Histological, Histochemical and Biochemical Studies of the Effect of Chronic Exposure of Noise Stress on the Kidney of Albino Rats

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Abstract: Twenty-five adult male albino rats were utilized to study the effect of noise stress for short and long durations on the kidney structure and functions of albino rats. After one week (short duration) of noise exposure, the biochemical data revealed a significant increase in serum albumin accompanied by a significant increase of body and kidney weights. A significant decrease of urine output was also detected. The renal tufts of the glomeruli were enlarged with increased cellularity filling the Bowman's space. The lining epithelium of the convoluted tubules was slightly swollen. After two weeks of rest, most of the glomeruli and the tubular elements were more or less like those of the control group. After two weeks (long duration) of noise exposure, the biochemical data revealed the same results as those shown in the animals which were exposed to noise for a short duration. However, the body weight of the animals revealed a highly significant increase accompanied by a highly significant decrease in the urine output. The glomerular capillaries of renal corpuscles were swollen, lobulated with increase of the mesengial matrix. The epithelial cells lining renal tubules exhibited cloudy swelling and vacuolar degeneration. After two weeks of rest, the majority of renal glomeruli revealed extensive glomerular retraction and degeneration. The epithelial cells lining the renal tubules showed necrotic changes and their lumina appeared packed with renal casts and cell debris. The thickened basement membranes surrounding both the Bowman's capsules and renal tubules exhibited an intense PAS reaction in rats exposed to long duration of noise stress. After one week of noise exposure, mercury-bromophenol blue staining of the kidney revealed a high protein content in the form of an intense blue colouration of the intact brush borders of the proximal convoluted tubules.
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Key words: Histological, Histochemical, Biochemical Studies, Noise Stress, Kidney, Albino Rats.

Introduction

Recently, many new forms of stresses and pollutants appeared in the world. Noise is one of the most modern and important stressful stimuli encountered nowadays especially in industrialized societies **(WHO, 2010).**

Noise is typically defined as an unwanted sound or a combination of sounds that may adversely affect people. Noise can induce physiological harm or pathological damage. The mechanism of damage from noise has yet to be fully understood, but research has demonstrated a multitude of factors including increased oxidative stress, vascular changes **(Evans et al., 1998 ; PHSHP report, 2000).**

There have been multiple studies that have investigated the relationship between noise, blood pressure, and myocardial damage **(Babisch, 2003; Sangeeta et al., 2009; Bao, 2011).** Other studies investigated the relationship of noise and blood pressure and stress response of cardiovascular system **(Evans et al., 1998 ; Wolfgang & Irene 2009; Kao et al., 2010)**.

Numerous studies recorded different pathological changes in renal functions following different types of stress **(Ahmed & Mazher, 2006; Moussa, 2005; Siems et al., 2001; Danielski et al., 2003)**. However, a little attention was paid to throw the light on the histological alterations that may occur in the kidney after chronic exposure to noise stress. Thus, the present study aims to determine the effects of exposure to noise stress for two different durations on the kidney histology, histochemistry and some biochemical parameters.

Material and Methods

Forty-two adult male albino rats (Rattus norvegicus), each weighing 130 $^{+}$ 10 grams were utilized in the present study. The animals were housed in specially designed wooden cages and were kept under similar constant airflow and at a convenient temperature during the whole period of experimentation. Rats were fed on a balanced laboratory diet, while water was allowed ad libitum.

The animals were divided into three groups, as follow **:**

Group I: This group included 6 animals used as control.

Group II: This group included 18 animals exposed to noise stress by using a special record to produce a

continuous noise of 100 decibels for 8 h / day for one week.

Group III: This group included 18 animals exposed to the same dose of noise stress (100 decibels for 8 h / day) for two weeks.

The last two groups were subdivided into two subgroups according to the time of sacrifice, where the animals of first subgroup were sacrificed immediately after the end of the period of noise exposure while animals of the second subgroup were sacrificed two weeks following the end of noise exposure.

Biochemical studies:

The body and kidney weights were recorded just before the beginning of study (day 0) and at the termination of the study (29 days).

Before scarifications, rats were individually housed in metabolic cages and urine was collected for estimating its volume (output/day).

Biochemical analysis of serum albumin was estimated according to **Drupt (1974).**

Histological study:

After fast dissection, the kidneys were rapidly excised and quickly trimmed into small pieces that were fixed in Carnoy's fixative and processed until paraffin blocks. Sections of 6 µm were prepared and stained with Harris haematoxylin and eosin **(Drury & Wallington, 1980)** to illustrate the histological alterations of the kidney.

Semi-quantitative analysis:

Histological alterations were scored in 100 fields/each group. Ten of them were randomly selected at 4oo magnification/each examined animal. The results were scored as percentage of the damaged tubules in the field examined as follows:

Mild damage: areas of tubular damage <25%.

Moderate damage: areas of tubular damage equalling 25%.

Severe damage: areas of tubular damage >50 %.

