

AIM OF THE WORK

This study aims to elucidate the physical properties and the predominant frequency of different kinds of noise obtained from different sources (traffic signals noise, industrial factory machine noise, and bread making machine); and studying its effects on mice. This study also dealt with the biochemical effects of noise on blood and brain and was confirmed by histological examination of mice's tissue.

MATERIALS AND METHODS

The following chapter describes the methodology used to study the non- auditory effects of the noise on mice as an experimental model.

The work plan includes using of three noise sources then demonstrating the predominant frequency (*) for each source, mice were exposed to each noise source or its predominant frequency to detect their effects.

The noise sources were recorded at:

1. Egyptian Copper factory.
2. Traffic light signal.
3. Furnace for bread backing.

The effects of these noise sources and the corresponding predominant frequency on mice groups were measured through:

1. Biochemical studies.
2. Behavioral study.
3. Histopathological studies.

N.B: For each recorded noise source, the most repeated pattern of the noise waves was selected as representing noise in this area.

The equipments used for carrying out this study are described below. The various performances of measurements are explained. The entire experimental setup for application of the three different noise attributes is enumerated and the data collected is described.

I. Materials:

I.1. Recording system:

Laptop internal microphone was used to record noise source.

I.2. Anechoic chamber:

Homemade 50 x 50 x 50 cm³ plywood box was made, lined with polyurethane foam (Fig.34) from all sides. The chamber contains illumination system and ventilation system.

(*) By the predominant frequency: we mean that frequency posses (holds) the highest amplitude in the selected pattern.

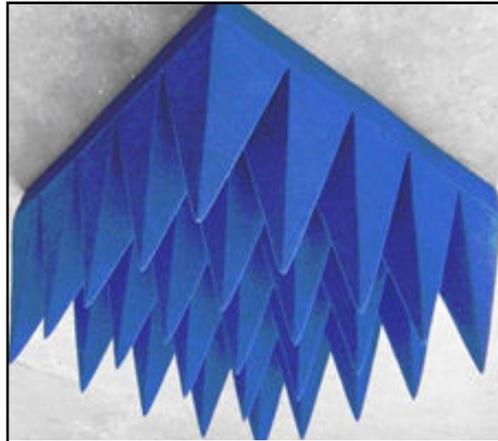


Fig.34: Sector of polyurethane foam used as a sound insulator.

I.3. Amplifier:

Public address amplifier (PA), Model: FUS-432RFL (Made in China) was used, with the following specifications: Root mean square output signal (RMS) =15W, and internal resistance = 4 Ω . The distortion value: < 0.5. Frequency response: 30 Hz- 18 KHz. Compatible with CD- VCD- DVD- Mp3- PC tunes.

I.4. Loud speaker:

15 W RMS loud speaker was installed on one side of the anechoic chamber, driven by the PA amplifier that can transmit the recorded specified noise with the required dB value.

I.5. Sound Level meter:

Noise level was measured by a SLM (Fig.35) (Lutron electronic enterprise Co., LTD.) Model: SL-4010, 35 to 130 dB, 3 ranges, Data hold, an instrument which responds to sound in approximately the same way as the human ear and gives reproducible measurement of sound level, Fig 36.

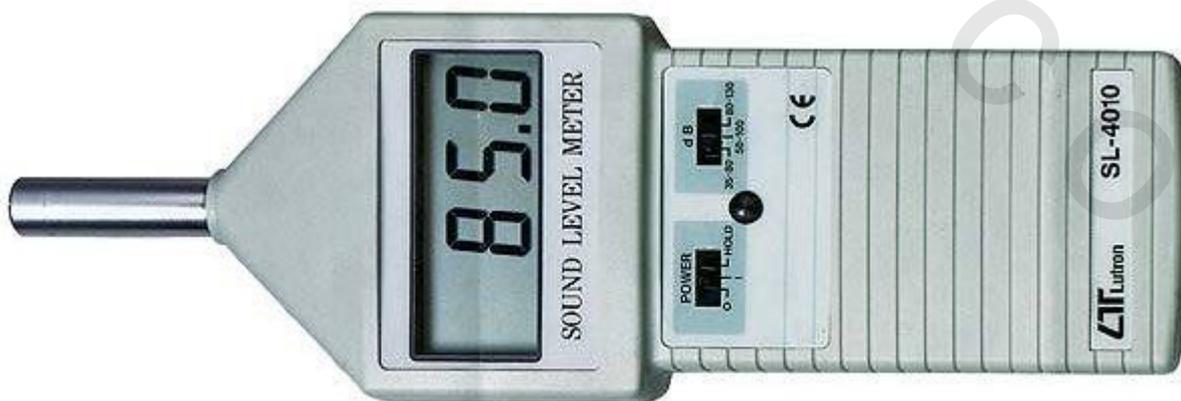


Fig.35: Sound level meter.

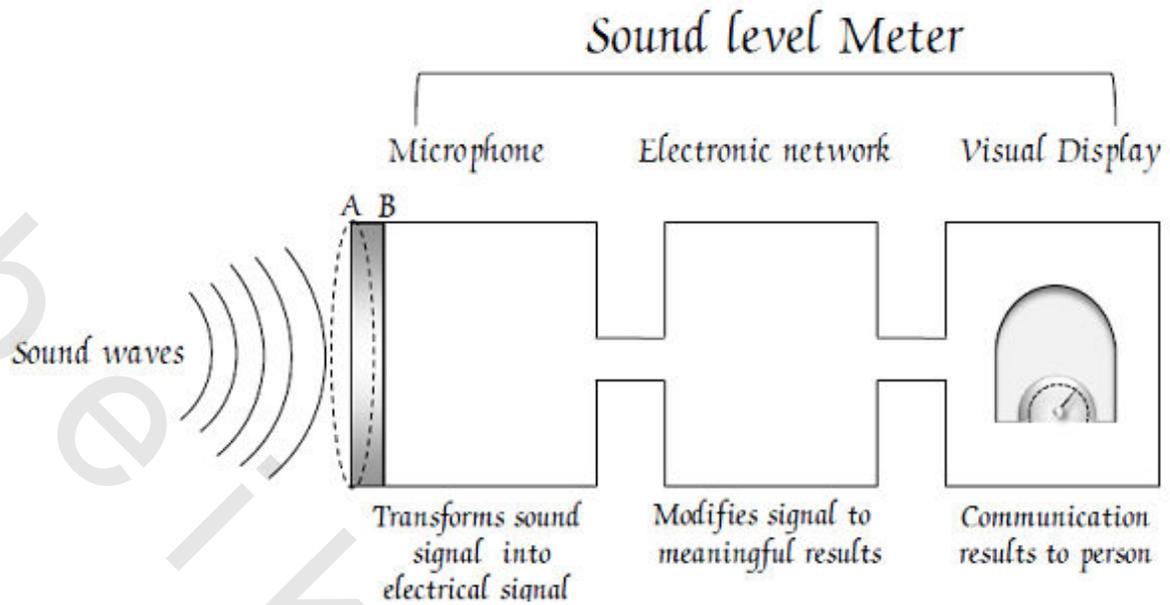


Fig.36: Sound level meter mode of action.

I.6. Study sites:

After a general survey of the city Alexandria Egypt, with special reference to the noise level. Noise-level was monitored in free flowing traffic condition four times a day, i.e. 6–7 A.M., 10–11 A.M., 5–6 P.M. and 9–10 P.M. According to the medium noise intensity, the recorded noise selected place was at $31^{\circ}12'19.3''N$ $29^{\circ}55'38.6''E$ at the time 10-11 AM. The Egyptian copper factory is at Hagar Elnawatya Street, Alexandria city (blanking press machine), and the baking furnace located at Alprince Street, Al-Ebrahymia, Alexandria city.

I.7. Selected sounds:

The recorded voices were analyzed using Audacity software program (version 2.0.5) <http://audacity.sourceforge.net>. Repeating area of the sound waves with the full characterization was obtained through the software analysis.

I.8. Selection of the predominant frequency of the used sound pattern:

The predominant frequency with its intensity in each sound pattern was obtained. The True, RTA software, (Real Time Audio Spectrum Analyzer) version 3.5 was suitable for such calculation.

I.9. Experimental sound:

The selected sound pattern as well as the predominant frequency was repeated to cover the exposure time applied to mice.

I.10. Mice groups:

130 mice weighted 25-30 gm were purchased from the animal house, Medical Research Institute, Alexandria University. The mice were kept in comfortable cages, well air conditioned; light system of 12 hr day/12 hr dark, food and water will be allowed freely. Use of experimental animals in the study was carried out in accordance with the ethical guidelines of Medical Research Institute Alexandria University (Appendix 2, Guiding Principles for Biomedical Research Involving Animals, 2011). The mice were divided into 13 groups; 10 mice were served as control while the rest 120 mice were divided equally into 12 exposed groups. The division process was completely at random.

I.11. Statistical analysis: ⁽¹⁵⁴⁻¹⁵⁵⁾

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. Quantitative data were described using range (minimum and maximum), mean, standard deviation and median. The distributions of quantitative variables were tested for normality using Kolmogorov-Smirnov test. If it reveals normal data distribution, parametric tests was applied. If the data were abnormally distributed, non-parametric tests were used. For normally distributed data, comparison between the studied groups was analyzed using F-test (ANOVA) and Post Hoc test (Scheffe). For abnormally distributed data, Kruskal Wallis test was used to compare between different groups and pair wise comparison was assessed using Mann-Whitney test. Significance of the obtained results was judged at the 5% level.

II. Methods:

II.1. Methods of using audacity software:

II.1.1. Plot spectrum:

Audacity software was used to record specified noise. Then we dealt with the selected audio (which is a set of sound pressure values at points in time) and converted it to a graph of frequencies (the horizontal scale in *Hz*) against *amplitudes* (the vertical scale in *dB*).

This is shown in the next example (Fig.37) followed in elucidating spectrum is of the word 'Audacity' spoken aloud, Plots are made using a mathematical *algorithm* known as a *Fourier Transform* or *FT*.

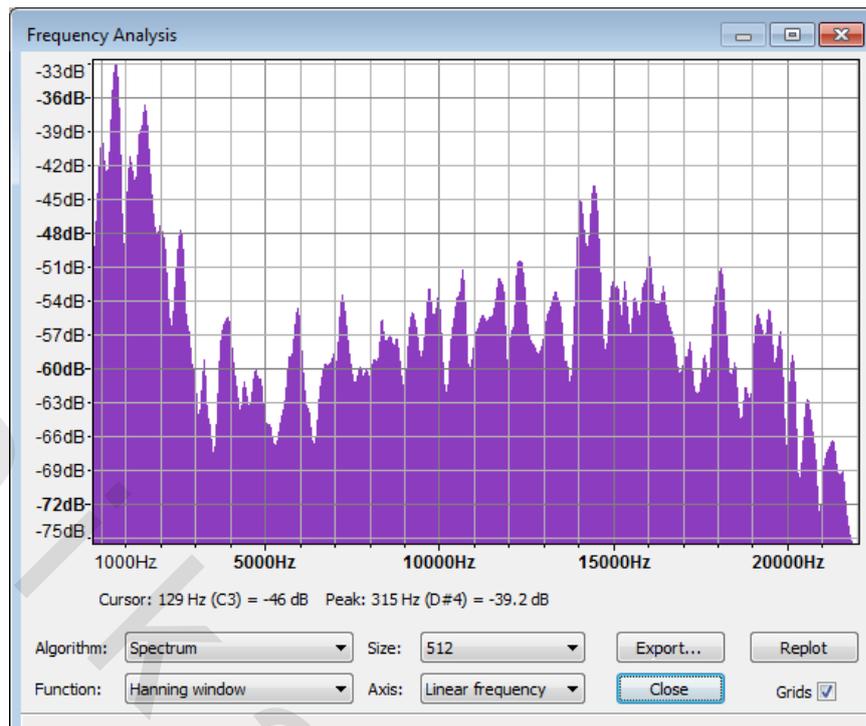


Fig.37: Spectrum is of the word 'Audacity' spoken aloud.

II.1.2. Standard autocorrelation:

These options measure to what extent the sound repeats itself. This is done by taking two copies of the audio and moving one forward by one sample. The two copies are then multiplied together, and all the values added up. This is repeated for two samples difference and so on, up to the number of samples in the size option. This gives a small result if the waveform is random (for example, noise) and a large result if it is repetitive (like a musical note). By looking at the peaks in the plot, the key frequencies present can be determined even if there is a lot of noise.

II.1.3. Cepstrum:

The cepstrum of an audio signal is related to the spectrum, but presents the rate of change in the different spectrum bands. It's particularly useful for properties of vocal tracks and is used, for example, in software to identify speakers by their voice characteristics.

II.2. Sounds

Sound pattern selection, Figs 38-40 show the amplitude– time plots of the selected patterns of the factory sound, traffic sound, and furnace sound signal in unexpanded mode, respectively (amplitude is measured in arbitrary units, and time in seconds).

The unexpanded plots contain screenshots of the entire sound, with a vertical line showing where the time-expanded sections (6, 5, 7 sec) were obtained.

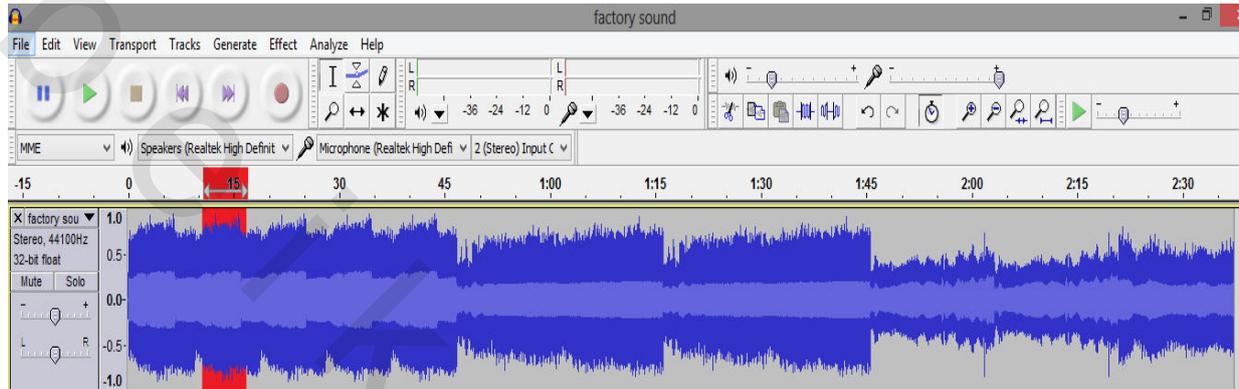


Fig.38: Factory sound.

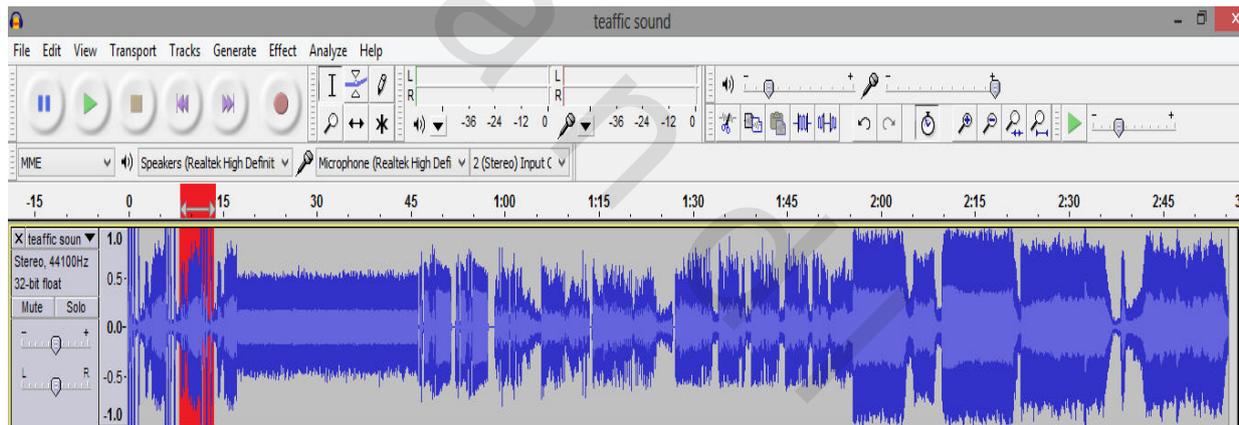


Fig.39: Traffic sound.

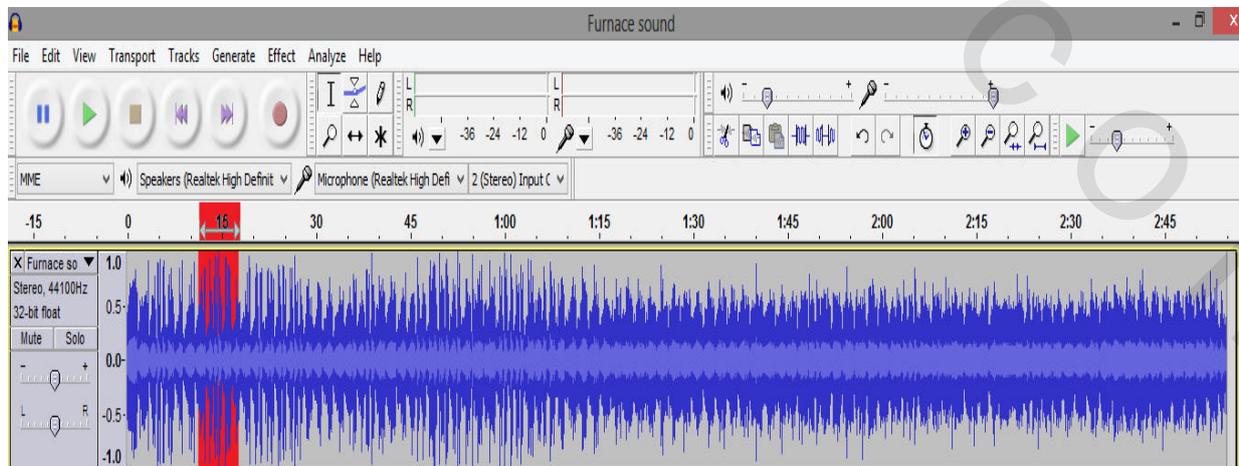


Fig.40: Furnace sound.

II.3. Mice groups:

The 130 mice were divided equally into 13 groups; one group serve as control group while the rest 12 groups were exposed to 3 types of noise sources (factory, traffic, and baking furnace). Each noise source was in form of both of all frequencies collection and its predominant frequency only.

The exposure to each noise collection and source predominant frequency was performed in both chronic and acute pattern as follow:

- **The acute effect:**

By exposing groups to each noise source (collection or predominant frequency) for 12 hr with intensity of 100 dB, for once.

- **The chronic effect:**

By exposing groups to each noise source (collection or predominant frequency) for 8 hr with intensity of 100 dB, for 21 d.

Tables 5 and 6 show the groups and the procedure of exposure for noise and its predominant frequency.

Table 5: Groups exposed to collection noise of different sources

Noise kind Exposure type	Factory noise	Traffic signal noise	Baking furnace noise	Control group
Acute exposure (12 hr for one time only).	GI _a	GII _a	GIII _a	G ₀
Chronic exposure 8hr/d for 21 d.	GI _c	GII _c	GIII _c	G ₀

Table 6: Groups exposed to predominant frequency of different sources

Noise kind Exposure type	Factory noise	Traffic signal noise	Baking furnace noise
Acute exposure (12 hr for one time only).	GI _{af}	GII _{af}	GIII _{af}
Chronic exposure (8hr/d for 21 d).	GI _{cf}	GII _{cf}	GIII _{cf}

Symbols in the above tables denote: G for group, 0, I, II, III for kind of noise used, a= acute exposure, c= chronic exposure. f = predominant frequency.

II.4. Experiment set-up:

- **Exposure System:**

The unit in which the mice were placed in order to be exposed to the noise, schematic diagram of anechoic chamber in which mice were placed to be exposed to a

certain type of noise, is illustrated in Fig.41. It consists of recording system, anechoic chamber and loud speaker.

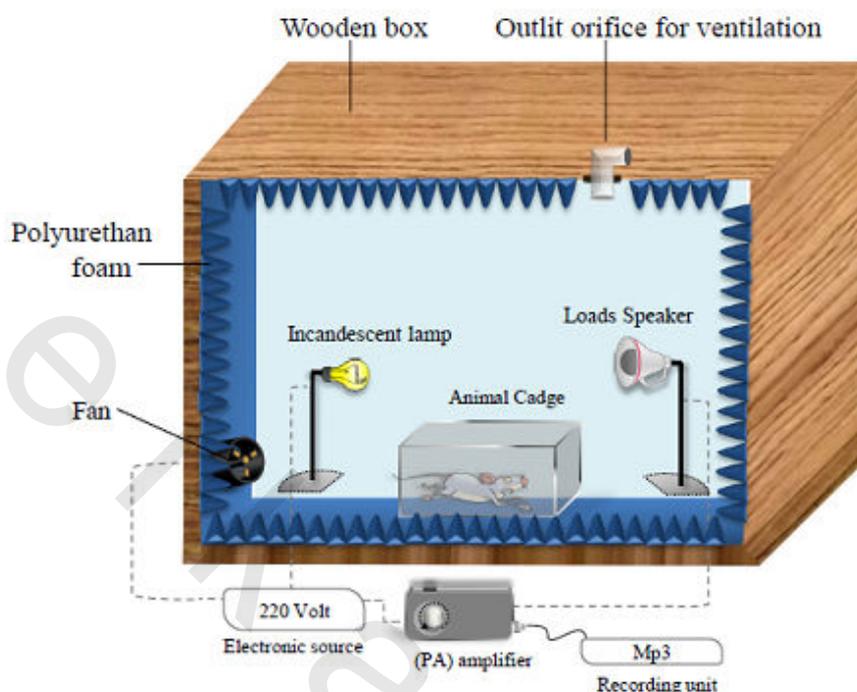


Fig.41: Exposure system (Anechoic chamber).

II.5. Biochemical studies:

II.5.1. Determination of serotonin (5-HT) level in brain tissue: ⁽¹⁵⁶⁾

Brain 5-HT levels were determined according to modification of the method of Schlumpf et al.

- **Principle:**

This method is based on extraction of monoamines by acidified butane. Treatment of the extract with a sensitive indicator O-Phthaldialdehyde (OPT) 20 mg% will form fluorescent compound with serotonin which could be measured at excitation wavelength 360 nm and emission wavelength 470 nm in the spectrofluorophotometer. Treatment of another portion of the extract with iodine in a weak acid medium results in reaction products which isomerise in alkaline medium in the spectrofluorophotometer at extraction wavelength 330 nm.

- **Chemicals:**

Hydrochloric acid 37%, n-Butanol, ethylene diamine tetra acetic acid (EDTA), Hydrated sodium acetate, Sodium hydroxid, Iodine crystals, Ethyl alcohol, Sodium sulphite, Glacial acetic acid, Potassium dihydrogen phosphate, Anhyrous disodium hydrogen phosphate, n-Heptane, Sodium acetate, Serotonincreatinine sulphate and OPT (was obtained from Sigma chemicals CO., U.S.A).

