Al-Azhar Med. J. *Vol. 45(3), July 2016, 571-583*
DOI: 10.12816/0033124/ORG. No. 96284 ***Issues_title_English:*** 2016 Vol.45 Issue 3

EFFECT OF CROWDING AND NOISE STRESSORS ON LIVER AND KIDNEY FUNCTIONS IN ADULT MALE ALBINO RAT

By

Gamal Ahmad Shawer, Shebl Ramadan Samaha, Ezz-Eldin Khalifa and Ahmad Shaaban Abd-El-Monsef

Medical Physiology Dpartment, Al-Azhar Faculty of Medicine

ABSTRACT

Background: Although the stress response can enhance the probability of survival in the face of true environmental threats, repeated activation in response to frequent or chronic stress can have serious pathological sequelae**. Objective**: This work was carried out to investigate the effect of crowding or noise stresses on some biochemical parameters and histological structure of the liver and kidney tissues of adult male albino rats after one month from chronic exposure to these environmental stressors. **Material and Methods**: Crowding was induced by multiplying the normal density by three. Noise was prepared by using 5 different sources of no harmonic sounds and high intensity music. Animals were divided into three equal groups:

- Control group.
- Rats exposed to noise for 30 days (over 90db, 4h/day).
- Rats exposed to crowding for 30 days.

At the end of experiment, blood samples were obtained for determination of:

1- Blood glucose.
2- Lipid profile (total cholesterol, TGs, HDL and LDL).
3- Liver functions tests (AST, ALT, total serum protein, serum albumin and serum bilirubin).
4- Kidney functions tests (blood urea, serum creatinine and uric acid).

Results: Both noise and crowding had similar effects on different measured parameters:

- A significant increase in blood glucose, total cholesterol, TGs, LDL, AST, ALT, total serum protein and serum creatinine.
- A significant decrease in serum albumin.

Exposure to crowding and noise led to many pathological changes in the liver tissues of rats. These changes included dilated congested central vein with irregular wall, vacuolated hepatocytes with destructive nuclei, increased numbers of von Kupffer cells and lymphocytic infiltration. Exposure of rats to crowding and noise showed several dystrophic changes in the kidney tissue. These changes included: highly atrophied glomeruli, faintly stained cells and nuclei of the convoluted tubules with wide lamina of the distal ones, ruptured brush borders of the proximal ones, thickened arterial walls with branched and corrugated walls of the congested vein.

Conclusion: Exposure to noise or crowding stressors showed many disturbances in liver and kidney parameters.

INTRODUCTION

Stress stimulates several adaptive hormonal responses, prominent among which are the secretion of catecholamines from the adrenal medulla, corticosteroids from the adrenal cortex, and adrenocorticotropin from the anterior pituitary (**Sabban and Kvetnansky, 2010).**

Noise and crowding are kinds of stresses which pervasive aspects resemble of many modern community and work environments. Acute noise or crowding exposure activated the autonomic and hormonal systems, leading to temporary changes such as increased blood pressure, heart rate and vasoconstriction. After prolonged exposure, susceptible individuals in the general population may develop permanent effects, such as hypertension and ischemic heart disease that are associated with exposures to high sound pressure levels. Other extra anural effects of noise include the impairments of rest, sleep and blood pressure (**Tomoyuki, 2004).**

This study aimed at clarifying the effect of each crowding and noise as specific stressors which have effects on the various metabolic processes and disturbance of some biochemical parameters and histological structures.

MATERIAL AND METHODS

Animals: A total number of forty two adult male albino rats of local strain were the model of the present work. This experiment was done at Mansura Nile Center for animal experiments. All rats were about the same age and healthy, their weight ranging between 140 - 180 gm (average weight 160 gm), they were kept in suitable cages (20 × 30 × 20 cm for every 3 rats) made of zinc material with network bases to clarify the waste products of rats. Rats were maintained on balanced standard rat's cubes with free water supply. They were left for two weeks for acclimatization in the laboratory room at comfortable temperature with natural light - dark cycle.

Rats were divided into three groups:

Group I (Control group): Twelve rats were kept in four cages (3 rats per cage).

Group II (Crowded group): Eighteen rates were kept in two cages (9 rats per cage).

Group III (Noised group): Twelve rats were kept in four cages (3 rats per cage).

Induction of crowding: Crowding was induced by multiplying the normal rat density by three. Normally, the cage was suitable for 3 adult rats. So, nine rats were kept per cage **(Armario *et al.*, 1987).**

Induction of noise: Noise was induced by exposure of the animals to 90 dB of prerecorded noise delivered via high volume setting stress speaker placed one meter from the cage for four hours daily in a separate room away from other rats for 30 consecutive days **(Waye *el al.*, 2002**).