The presence of luminal debris, hyaline casts, cytoplasmic vacuolization and nuclear changes were used as evidence on tubule damage **(Shah & Walker, 1988).** Semiquantitative measurements were carried out using image analyzer (Super Eye-Heidi Soft), Diagnostic Pathology Department, Jubail Royal Hospital, Kingdom of Saudi Arabia.

Histochemical study:

For the histochemical study, small pieces of kidney were fixed in Carnoy's fluid and then processed and sectioned to get 6 µm thick paraffin sections. Then, the following methods were applied:

1. Periodic acid Schiff's (PAS) technique for the demonstration of polysaccharides **(Hotchkiss, 1948).** The nuclei counterstained by Harris haematoxylin.

2. Mercury bromophenol blue technique for the demonstration of total protein **(Mazia et al., 1953).**

Statistical analysis:

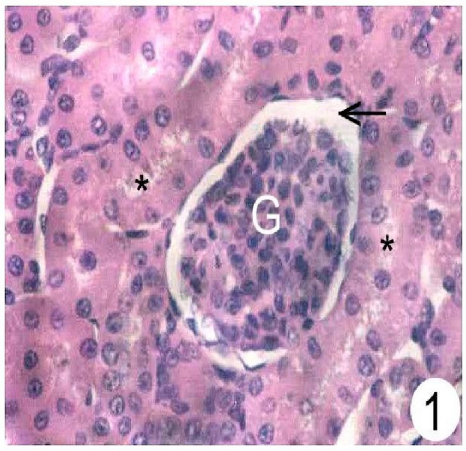
The statistical data included body weight, kidney weight, urine output and serum albumin level**.** Data were expressed as arithmetic mean $^{+}$ standard deviation (SD) .Student t-test was used to test the significant change of each parameter of exposed animal in comparison to control group. Statistical analysis of the data was performed by Med Cale software for medical statistics **(Schoonjans et al., 1995).**

Results

Histological Observations:

Group I (Control):

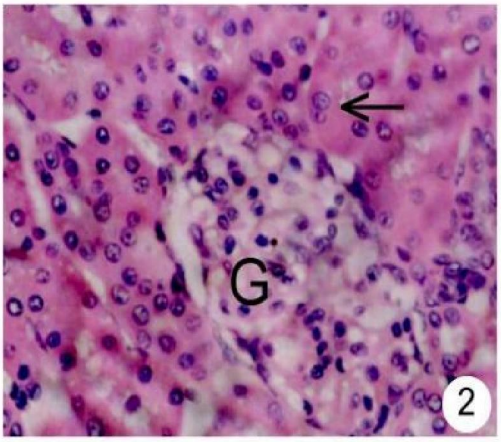
The glomeruli of the control rats showed a normal histological picture, where both visceral and peripheral layers of renal capsules were clearly identified and the Bowman's spaces were clearly visible. The lining epithelia of both the convoluted and collecting tubules were normal in shape, having granular acidophilic cytoplasm and normal nuclei **(Fig. 1).**



(Fig.1): A photomicrograph of a kidney section a control rat showing the normal histological configuration of the kidney. The photo shows a glomerulus **(G)**, Bowman's space **(arrow)** and the lumina of the proximal convoluted tubules **(*)**. **(H&E Stain X400)**

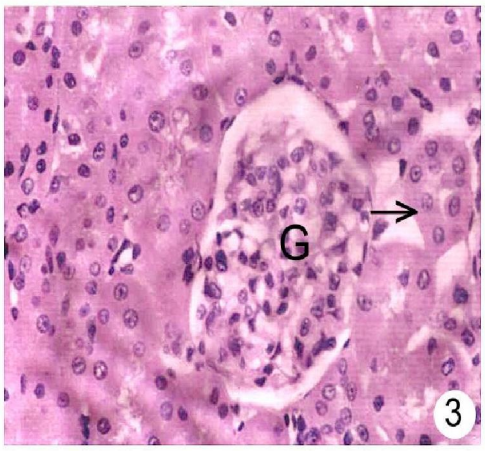
Group II*:*

In animals of subgroup A which were sacrificed immediately after the end on noise exposure, showed that the glomerular tufts were enlarged with increased cellularity to fill the Bowman's space. The lining epithelium of the convoluted tubules was slightly swollen and showed smudgy appearance. Some of the cells of such tubules lost their boundaries and brush borders **(Fig. 2).**



(Fig.2): A photomicrograph of a kidney section of a rat after one week of exposure to noise stress **(of subgroup II A)** showing an enlarged glomerular tuft **(G)** filling Bowman's space and the swollen epithelial cells **(arrow)** lining the proximal convoluted tubules. **(H&E Stain X400)**

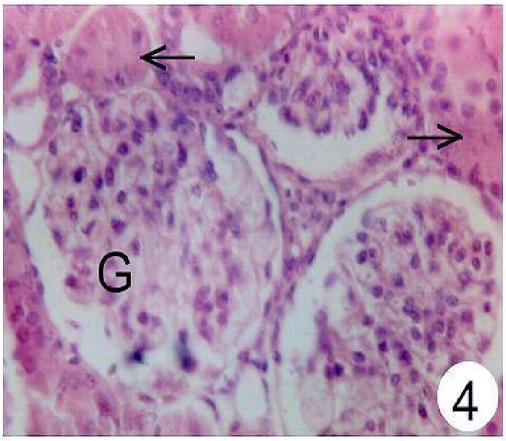
In animals of subgroup B, most of the glomeruli and the elements of the kidney tubules were more or less similar to those of the control group. However, few areas of renal cortex revealed some dilatation of Bowman's spaces **(Fig. 3).**



