatic or thyroid dysfunction, cardiovascular disorders, with signs and symptoms of acute inflammatory disease and CRP > 10 mg/L, with a history of alcohol consumption and smoking, and those who used any medications were excluded from the study. Using the World Health Organization growth reference 5-19 years [5] adolescents were categorized as normal weight ($-2SD \leq$ BMI z-score $\leq +1SD$), and overweight ($+1SD <$ BMI z-score $< +2SD$) or obese (BMI z-score$\geq +2$ SD).

RESULTS AND DISCUSSION

Overweight/obese girls displayed higher serum CRP, sTfR and ferritin levels ($p<0.001$, $p=0.013$ and $p=0.034$, respectively), but lower MCHC ($p=0.008$) as compared with normal weight group. However, there was no difference in serum transferrin and iron level, RBC, hemoglobin, Hct, MCV and MCH between groups. In all girls, serum ferritin correlated positively with body weight and BMI z-score ($p=0.043$ and $p=0.030$, respectively). sTfR correlated positively with body weight and BMI z-score ($p=0.023$ and $p=0.029$, respectively), RBC ($p=0.005$), and negatively with MCV, MCH and MCHC ($p=0.011$,