• **Reagents:**

- 1- Hydrochloric acid –Butanol: was prepared by addition of 0.85 ml of 37% HCl to one liter (L) of n-butanol.
- 2- 0.1 M HCl: 10 ml of 37% HCl was completed to one L with bidistilled water.
- 3- 0.4 M HCl: 40 ml of 37% HCl was completed to one L with bidistilled water.
- 4- Ethylene diamine tetra acetic acid disodium (EDTA) / sodium acetate buffer pH 6.9: sodium acetate solution was first prepared by dissolving 13.6 g of hydrated (3H₃O) sodium acetate in 100 ml distilled water. Then the buffer was prepared by dissolving 3.72 g of hydrated EDTA (2H₂O) in 90 ml of sodium acetate solution. The pH was adjusted to 6.9 by addition of 10 M (NaOH) solution. The volume was completed to 100 ml by water or sodium acetate solution.
- 5- 0.1 M iodine solution: it was prepared by dissolving 1.27 g iodine crystals in 100 ml of 90% ethyl alcohol.
- 6- Sodium sulphite in 5 M NaOH solution: 5 M NaOH solution was prepared by dissolving 20 g NaOH in 100 ml bidistilled water. 0.5 g sodium sulphite was dissolved in 2 ml distilled water then 18 ml of 5 M NaOH was added.
- 7- 10 M acetic acid: 60 ml of glacial acetic acid (molecular weight=60.05) was completed to 100 ml with bidistilled water.
- 8- 5-HT standard solution: a stock of 5-HT creatinine sulphate containing 100 µg base/ml was prepared in 0.01 M HCl and stored at -20 °C. an external standard was freshly prepared by diluting the standard stock solution with 0.1 M HCl to obtain a final concentration of 0.1 µg/ ml of 5-HT. one ml of the diluted solution as external standard.
- 9- 0.05 M phosphate buffer pH 7: 2.7 potassium dihydrogen phosphate and 4.39 g of anhydrous sodium hydrogen phosphate were dissolved in one L bidistilled water, pH was adjusted with 10 M NaOH solution or concentrated HCl.
- 10- 10 M NaOH solution: 40 g NaOH was dissolved in 100 ml bidistilled water.
- 11- OPT: 20 mg% in concentration HCl.

• **Experimental procedure:**

- 1- 0.1 mg of brain tissue was homogenized in 2 ml HCl-butanol in Micro-Dismembrator homogenizer.
- 2- The homogenate was centrifuged for 10 min at 6000 rpm in a cooling centrifuge at 0°C.
- 3- An aliquot of 1.6 ml of the supernatant fluid was transferred to a 10 ml-glass stoppered centrifuge tube containing 0.2 ml n-heptane and 0.5 ml of 0.1 M HCl.

- 4- The tube was shaken mechanically for 10 minutes, centrifuged at 0°C and then the organic phase was discarded.
- 5- The supernatant fluid (0.2 ml) was taken.

• **Serotonin assay:**

- 1- 0.125 ml of OPT reagent was added to a tube containing 0.1 ml of the supernatant fluid and mixed well.
- 2- Then the mixture was allowed to boil in a boiling water bath for 10 minutes and cooled under tap water.
- 3- When the sample reached room temperature, it was transferred to quartz cuvette for the determination of serotonin level.
- 4- The fluorescence was read in the spectrofluorometer at excitation wavelength 360 nm and emission wavelength 470 nm.
- 5- A blank experiment for serotonin was carried out by assaying two tubes containing 0.2 ml of 0.2 N acetic acid.
- 6- The standard curve for serotonin was made using different concentrations of the standard serotonin solutions which were subjected to the same methods of determination (Fig.42).

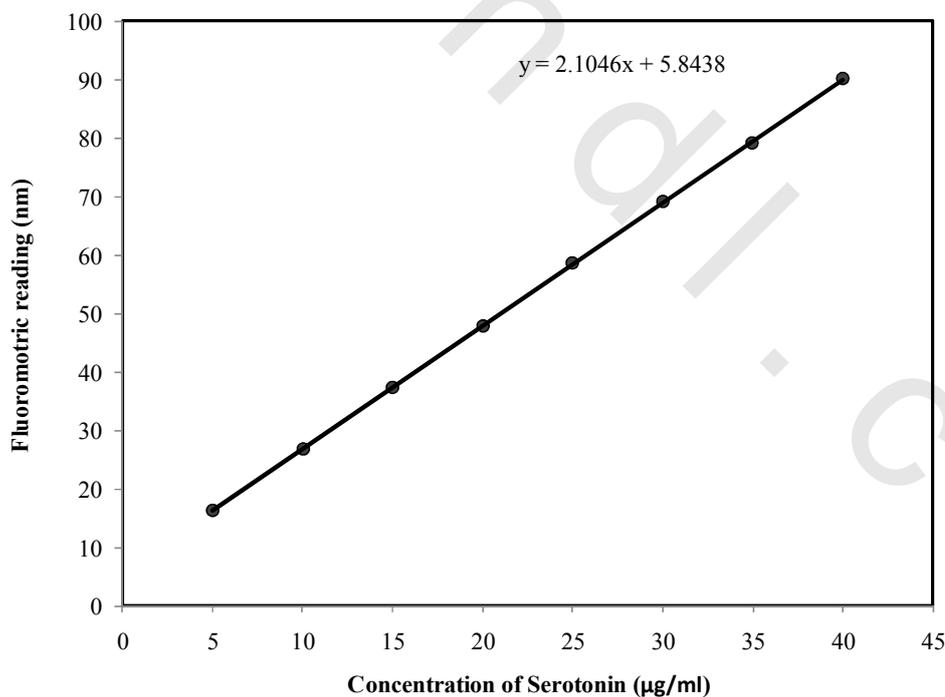


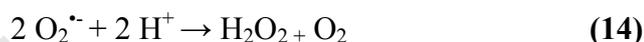
Fig.42. Standard curve of serotonin.

II.5.2. Assay of superoxide dismutase (SOD) specific activity: ⁽¹⁵⁷⁾

The activity of SOD was assayed in brain homogenate as described by Marklund S and Marklund G.

- **Principals:**

This methods depends on the spontaneous auto-oxidation of pyrogallol at alkaline phosphate resulting in the production of superoxide anion radical $O_2^{\cdot-}$, which in turn enhances auto-oxidation of pyrogallol. Auto-oxidation is manifested as an increase in absorbance 420 nm. The presence of SOD in the reaction medium leads to removal of $O_2^{\cdot-}$, Thereby inhibiting auto-oxidation of pyrogallol. This inhibition was linear up to 50% of the rate of the reaction.



- **Reagents:**

1. 50 mM Tris-HCl buffer containing 1 mM diethylenetriaminepentacetic acid (DTPA), the pH of which was adjusted to 8.2 with NaOH solution.
2. 20 mM Pyrogallol (1, 2, 3-benzenetriol) in 10 mM hydrochloric acid.
3. SOD standard solution was prepared in distilled water at a concentration of 5ng/ μ l.

- **Procedure:**

The reaction was carried out at 30°, 1ml of Tris-HCl buffer was added followed by 10 μ l of pyrogallol and then 10 μ l of diluted brain homogenate (1:10). The change in the absorbance at 420 nm was determined through one min (ΔA /min) using spectrophotometer. A blank was similarly treated but without adding tissue homogenate.

Standard curve (Fig.43) was made by the same procedure for the sample using different volumes of the standard SOD solution (5, 10, 15, 20 μ l.... ect).

- **Calculation:**

The percentage inhibition of pyrogallol autoxidation by standard SOD was determined from the equation:

$$100 - \frac{\Delta A (\text{Sample or Standard})}{\Delta A (\text{Blank})} \times 100 \quad (15)$$

From the standard curve, one unit of SOD activity equals 104 ng. The specific activity of SOD in the sample as ng/mg protein was calculated by dividing the value of SOD as ng/ml by protein concentration in the sample (mg protein/ml).

One unit of SOD activity is defined as the amount of enzyme required to inhibit the pyrogallol auto-oxidation by 50%. From the standard curve, one unit of SOD activity equals 111 ng. Therefore, the enzyme specific activity in units/mg protein (U/mg protein) was obtained by dividing the value in ng/mg protein by 111.

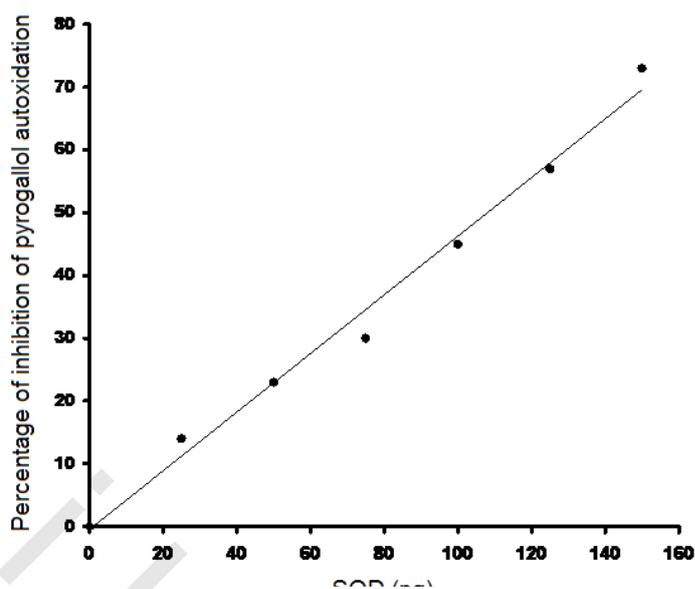


Fig.43: Standard curve of SOD.

II.5.3. Determination of malondialdehyde: ⁽¹⁵⁸⁾

Malondialdehyde in whole homogenate was determined according to the method of Draper and Hadley.

- **Principle:**

The sample under test is heated with thiobarbituric acid (TBA) at low pH. The resulting pink chromogen has a maximal absorbance at 532nm.

- **Chemicals:**

1. Sodium dodecyl sulphate (SDS) 8.1 % in distilled water.
2. Acetic acid 20%. Its pH was adjusted to 3.5 with 1N sodium hydroxide.
3. Thiobarbituric acid (TBA) 0.8 % in distilled water.
4. n-Butanol.

- **Procedure:**

An aliquot of 0.1 ml of the sample was pipetted into a tube containing an equal volume of SDS solution. This was followed by the addition of 0.75 ml acetic acid, 0.75 ml of TBA and 0.3 ml of distilled water. The contents of the tubes were then mixed with a vortex. The tubes were incubated in a boiling water bath for 1 hr then cooled to room temperature. An aliquot of 0.5 ml of distilled water was added to each tube followed by the addition of 2.5ml n-butanol. The contents of the tubes were vigorously mixed with a vortex then rotated in a centrifuge at 2500xg for 10 min. Absorbance of the organic layer was read at 532nm in a spectronic 21 spectrophotometer against a blank prepared and treated exactly like the sample however, containing phosphate buffer solution instead of the sample.

The concentration (nmole/ml) of MDA in sample was obtained from a standard curve (Fig.44) made by preparing serial dilutions of tetramethoxypropane (TMP), 1,2,4,6,8,12 nmole/ml, (Aldrich Chemical Co., Milwaukee, Wisconsin, USA) in ethanol and treating them like the sample. Results were expressed as nmole MDA/mg protein by dividing the concentration of MDA in the sample by the protein concentration in the same sample.

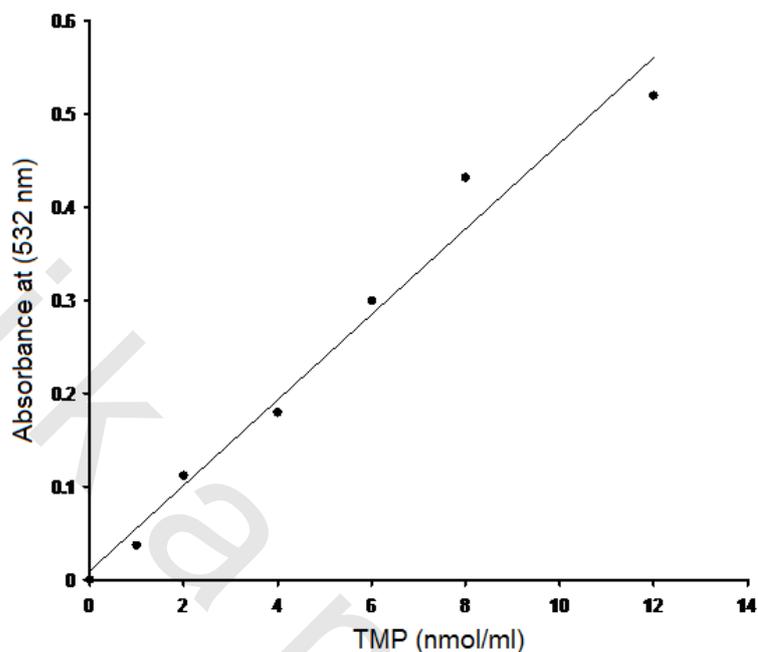


Fig.44: Standard curve of MDA.

II.5.4. Determination of total protein: ⁽¹⁵⁹⁾

- **Principle:**

A modification of the method of Lowry et al. was used for the determination of protein in the samples. The color produced is thought to be due to a complex between the alkaline copper-phenol reagent and tyrosine and tryptophan residues of the protein in the sample. The protein concentration in each sample was estimated by referring to a standard curve (Figure 16) which was constructed using bovine serum albumin.

- **Reagents:**

1. Sodium hydroxide 0.1M.
2. Sodium carbonate (anhydrous) 2% in 0.1M NaOH.
3. K/Na tartarate 2%.
4. Lowry C reagent: prepared immediately before use by mixing volumes of sodium carbonate, K/Na tartarate and copper sulphate reagent in a ratio: 100: 1: 1.
5. Folin-Ciocalteu reagent. The working reagent was prepared by diluting the stock reagent 1: 1 (V/V) with distilled water immediately before use.

- **Procedure:**

The sample was diluted in distilled water (1: 10). Aliquots of 10 μ l of diluted samples were mixed with 2.5ml of Lowry C reagent. After incubation for 10 minutes at room temperature, 0.25ml of working Folin-Ciocalteu's reagent was added. The tubes were then mixed and incubated in a dark place for one hour at room temperature, after which the absorbance was read at 695 nm using spectronic a 21 spectrophotometer. A blank containing phosphate buffered saline instead of the sample was treated similarly.

- **Calculation:**

The protein concentration in each sample was estimated by referring to the standard curve (Fig.45) which was constructed using concentration of bovine serum albumin (BSA) (25 μ g/ml).

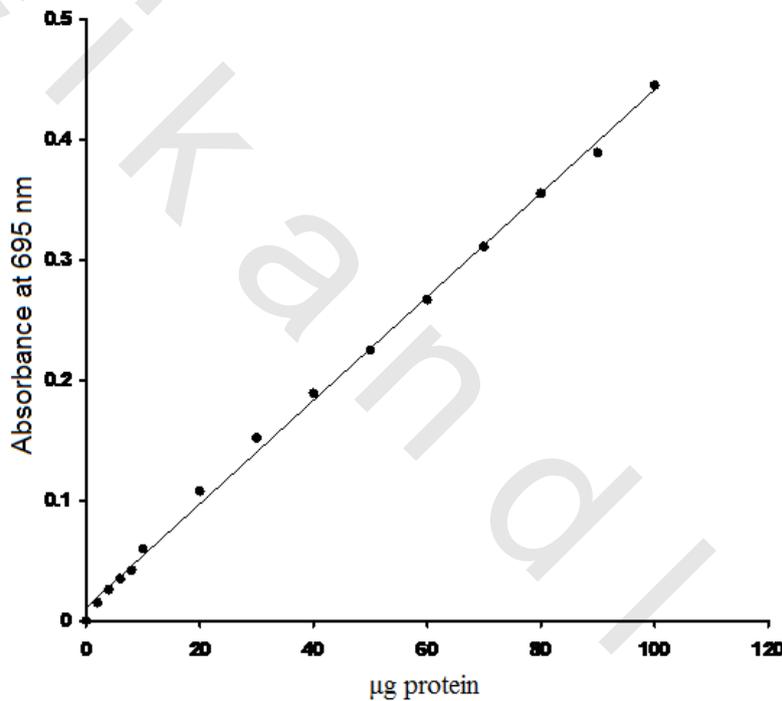


Fig.45: Standard curve of total protein.

II.5.5. Glucose level measurement: ⁽¹⁶⁰⁾

- **Principle:**

The glucose assay uses the glucose oxidase-peroxide reaction for determination of glucose concentrations by generation of a pink dye with an optimal absorption at 514 nm.

- **Sample preparation:**

Glucose level was measured in blood serum:

1. Collect the blood without adding anticoagulant.
2. Allow blood to clot for 30 minutes at 25 °C.
3. Centrifuge the blood at 2000 x g for 15 minutes at 4 °C.
4. Pipette off the yellow serum layer without disturb the white buffy layer.
5. Dilute serum 1:5 with diluted Assay Buffer before assaying.

- **Procedure:**

1. Flavin adenine dinucleotide (FAD) dependent enzyme glucose oxidase was added to serum sample, oxidizing the glucose in the sample to δ -gluconolactone with reduced form of glucose oxidase.
2. Reduced form of glucose oxidase is regenerated to its oxidized form by molecular oxygen to produce hydrogen peroxide.
3. Finally, with horseradish peroxidase as a catalyst, hydrogen peroxide reacts with 3,5-dichloro-2-hydroxybenzensulfonic acid and 4-aminoantipyrine (also called 4-aminophenazone) to generate a pink dye with an optimal absorption at 514 nm.

- **Calculation:**

The concentration (mg/dl) of glucose in sample was obtained from a standard curve (Fig. 46)

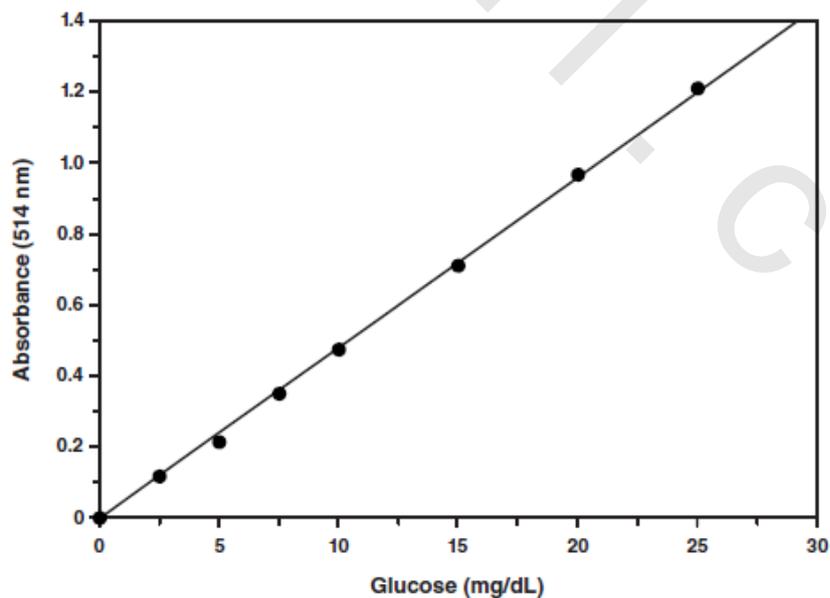


Fig.46: Standard curve of glucose.

II.5.6. White blood cells count: ⁽¹⁶¹⁾

- **Sample preparation:**

WBCs count was measured in blood plasma:

1. Collect the blood using an anticoagulant (EDTA).
2. Centrifuge the blood at 700 - 1000 x g for 10 min at 4 °C.
3. Pipette off the yellow plasma layer without disturb the white buffy layer.
4. Dilute serum 1:5 with diluted Assay Buffer before assaying.

- **Procedure:**

WBCs count performed by an automated analyzer. The cell counting component counts the numbers and types of different cells within the blood. The results are printed out or sent to a computer for review.

II.6. Behavioral study:

The Novel Object Recognition (NOR) task is used to evaluate cognition, particularly recognition memory. This test is based on the spontaneous tendency of rodents to spend more time exploring a novel object than a familiar one. The choice to explore the novel object reflects the use of learning and recognition memory.

II.6.1. Procedure:

The task procedure consists of three phases: habituation, familiarization, and test phase.

II.6.1.1. The habituation phase: ⁽¹⁶²⁾

- Each mouse was allowed freely exploring the open-field arena (Fig.47) in the absence of objects.
- Habituation was held in one session for 10 min.
- The mice was then removed from the arena and placed in its holding cage.

II.6.1.2. The familiarization phase: ⁽¹⁶³⁾

- Each mouse was placed in the open-field arena containing two identical sample objects ($X+X$).
- Familiarization was held in three sessions, each for 10 min. Sessions were separated by three minutes interval where the mice was returned back to its cage.
- To prevent coercion to explore the objects, rodents were released against the center of the opposite wall with its back to the objects.
- After a retention interval of about 15 min, the test was performed.

II.6.1.3. The test phase: ⁽¹⁶⁴⁾

- The mice were returned to the open-field arena with two objects, one is identical to the sample and the other is novel ($X + Y$).
- Test time was three min.
- The time spent exploring each object and the recognition index was recorded.



Fig.47: The open field arena. (a) The familiarization phase, (b) The test phase.

II.6.2. Analysis of results:

Results were analyzed by calculating the *Recognition Index*. This technique involves dividing the time spent exploring the novel object (T_N) by the total time spent exploring either objects ($T_N + T_F$), yielding % Novel exploration or recognition index.

- **Recognition Index (RI):**

It equals the time spent investigating the novel object relative to the total object investigation, and it is the main index of retention. ^(162, 165)

$$[RI = T_N / (T_N + T_F)] \quad (16)$$

Apparatus used was an open box made of wood $65 \times 45 \times 65$ cm, rectangular in shape. Objects that have been used in the NOR test vary widely in shapes, sizes, textures, materials, colors, and appearance.

After each session of the NOR, the arena and objects were cleaned using 10% ethanol solution to ensure that behavior of animals was not guided by odor cues. ⁽¹⁶⁵⁾

II.7. Histopathological studies:

II.7.1. Instruments:

- **Cryostat:**

(SLEE MAINZ; type MEV) cryostat with a stainless steel knife. This device used in

process called "cryosectioning" at which the tissue can be frozen and sectioned at a low temperature (-60: -30 °C), on the refrigerator cabinet contain microtome.

- **Light microscope:**

Type: BX41; Olympus America Inc. Has wide objective magnification power, fitted with precise-motion stage which is available for left and right hand use, with stage tension adjustable to suit individual preferences.

- **Digital camera:**

(CAMEDIA C-7070, U-CMAD3, Olympus Company with high performance 7.1 megapixel colored camera system megapixel colored camera system.

II.7.2. Histological method: ⁽¹⁶⁶⁾

- **Preparation of frozen section:**

The brain and small intestine were used to prepare a frozen section. It was placed in a holder in a refrigerated chamber (-60 degrees). When a wheel was turned, the holder advanced towards the blade by the required thick (10 µm) of section during this process the older also moved up and down, to bring the edge of the section down on the top of the blade and pull up the section to the room temperature slide.

- **Procedure:**

The section was fixed at a 10% formaldehyde, washed in dis. H₂O. The nuclear fast red was used as a counter stain, then left the slide to dry and mounted by glycerine-gel and covered by cover slip to be observed under light microscope, and then a photograph was taken with digital compact camera.

RESULTS

I. Analysis of different sources of noise:

I.1. Factory noise:

I.1.1. Band selection and expansion

Figures 48(a,b) show amplitude–time plots in both, time-unexpanded and time-expanded modes respectively. Amplitude is measured in arbitrary units, and the time in sec. The unexpanded plot contains screenshot of the entire recorded factory-signal sound, with a vertical red line showing where the time-expanded sections were selected.