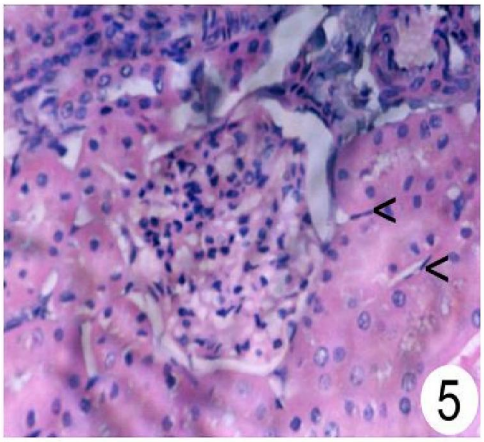
(Fig.3): A photomicrograph of a kidney section of a rat exposed to short duration of noise stress **(of subgroup II B)** showing a more or less normal glomerulus **(G)** and epithelial cells lining the convoluted tubules **(arrow). (H&E Stain X400)**

Group III*:*

In animals of subgroup A, the histological examination revealed high cellularity, lobulated and congested glomeruli. The glomerular capillaries of renal corpuscles were swollen and displayed increase of the mesengial matrix that showed adhesion to the visceral cells of Bowman's capsules. The epithelial cells lining the convoluted and collecting tubules of both cortex and medulla exhibited cloudy swelling and vacuolar changes **(Fig. 4).** Moreover the epithelial cells lining the proximal convoluted tubules lost their boundaries and had damaged brush borders. Numerous compressed endothelial cells were observed in the interstitial spaces inbetween the renal tubules **(Fig. 5).**



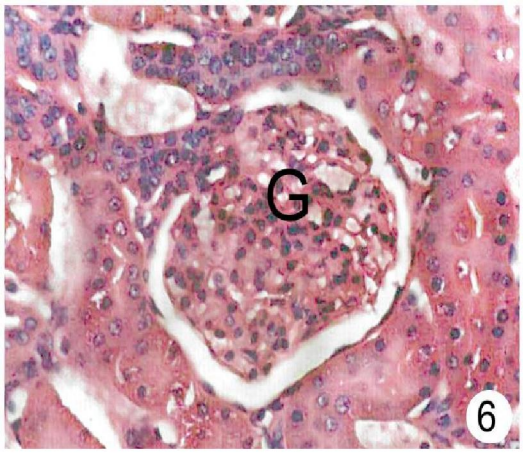
(Fig.4): A photomicrograph of a kidney section of a rat exposed to long duration of noise stress **(of subgroup III A)** showing swollen, lobulated and congested glomeruli **(G)** and swollen epithelial cells lining the convoluted tubules **(arrow). (H&E Stain X400)**



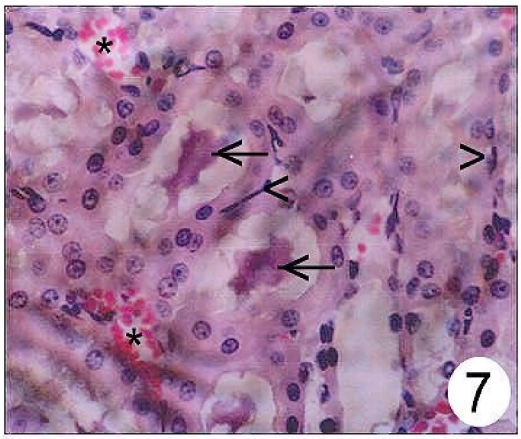
(Fig.5): A photomicrograph of a kidney section of a rat exposed to long duration of noise stress **(of subgroup III A)** showing that the epithelial cells lining the proximal convoluted tubules lost their boundaries and numerous compressed endothelial cells **(arrowheads)** are shown inbetween the renal tubules. **(H&E Stain X400)**

In animals of subgroup B, the majority of renal glomeruli revealed extensive glomerular retraction or atrophy with extremely widened Bowman's spaces**.** Also, some glomeruli showed local degeneration**.** The epithelial cells lining the

convoluted and collecting tubules were partially detached and showed necrotic changes **(Fig. 6)**; their lumina appeared packed with renal casts and cell debris. Numerous compressed endothelial cells, blood cells and haemolysis were observed between the renal tubules **(Fig. 7)**.



(Fig.6): A photomicrograph of a kidney section of a rat exposed to long duration of noise stress **(of subgroup III B)** showing a retracted glomerulus **(G)** and a wide Bowman's space **(arrow). (H&E Stain X400)**



(Fig.7): A photomicrograph of a kidney section of a rat exposed to long duration noise stress **(of subgroup III B)** showing the collecting tubule's lumina packed with renal casts **(arrows)** , numerous compressed endothelial cells **(arrowheads)** and blood cells between the renal tubules(*).**(H&E Stain X400)**

Semiquantitative results:

Table (1): Semi-quantitative analysis of renal histological alterations.