Blood sampling: At the end of experimental period, blood samples were obtained from each rat, centrifuged at 5000 rpm for 10 minutes to separate sera which were collected and stored frozen at -20?C until assayed for determination of:

1- Blood glucose **(Tietz, 2011)**.

2- Lipid profile: Total cholesterol **(Tietz, 2011)**, triglycerides **(Fossati and Prencipe, 1982)**, HDL cholesterol **(Widhaim and Pakosta, 1991)**, and LDL cholesterol **(Viikari, 1976)**.

3- Liver functions tests: AST and ALT **(Reitman and Frankel, 1957)**, total serum protein **(Tietz, 2011)**, serum albumin**(Doumas, 1971)**, and serum bilirubin ***(*Jendrassik, 1938)**.

4- Kidney functions tests: Urea **(Patton and Crouch, 1977)**, creatinine **(Henry, 1974)**, and uric acid **(Tietz, 2011).**

Histopathological study: At the end of the experimental period, the anesthetized rats were killed by intra-cardiac perfusion of 10% formalin solution **(Yüksek et al., 2009).** The abdominal cavities were opened, and then livers and the kidneys were exposed, dissected and excised. Samples were kept in 10% formalin solution. Paraffin blocks were made and different sections at different levels were obtained. Slides were stained with hematoxyline and eosine (Hx and E) and pass stains, and examined using a light microscope.

RESULTS

Changes in blood glucose and lipid profile:

When compared to the crowded group, induction of noise led to insignificant increase in the mean value of blood glucose level from 134.00 ± 2.8 mg/dl to 149.10 ± 2.42 mg/dl (+11.26%), insignificant increase in the mean value of blood cholesterol level from 151.22 ± 1.68 mg/dl to 168.45 ± 1.2 mg/dl (+11.40%), insignificant decrease in the mean value of triglycerides level from 124.11 ± 0.96 mg/dl to 103.31 ± 1.54 mg/dl (+16.76%), insignificant increase in the mean value of HDL level from 70.06 ± 0.66 mg/dl to 76.91 ± 0.37 mg/dl (+9.79%) and insignificant increase in the mean value of LDL level from 80.17 ± 0.65 mg/dl to 91.37 ± 0.79 mg/dl (+13.97% - Table 1).

Table (1): Changes in blood glucose and lipid profile (Mean ± SE).

Groups / Parameters	Control (a) (n=12)	Crowded (b) (n=18)	Noised (c) (n=12)	% Change (a-b)	% Change (a-c)	% Change (b-c)
Blood Glucose(mg/dl)	**101.7 ± 3.25**	**134.00 ± 2.8 ***	**149.1 ± 2.42 ***	**+ 31.8**	**+ 46.61**	**+ 11.26**
Cholesterol(mg/dl)	**119.67 ± 2.58**	**151.22 ± 1.68***	**168.45 ± 1.2***	**+ 26.36**	**+ 40.76**	**+ 11.4**
TGs. (mg/dl)	**66.59 ± 0.59**	**124.11 ± 0.96***	**103.31 ± 1.54***	**+ 86.37**	**+ 55.14**	**- 16.76**
HDL(mg/dl)	**65.28 ± 1.09**	**70.06 ± 0.66**	**76.91 ± 0.37**	**+ 7.32**	**+ 17.82**	**+ 9.77**
LDL(mg/dl)	**50.97 ± 0.47**	**80.17 ± 0.65***	**91.37 ± 0.79 ***	**+ 57.28**	**+ 79.26**	**+ 13.97**

- n: number of rats.
- *: significant.

Changes in liver functions:

When compared to the crowded group, induction of noise led to insignificant decrease in the mean value of AST level from 31.6 ± 1.93 u/l to 31.1 ± 1.92 u/l (-1.58%), insignificant decrease in the mean value of ALT level from 59.3 ± 2.17 u/l to54.2 ± 3.45 u/l (- 8.6%),insignificant increase in the mean value of albumin level from 3.55 ± 0.08 g/dl to 3.75 ± 0.14 g/dl (+5.63%), insignificant increase in the mean value of total protein level from 7.6 ± 0.07 g/dl to 7.75 ± 0.2 g/dl (+1.97%) and insignificant decrease in the mean value of total bilirubin level from 0.387± 0.01 mg/dl to 0.385 ± 0.007 mg/dl (- 0.52% -Table 2).