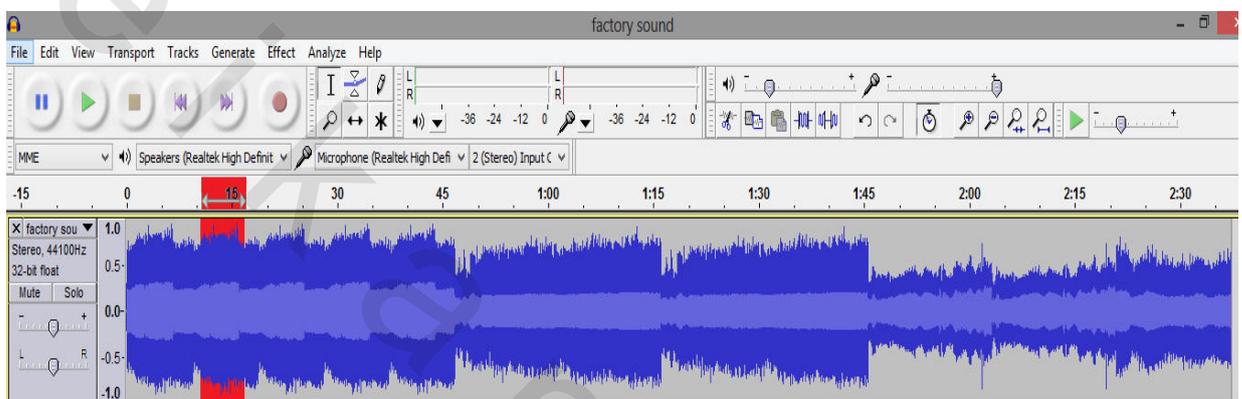


Fig.48a: Factory noise amplitude-time plot in unexpanded mode. (The axis is in a logarithmic scale)

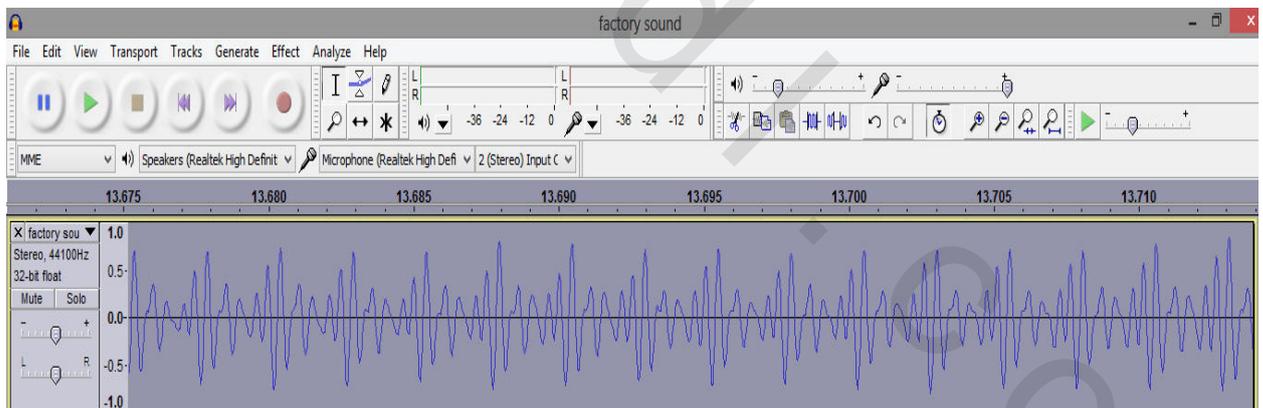


Fig.48b: Expanded band of the selected portion of the recorded factory noise.

I.1.2. Predominant frequency for factory sound:

The predominant frequency was determined using the unexpanded plot shown in Fig 48a, which contains the entire recorded factory signal sound. The Real Time Analyzer was used in this analysis to plot the frequency-intensity diagram as seen in Fig.49. Clearly Figure 49 shows the distribution of frequency-intensity, the 2 kHz has 100 dB which represent the selected predominant frequency as defined before.

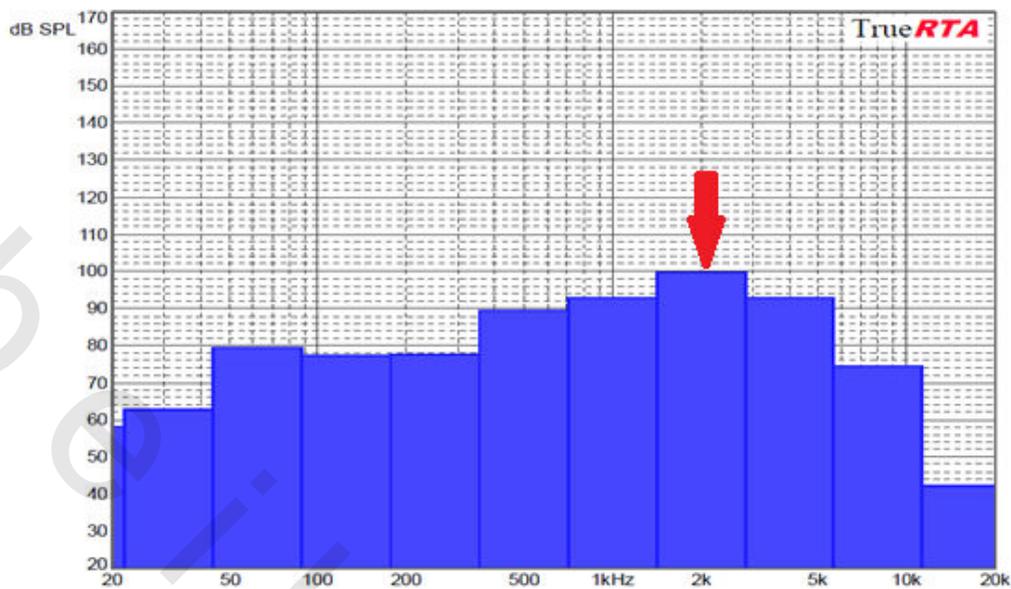


Fig.49: Factory noise frequency-intensity diagram. The X-axis represents the sound frequency in Hz, while the Y-axis represents the sound intensity in (dB SPL).

I.1.3. Cepstrum analysis:

The cepstrum diagram (Fig.50) of the selected sound band shows sporadic very high spikes of sounds such as other sound of other machines that are closed to the main machine source and workers speech or movement.

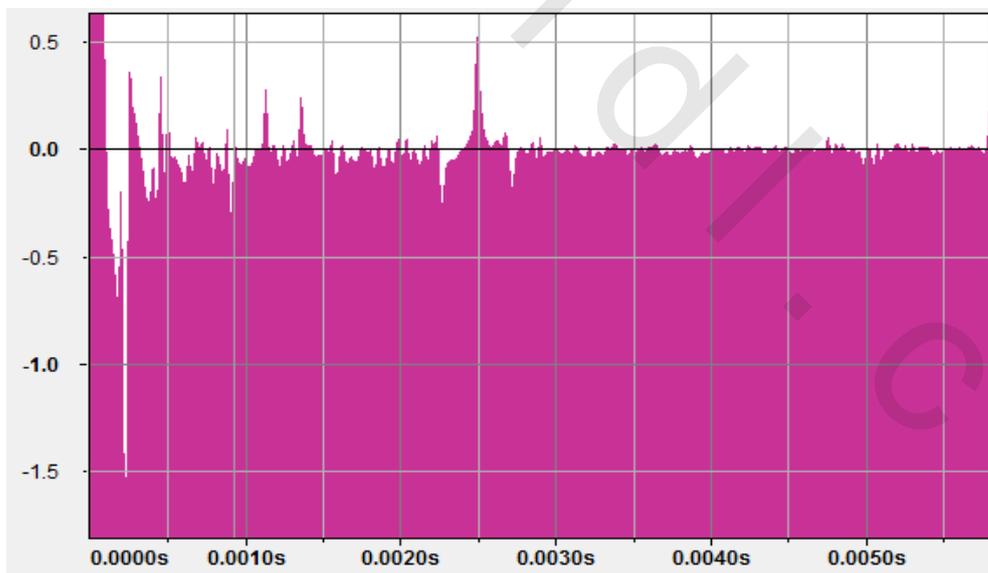


Fig.50: Factory noise signal cepstrum analysis. The horizontal axis is the time in seconds and the vertical axis is the magnitude of the cepstrum (function). The sound of other machines that are closed to the main machine source and/or workers speech and movement are separated to left of the diagram as a characteristic band, while the constant annoying factory signal sound appears, at the rest of the x-axis.

I.1.4. Standard autocorrelation:

As it is already known that autocorrelation measures, to what extent the sound repeats itself. In other words how many identical frequencies in the used sound sample. From the autocorrelation Figure (Fig.51) of the selected sound band the higher level sound intensity is separated at the left side of the plot (unrepeated), while the continuous factory sound repeated in eight distinct bands.

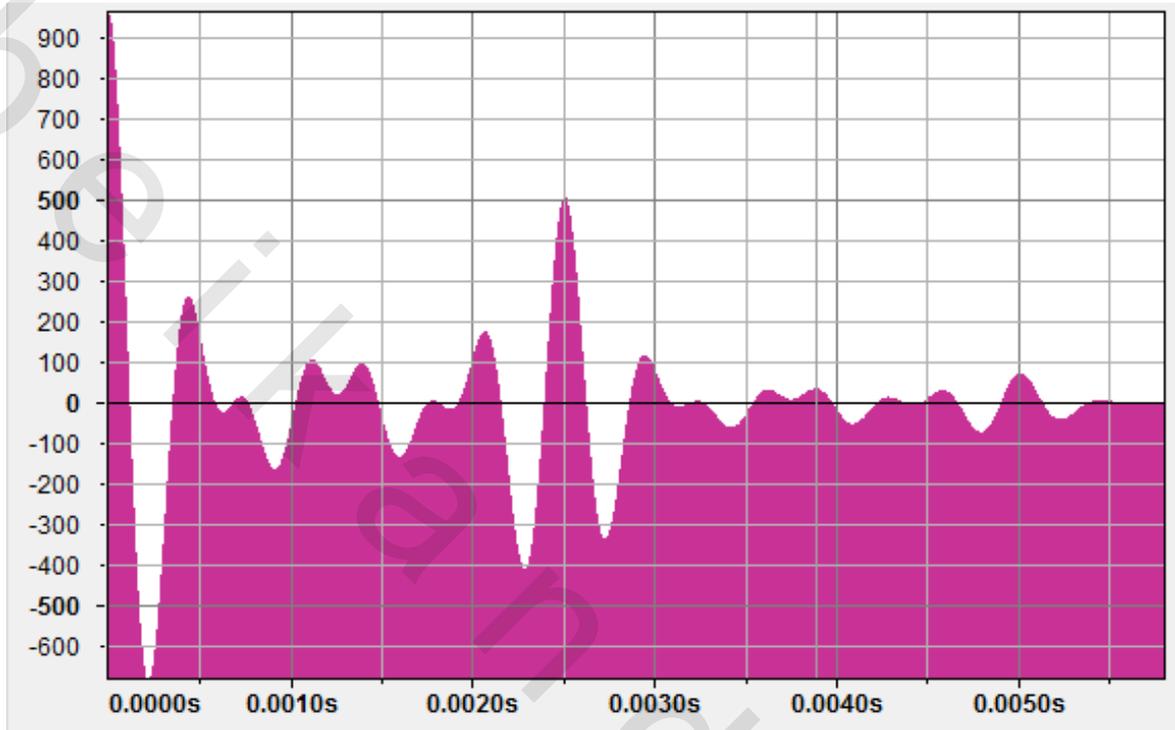


Fig.51: The standard autocorrelation of the recorded factory noise signal. The horizontal axis is the time in second; the vertical axis is the value of the autocorrelation function.

I.2. Traffic noise:

I.2.1. Band selection and expansion:

Figures 52 (a,b) show amplitude–time plots in both time-unexpanded and time-expanded modes respectively. Amplitude is measured in arbitrary units, and the time in seconds. The unexpanded plot contains screenshot of the entire recorded traffic-signal sound, with a vertical red line showing where the time-expanded sections.

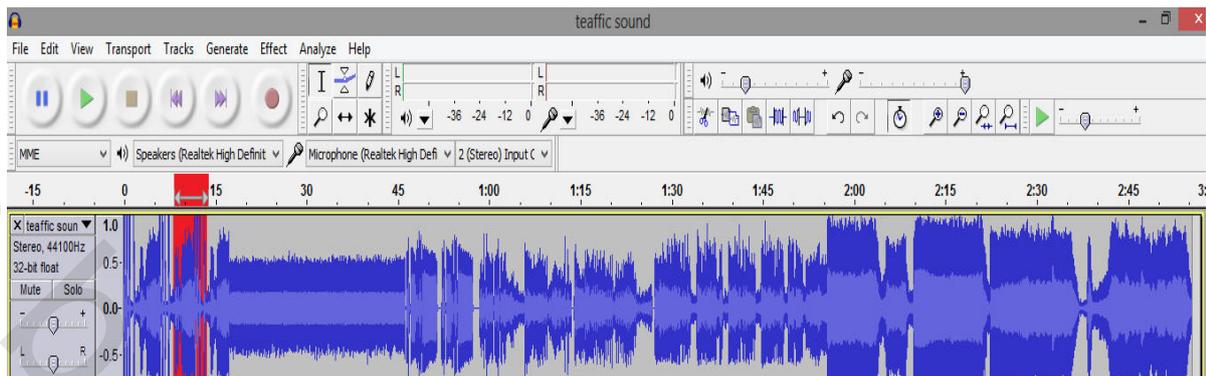


Fig.52a: Traffic noise amplitude-time plot in unexpanded mode. (The axis is in a logarithmic scale).

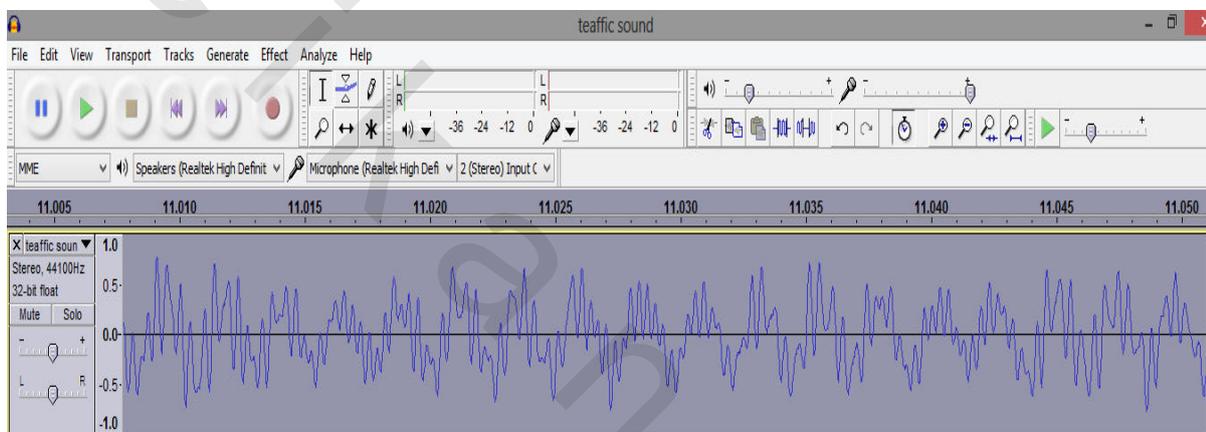


Fig.52b: Expanded band of the selected portion of the recorded traffic noise.

I.2.2. Predominant frequency for traffic sound:

The predominant frequency was determined using the unexpanded plot shown in Fig 52a, which contains the entire recorded traffic signal sound. The Real Time Analyzer was used in this analysis to plot the frequency-intensity diagram as seen in Fig.53. Figure 53 shows the distribution of frequency-intensity, clearly the 1 kHz has 100dB which represent the selected predominant frequency as defined before.

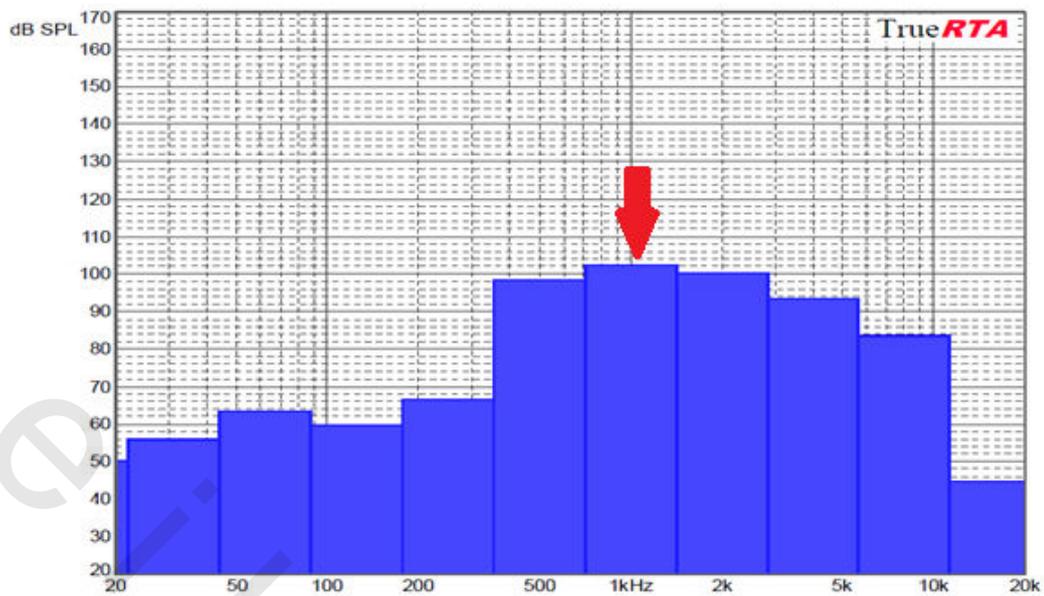


Fig.53: Traffic noise frequency-intensity diagram. The X-axis represents the sound frequency in Hz, while the Y-axis represents the sound intensity in (dB SPL).

I.2.3. Cepstrum analysis:

The cepstrum diagram shows (Fig.54) sporadic very high spikes of sounds such as police or ambulance alarms overlapped on constant annoying sounds due motors and cars movement.

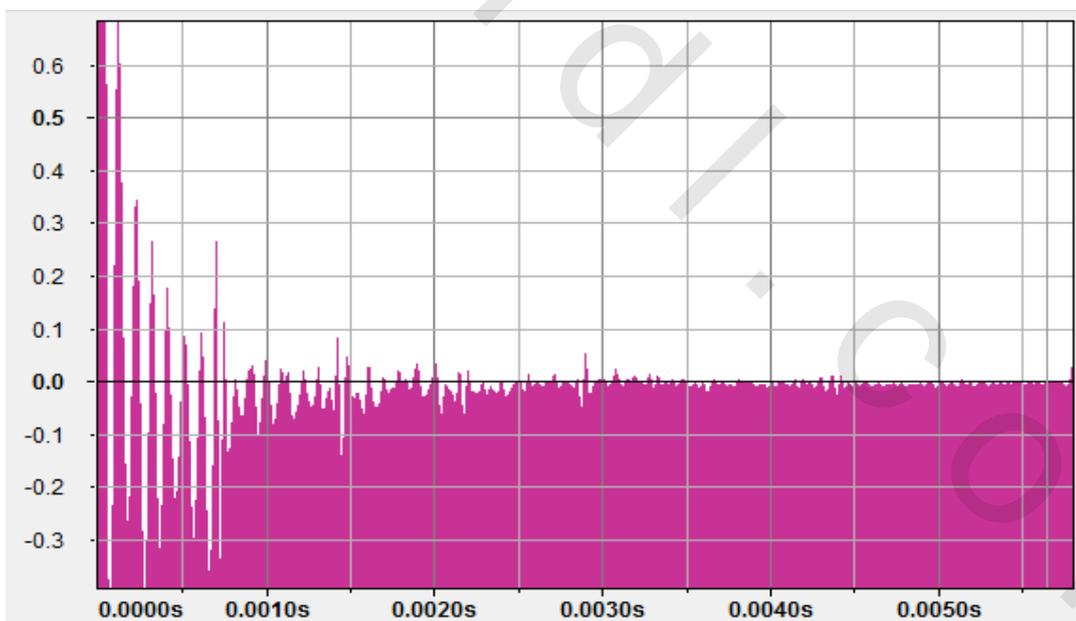


Fig. 54: Traffic noise signal cepstrum analysis. The horizontal –axes is the time in units of seconds and the vertical axis is the magnitude of the cepstrum (function). The sound of police and/or ambulance serine are separated to left of the diagram as a characteristic band, while the constant annoying traffic signal sound appears, at the rest of the x-axis.

I.2.4. Standard autocorrelation:

From the autocorrelation Figure (Fig.55) the higher level sound intensity is separated at the left side of the plot (unrepeated), while the continuous traffic sound repeated in three distinct bands.

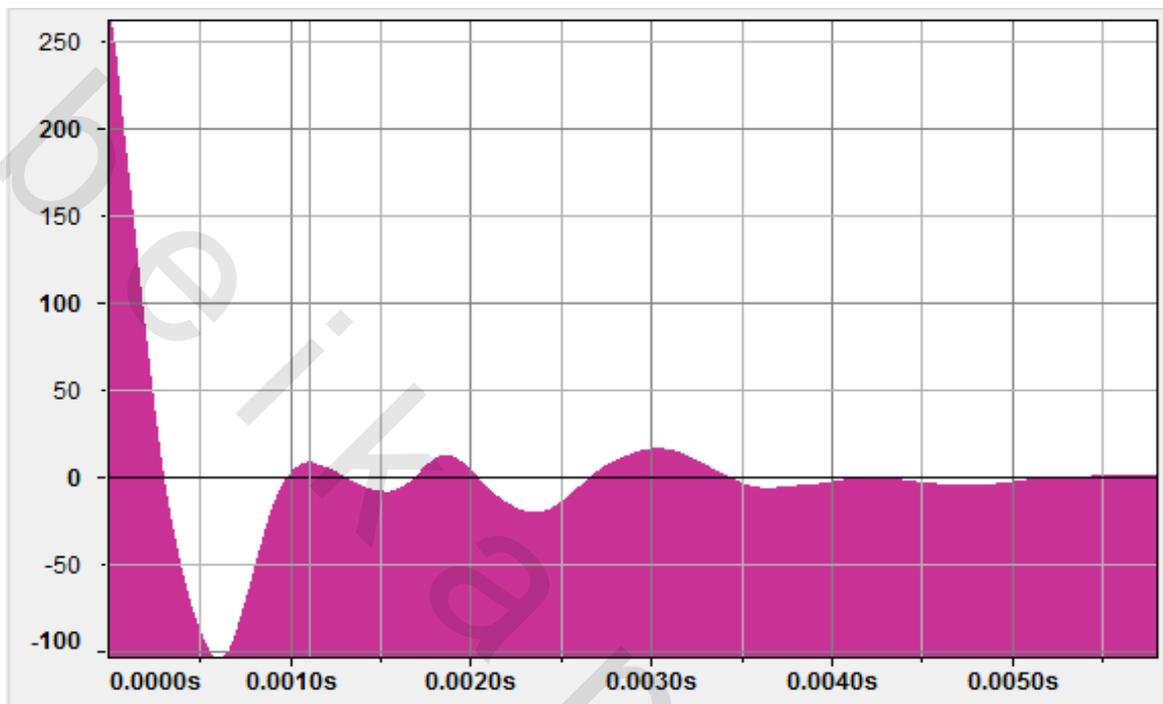


Fig.55: The standard autocorrelation of the recorded traffic signal noise. The horizontal axis is time in second; the vertical axis is the value of the autocorrelation function.

I.3. Furnace noise:

I.3.1. Band selection and expansion:

Figures 56 (a,b) show amplitude–time plots in both time-unexpanded and time-expanded modes respectively. Amplitude is measured in arbitrary units, and the time in seconds. The unexpanded plot contains screenshot of the entire recorded furnace-signal sound, with a vertical red line showing where the time-expanded sections.