Group / Parameter	control	Group II a	Group II b	Group III a	Group III b
No Change	95	77	92	21	18
Mild Change in the tubule	5	20	6	61	22
Moderate Change in the tubule	0	3	2	14	31
Severe in the tubule	0	0	0	4	29
Total	100	100	100	100	100

Histochemical results:

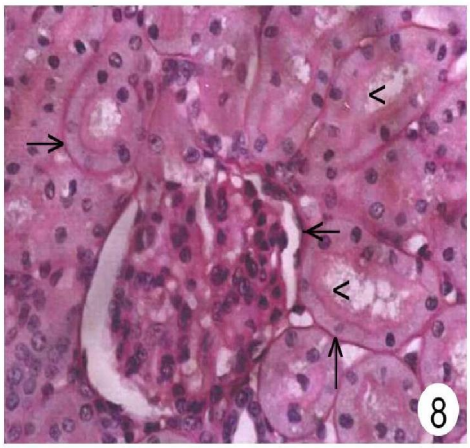
PAS reaction:

Group I (Control):

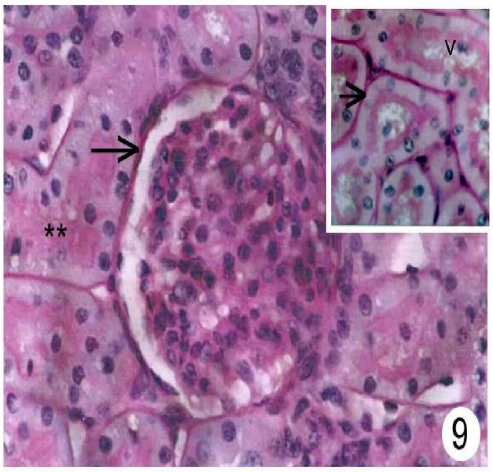
The PAS-stained sections revealed purple colour of the brush border of the proximal convoluted tubules and basement membranes of both Malpighian corpuscles and all the renal tubules **(Fig. 8)**.

Group II:

In animals of subgroup A, many of the proximal convoluted tubules appeared with intact brush borders; however some of them showed that PAS-positive brush borders were partially lost. A slight increase was observed in the thickness of the basement membranes surrounding both the Bowman's capsules and the renal tubules **(Fig.9)**.

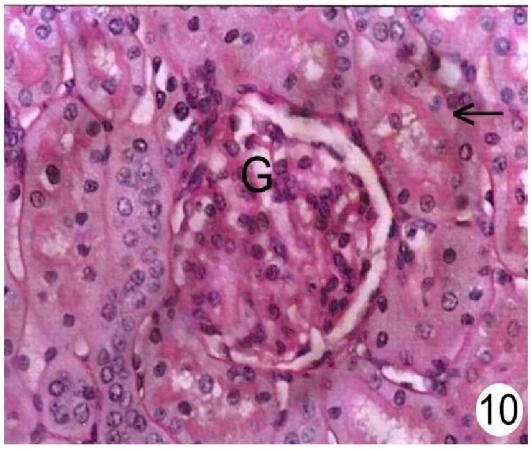


(Fig.8): A photomicrograph of a kidney section of control rat showing the normal PAS-positive brush border of the proximal convoluted tubules **(arrowheads)** and basement membranes **(arrows)**. **(PAS reaction counter with H&E stain X400)**



(Fig.9): A photomicrograph of a kidney section of a rat after one week of exposure to noise stress **(of subgroup II A)** showing intact brush borders of some proximal convoluted tubules **(**);** others lost brush borders **(arrowheads)** and display slightly thickened basement membranes **(arrows)**. **(PAS reaction counterstained with H&E stain X400)**

In animals of subgroup B, the PAS-positive materials of both the renal glomeruli and the renal tubules elements appeared more or less similar to those of the control group **(Fig. 10)**.



(Fig.10): A photomicrograph of a kidney section of a rat exposed to short duration of noise stress **(of subgroup II B)** showing a more or less normal distribution of the PAS-positive materials of both the renal glomeruli **(G)** and the renal tubules **(arrow). (PAS reaction counterstained with H&E stain X400)**

Group III:

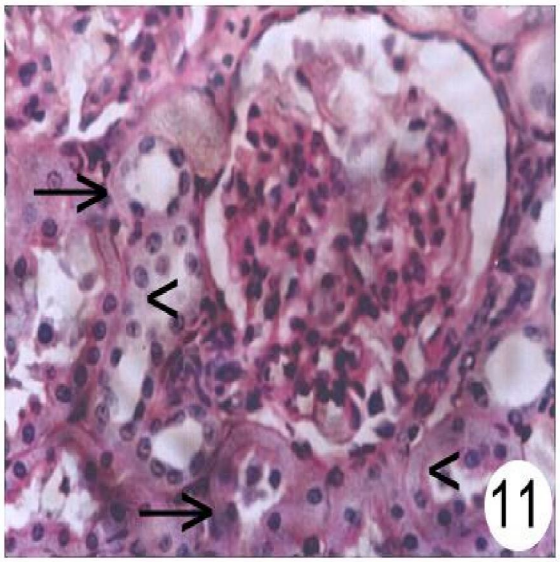
In animals of subgroup A, the PAS positive reaction showed that the brush borders of most of the proximal convoluted tubules were completely lost and their basement membranes appeared faintly stained and ill-defined as compared with the control sections **(Fig. 11)**.