Table (2): Changes in liver functions (Mean ± SE).

Groups / Parameters	Control (a) (n=12)	Crowded (b) (n=18)	Noised (c) (n=12)	% Change (a-b)	% Change (a-c)	% Change (b-c)
AST(u/l)	**28.00 ± 1.25**	**31.6 ± 1.93***	**31.1 ± 1.92***	**+ 12.9**	**+ 11.07**	**- 1.58**
ALT(u/l)	**39.6 ± 1.13**	**59.3 ± 2.17***	**54.2 ± 3.45***	**+ 49.74**	**+ 36.86**	**- 8.6**
Albumin (g/dl)	**4.21 ± 0.16**	**3.66 ± 0.08***	**3.75 ± 0.14***	**- 13.06**	**- 10.42**	**+ 5.36**
Total Protein (g/dl)	**6.9 ± 0.13**	**7.6 ± 0.07***	**7.75 ± 0.2***	**+ 10.15**	**+ 12.32**	**+ 1.97**
Bilirubin (mg/dl)	**0.375 ± 0.011**	**0.387 ± 0.1**	**0.385 ± 0.007**	**+ 3.2**	**+ 2.7**	**- 0.52**

- n: number of rats.
- *: significant.

Exposure to crowding led to dilated congested central vein with irregular wall, vacuolated hepatocytes with destructive nuclei, increased numbers of von Kupffer cells and lymphocytic infiltration. Highly decreased PAS +ve materials were noticed in hepatocytes of the central and the portal areas of liver tissue of rats exposed to crowding (Fig. 1, 2 and 3).

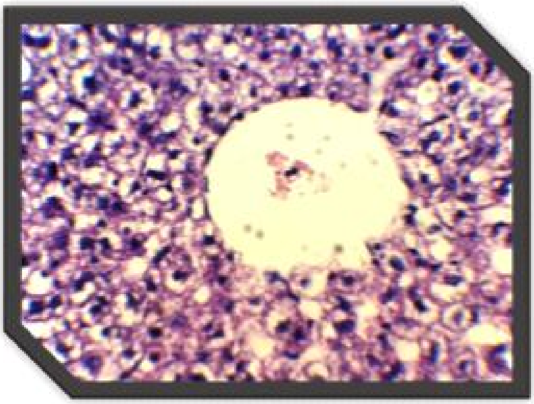

Fig. (1): Liver tissue of crowded group showing dilated congested central vein with irregular wall (black arrow), vacuolated hepatocytes with destructive nuclei (yellow arrow),von Kupffer cell (green arrow) and lymphocytic infiltration (blue arrow) **(H&E ×400).**

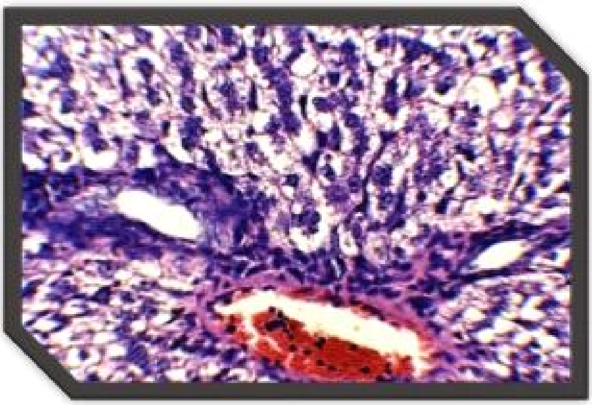

Fig. (2): Liver tissue of crowded group (GII) showing dilated congested portal vein (black arrow), vacuolated hepatocytes (yellow arrow), thick wall congested hepatic artery (violet arrow) and lymphocytic infiltration (blue arrow) **(H&E ×400).**

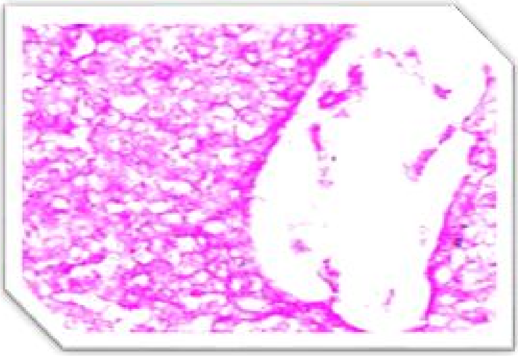

Fig. (3): Liver tissue of crowded group (GII) showing PAS +ve material in the wall of dilated central vein (black arrow) and accumulating in periphery of vacuolated hepatocytes (yellow arrow) **(PAS x400).**

Exposure to noise stress caused dilated congested central vein, vacuolated hepatocytes with destructive nuclei, obliterated sinusoidal space, increased numbers of von Kupffer cells and lymphocytic infiltration. Hepatocytes were poorly stained in the central area, and increased stain affinity of PAS +ve materials in the portal area of the liver tissue of a rat exposed to noise as in figure (Fig. 4, 5 and 6).