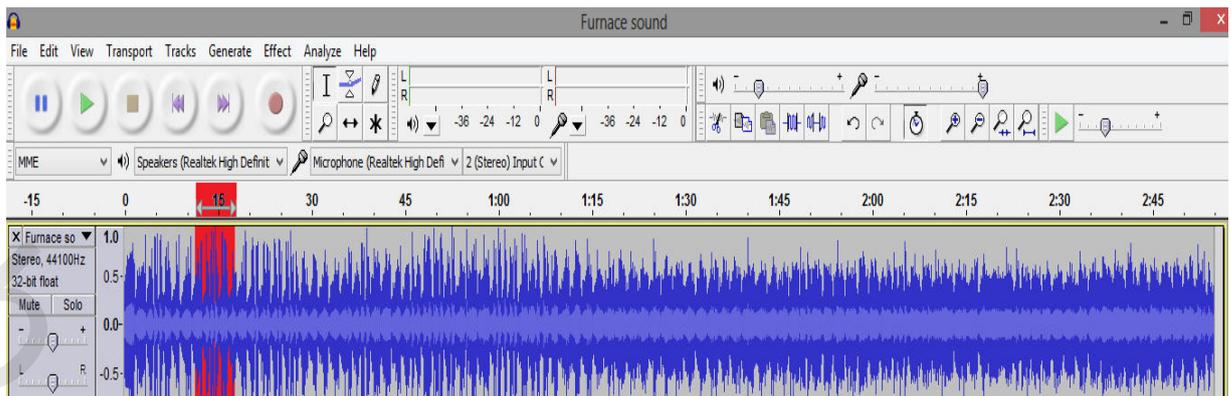


Fig.56a: Furnace noise amplitude-time plot in unexpanded mode. (The axis is in a logarithmic scale)

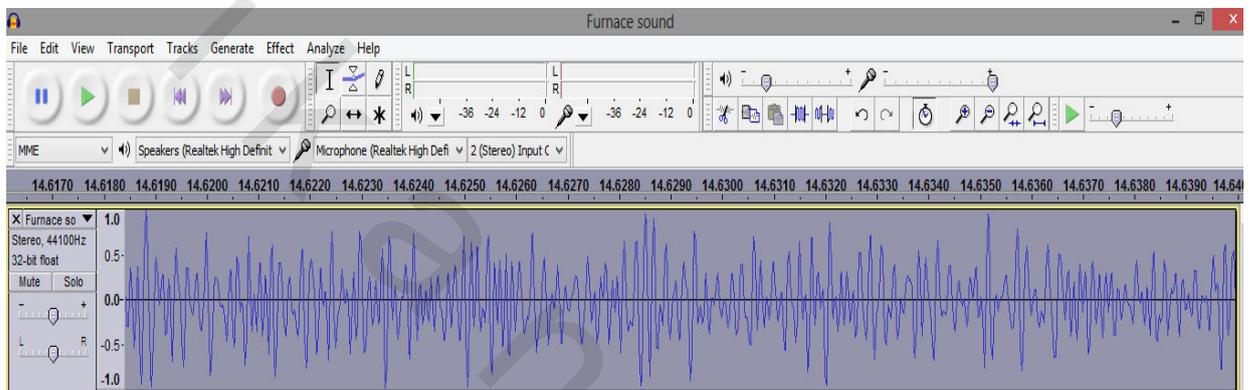


Fig.56b: Expanded band of the selected portion of the recorded furnace noise.

I.3.2. Predominant frequency for furnace sound:

The predominant frequency was determined using the unexpanded plot shown in Fig. 56a, which contains the entire recorded furnace signal sound. The Real Time Analyzer was used in this analysis to plot the frequency-intensity diagram as seen in Fig.57. Figure 57 shows the distribution of frequency-intensity, clearly the 5 kHz has around 100 dB which represent the selected predominant frequency as defined before.

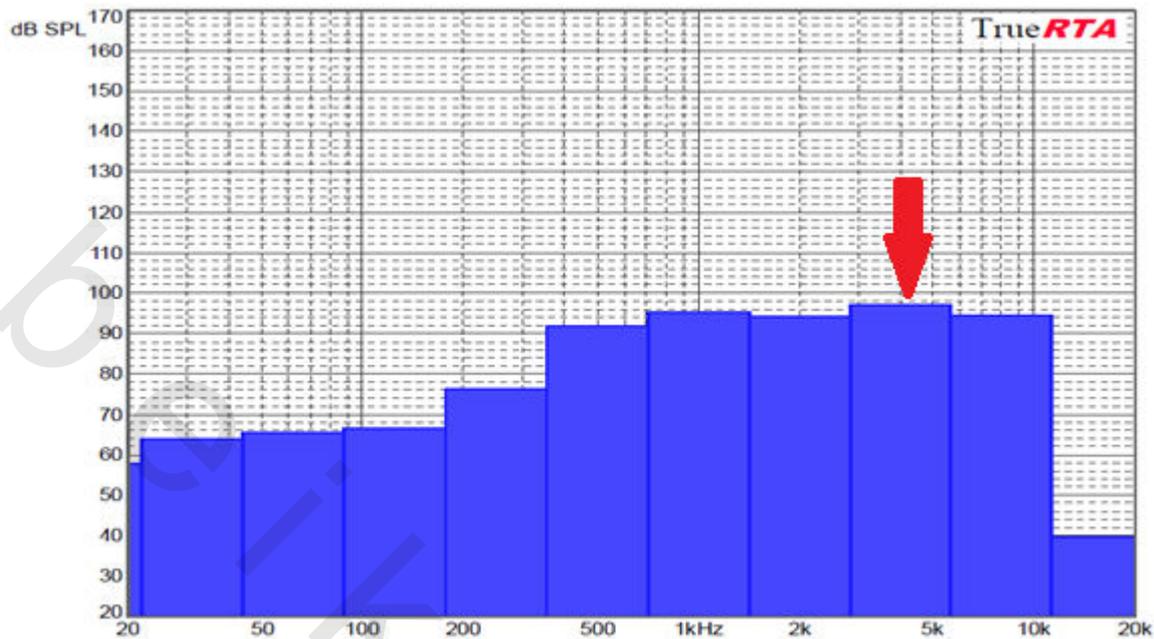


Fig.57: Furnace noise frequency-intensity diagram. The X-axis represents the sound frequency in Hz, while the Y-axis represents the sound intensity in (dB SPL)

I.3.3. Cepstrum:

The cepstrum diagram (Fig.58) shows sporadic very high spikes of sounds such as workers, sales and customers speech and outdoor cars movement.

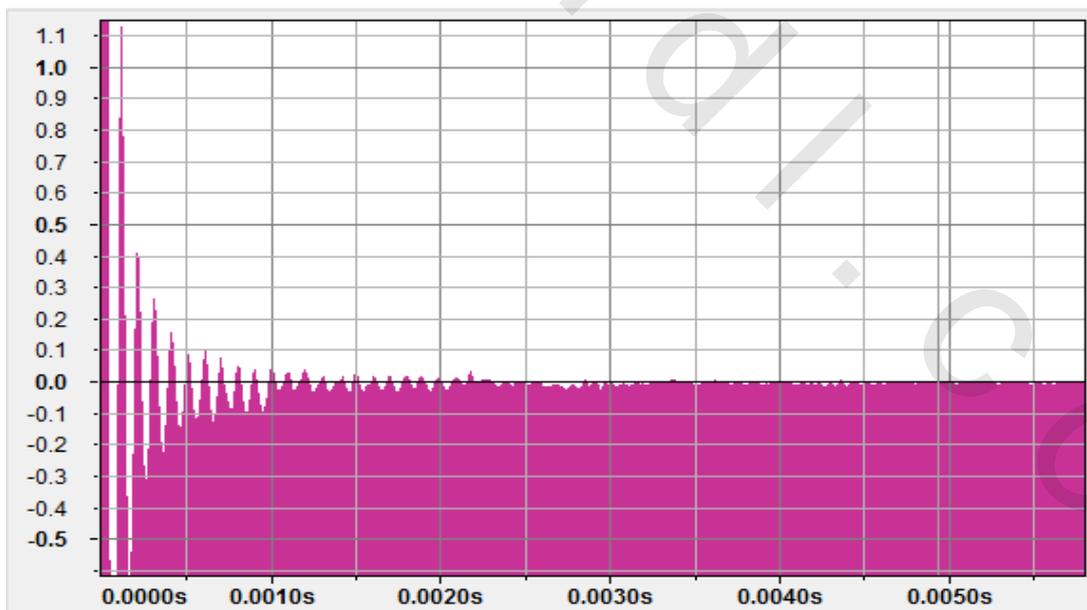


Fig.58: The furnace noise signal cepstrum analysis. The horizontal axis is in seconds and the vertical axis is the magnitude of the cepstrum (function). The sound of workers, sales and customers speech and/or outdoor cars movement are separated to left of the diagram as a characteristic band, while the constant annoying furnace signal sound appears, at the rest of the x-axis.

I.3.4. Standard autocorrelation:

From the autocorrelation Figure (Fig.59) the higher level sound intensity is separated at the left side of the plot (unrepeated), while the continuous furnace sound repeated in five distinct bands.

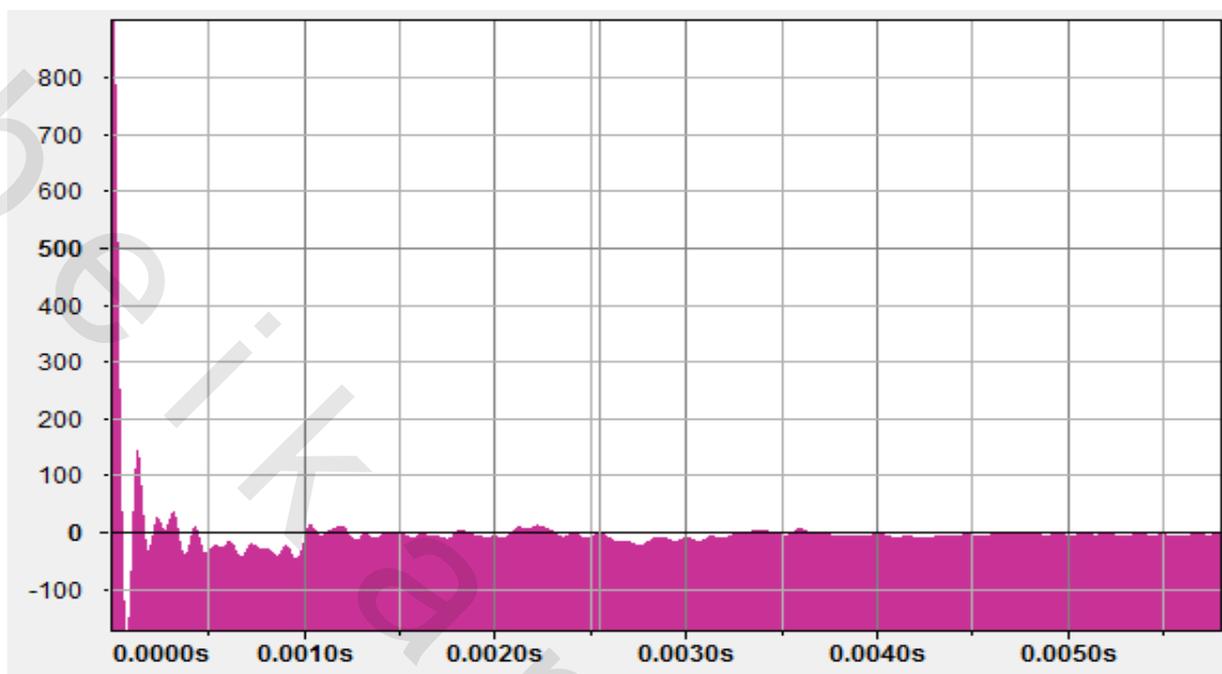


Fig.59: The standard autocorrelation of the recorded furnace signal noise. The horizontal axis is time in second; the vertical axis is the value of the autocorrelation function.

II. Biochemical and behavioral studies:

The studied biochemical and behavioral parameters that were measured after exposure to different noise types (factory, traffic, and furnace) were:

- 1- Blood glucose level.
- 2- WBCs counts.
- 3- Superoxide dismutase (SOD) level.
- 4- Malondialdehyde (MDA) level.
- 5- Serotonin level.
- 6- Recognition index (cognitive function) was calculated and correlated to glucose, SOD and MDA.
- 7- Histopathological changes on both brain and small intestine.

All the studied groups were 10 mice and classified as:

- G0: control exposed (unexposed group).
- GIa: factory acute collection group exposure.
- GIIa: traffic acute collection group exposure
- GIIIa: furnace acute collection group exposure
- GIaf: factory acute predominant frequency group exposure.
- GIIaf: traffic acute predominant frequency group exposure.
- GIIIaf: furnace acute predominant frequency group exposure.
- GIc: factory chronic collection group exposure.
- GIIc: traffic chronic collection group exposure.
- GIIIc: furnace chronic collection group exposure.
- GIcf: factory chronic predominant frequency group exposure.
- GIIcf: traffic chronic predominant frequency group exposure.
- GIIIcf: furnace chronic predominant frequency group exposure.

II.1. Biochemical and psychological parameter analysis for all exposed groups:

Tables 7-12 are the master tables in which all the studied groups are compared together for the studied biochemical and behavioral parameters.

II.1.1. Blood glucose levels (mg/dl):

Blood glucose levels (mg/dl) were measured in all groups. It's clear that, all experimental groups were suffering from reduction in glucose levels (hypoglycemia) compared to control group (87.40 ± 3.92 mg/dl) at ($p < 0.001$). The lowest reduction was noticed in group exposed to factory acute collection group (GIa) (42.60 ± 8.71 mg/dl), while the highest reduction in the blood glucose level was shown after traffic chronic predominant frequency (GIIa) exposure (8 ± 2.11 mg/dl), as shown in Table: 7, and Fig. 60.

Table (7): Effect of noise exposure on the blood glucose level (mg/dl)

	G0	G1a	G1Ia	G1IIa	G1af	G1Iaf	G1IIaf	G1c	G1Ic	G1IIc	G1cf	G1Icf	G1IIcf	F	p
Min.	82.0	32.0	27.0	22.0	27.0	14.0	13.0	24.0	20.0	18.0	12.0	5.0	14.0	171.202*	<0.001*
Max.	92.0	53.0	45.0	34.0	31.0	21.0	28.0	37.0	27.0	31.0	25.0	11.0	20.0		
Mean	87.40	42.60	35.60	29.60	28.60	19.0	21.80	30.80	22.80	24.40	18.60	8.0	17.20		
SD.	3.92	8.71	6.62	4.35	1.43	2.75	5.39	4.69	2.62	5.68	4.79	2.11	2.44		
Median	87.0	45.0	34.0	31.0	28.0	20.0	23.0	32.0	22.0	24.0	18.0	8.0	18.0		
p		<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*		

F: F test (ANOVA).

p: p value for Post Hoc Test (LSD) for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

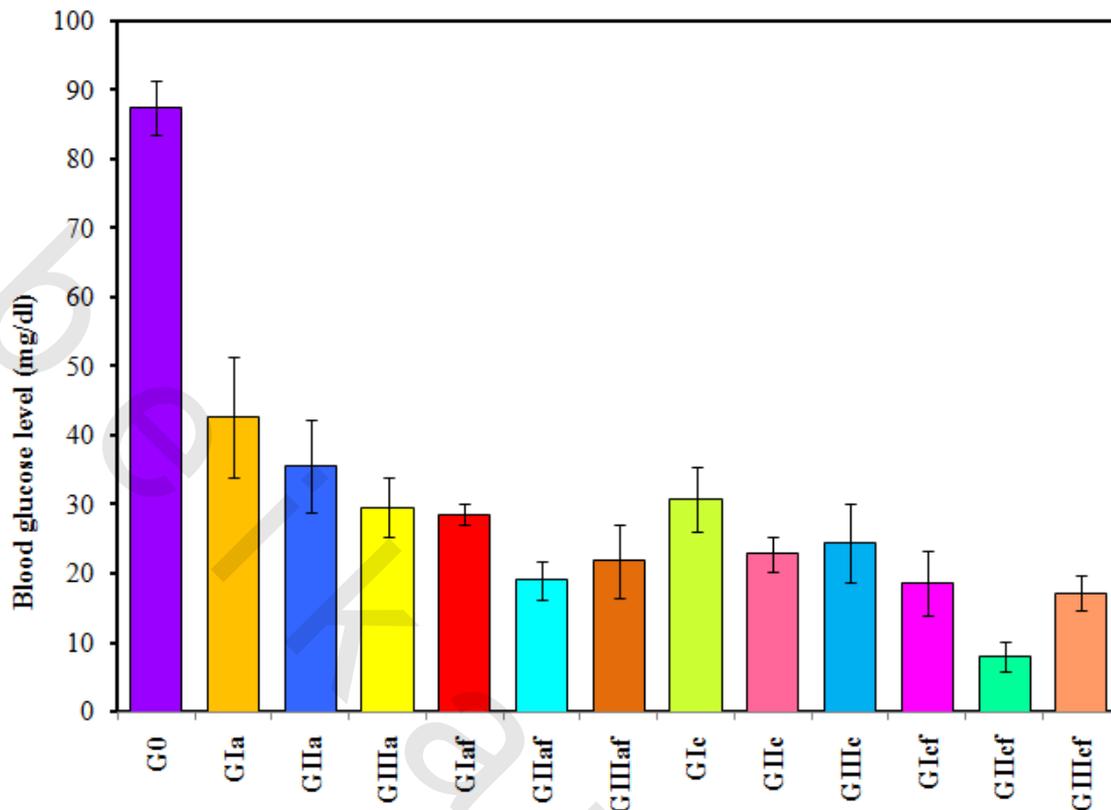


Fig.60. Effect of noise exposure on the blood glucose level (mg/dl).

II.1.2. WBCs counts (cells/ μ l):

Total and differential WBCs counts were measured in all experimental groups. All groups show no significant difference in comparison to control group, in case of differential WBCs count. According to total WBCs counts (cells/ μ l). The control group (G0) mean value was $(11520 \pm 1761.8 \text{ cells}/\mu\text{l})$, all groups show significant decrease than control, $p < 0.001$. The lowest decrease in WBCs counts occurred after acute exposure by factory collection dose (GIIIa) $(8440 \pm 497.1 \text{ cells}/\mu\text{l})$, while the highest decrease in the WBCs counts was done by traffic chronic predominant frequency exposure (GIIcf), $(3450 \pm 462.4 \text{ cells}/\mu\text{l})$. Table: 8, and Fig. 61.

Table (8): Effect of noise exposure on the WBCs counts (cells/ μ l)

	G0	G1a	G1b	G1c	G1d	G1e	G1f	G1g	G1h	G1i	G1j	G1k	G1l	χ^2_{KW}	p
Min.	9700.0	6100.0	5860.0	7900.0	5900.0	3200.0	6900.0	3220.0	3600.0	5100.0	3350.0	3110.0	4070.0	103.416*	<0.001*
Max.	14500.0	8400.0	6760.0	9100.0	7400.0	6100.0	7900.0	7120.0	6100.0	9500.0	4250.0	4110.0	5970.0		
Mean	11520.0	7420.0	6200.0	8440.0	6500.0	5120.0	7300.0	5400.0	4560.0	6700.0	3650.0	3450.0	5100.0		
SD.	1761.8	847.0	368.8	497.1	592.5	1069.6	394.4	1742.8	1096.7	1559.2	333.3	462.4	788.9		
Median	11500.0	7300.0	5960.0	8200.0	6200.0	5300.0	7400.0	5820.0	4000.0	6200.0	3550.0	3210.0	5220.0		
p		<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*		

χ^2 : Chi square for Kruskal Wallis test.

p: p value for Mann Whitney test for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

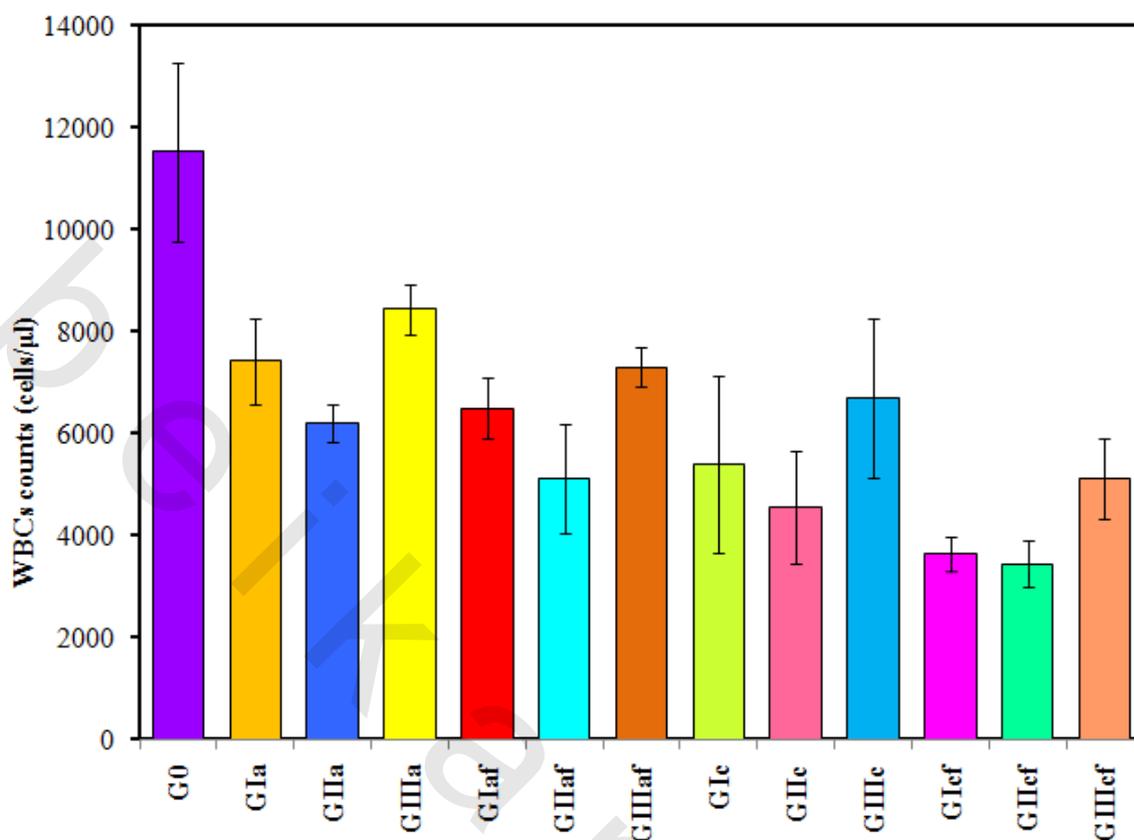


Fig.61: Effect of noise exposure on the WBCs counts (cells/μl).

II.1.3. SOD levels (U/mg protein):

SOD levels (U/mg protein) were measured in all experimental groups. Control group (G0) mean value was $(1.99 \pm 0.54 \text{ U/mg})$. All acute groups whether collection or predominant frequency show no significance difference than control group. While all chronic groups whether collection or predominant frequency show significant decrease than control, $p < 0.001$. The lowest decrease in SOD was in factory chronic collection exposure group (GIc) ($0.9 \pm 0.07 \text{ U/mg}$), while the highest decrease on its level was done after chronic exposure to furnace predominant frequency dose (GIIIcf) ($0.69 \pm 0.15 \text{ U/mg}$). Table: 9, Fig.62.