In animals of subgroup B, the PAS-stained sections showed a strong PAS reaction of the thickened basement membranes surrounding both the Bowman's capsules and the renal tubules as compared with the control sections **(Fig.12)**. Moreover, considerable deposits of hyaline materials were detected in the collecting tubules and/or interstitial tissues **(Fig.13)**.

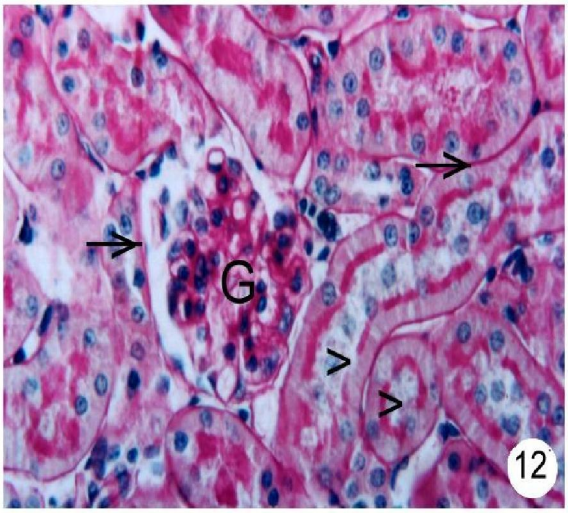
Mercury bromophenol blue staining:

Group I (Control):

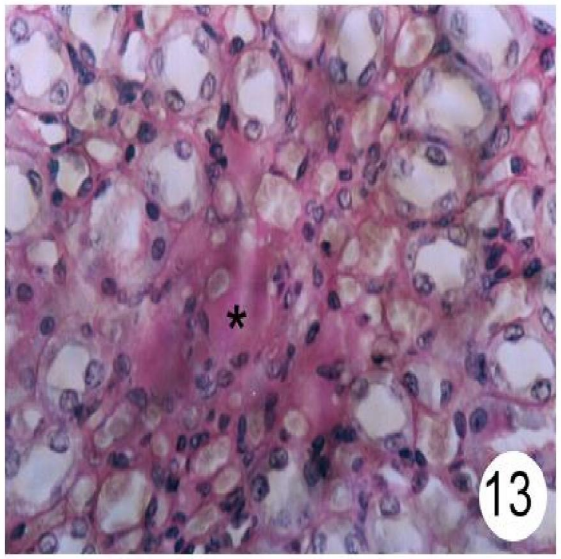
The renal glomeruli displayed moderate protein content. The epithelium lining the renal tubules display a dark blue colouration of proteinic particles diffused homogeneously in the cytoplasm of their cells **(Fig. 14).**



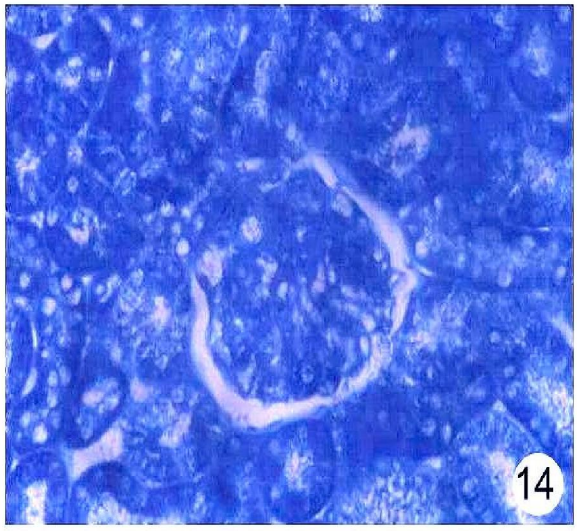
(Fig.11): A photomicrograph of a kidney section of a rat after two weeks of exposure to noise stress **(of subgroup III A)** showing the degenerated brush borders of most of the proximal convoluted tubules **(arrowheads)** and ill-defined basement membranes **(arrow). ((PAS reaction counterstained with H&E stain X400)**



(**Fig.12):** A photomicrograph of a kidney section of a rat exposed to long duration of noise stress **(of subgroup III B)** showing a strong PAS-reaction of the thickened basement membranes surrounding both the Bowman's capsules **(arrow)** and the proximal convoluted tubules **(arrowheads)** which lost the brush borders**. (PAS reaction counterstained with H&E stain X400)**