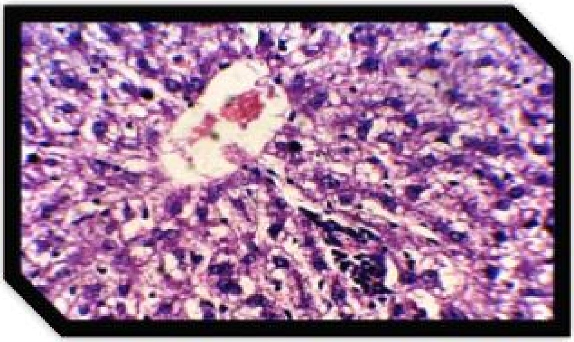

Fig. (4): Liver tissue of noised group (GIII) showing dilated congested central vein (black arrow), vacuolated hepatocytes with destructive nuclei (yellow arrow), obliterated sinusoidal space (green arrow), von Kupffer cell (violet arrow) and lymphocytic infiltration (blue arrow) **(H&E ×400).**

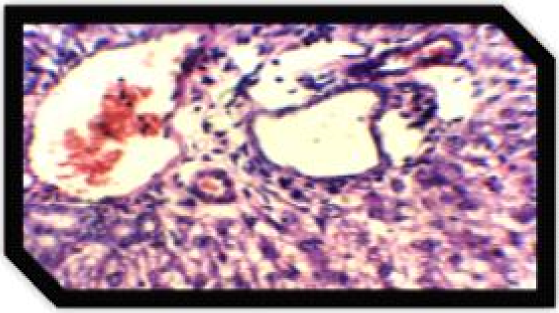

Fig. (5): Liver tissue of noised group (GIII) showing porta heptes with dilated congested portal vein (black arrow), vacuolated hepatocytes (yellow arrow), congested hepatic artery (violet arrow) and lymphocytic infiltration (blue arrow) **(H&E ×400).**

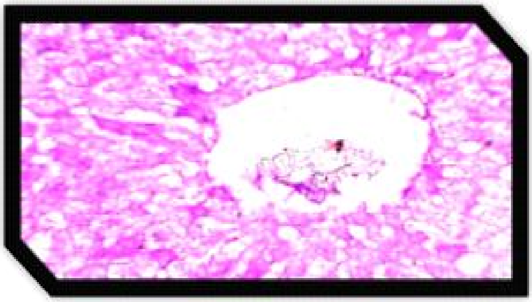

Fig. (6): Liver tissue of noised group (GIII) showing PAS +ve material in the wall of dilated central vein (black arrow) and vacuolated hepatocytes (yellow arrow) **(PAS x400).**

Changes in kidney functions:

When compared to the crowded group, induction of noise led to insignificant increase in the mean value of uric acid level from 3.12 ± 0.05 mg/dl to 3.49 ± 0.14 mg/dl (+11.86%), insignificant decrease in the mean value of urea level from 27.77 ± 0.22 mg/dl to 26.04 ± 0.07 mg/dl (-6.23%) and significant decrease in the mean value of creatinine level from 1.76 ± 0.02 mg/dl to 1.58 ± 0.07 mg/dl (-10.22% - Table 3).

Table (3): Changes in kidney functions (Mean ± SE).

Parameters \ Groups	Control (a) (n=12)	Crowded (b) (n=18)	Noised (c) (n=12)	% Change (a-b)	% Change (a-c)	% Change (b-c)
Uric acid(mg/dl)	3.4 ± 0.16	3.48 ± 0.05	3.49 ± 0.14	+ 2.35	+ 2.64	+ 11.86
Urea(mg/dl)	28.66 ± 0.13	27.77± 0.22	26.0 ± 0.07	- 3.1	- 9.28	- 6.23
Creatinine(mg/dl)	0.58 ± 0.02	1.76 ± 0.02*	1.85±0.07*	+ 203.45	+ 218.96	- 10.22

- n: number of rats.
- *: significant.