Table (9): Changes on the SOD level (U/ mg protein) after noise exposure

	G0	G1a	G11a	G111a	G1af	G11af	G111af	G1c	G11c	G111c	G1cf	G11cf	G111cf	F	p
Min.	1.59	1.41	1.82	1.03	1.49	1.70	1.44	0.83	0.60	0.78	0.60	0.43	0.46	35.377*	<0.001*
Max.	3.04	2.54	2.60	2.24	2.36	2.60	2.79	0.98	1.10	0.93	0.93	0.84	0.90		
Mean	1.99	2.03	2.12	1.78	1.80	1.93	1.79	0.90	0.88	0.88	0.86	0.70	0.69		
SD.	0.54	0.43	0.31	0.45	0.33	0.30	0.41	0.07	0.13	0.07	0.11	0.19	0.15		
Median	1.75	2.18	1.93	2.01	1.78	1.80	1.68	0.86	0.93	0.93	0.92	0.84	0.70		
p		0.795	0.372	0.132	0.164	0.635	0.141	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*		

F: F test (ANOVA).

p: p value for Post Hoc Test (LSD) for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

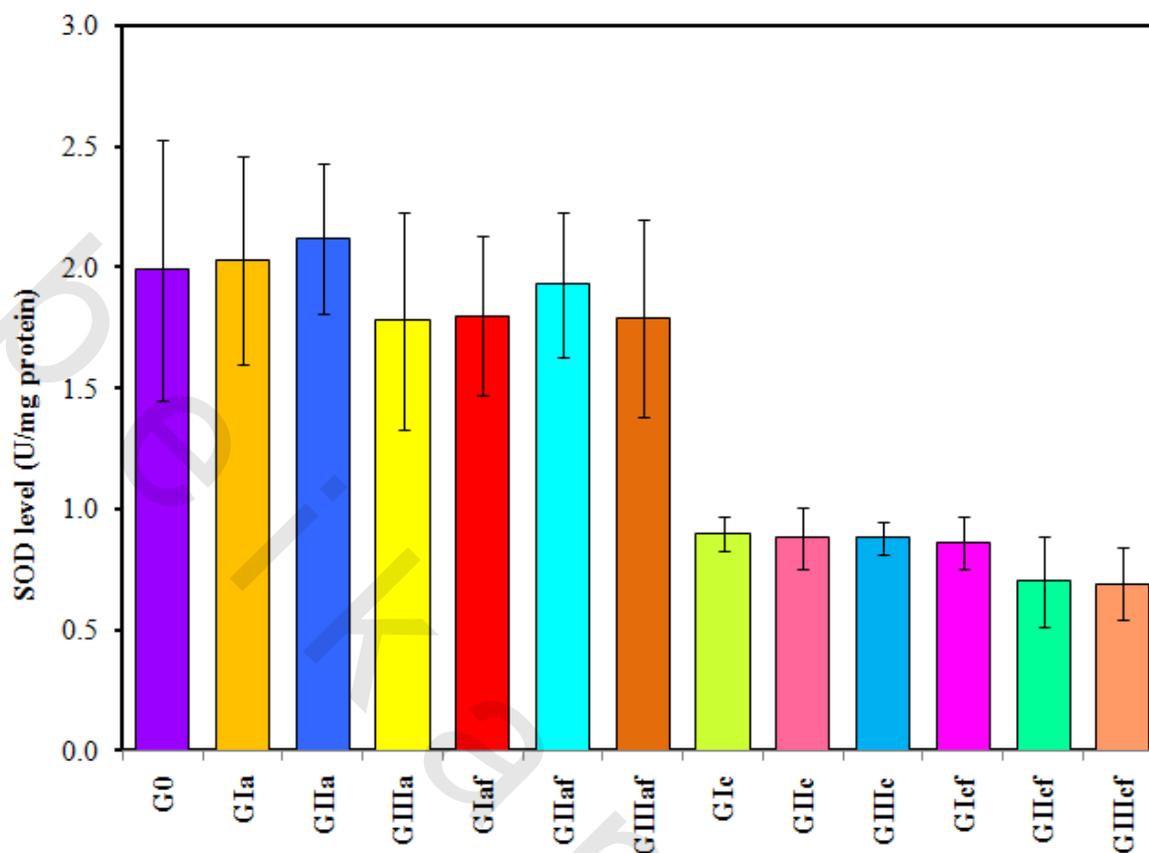


Fig.62. Effect of noise exposure on the SOD level (U/ mg protein).

II.1.4. MDA levels (nmol/mg protein):

MDA levels (nmol/mg protein). were measured in all experimental groups. Control group (G0) mean value was (0.09 ± 0.03) . All acute groups whether collection or predominant frequency show no significance difference than control group. While all chronic groups whether collection or predominant frequency show significance increase than control, $p < 0.001$. The lowest increase in MDA noticed at both chronic exposures to factory and traffic collection doses (GIc, GIIc) $(0.15 \pm 0.02 \text{ nmol/mg})$ for both groups, while the highest increase in MDA level was occurred after chronic exposure to furnace predominant frequency (GIIIcf) $(0.19 \pm 0.04 \text{ nmol/mg})$. Table: 10, Fig.63.

Table (10): Effect of noise exposure on the MDA level (nmol/mg protein)

	G0	G1a	G1Ia	G1IIa	G1af	G1Iaf	G1IIaf	G1c	G1Ic	G1IIc	G1cf	G1Icf	G1IIcf	χ^2 ^{KW}	p
Min.	0.05	0.05	0.06	0.04	0.04	0.04	0.03	0.12	0.12	0.15	0.13	0.14	0.15	104.195*	<0.001*
Max.	0.12	0.09	0.10	0.08	0.10	0.09	0.10	0.18	0.17	0.20	0.21	0.19	0.25		
Mean	0.09	0.07	0.08	0.06	0.07	0.07	0.07	0.15	0.15	0.17	0.17	0.17	0.19		
SD.	0.03	0.01	0.01	0.01	0.02	0.02	0.03	0.02	0.02	0.01	0.03	0.02	0.04		
Median	0.08	0.08	0.08	0.07	0.06	0.07	0.08	0.15	0.15	0.17	0.15	0.17	0.17		
p		0.486	0.759	0.106	0.168	0.125	0.401	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*		

χ^2 : Chi square for Kruskal Wallis test.

p: p value for Mann Whitney test for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

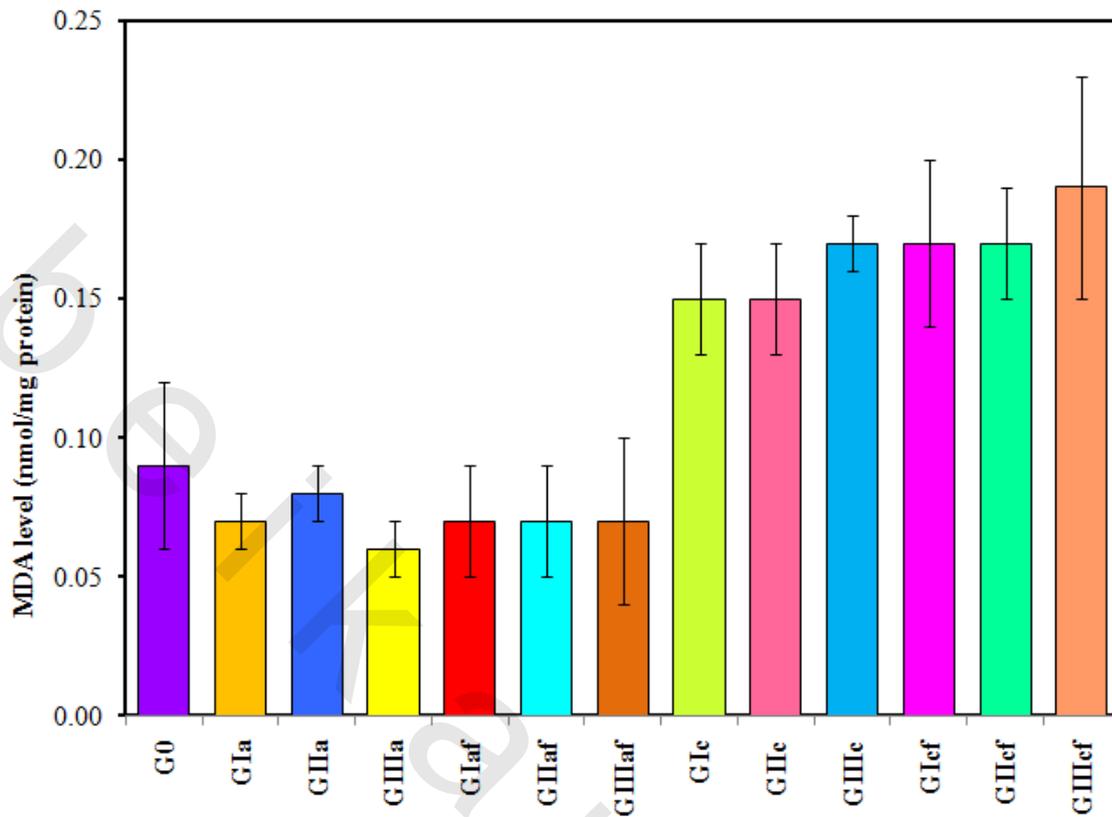


Fig.63. Effect of noise exposure on the MDA level (nmol/mg protein).

II.1.5. Serotonin levels ($\mu\text{g/ml}$):

Serotonin levels ($\mu\text{g/ml}$) were measured in all experimental groups. Control group (G0) mean value was $(0.37 \pm 0.07 \mu\text{g/ml})$. No group shows any significance difference than control group. Table: 11, Fig.64.

Table (11): Effect of noise exposure on the serotonin level ($\mu\text{g}/\text{ml}$)

	G0	G1a	G1b	G1c	G1d	G1e	G1f	G1g	G1h	G1i	G1j	G1k	G1l	F	P
Min.	0.31	0.28	0.27	0.23	0.29	0.33	0.29	0.33	0.24	0.29	0.32	0.25	0.23	3.336*	<0.001*
Max.	0.50	0.49	0.42	0.45	0.42	0.47	0.45	0.49	0.40	0.41	0.50	0.43	0.41		
Mean	0.37	0.39	0.36	0.33	0.34	0.40	0.37	0.40	0.32	0.34	0.41	0.34	0.32		
SD.	0.07	0.06	0.05	0.07	0.04	0.05	0.06	0.05	0.05	0.04	0.06	0.05	0.05		
Median	0.33	0.40	0.37	0.33	0.34	0.41	0.38	0.39	0.32	0.34	0.44	0.35	0.32		
p		0.359	0.690	0.121	0.319	0.131	0.873	0.152	0.068	0.402	0.057	0.300	0.103		

F: F test (ANOVA).

p: p value for Post Hoc Test (LSD) for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

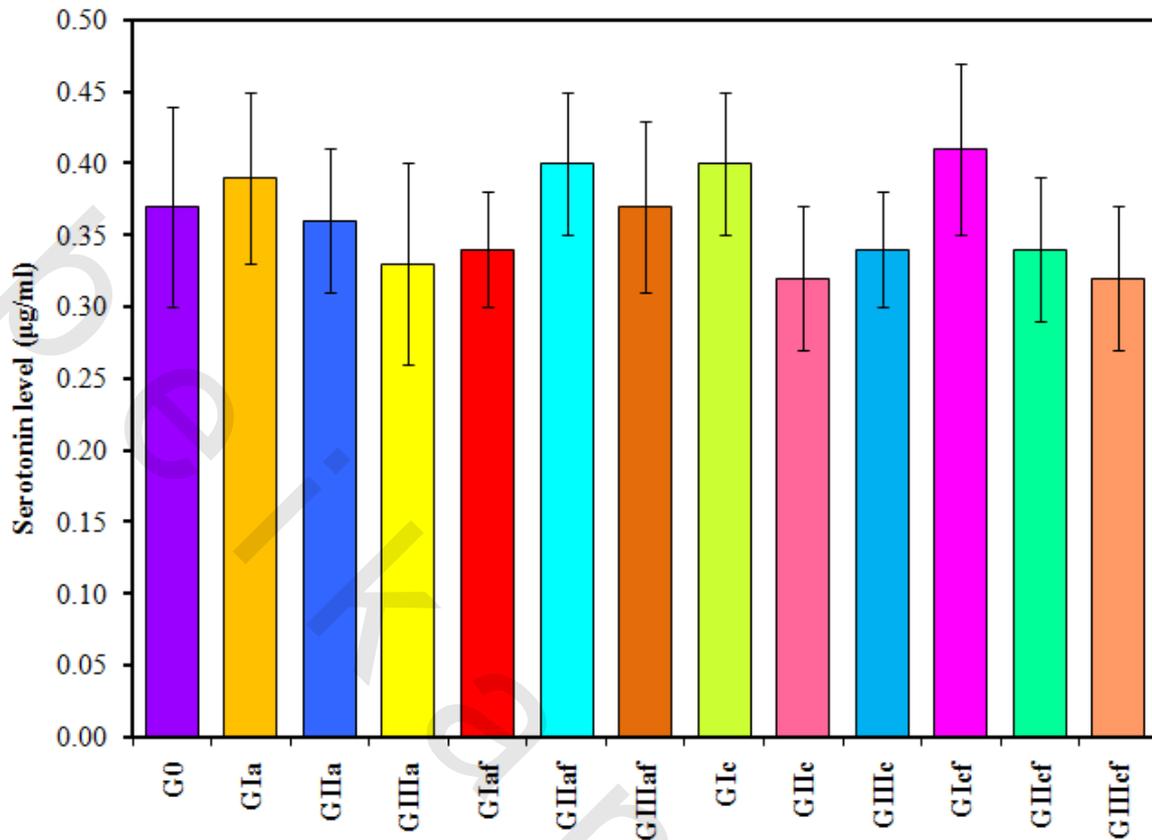


Fig.64. Effect of noise exposure on the serotonin level (µg/ml).

II.1.6. Recognition Index values:

Recognition Index as a parameter of short memory was calculated in all groups. It's clear that there was no significant difference between control group (89.20 ± 1.23) and all groups exposed to acute noise dose (whether collection or predominant frequency). On the other hand all chronic groups (collection & predominant frequency) showed significant reduction in the RI as compared to control one ($p < 0.001$) as shown in Table 12, Fig.65.

Table (12): Effect of noise exposure on the Recognition Index

	G0	G1a	G1b	G1c	G1d	G1e	G1f	G1g	G1h	G1i	G1j	G1k	G1l	F	P
Min.	88.0	84.0	78.0	82.0	86.0	81.0	82.0	54.0	57.0	56.0	53.0	52.0	54.0		
Max.	91.0	93.0	91.0	90.0	91.0	93.0	92.0	68.0	64.0	66.0	64.0	62.0	62.0		
Mean	89.20	87.40	86.40	87.20	88.80	87.40	86.80	60.00	59.60	60.00	58.60	58.40	59.20	165.617*	<0.001*
SD.	1.23	3.44	4.72	2.94	1.81	4.65	3.74	4.94	2.55	3.83	4.25	3.81	2.94		
Median	89.0	86.0	86.0	88.0	89.0	88.0	87.0	60.0	59.0	59.0	60.0	60.0	60.0		
P		0.268	0.086	0.218	0.805	0.268	0.140	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*		

F: F test (ANOVA).

p: p value for Post Hoc Test (LSD) for comparing between G0 and each other group.

*: Statistically significant at $p \leq 0.05$.

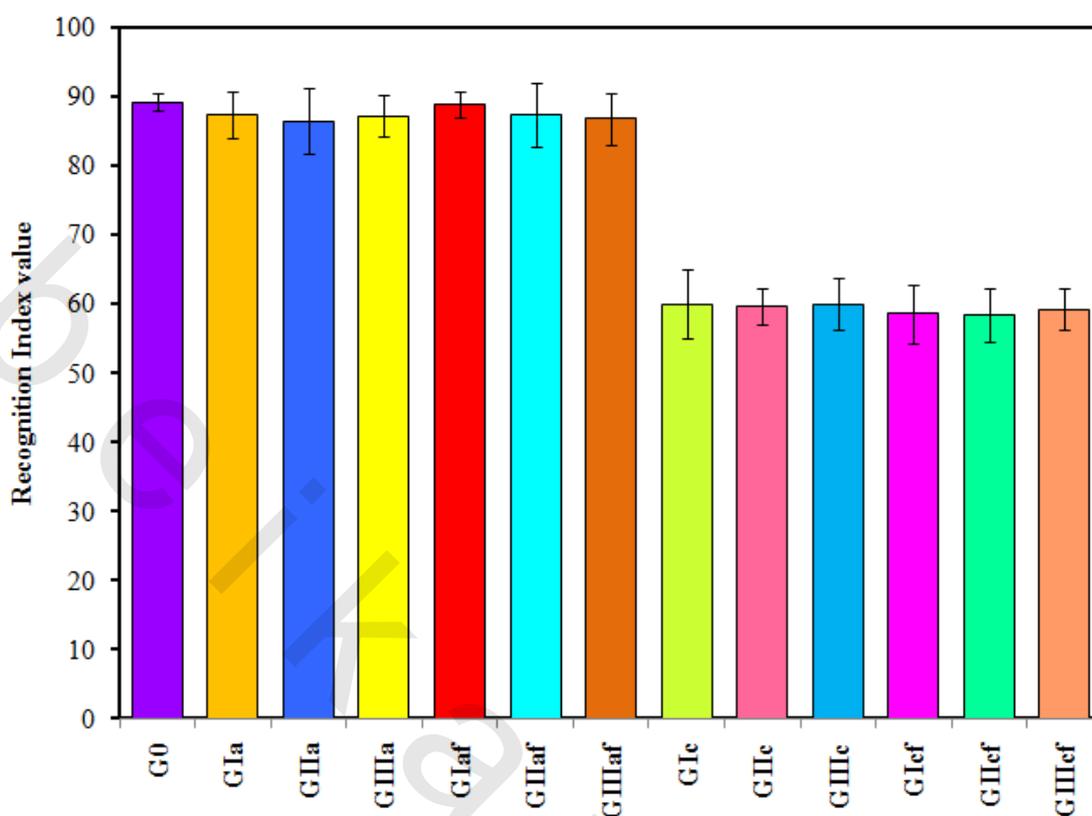


Fig.65: Effect of noise exposure on the Recognition Index.

II.2. Alternations associated with different types of noise individually:

II.2.1. Alternations associated with factory noise exposure:

Table: 13 shows parameters measured after exposure to factory noise acute collection (GIa), acute predominant (GIaf), chronic collection (GIc), and chronic predominant (GIcf) groups in comparison to control group (G0) and to each other.

- **Glucose levels (mg/dl) as measured for mice after exposure to factory noise:**

Blood glucose levels (mg/dl) were measured in all groups. In control group (G0) its mean value was $(87.40 \pm 3.92 \text{ mg/dl})$, while the mean values were $(42.60 \pm 8.71 \text{ mg/dl})$ and $(28.60 \pm 1.43 \text{ mg/dl})$ in acute collection group (GIa) and acute predominant group (GIaf) respectively. Both groups show significant decreased in comparison to the control group, $p < 0.001$. Also, blood glucose mean value in chronic collection group (GIc) was $(30.80 \pm 4.69 \text{ mg/dl})$, and $(18.60 \pm 4.79 \text{ mg/dl})$ in chronic predominant group (GIcf), blood glucose level in the two latter groups were also significantly decreased than the control group, $p < 0.001$, as shown in Fig.66.

There was significant decrease in level between both the GIaf and the GIa, and between the GIcf and GIc, $p < 0.001$. In accordance, the results showed significant difference between two similar tones upon different patterns of exposure whether acute or chronic, in a way that blood glucose in GIcf was significantly decreased than the GIaf and similarly GIc group was significantly decreased than the GIa, $p < 0.001$.

- **WBCs counts (cells/ μ l) of mice exposed to factory noise:**

WBCs counts (cells/ μ l) were measured in all groups. In G0 its mean value was (11520 \pm 1761.8 cells/ μ l), while the mean value was (7420 \pm 847 cells/ μ l), (6500 \pm 592.5 cells/ μ l), (5400 \pm 1742.8 cells/ μ l), and (3650 \pm 333.33 cells/ μ l) in GIa and GIaf, GIc, and GIcf respectively. All groups show significant decreased in comparison to the G0, $p < 0.001$. as shown in Fig.67.

There was significant difference in WBCs count between both the GIaf and GIa, and between GIcf and GIc, $p < 0.05$. The results also shows significant difference between GIa, and GIc and also between GIcf, and GIaf, $p < 0.001$.

- **SOD levels (U/mg protein) as measured for mice exposed to factory noise:**

The SOD levels (U/mg protein) were measured in all groups. In G0 its mean value was (1.99 \pm 0.54 U/mg), while the mean values were (2.03 \pm 0.43 U/mg) and (1.80 \pm 0.33 U/mg) in GIa and GIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, SOD mean value in GIc was (0.90 \pm 0.07 U/mg), and (0.86 \pm 0.11 U/mg) in GIcf, SOD values in the two latter groups were significantly decreased than the G0, $p < 0.001$. as shown in Fig.68.

There was no significant difference neither between the GIaf and the GIa, nor between the GIcf and the GIc. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that SOD in GIcf was significantly decreased than the GIaf and similarly GIaf was significantly decreased than the GIa, $p < 0.001$.

- **MDA levels (nmol/mg protein) as measured for mice exposed to factory noise:**

The MDA levels (nmol/mg protein) were measured in all groups. In G0 its mean value was (0.09 \pm 0.03 nmol/mg), while the mean value was (0.07 \pm 0.01 nmol/mg) and (0.07 \pm 0.02 nmol/mg) in GIa and GIaf respectively. The latter 2 groups show no significant difference in comparison to the G0. On the other hand, MDA mean value in GIc was (0.15 \pm 0.02 nmol/mg), and (0.17 \pm 0.03 nmol/mg) in GIcf, MDA values in the two latter groups were significantly increased than the G0, $p < 0.001$, Fig.69.

There was no significant difference neither between the GIaf and GIa groups, nor between the GIcf and the GIc groups. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that MDA in GIcf was significantly increased than the GIaf and similarly GIaf was significantly increased than the GIa, $p < 0.001$.

- **Serotonin levels ($\mu\text{g/ml}$) as measured for mice exposed to factory noise:**

The Serotonin levels ($\mu\text{g/ml}$) were measured in all groups. In G0 its mean value was (0.37 ± 0.07), while the mean value was ($0.39\pm 0.06 \mu\text{g/ml}$), ($0.34\pm 0.04 \mu\text{g/ml}$), ($0.40\pm 0.05 \mu\text{g/ml}$), and ($0.41\pm 0.06 \mu\text{g/ml}$) in GIa, GIaf, GIc, and GIcf respectively. All groups show no significant difference in comparison to the G0. As shown in Fig.70.

- **Recognition index value as calculated for mice after exposure to factory noise:**

The Recognition index, as a parameter for short term memory, was calculated in all groups. In G0 its mean value was (89.20 ± 1.23), while the mean value was (87.40 ± 3.44) and (88.80 ± 1.81) in GIa and GIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, RI mean value in chronic collection group (GIc) was (60.0 ± 4.94), and (58.60 ± 4.25) in GIcf, RI values in the two latter groups were significantly decreased than the G0, $p < 0.001$. as shown in Fig.71.

There was no significant difference neither between the GIaf and the GIa, nor between the GIcf and the GIc. In contrast, the results showed significant difference between two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIcf was significantly decreased than the GIaf and similarly GIaf was significantly decreased than the GIa, $p < 0.001$.