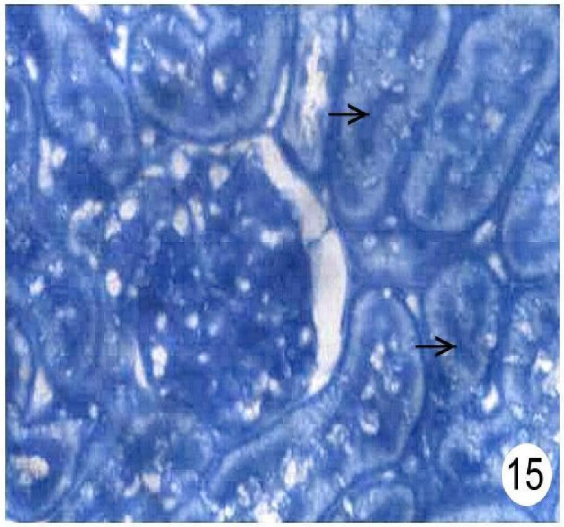
(Fig.13): A photomicrograph of a kidney section of a rat exposed to long duration of noise stress (**of subgroup III B)** showing deposits of hyaline materials **(*)** in collecting tubules and/or interstitial tissues. **(PAS reaction counterstained with H&E stain X400)**



(Fig.14): A photomicrograph of a kidney section of a control rat showing the normal distribution of total protein contents**. (Mercury bromophenol blue method X400)**

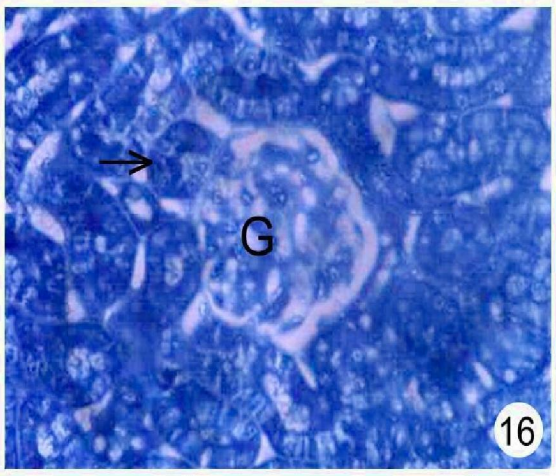
Group II:

In animals of subgroup A, the glomerular tufts and the epithelial cells lining the renal tubules showed a slight decrease in their proteinic contents. Many of the proximal convoluted tubules appeared with intact brush borders revealing an intense dark blue colouration **(Fig. 15).**



(Fig.15): A photomicrograph of a kidney section of rat after one week of exposure to noise stress **(of subgroup II A)** showing a slight decrease in their proteinic contents and the intact brush borders **(arrow)** of the proximal convoluted tubules, these display an intense blue colour. **(Mercury bromophenol blue method X400)**

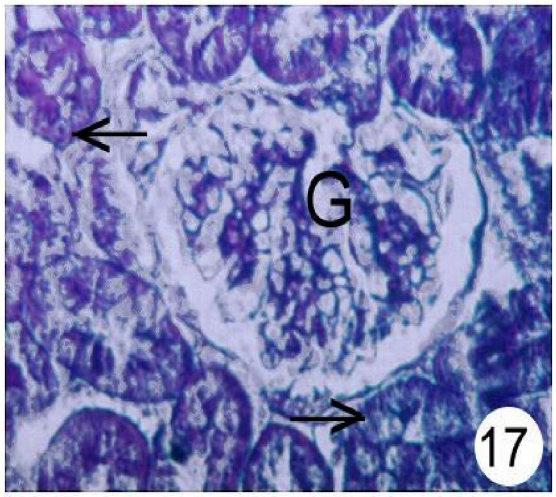
In animals of subgroup B, the stained proteinic materials of both the renal glomeruli and the renal tubules elements appeared more or less like those of the control group **(Fig. 16).**



(Fig.16): A photomicrograph of a kidney section of a rat exposed to short duration of noise stress **(of subgroup II B)** showing almost normal distribution of the positive total protein materials of both the renal glomeruli **(G)** and the renal tubules **(arrow). (Mercury bromophenol blue method X400)**

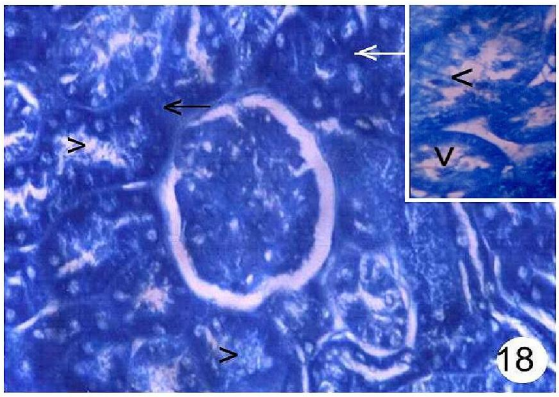
Group III:

In animals of subgroup A, a marked decrease in the proteinic contents was observed in both the glomerular tufts and the epithelial cells lining the renal tubules. These cells appeared faintly stained, vacuolated and pocessed dark blue intensely-stained pyknotic nuclei **(Fig.17)**.