Exposure of rats to crowding showed several dystrophic changes in the kidney tissue including highly atrophied glomeruli, faintly stained cells and nuclei of the convoluted tubules with wide lamina of the distal ones, ruptured brush borders of the proximal ones, thickened arterial walls with branched and corrugated walls of the congested vein. Kidney tissue of crowded group showed showing increase of PAS positive materials in the renal tissue, Bowman's membrane brush border and basement membrane of the renal tubules and glomeruli (Fig. 7, 8 and 9).

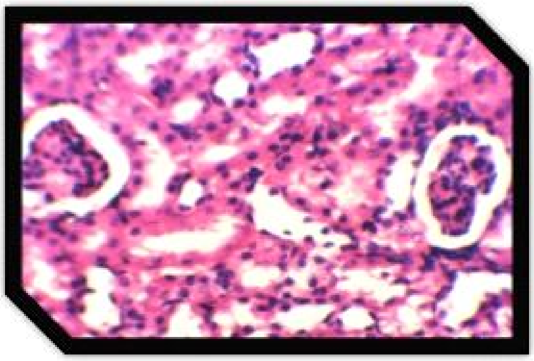

Fig. (7): Renal cortex of crowded group (GII) showing shrunken glomeruli tuft (black arrow), with wide sub-capsular space (green arrow), areas of cellular hyperplasia (yellow arrow), vacuolated cytoplasm of renal tubules (blue arrow) and lymphocytic infiltration (violet arrow) **(H&E ×400).**

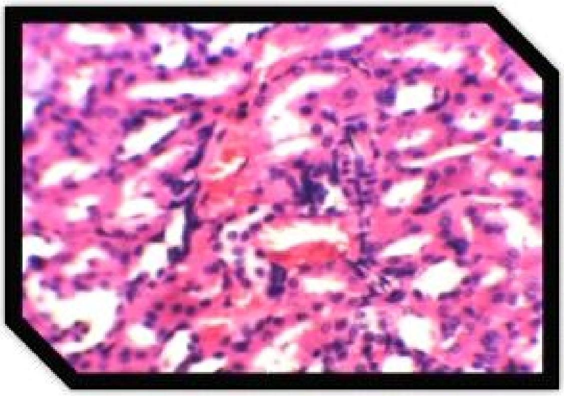

Fig. (8): Renal cortex of crowded group (GII) showing congested blood vessels (black arrow) with hemorrhagic areas (blue arrow), areas of cellular hyperplasia (yellow arrow), vacuolated cytoplasm of renal tubules (blue arrow) and lymphocytic infiltration (violet arrow) **(H&E ×400).**

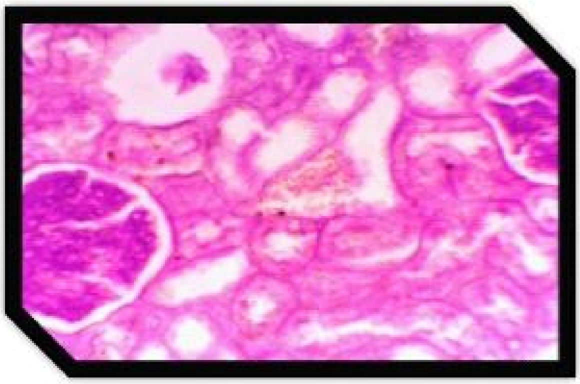

Fig. (9): Kidney of crowded group (GII) showing increase of PAS positive materials in the renal tissue, bowman's membrane (black arrow), brush border (yellow arrow) and basement membrane (blue arrow) of the renal tubules and glomeruli (green arrow) **(PAS stain x400).**

Exposure of rats to noise showed shrunken glomeruli tuft with wide sub-capsular space, proximal tubules loss its characteristic shape, vacuolated cytoplasm of renal tubules, hemorrhagic areas, lymphocytic infiltration and patchy necrosis. Kidney tissue of noised group showed poor stain affinity of PAS +ve materials with moderately stained tunica media and adventitia of the highly distorted renal artery compared with the control group. Poorly stained glomeruli of the kidney cortex of noised group were detected as in figure (Fig. 10, 11 and 12).