Table (13): Parameters measured after exposure to factory noise

Parameters		Control (G ₀)	Acute		Chronic	
			Collection (G _{1a})	Predominant (G _{1af})	Collection (G _{1c})	Predominant (G _{1cf})
Glucose (mg/dl)	Min.	82.0	32.0	27.0	24.0	12.0
	Max.	92.0	53.0	31.0	37.0	25.0
	Mean±SD	87.40±3.92	42.60 ^a ±8.71	28.60 ^{ab} ±1.43	30.80 ^{ac} ±4.69	18.60 ^{abc} ±4.79
WBCs (cells/ μ l)	Min.	9700.0	6100.0	5900.0	3220.0	3350.0
	Max.	14500.0	8400.0	7400.0	7120.0	4250.0
	Mean±SD	11520±1761.8	7420 ^a ±847.0	6500 ^{ab} ±592.5	5400 ^{ac} ±1742.8	3650 ^{abc} ±333.3
SOD (U/mg)	Min.	1.59	1.41	1.49	0.83	0.60
	Max.	3.04	2.54	2.36	0.98	0.93
	Mean±SD	1.99±0.54	2.03±0.43	1.80±0.33	0.90 ^{ac} ±0.07	0.86 ^{ac} ±0.11
MDA (nmol/mg)	Min.	0.05	0.05	0.04	0.12	0.13
	Max.	0.12	0.09	0.10	0.18	0.21
	Mean±SD	0.09±0.03	0.07±0.01	0.07±0.02	0.15 ^{ac} ±0.02	0.17 ^{ac} ±0.03
Serotonin (μ g/ml)	Min.	0.31	0.28	0.29	0.33	0.32
	Max.	0.50	0.49	0.42	0.49	0.50
	Mean±SD	0.37±0.07	0.39±0.06	0.34±0.04	0.40±0.05	0.41±0.06
RI	Min.	88.8	84.0	86.0	54.0	53.0
	Max.	91.0	93.0	91.0	68.0	64.0
	Mean±SD	89.20±1.23	87.40±3.44	88.80±1.81	60.0 ^{ac} ±4.94	58.60 ^{ac} ±4.25

a: Significantly different from control group, $P < 0.001$.

b: Predominant is significantly different from collection within the same pattern of noise exposure (acute, or chronic), $p \leq 0.001$ unless otherwise indicated.

c: Chronic is significantly different from acute exposure to the same noise tone (collection, or predominant), $p \leq 0.001$.

b: In WBCs $p < 0.05$

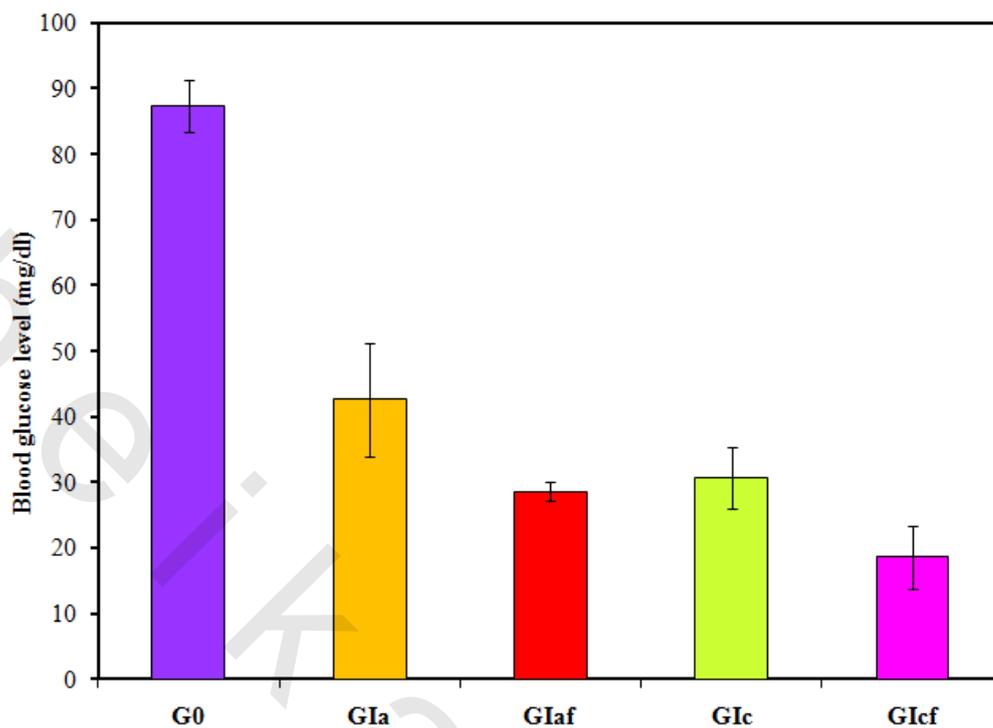


Fig.66: Mean values of blood glucose level (mg/dl) related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group(G0).

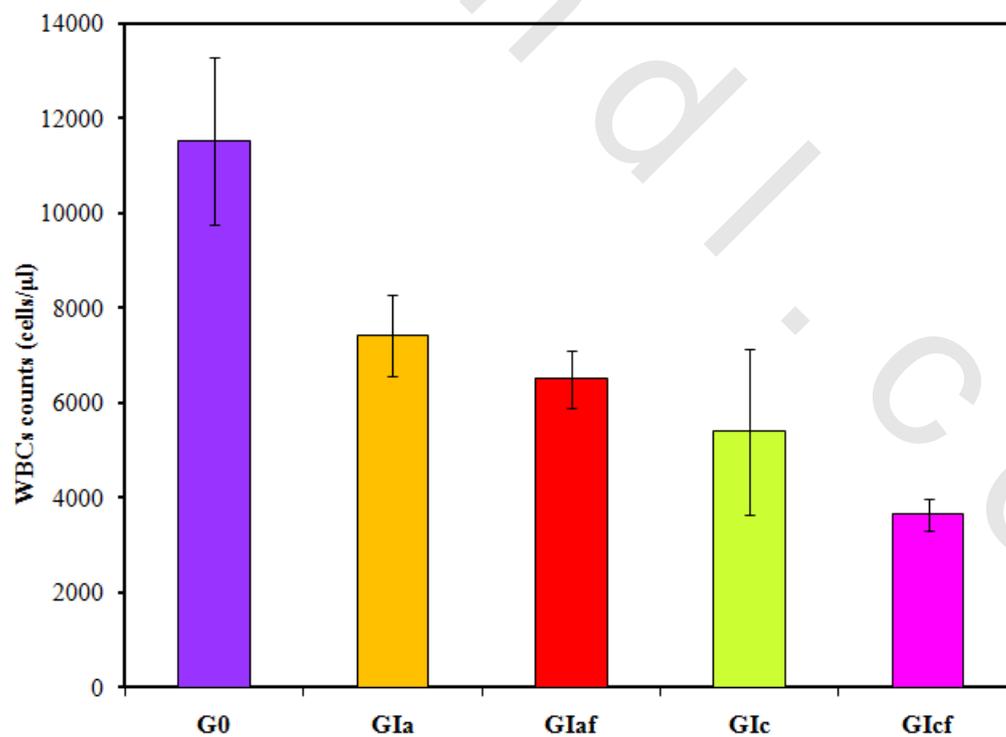


Fig.67: Mean of WBCs counts (cells/μl) related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group (G0).

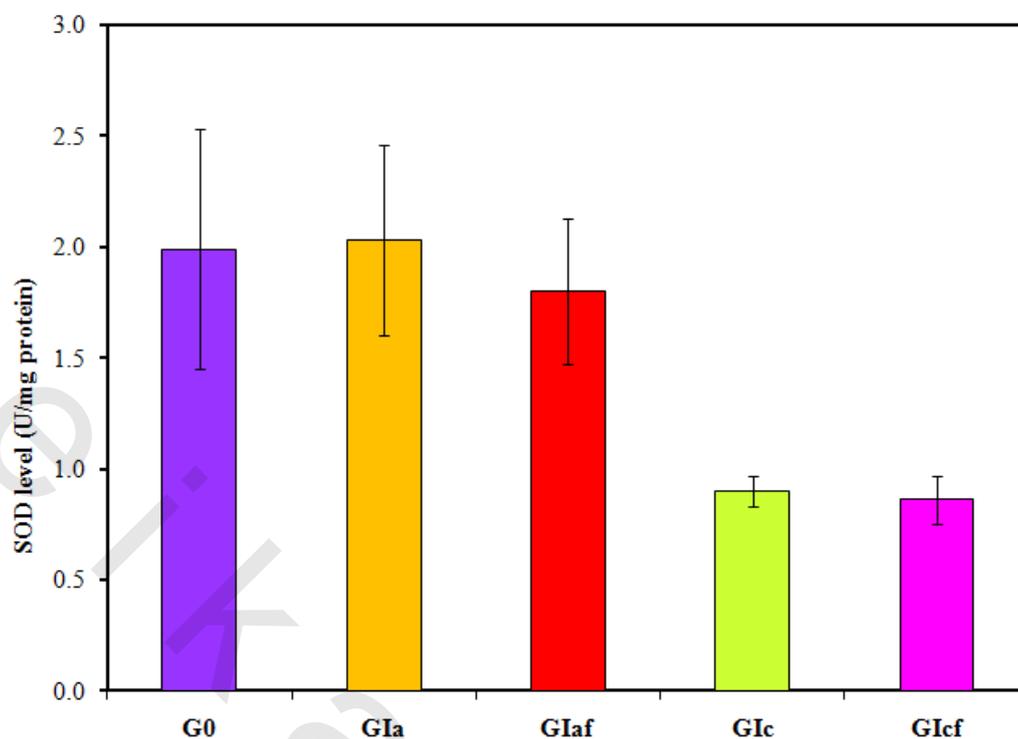


Fig.68: Mean values of SOD (U/mg protein) related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group (G0).

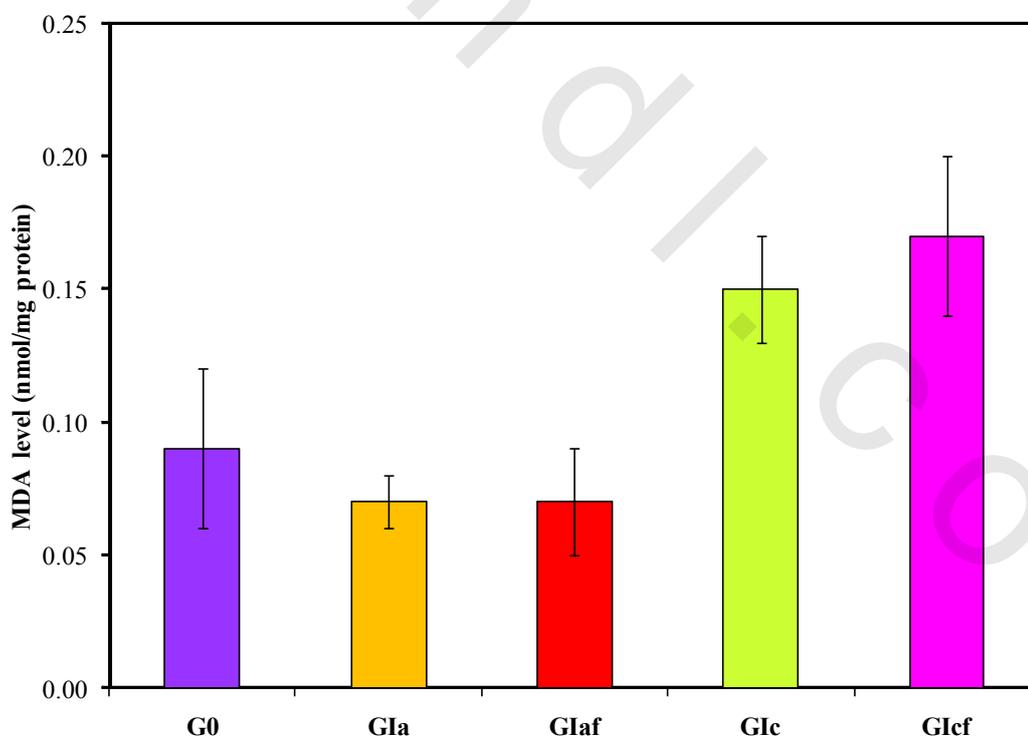


Fig.69: Mean values of MDA (nmol/mg protein) related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group (G0).

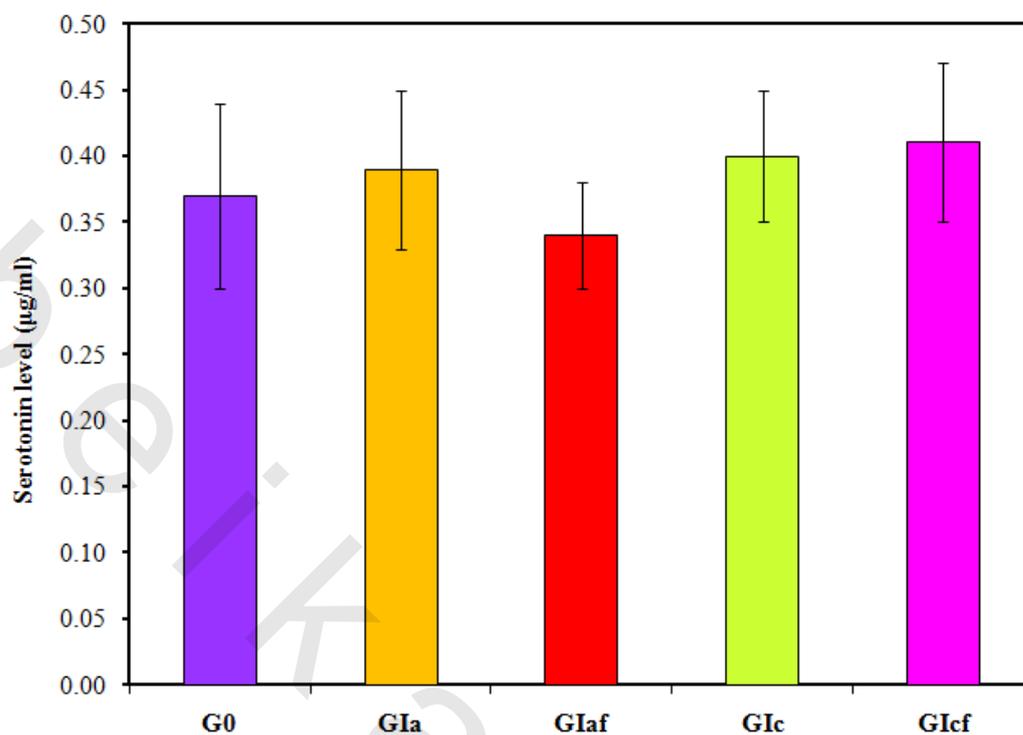


Fig.70: Mean values serotonin (µg/ml) related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group (G0).

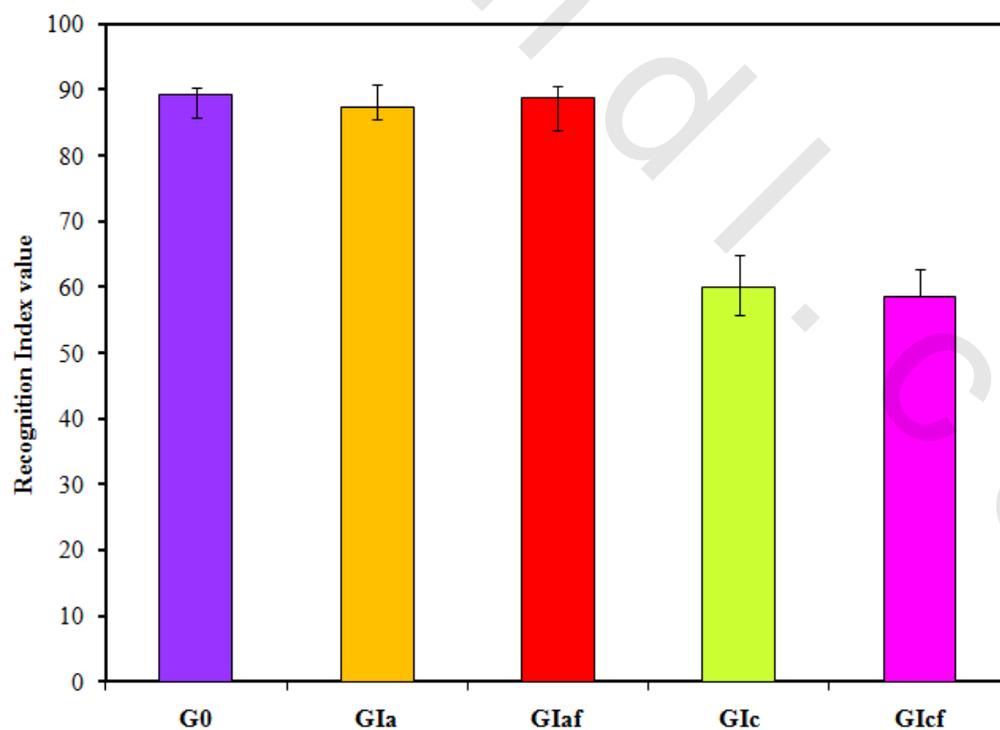


Fig.71: Mean values of RI related to different factory groups (GIa, GIaf, GIc, GIcf) in comparison to control group (G0).

II.2.2. Alternations associated with traffic noise exposure:

Table: 14 shows parameters measured after exposure to traffic noise acute collection (GIIa), acute predominant (GIIaf), chronic collection (GIIc), and chronic predominant (GIIcf) groups in comparison to control group (G0) and to each other.

- **Blood glucose levels (mg/dl) as measured for mice exposed to traffic noise:**

Blood glucose levels (mg/dl) were measured in all experimental groups. In control group (G0) its mean value was (87.40±3.92 mg/dl), while the mean values were (35.60±6.62 mg/dl) and (19.0±2.75 mg/dl) in acute collection group (GIIa) and acute predominant group (GIIaf) respectively. Also, blood glucose mean value in chronic collection group (GIIc) was (22.80 ±2.62 mg/dl), and (8.0±2.11 mg/dl) in chronic predominant group (GIIcf), glucose values in the all groups were also significantly decreased than the control group, $p < 0.001$. As shown in Fig.72.

There was significant decrease between both the GIIaf and the GIIa groups, and between the GIIcf and the GIIc, $p < 0.001$. on the other hand, the results showed significant difference between two similar tones upon different patterns of exposure whether acute or chronic, in a way that glucose in GIIaf was significantly decreased than the GIIaf and similarly GIIc group was significantly decreased than the GIIa group $p < 0.001$.

- **WBCs counts (cells/ μ l) of mice exposed to traffic noise:**

WBCs counts (cells/ μ l) were measured in all groups. In G0 its mean value was (11520±1761.8 cells/ μ l), while the mean value was (6200±368.88 cells/ μ l) and (5120±1069.6 cells/ μ l) (4560 ±1096.7 cells/ μ l), and (3450±462.4 cells/ μ l) in GIIa, GIIaf, GIIc, and GIIcf respectively. All groups show significant decreased in comparison to the G0, $p < 0.001$. As shown in Fig.73.

There was significant difference in WBCs count between both the GIIaf and GIIa, and between GIIcf and GIIc $p < 0.05$. The results also shows significant difference between GIIa, and GIIc and also between GIIcf, and GIIaf, $p < 0.05$.

- **SOD levels (U/mg protein) as measured for mice exposed to traffic noise:**

The SOD levels (U/mg protein), were measured in all groups. In G0 its mean value was (1.99±0.54 U/mg), while the mean value was (2.12±0.31 U/mg) and (1.93±0.30 U/mg) in GIIa and GIIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, SOD mean value in GIIc was (0.88 ±0.13 U/mg), and (0.70±0.19 U/mg) in GIIcf, SOD values in the two latter groups were significantly decreased than the G0, $p < 0.001$. as shown in Fig.74.

There was no significant difference neither between the GIIaf and GIIa groups, nor between the GIIcf and the GIIc groups. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIcf was significantly decreased than the GIIaf and similarly GIIaf was significantly decreased than the GIIa, $p < 0.001$.

- **MDA levels (nmol/mg protein) as measured for mice exposed to traffic noise:**

The MDA levels (nmol/mg protein), were measured in all groups. In G0 its mean value was (0.09±0.03 nmol/mg), while the mean value was (0.08±0.01 nmol/mg) and (0.07±0.02 nmol/mg) in GIIa and GIIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, MDA mean value in GIIc was (0.15 ±0.02 nmol/mg), and (0.17 ±0.02 nmol/mg) in GIIcf, MDA values in the two latter groups were significantly increased than the G0, $p<0.001$. as shown in Fig75.

There was no significant difference neither between the GIIaf and GIIa groups, nor between the GIIcf and the GIIc groups. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIcf was significantly decreased than the GIIaf and similarly GIIaf was significantly decreased than the GIIa, $p<0.001$.

- **Serotonin levels (µg/ml) in traffic noise exposed groups:**

The serotonin levels (µg/ml) were measured in all groups. In G0 its mean value was (0.37±0.07 µg/ml), while the mean value was (0.36±0.05 µg/ml), (0.40±0.05 µg/ml), (0.32 ±0.05 µg/ml), and (0.34±0.05 µg/ml) in GIIa, GIIaf, GIIc, and GIIcf respectively. All groups show no significant difference in comparison to the G0. As shown in Fig.76.

- **Recognition index value in traffic noise exposed groups:**

The Recognition index (short term memory parameter), was calculated in all groups. The mean values of RI in mice exposed to acute noise (collection and predominant) were (86.40±4.72) and (87.40±4.65) respectively in comparison with control (89.20±1.23). On the other hand the mean RI values as calculated for mice exposure to chronic noise (collection and predominant) were (59.60 ±2.55), and (58.40±3.81), all show significant difference with control group at ($p<0.001$), Fig.77.

There was no significant difference neither between the GIIaf and GIIa groups, nor between the GIIcf and the GIIc groups. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIcf was significantly decreased than the GIIaf and similarly GIIaf was significantly decreased than the GIIa, $p<0.001$.

Table (14): Parameters measured after exposure to traffic noise

Parameters		Control (G ₀)	Acute		Chronic	
			Collection (GII _a)	Predominant (GII _{af})	Collection (GII _c)	Predominant (GII _{cf})
Glucose (mg/dl)	Min.	82.0	27.0	14.0	20.0	5.0
	Max.	92.0	45.0	21.0	27.0	11.0
	Mean±SD	87.40±3.92	35.60 ^a ±6.62	19.0 ^{ab} ±2.75	22.80 ^{ac} ±2.62	8.0 ^{abc} ±2.11
WBCs (cells/μl)	Min.	9700.0	5860.0	3200.0	3600.0	3110.0
	Max.	14500.0	6760.0	6100.0	6100.0	4110.0
	Mean±SD	11520±1761.8	6200 ^a ±368.8	5120 ^{ab} ±1069.6	4560 ^{ac} ±1096	3450 ^{abc} ±462.4
SOD (U/mg)	Min.	1.59	1.82	1.70	0.60	0.43
	Max.	3.04	2.60	2.60	1.10	0.84
	Mean±SD	1.99±0.54	2.12±0.31	1.93±0.30	0.88 ^{ac} ±0.13	0.70 ^{ac} ±0.19
MDA (nmol/mg)	Min.	0.05	0.06	0.04	0.12	0.14
	Max.	0.12	0.10	0.09	0.17	0.19
	Mean±SD	0.09±0.03	0.08±0.01	0.07±0.02	0.15 ^{ac} ±0.02	0.17 ^{ac} ±0.02
Serotonin (μg/ml)	Min.	0.31	0.27	0.33	0.24	0.25
	Max.	0.50	0.42	0.47	0.40	0.43
	Mean±SD	0.37±0.07	0.36 ± 0.05	0.40 ± 0.05	0.32 ± 0.05	0.34 ± 0.05
RI	Min.	88.8	78.0	81.0	57.0	52.0
	Max.	91.0	91.0	93.0	64.0	62.0
	Mean±SD	89.20±1.23	86.40±4.72	87.40±4.65	59.60 ^{ac} ±2.55	58.40 ^{ac} ±3.81

a: Significantly different from control group, P< 0.001.

b: Predominant is significantly different from collection within the same pattern of noise exposure (acute, or chronic), p≤ 0.001, unless otherwise indicated.

c: Chronic is significantly different from acute exposure to the same noise tone (collection, or predominant), p<0.001, unless otherwise indicated.

b,c: In WBCs p< 0.05.