(Fig.17): A photomicrograph of a kidney section of a rat after two weeks of exposure to noise stress **(of subgroup III A)** showing a marked decrease in the proteinic contents in both the glomerular**(G)** tufts and the vacuolated epithelial cells lining the renal tubules **(arrow). (Mercury bromophenol blue method X400)**

In animals of subgroup B, the sections revealed a strong stainability with bromophenol blue in the atrophied epithelial cells line both the collecting and convoluted tubules. Darkly blue stained granular casts and cell debris were accumulated in the lumina of the proximal convoluted tubules and in the interstitial tissue **(Fig.18)**.



(Fig.18): A photomicrograph of a kidney section of a rat exposed to long duration of noise stress **(of subgroup III B)** showing a strong stainability of total protein contents in the atrophied renal epithelial cells **(arrows) and** granular casts and cell debris in the lumens of renal tubules **(arrowheads)** . **(Mercury bromophenol blue method X400)**

Table (2): The biochemical statistical data of rats exposed to noise stress.

Group Parameter	control	*Group II* a	*Group II* b	*Group III* a	*Group III* b
body weight/g	110 ± 10	127 ± 6*	119 ± 8	139 ± 67**	143 ± 2**
Kidney weight/g	1.28 ± 0.4	1.38 ± 0.3*	1.23 ± 0.9	1.51 ± 0.4**	1.1 ± 0.2*
urine out put/ml/100g b. w./day)	5.4 ± 0.5	4.2 ± 0.4*	5.1 ± 0.2	4.6 ± 0.3*	4.4 ± 0.3*
Albumin g/dl	3.1± 0.5	2.2 ± 0.4*	3.2± 0.1	2.0± 0.4*	1.51± 0.2**

All data are Mean value± Standard error. N= 6 animals of each group.
* Significant at ($p < 0.05$). ** Highly significant at (< 0.01).

Discussion

Although numerous investigations have been carried out on different pollutants, noise stress is the least illustrated or understood. The different pathological changes produced by noise are governed by sympathetic nervous system and appear to be similar to those produced by other physical and chemical stresses **(Dejoy 1984; Kjellberg 1990).**

Initially under the conditions of the present study, both glomeruli and renal tubules seemed to be sensitive to the noise stress. However, the renal tubules largely returned to the normal histological pattern in rats which were exposed to noise for one week (short duration) after two weeks of rest following the end of exposure. This recovery probably occurred because of active tubular regenerative processes, whereas the glomeruli lesions persisted and evolved towards some membranous changes in case of long exposure periods **(Marino & Becker 1977).**

The reversibility of renal changes was not detected when exposure to noise stress has been omitted for two weeks. This is based on, multiple doses of noise exposure at a constant value of intensity is not completely reversible and appeared more or less to be permanent due to severe degenerative changes which are difficult to be completely recovered.

Another explanation is that prolonged exposure to stressful stimuli may have caused prolonged rise in blood pressure leading to circulatory adaptation and permanent hypertension **(Andren et al., 1980; Pereet, 1982; Van Kempen, et al., 2002).**

In view of the present results, it was found that the experimental animals which were exposed to noise stress for two weeks (long duration) did not reveal signs of reversibility after two weeks of rest following the end of exposure. The kidney's tissues still showed advanced degree of glomerular shrinkage or collapse, renal tubules damage, in addition to the presence of interstitial blood cells and some areas of haemolysis.

Nekhoroshev & Glinchikov (1991) recorded micropunctate hemorrhages under the effect of white noise stress for 7 days.

Also, **Van Dijkken, et al. (1992)** reported that animals exposed to chronic stress showed congestion and haemorrhage in all the body organs due to permanent rise in arterial blood pressure. Thus, the present histological alterations-that attributed to a sustained elevation of blood pressure-may be associated with the risk of hypertension.

Some other studies have suggested that occupational noise exposure is associated with a sustained elevation of blood pressure **(Nawaz &Hasnain 2010 ; Tomei et al., 2010)** or with a higher risk of hypertension **(Sbihi et al., 2008),** but other studies have not revealed any significant interaction **(Talbott et al., 1985 ; Inoue et al., 2005).**

Andren et al. (1979) proved that noise influences the renal handling of salts. This effect was believed to be due to an isolated secretion of oxytocin without concomitant secretion of vasopressin. Moreover, **Koepoke, et al., (1988)** and **Koepoke (1989)** stated that environmental stress increases renal sympathetic nerve activity and decreases urinary sodium excretion in both normotensive and hypertensive rats through increased renal tubular reabsorption of sodium and water.

Other studies postulated the cause of renal changes after noise exposure; yet, the most acceptable one was the occurrence of hypertension. This hypertension is believed to be due to the elevation of suprarenal hormones level.

Noise as a stressor leads to increased release of the stress hormones including epinephrine, nor-epinephrine and adrenal steroids**,** thus, causing elevation of sodium- water retention and increase the arterial blood pressure **(Andren et al., 1979; Brandenberger et al., 1980; Lai &Carino, 1990).** Moreover, **Babisch (2003) and Seema et al. (2010)**

recorded that acute and chronic exposure to noise stress causes changes in neuroendocrine, psychological and behavioral functions.