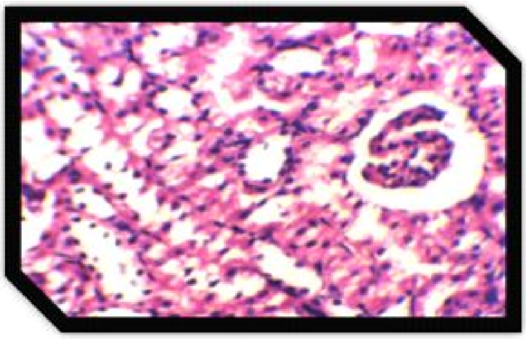

Fig. (10): A photomicrograph of a section from the renal cortex of noised group (GIII) showing shrunken glomeruli tuft (black arrow), with wide sub-capsular space (green arrow), proximal tubules loss its characteristic shape (yellow arrow), vacuolated cytoplasm of renal tubules (blue arrow) and lymphocytic infiltration (violet arrow) **(H&E ×400).**

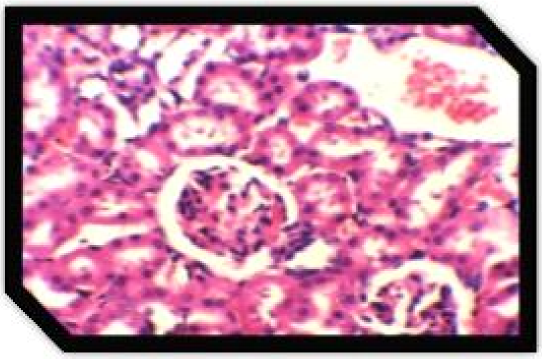

Fig. (11): Renal cortex of noised group (GIII) showing shrunken glomeruli tuft (black arrow), with wide sub-capsular space (green arrow), hemorrhagic areas (blue arrow) and patchy necrosis (yellow arrow) **(H&E ×400).**

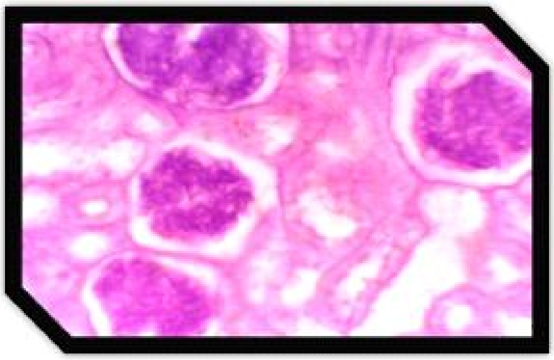

Fig. (12): Kidney of noised group (GIII) showing increase of PAS positive materials in the renal tissue, bowman's membrane (black arrow), brush border (yellow arrow) and basement membrane (blue arrow) of the renal tubules and glomeruli (green arrow) **(PAS stain x400).**

DISCUSSION

This work was carried out to investigate the effect of crowding and noise as chronic stressors on some physiological, biochemical parameters and histological changes in the liver and kidney of adult male albino rats.

The present work showed significant increase in blood glucose level in the stressed groups. **Nayanatra *et al. (*2006)** found that there was significant increase in blood glucose level in experimental rats exposed stress. Hyperglycemia in this study could be due to excess secretion of stress hormones in response to chronic stressful conditions. Cortisol role is to provide glucose to the body through utilization of protein stores. This quick delivery of glucose prepares the body for the fight or flight mechanism. When the body is in a persistent stressful state, cortisol is supplying the body with essential blood glucose. It did this by stimulating the liver to make glucose from protein and fat through gluconeogenesis process **(Aronson, 2009).**

Cortisol plays an important role in glycogenolysis **(Robert *et al.*, 2009).** Cortisol also lowers the use of glucose by stimulating the lymphatic tissue, fat tissue and muscle to use free fatty acids instead of glucose for energy. At the same time, cortisol also reduces the effects of insulin. Therefore, insulin is unable to perform its regular function of maintaining normal glucose levels **(Piroli *et al.*, 2004).**

Hyperglycemia of animals group exposed to stressors in this work could be one of the possible mechanisms underlying the pathogenesis of diabetes mellitus with stress. Hyperglycemia, if persisted, may exhaust the β cells of Langerhans, leading to insulin deficiency and diabetes mellitus **(Radahmadi *et al.*, 2006).**

The present work showed significant increase in total serum cholesterol, serum triglycerides, and LDL with insignificant increase in HDL in adult male rats exposed to stressors. Plasma lipid response to stress varies from one stressor to another according to severity and combination of more than stressors and from organism to another **(Cohen*et al.*, 2006).** This hyperlipidemia may be due to the enhanced lipolysis that was secondary to an increase in circulating catecholamine levels. The increase in serum triglycerides may be attributed to increased hepatic triglyceride synthesis **(Willis *et al.*, 2009)**. **Vogel*et al.* (2003)** mentioned that stress induces increase in plasma levels of cholesterol and triglycerides. **Radahmadi *et al.* (2006)** mentioned that all types of plasma cholesterol levels increase in response to stress particularly LDL-C which constitutes the bad type of cholesterol. **Willis *et al.* (2009)** mentioned that total plasma cholesterol increased in the stressed animals maintained on standard laboratory diets.It is also possible that the increase in serum triglycerides was due to impairment in plasma triglyceride clearance, since catecholamine inhibits the enzyme lipoprotein lipase which determines the plasma triglyceride clearance **(Hücking *et al.*, 2003).**