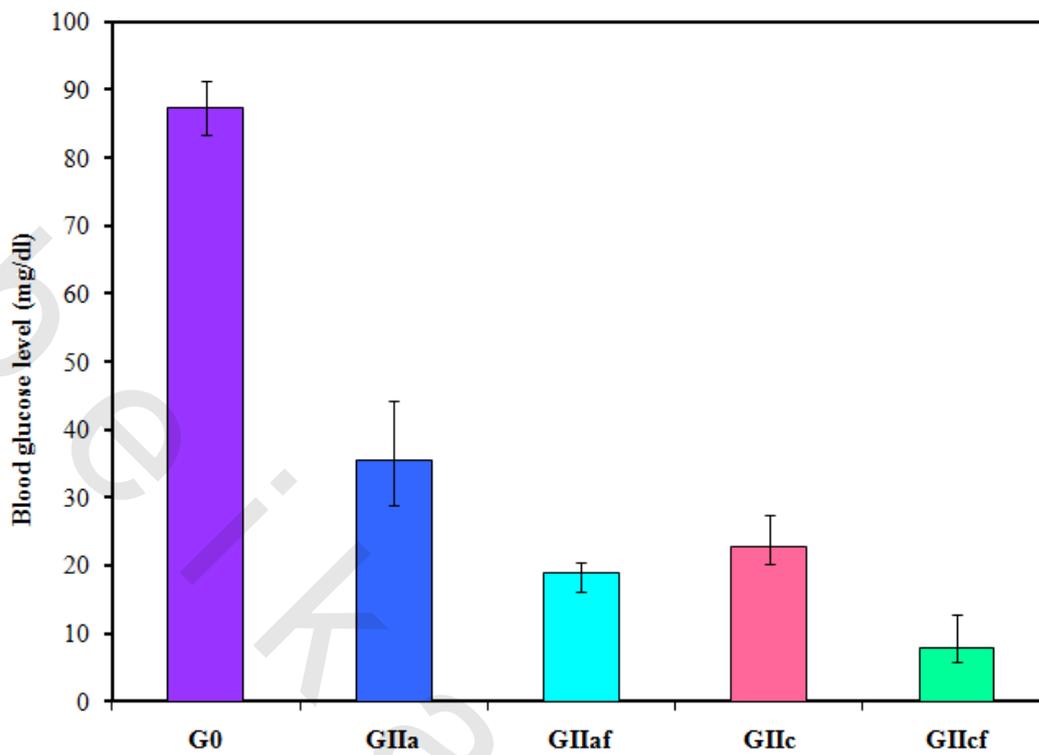


Fig.72: Mean values of blood glucose (mg/dl) related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control (G0).

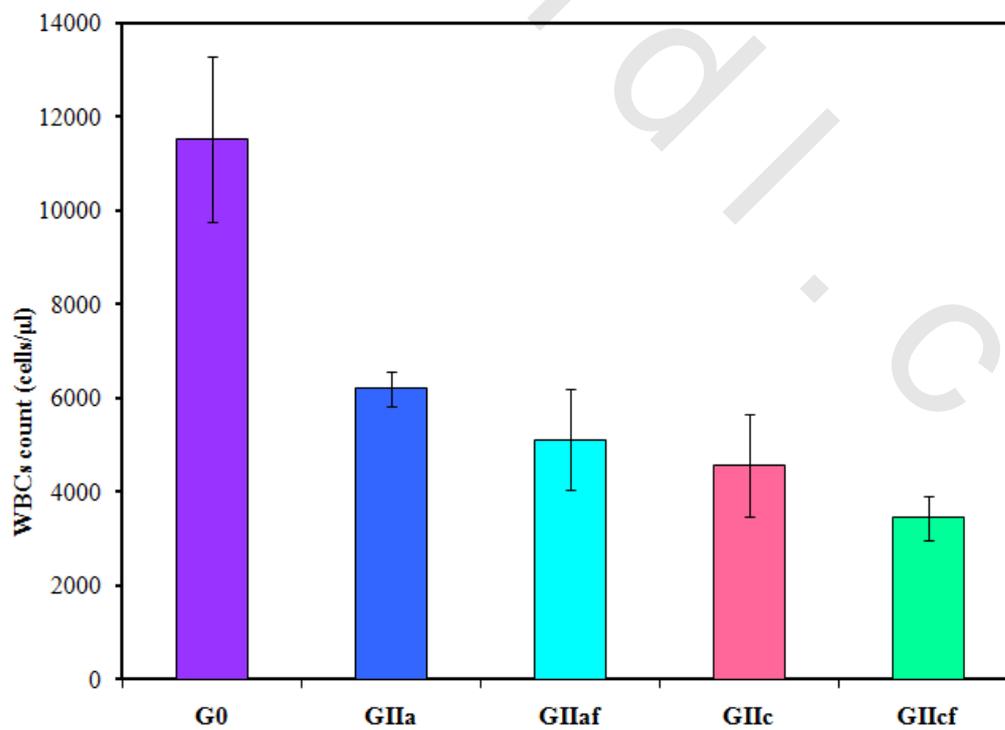


Fig.73: Mean counts of WBCs (cells/μl) related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control (G0).

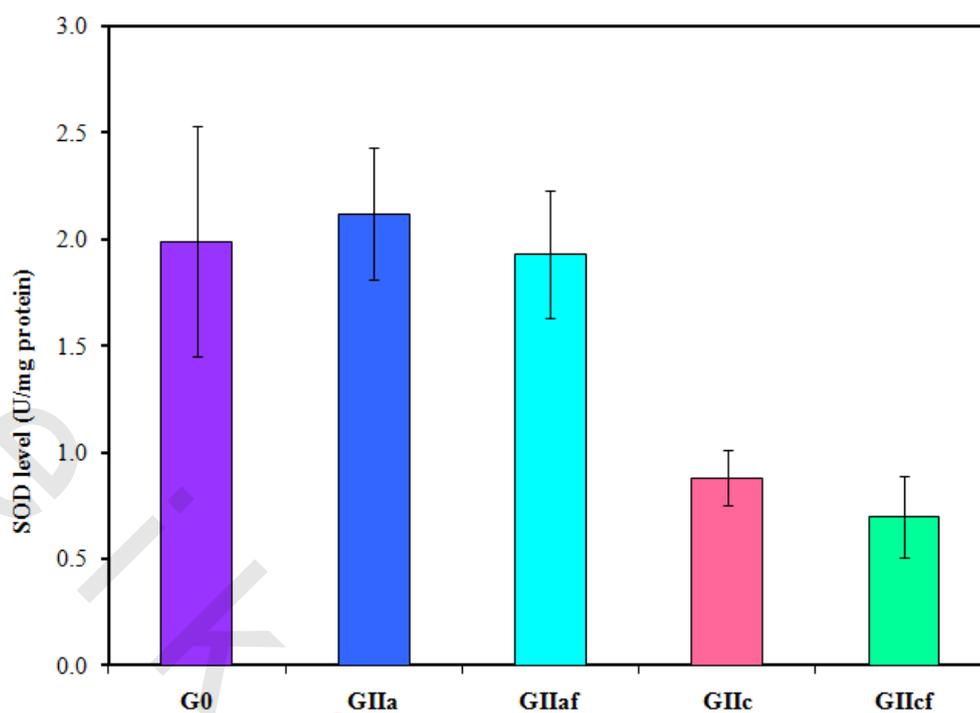


Fig.74: Mean values of SOD (U/mg protein) related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control (G0).

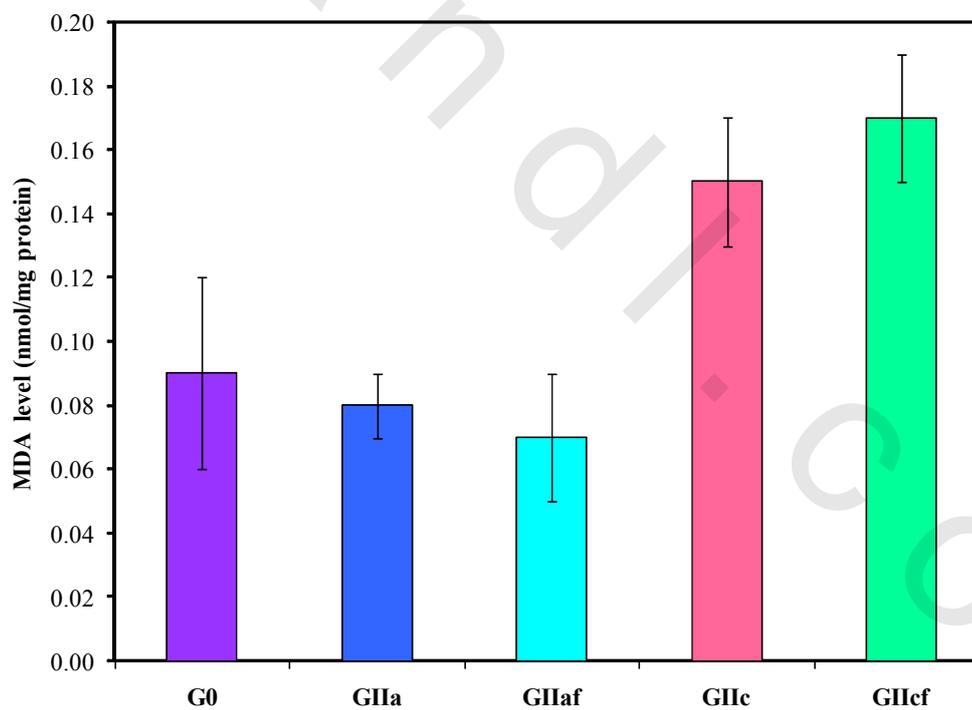


Fig.75: Mean values of MDA (nmol/mg protein) related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control (G0).

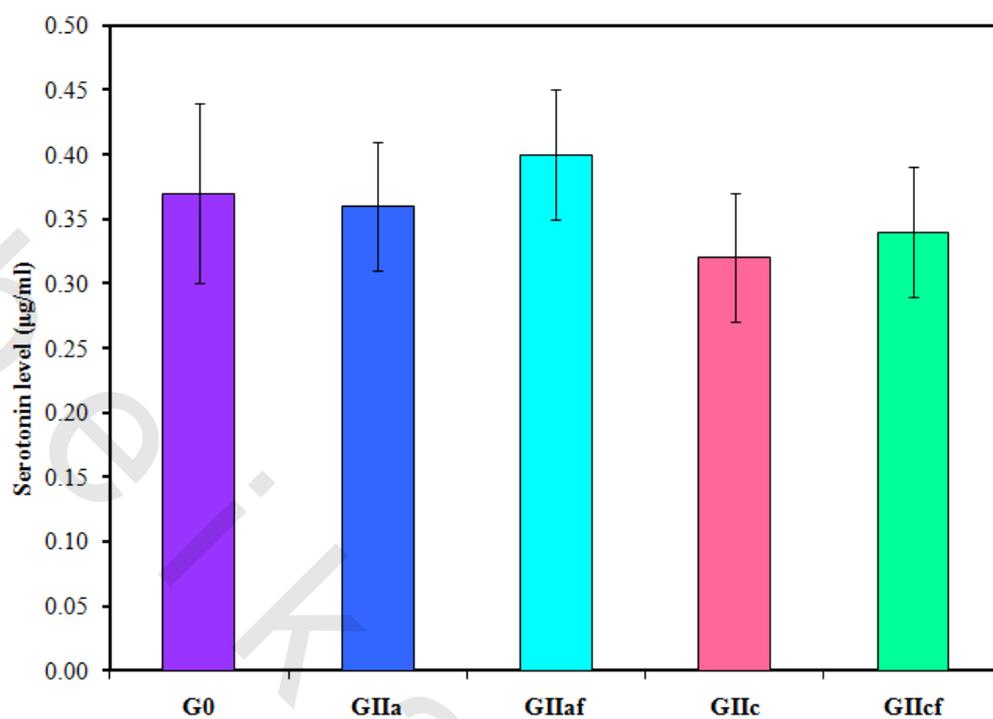


Fig.76: Mean values of serotonin ($\mu\text{g/ml}$) related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control (G0).

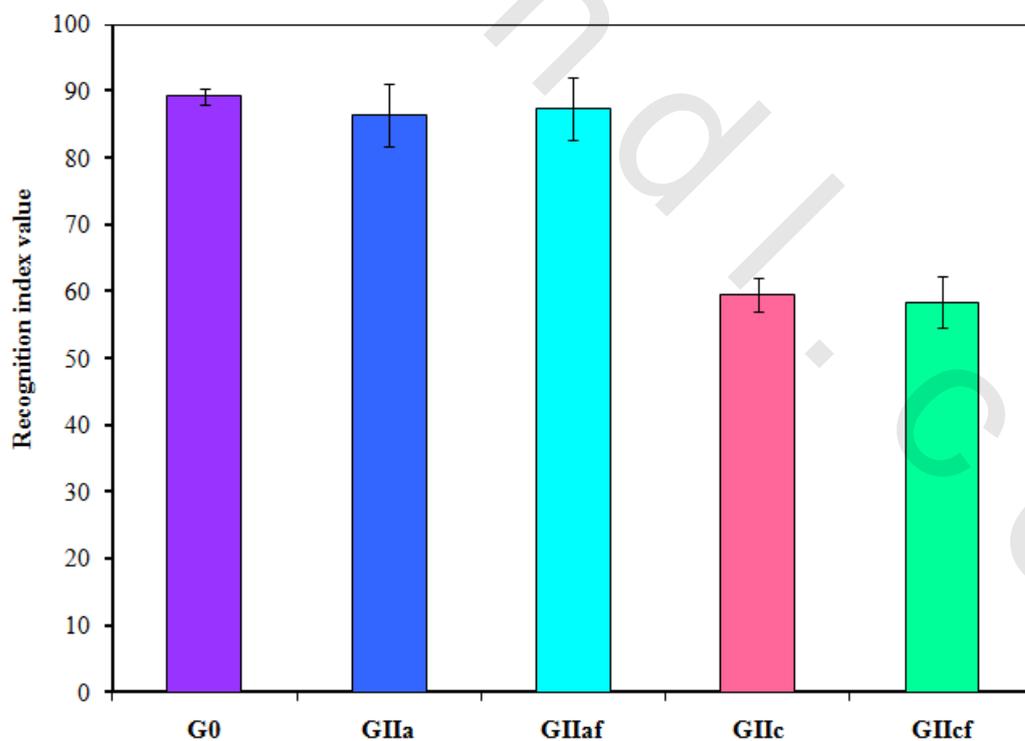


Fig.77: Mean values of RI related to different traffic groups (GIIa, GIIaf, GIIc, GIIcf) in comparison to control group (G0).

II.2.3. Alternations associated with furnace noise exposure:

Table: 15 shows parameters measured after exposure to furnace noise acute collection (GIIIa), acute predominant (GIIIaf), chronic collection (GIIIc), and chronic predominant (GIIIcf) groups in comparison to control group (G0) and to each other.

- **Blood glucose levels (mg/dl) as measured for mice exposed to furnace noise:**

Blood glucose levels (mg/dl) were measured in all experimental groups. In control group (G0) its mean value was (87.40±3.92 mg/dl), while the mean value was (29.60±4.35 mg/dl) and (21.80±5.39 mg/dl) in acute predominant group (GIIIa) and acute predominant group (GIIIaf) respectively. Both groups show significant decreased in comparison to the control group, $p < 0.001$. Also, blood glucose mean value in chronic collection group (GIIIc) was (22.80 ±2.62 mg/dl), and (8.0±4.792.11 mg/dl) in chronic predominant group (GIIIcf), glucose level in the two latter groups were significantly decreased than the control group, $p < 0.001$. as shown in Fig.78.

There was significant decrease between both the GIIIaf and the GIIIa groups, and between the GIIIcf and the GIIIc, $p < 0.001$. on the other hand, the results showed significant difference between two similar tones upon different patterns of exposure whether acute or chronic, in a way that glucose in GIIIaf was significantly decreased than the GIIIaf and similarly GIIIc group was significantly decreased than the GIIIa group $p < 0.001$.

- **WBCs counts (cells/μl) of mice exposed to furnace noise:**

WBCs counts (cells/μl) were measured in all experimental groups. In G0 its mean value was (11520±1761.8 cells/μl), while the mean value was (8440±497.1 cells/μl), (7300±394.4 cells/μl), (6700 ±1559.2 cells/μl), and (5100 ±788.9 cells/μl) in GIIIa, GIIIaf, GIIIc, GIIIcf respectively. WBCs values in the all 4 groups were significantly decreased than the G0, $p < 0.001$. as shown in Fig.79.

There was significant difference in WBCs count between both the GIIIaf and GIIIa, and between GIIIcf and GIIIc $p < 0.05$. The results also shows significant difference between GIIIa, and GIIIc and also between GIIIcf, and GIIIaf, $p < 0.05$.

- **SOD levels (U/mg protein) as measured for mice exposed to furnace noise:**

The SOD levels (U/mg protein) were measured in all experimental groups. In G0 its mean value was (1.99±0.54 U/mg), while the mean value was (1.78±0.45 U/mg) and (1.79±0.41 U/mg) in GIIIa and GIIIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, SOD mean value in GIIIc was (0.88 ±0.07 U/mg), and (0.69±0.15 U/mg) in GIIIcf, SOD values in the two latter groups were significantly decreased than the G0, $p < 0.001$. as shown in Fig.80.

There was no significant difference neither between the GIIIaf and the GIIIa, nor between the GIIIcf and the GIIIc. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIIcf was significantly decreased than the GIIIaf and similarly GIIIc was significantly decreased than GIIIa, $p < 0.001$.

- **MDA levels (nmol/mg protein) as measured for mice exposed to furnace noise:**

The MDA levels (nmol/mg protein), were measured in all experimental groups. In G0 its mean value was (0.09±0.03 nmol/mg), while the mean value was (0.06±0.01 nmol/mg) and (0.07±0.02 nmol/mg) in GIIIa and GIIIaf respectively. Both groups show no significant difference in comparison to the G0. On the other hand, MDA mean value in GIIIc was (0.17 ±0.01 nmol/mg), and (0.19 ±0.04 nmol/mg) in GIIIcf, MDA values in the two latter groups were significantly increased than the G0, $p \leq 0.001$. as shown in Fig.81.

There was no significant difference neither between the GIIIaf and the GIIIa, nor between the GIIIcf and the GIIIc. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIIcf was significantly decreased than the GIIIaf and similarly GIIIc was significantly decreased than GIIIa, $p < 0.001$.

- **Serotonin levels (µg/ml) as measured for mice exposed to furnace noise:**

The serotonin levels (µg/ml) were measured in all experimental groups. In G0 its mean value was (0.37±0.07 µg/ml), while the mean value was (0.33±0.07 µg/ml), (0.37±0.06 µg/ml), (0.34 ±0.04 µg/ml), and (0.32±0.05 µg/ml) in GIIIa , GIIIaf, GIIIc, and GIIIcf respectively. All groups show no significant difference in comparison to the G0, $p < 0.001$, Fig.82.

- **Recognition index value as calculated for mice after exposure to furnace noise:**

The Recognition index (short term memory parameter), was calculated for all experimental groups. The mean values of RI in mice exposed to acute noise (collection and predominant) were (87.20±2.94) and (86.8±3.74) respectively in comparison with control (89.20±1.23). On the other hand the mean RI values as calculated for mice exposure to chronic noise (collection and predominant) were (60.0 ±3.83), and (59.20±2.94), all show significant difference with control group at ($p < 0.001$), Fig.83.

There was no significant difference neither between the GIIIaf and the GIIIa, nor between the GIIIcf and the GIIIc. In contrast, the results showed significant difference between any two similar tones upon different patterns of exposure whether acute or chronic, in a way that RI in GIIIcf was significantly decreased than the GIIIaf and similarly GIIIc was significantly decreased than GIIIa, $p < 0.001$.

Table (15): Parameters measured after exposure to furnace noise

Parameters		Control (G ₀)	Acute		Chronic	
			Collection (GIII _a)	Predominant (GIII _{af})	Collection (GIII _c)	Predominant (GIII _{cf})
Glucose (mg/dl)	Min.	82.0	22.0	13.0	18.0	14.0
	Max.	92.0	34.0	28.0	31.0	20.0
	Mean±SD	87.40±3.92	29.60 ^a ±4.35	21.80 ^{ab} ±5.39	24.4 ^{ac} ±5.68	17.20 ^{abc} ±2.44
WBCs (cells/μl)	Min.	9700.0	7900.0	6900.0	5100.0	4070.0
	Max.	14500.0	9100.0	7900.0	9500.0	5970.0
	Mean±SD	11520±1761.8	8440 ^a ±497.1	7300 ^{ab} ±394.4	6700 ^{ac} ±1559.2	5100 ^{abc} ±788.88
SOD (U/mg)	Min.	1.59	1.03	1.44	0.78	0.46
	Max.	3.04	2.24	2.79	0.93	0.90
	Mean±SD	1.99±0.54	1.78±0.45	1.79±0.41	0.88 ^{ac} ±0.07	0.69 ^{ac} ±0.15
MDA (nmol/m)	Min.	0.05	0.04	0.03	0.15	0.15
	Max.	0.12	0.08	0.10	0.20	0.25
	Mean±SD	0.09±0.03	0.06±0.01	0.07±0.03	0.17 ^{ac} ±0.01	0.19 ^{ac} ±0.04
Serotonin (μg/ml)	Min.	0.31	0.23	0.33	0.29	0.23
	Max.	0.50	0.45	0.47	0.41	0.41
	Mean±SD	0.37±0.07	0.33 ± 0.07	0.40 ± 0.05	0.34 ± 0.04	0.32 ± 0.05
RI	Min.	88.8	82.0	82.0	56.0	54.0
	Max.	91.0	90.0	92.0	66.0	62.0
	Mean±SD	89.20±1.23	87.20±2.94	86.8±3.74	60.0 ^{ac} ±3.83	59.20 ^{ac} ±2.94

a: Significantly different from control group, P< 0.001.

b: Predominant is significantly different from collection within the same pattern of noise exposure (acute, or chronic), p≤ 0.001 unless otherwise indicated.

c: Chronic is significantly different from acute exposure to the same noise tone (collection, or predominant), p<0.001.

b, c: In WBCs p< 0.05.