Swollen renal tubules with intact or damaged brush borders revealed a slight decrease in PAS reaction. However, the thickened basement membranes surrounding both the Bowman's capsule and renal tubules showed intense PAS reaction.

It is well known that renal blood flow is decreased during stress and it is equally recognized that this ischemia is due to renal arterial vasoconstriction **(Dastron &Deutsches, 1975).** Thus, the advanced degree of glomerular shrinkage or collapse and the damages of the renal tubules may also be attributed to ischemia.

Hypoxia, causing decrease in the cellular ATP and associated with an increase in cAMP, could stimulate the phosphorylase enzyme activity responsible for glycogenolysis in order to maintain the cell's energy sources by generating ATP from glycogen **(Cotran et al., 1994).**

Moreover, the hormonal changes reported on exposure to noise stress could cause increased secretion of adrenaline and noradrenaline which in turn increases glycogenolysis and / or inhibits glycogenesis **(Dixey&Rein, 1982)**

The biochemical measurements of the present study showed that both the relative body and kidney weights of noise exposed animals for one week displayed a significant increase ($p < 0.05$) than that of the control group. These increased values gradually returned to the normal level after two weeks of rest. However the relative body and kidney weights of noise exposed animals for two weeks showed a highly significant increase ($p < 0.01$) than that of the control group. Such increases of body and kidney weights seemed to be proportional with the decreased levels of urine output of the same animal groups. These changes may also be attributed to the elevation of blood pressure which increases the water and sodium retention.

On the contrary, the kidney weight did not return to the normal value after two weeks of rest in animals which were exposed to noise to a long duration but showed a significant decrease ($p < 0.05$) as compared to the control rats. Also, the urine output still revealed a significant decrease ($p < 0.05$) value as compared with that of the control rats. These changes may be attributed to the prolonged elevation of blood pressure which in turn affects the kidney tissues leading to renal atrophy. Similar results were recorded by **Bennett et al. (1996)** in cases of renal toxicity. They related these changes to tubular atrophy and tubule-interstitial ischemia.

The advanced degrees of glomerular and tubular damage depicted in the present study support this explanation, where the glomerular injury was proportionate to the severity of the hypertension **(Fujihara et al., 1994).**

The increase of thickness of basement membranes depicted in the present study may be attributed to glomeruli and renal tubules shrinkage or collapse, as the same amount of basal lamina came to accumulate in a smaller volume of retracted tissues **(Ghadially, 1982).**

The biochemical measurements of serum albumen showed significant decreases ($p < 0.05$) in both animals exposed to noise stress for short and long durations. This decreased levels accompanied with a significant decrease in the body weights of animals of the same groups. This significant decrease ($p < 0.05$) was also accompanied with a similar decrease in the urine output.

The serum albumin level returned to the relative normal value after two weeks of rest in animals exposed to noise for short duration. However, excessive loosing of albumin was recorded in animals exposed to noise for long duration and showed highly significant decrease values ($p < 0.01$) as compared with the control group.

In this respect, **Howie (1986)** suggested that the glomerular adhesions with Bowman's capsules are secondary to the lack of proteins or to some factors associated with their synthesis. Moreover, **Lawrence& Brewer (1982)** found that increased albumin glomerular filtration was shown to cause fusion of glomerular epithelium in female rats with hyperalbuminurea. They postulated that protein droplets acts by stimulating glomerular epithelial cells endocytosis with the result that these protein droplets accumulate within the epithelial cells so causing marked cytoplasmic swelling which forces the glomerular foot processes to spread out and fuse together.

Glassock (1985) reported that the glomeruli from heavily proteinuric rats showed mesengial cells hypercellularity and mesengial matrix expansion. Thus, the glomerular adhesion may be attributed to certain damage of the glomerular cells which may lead to increased influx of macromolecular substances into glomerular mesengium causing overload of mesengial matrix. So, **Neill et al. (1991)** and **Bains et al.(1997)** stated that glomerular injury can be identified functionally as proteinuria, particularly in the presence of high molecular weight proteins which are normally non-filterable. Thus, whenever there is heavy proteinuria, the glomerular epithelial cells show dramatic morphological changes which clearly demonstrate changes in cell adhesion.

The protein staining of the rat's kidney after one week of exposure to noise stress revealed an intense dark blue colouration of the intact brush

borders; this supports the previous opinions. The biochemical test of serum albumin also supports this opinion and indicates the increase of protein losing. Thus, in view of the present study, the histological lesions and histochemical alterations induced by noise stress were in good agreement to the biochemical measurements.

In conclusion, the semiquantitative analysis of renal histological alterations observed in the present study revealed the reversibility of renal changes when the exposure has been emitted for short duration. However, as exposure to noise was emitted to long duration, renal changes appear to be severe and permanent. It is directly and irreversibly affects the renal functions and causes permanent histopathological changes. However, the short duration of exposure insignificantly affects the renal functions and causes temporary and reversible histological alterations. It is recommended that all workers exposed to noise must avoid the continuous exposure to acute noise stress for long durations. Also, they must be periodically subjected to check up for their renal function tests, blood pressure and educated about the value of using ear protectors.

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