Un and Myung (2014) observed that hyperlipidemia under chronic environmental stressors may be due to decreased insulin secretion. Insulin has an antagonistic effect upon catecholamine mediated lipolysis.

The present study showed significant increase in serum alanine transaminase (ALT), aspartate aminotransferase (AST), serum total protein, and serum albumin in adult male rats exposed to stress. This may be attributed to excessive release of such enzymes from the damaged liver cells into the blood circulation **(Maisa, 2012). Nayanatara *et al.* (2006)** mentioned that the increase in aspartate aminotransferase (AST) and alanine transaminase (ALT) in rats exposed to many chronic environmental stressors showed an intimate relation to the cell damage, necrosis, and increased the permeability of the cell membrane. Significant increase in serum total protein level in adult male rats of stressed groups. This may be due to catabolic effect of stress hormones especially glucagon and cortisol **(Alba *et al.*, 2011).** Increased insulin secretion under stress may play a role in the elevation of total serum protein. Insulin hormones increase the rate of protein synthesis, increase cellular protein, and enhance active transport of many of the amino acids into the cells to inhibit the protein catabolism **(Vanhorebeek and Van Den, 2004).**

Prevention of insulin release with somatostatin enhances the ability of cortisol to increase plasma gluconeogenic precursors (lactate, alanine, and pyruvate). Also, the anabolic effects of insulin partially block the catabolic effect of corticosteroids. So, increase in the total serum protein under stress may be due to increasing insulin concentration in plasma after administration of corticosteroids, or due to over-activity of the pancreatic cells in response to stress **(Elijah *et al.*, 2015).**

The present work showed significant hypo-albuminemia in adult male rats exposed to noise stress may be due to malnutrition; this also may denote kidney and liver affection by stress where this finding is usually associated with structure hepatic and kidney damage. It has been showed that, during the alarm reaction, the usual liver function tests reveal a marked hepatic insufficiency **(Willis *et al.*, 2009)**.

Impaired binding function of albumin has been demonstrated in end-stage liver disease. This and other functional disturbances of albumin may be related to oxidative stress which is believed to play an important role in the pathogenesis of liver failure as well as sepsis **(Manikandan *et al.*, 2006)**.

Ourhistopathological resultsin rats of stressed groups showed an increased lymphocytes and Kupffer cells in the portal areas, dilated congested portal vein and sinusoidal spaces, and vacuolated hepatocytes with pyknotic nuclei.Since Kupffer cells produce cytokines inflammatory mediators, these mediators may be released in response to an increase in sympathetic activity due to stress and leading to liver cell damage and necrosis **(Dawei *et al.*,2014).**

Exposure to stressors caused many changes in the liver tissue of rats. These changes include increased lymphocytes and Kupffer cells in the portal areas, dilated congested portal vein and sinusoidal spaces, and vacuolated hepatocytes with pyknotic nuclei. **Fatma *et al.* (2010)** stated that principal effect of stress on the liver is related solely to changes in hepatic blood flow specifically. This hypothesis suggested that emotional stress leads to vasospasm and centrilobular hypoxia, and ultimately to liver damage.

Highly affected endothelial lining of blood vessels of the liver tissue post-exposure to stress for a long time were observed by **Filip*et al.* (2004).** It was suggested that stress influenced hepatic blood flow by inducing vasospasm and centrilobular hypoxia, leading to liver damage **(Chida *et al.*, 2006).**

The present study showed significant increase in serum creatinine in adult male rats exposed to stress. This may result from kidney function impairment such as generation in the proximal convoluted tubules **(Senior, 2009)**. **Matsumoto *et al.* (2009)** recorded an increase in creatinine serum in rat exposed to stress which may be due to increase catabolism in muscle and tissue that appear to act as a stimulus to synthesis of more serum creatinine. According to **Agarwal (2005),** stressors lead to oxidative stress which contributes to renal injury. This injury seems to be predominantly localized to the renal proximal tubules, and this injury was realized in the destructed brush order of proximal convoluted tubules observed in the present study.

The histological damage might result from an increase in the process of lipid peroxidation and decreased activity of antioxidant enzymes of the body with the consequent damage of cellular membranes **(Sha *et al.*, 2015).** Exposure of rats to stressors showed several dystrophic changes in the kidney tissue compared with the control group. These changes included highly atrophied glomeruli, faintly stained cells and nuclei of the convoluted tubules with wide lamina of the distal ones, ruptured brush borders of the proximal ones, thickened arterial walls with branched and corrugated walls of the congested vein. According to **Agarwal (2005),** stresses led to oxidative stress which contributes to renal injury. This injury seems to be predominantly localized to the renal proximal tubules, and this injury was realized in the destructed brush borders of proximal convoluted tubules observed in the present study. Signs of improvement were observed in the kidney cortex of rats exposed to stressors and treated with the antidepressant drugs.

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تأثير ضغوط الزحام والضوضاء على وظائف الكبد والكلى فى ذكور الجرذان البيضاء البالغة

جمال أحمد شاور – شبل رمضان سماحة – عز الدين خليفة – أحمد شعبان عبد المنصف

قسم الفسيولوجيا الطبية – كلية طب الأزهر

خلفية البحث: التعرض للضغوط اليومية فى حياة الإنسان يسبب الكثير من المشاكل الصحية ويكون له رد فعل في جسم الإنسان ، ومن هذه الضغوط الإزدحام والضوضاء التى تؤثر على صحة وقدرة وكفاءة الإنسان.

الهدف من البحث: دراسة تأثير إثنين من الضغوط التى يتعرض لها الإنسان فى حياته اليومية وهما الازدحام والضوضاء على وظائف الكبد والكلى.

مواد وطرق البحث: أجرى البحث على ذكور الجرذان البيضاء البالغة. وقسمت الحيوانات إلى ثلاث مجموعات كالآتى.

- مجموعة ضابطة (عددها 12 جرذ).
- مجموعة تعرضت للضوضاء فقط أكثر من 90 ديسبل لمدة 30 يوماً (عددها 18 جرذ).
- مجموعة تعرضت للزحام فقط 30 يوماً (عددها 12 جرذ).

وكانت كثافة الجرذان فى المجموعة المعرضة للازدحام ٣ أضعاف المجموعة الضابطة، أما بالنسبة للضوضاء فقد تم تحضير خمسة أصوات من مصادر مختلفة. وقد تم وضع هذه الحيوانات تحت تأثير الزحام والضوضاء لمدة 30 يوماً، ثم تم أخذ الأمصال فى نهاية هذه المدة لتحديد مستوى القياسات الآتية:

- نسبة السكر بالدم.
- وظائف الكبد.
- وظائف الكلى.
- البروتين الكلي
- قياس الدهون.

أما بالنسبة للدراسة النسيجية والكيمانسيجية، فلقد أخذت أجزاء صغيرة من كبد وكلي الجرذان من كل المجموعات، وقد تم عمل قطاعات شمعية بالطرق المعروفة وصبغها بعدة صبغات.

نتائج البحث: عند التعرض إلى الضغط سواء كان زحام أو ضوضاء فقد كانت هناك زيادة إحصائية فى كل من نسبة السكر بالدم، ووظائف الكبد، ونسبة الكرياتينين، والبروتين الكلي، والدهون الكلية والثلاثية والكوليسترول منخفض الكثافة. وعلي الجانب الاخر كان هناك نقصا ذا دلالة إحصائية في نسبة الأليومين. وقد أدى التعرض لضغط الإزدحام والضوضاء إلى ظهور العديد من التغيرات النسيجية فى كبد الجرذان منها تحطم بعض الخلايا الكبدية، وتمدد الأوعية الدموية وإحتقانها، كما ظهر الإرتشاح الخلوي فى وحول المناطق البابيه. أما النسيج الكلوى فقد عانى من التضخم الشديد لجدار الأوعية الدموية، وخصوصا الشرايين التي إحتوت على كريات دموية متحللة، ومناطق نزف داخلية عديدة، وإتساع فجوات الأنيبيبات الملتوية البعيدة مع تحطم بعض خلاياه وتزايد خلايا كوفر.

الإستنتاج: ينصح بالإبتعاد عن مناطق الإزدحام والضوضاء ولو لفترات متقطعة فى حياة الإنسان لاستعادة الحياة النفسية الطبيعية لتخفيف التغيرات الفسيولوجية والنسيجية الناجمة عن التعرض لهذه الضغوط اليومية.