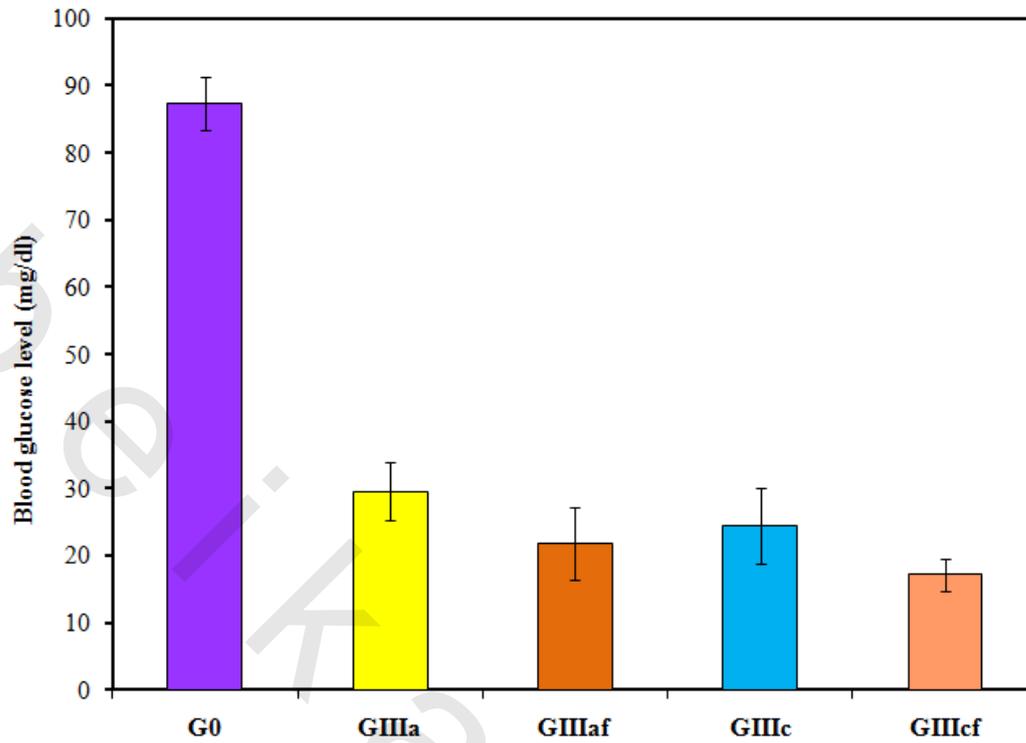


Fig.78: Mean values of blood glucose level (mg/dl) related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

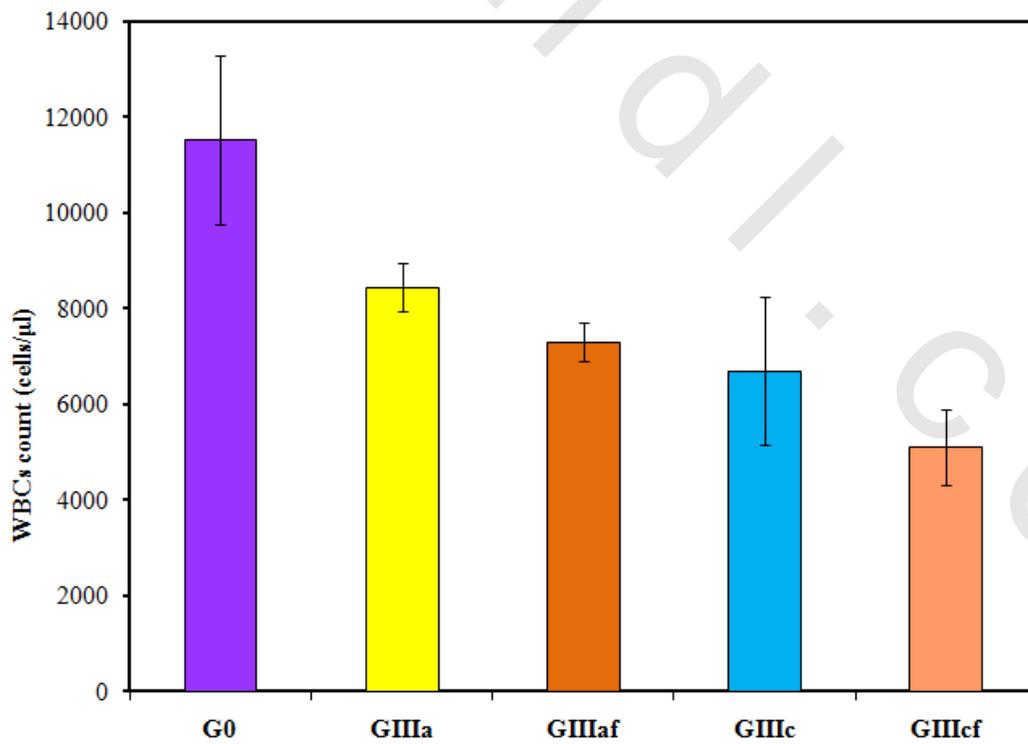


Fig.79: Mean values of WBCs (cells/μl) counts related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

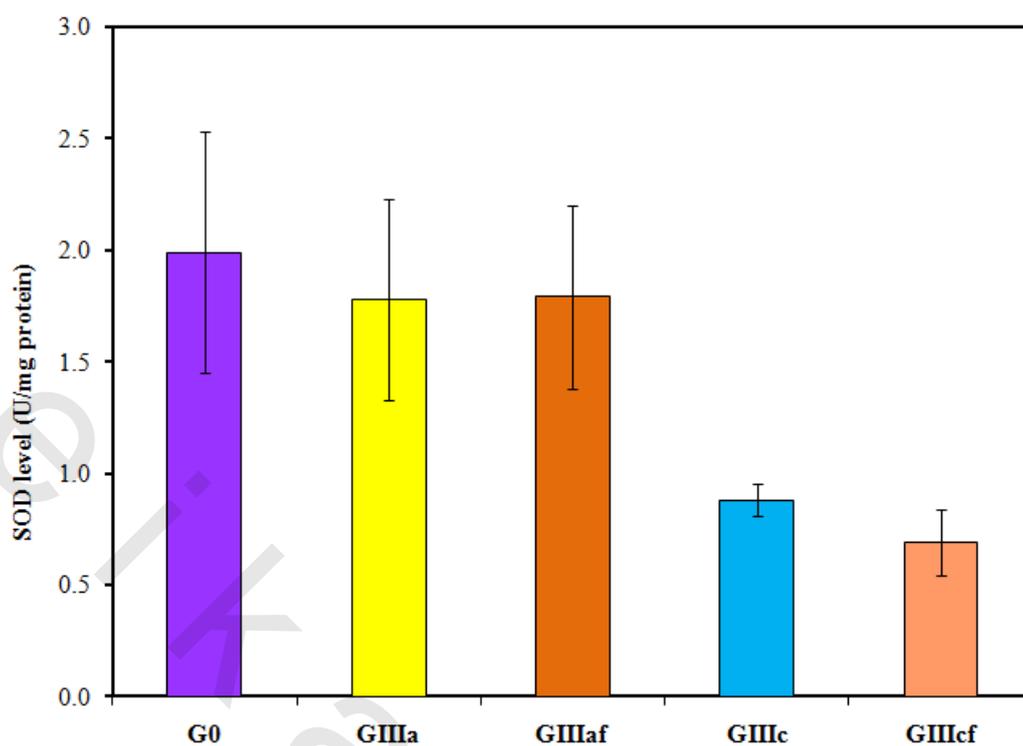


Fig.80: Mean values of SOD (U/mg protein) related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

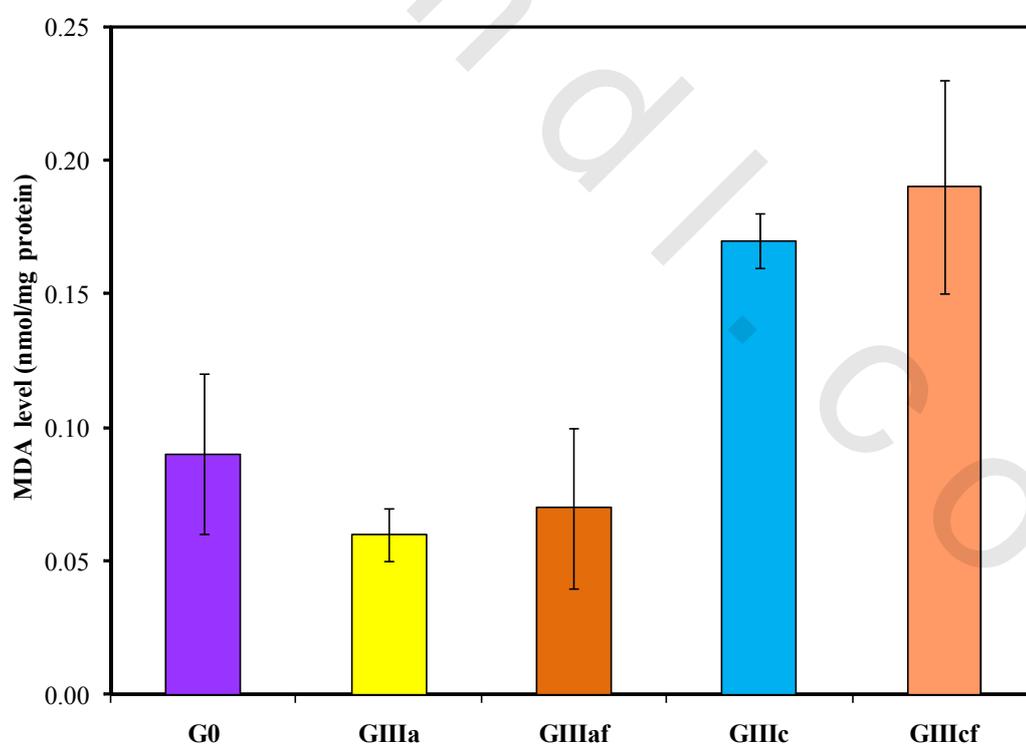


Fig.81: Mean values of MDA (nmol/mg protein) related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

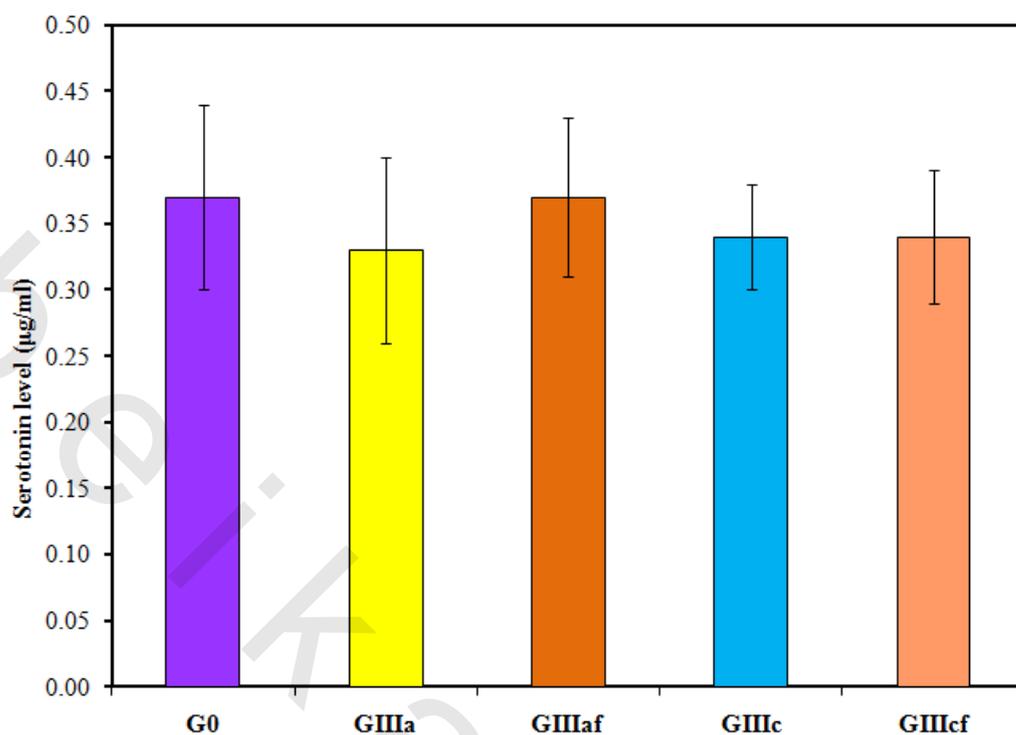


Fig. 82: Mean values of serotonin ($\mu\text{g/ml}$) related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

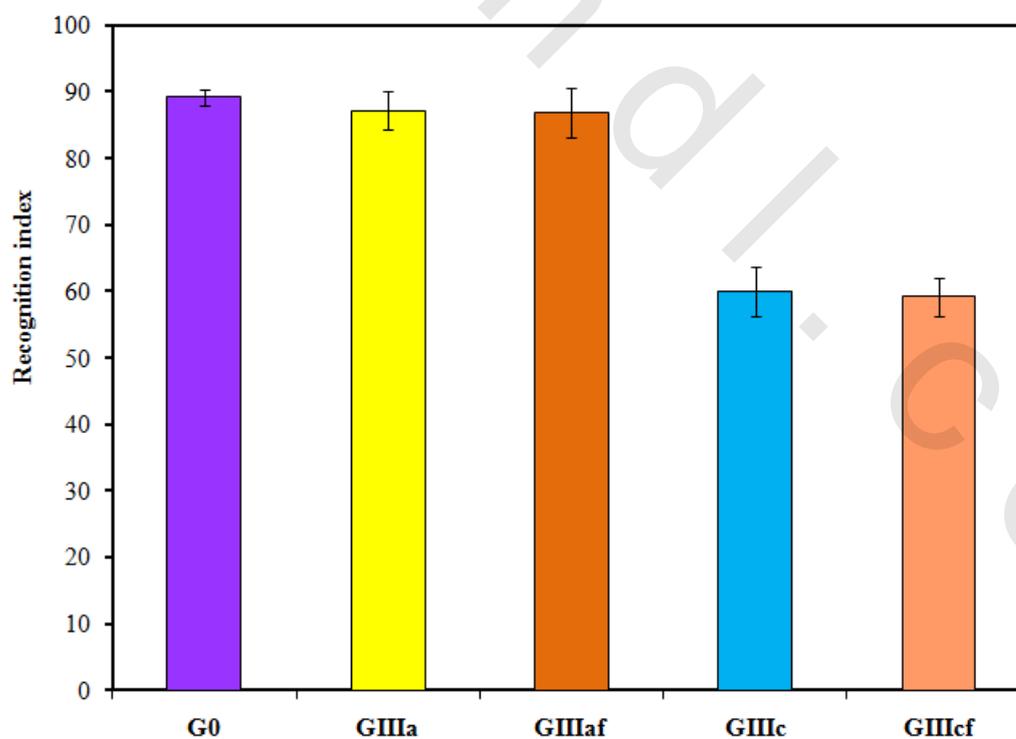


Fig.83: Mean values of RI related to different furnace groups (GIIIa, GIIIaf, GIIIc, GIIIcf) in comparison to control (G0).

II.3. Correlation studies

Correlation between Recognition index and glucose, SOD, MDA was carried, and it was found that there are positive correlation between RI and glucose also between RI and SOD, but there are negative correlation between RI and MDA. (Figs. 84-86)

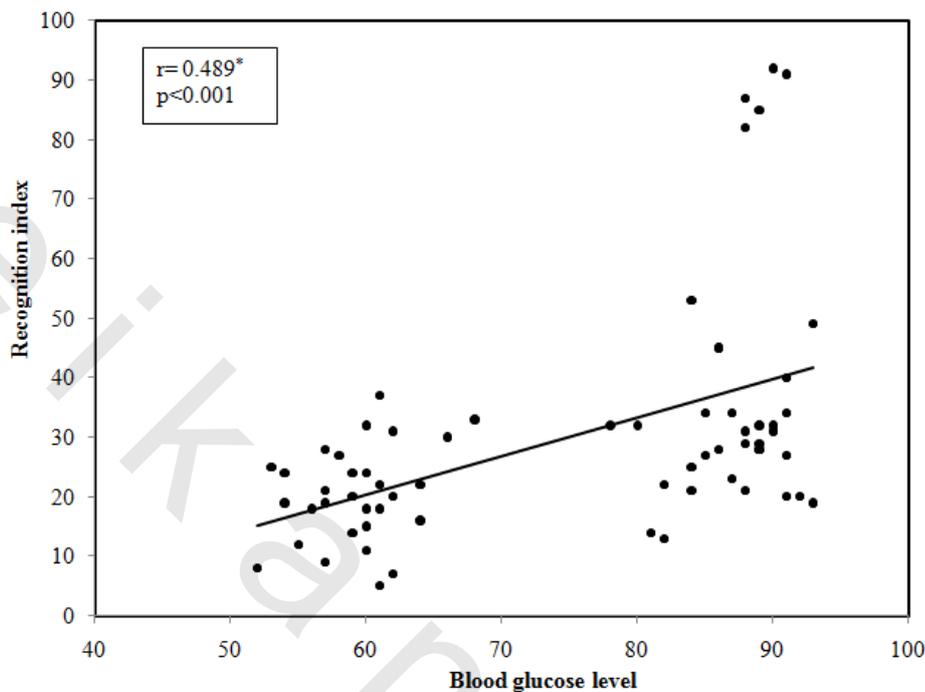


Fig.84: Correlation between recognition index and glucose.

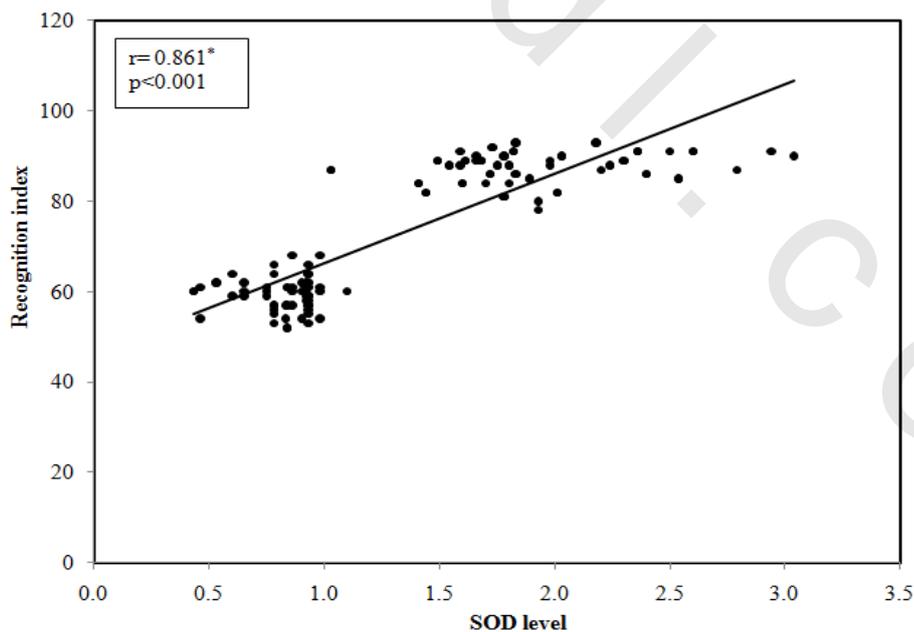


Fig.85: Correlation between recognition index and SOD.

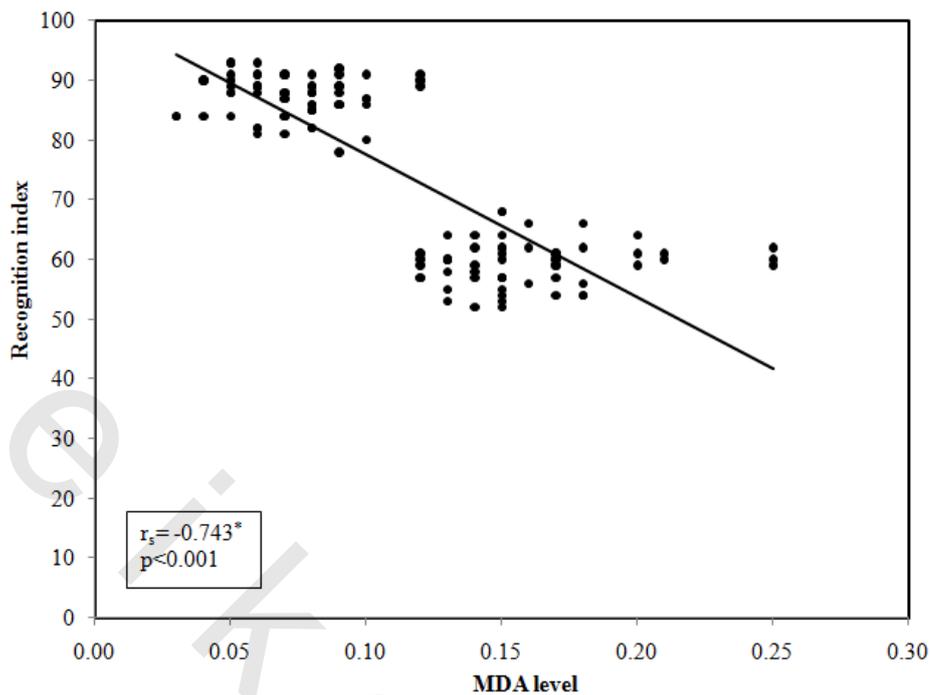


Fig.86: Correlation between recognition index and MDA.

III. Histopathological studies:

The histopathological alternations in brain and small intestine of mice exposed to different modalities of noise in comparison with control (unexposed mice) were studied.

Figures (87-88) show the histological structure of normal brain and small intestine of unexposed mice.

Figures (89 - 112) show the histopathological alternation occur in brain and small intestine of mice exposed to noise. These changes are ranged from mild to moderate as mentioned in Table (16).

Table (16): Changed in brain and small intestine as result of noise exposure ranged from mild to moderate

	G ₀	G _{Ia}	G _{IIa}	G _{IIIa}	G _{Iaf}	G _{IIaf}	G _{IIIaf}	G _{Ic}	G _{IIc}	G _{IIIc}	G _{Icf}	G _{IIcf}	G _{IIIcf}
Brain:													
• Vaculation	-	M	M	M-D	M	D	M	M	D	D	D	M-D	D
• Astrocytosis	-	M	M	M-D	M	D	M	D	D	D	D	M-D	M
• Condensed chromatin	-	-	-	+	-	+	-	-	+	-	-	+	-
Intestine:													
• Inflammation	-	D	M-D	M-D	M	M	M-D	M-D	D	D	D	D	D
• Edema	-	M	M	M	M	M-D	M	M	-	D	M	D	M
• Villi distortion	-	-	M	M	-	M	M	M	M	M	M	M	D

M: mild
D: moderate
M-D: mild to moderate
+ : Confirmed factor

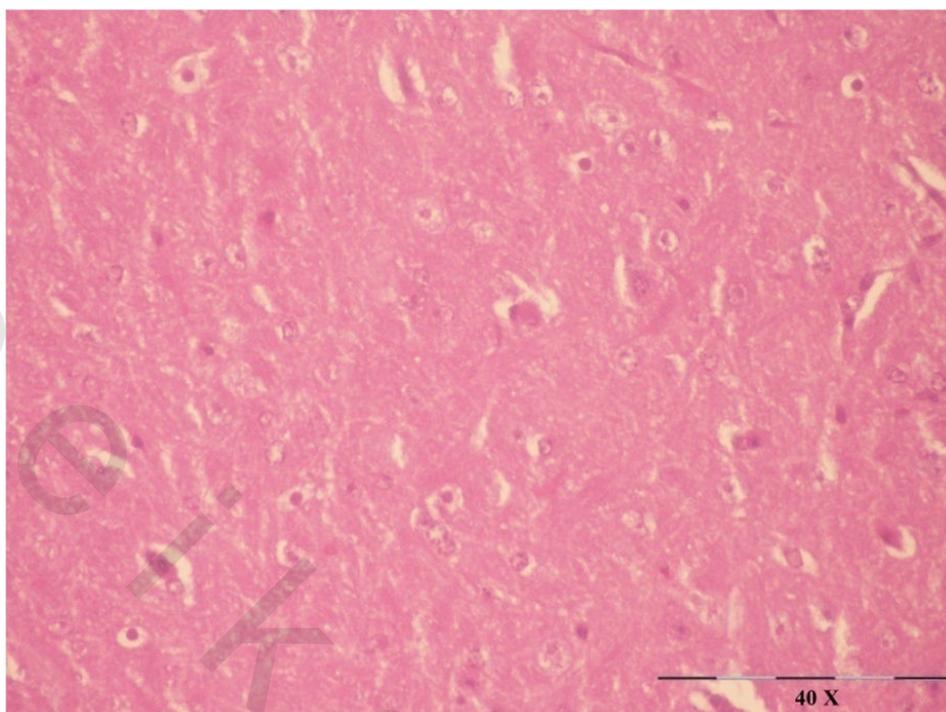


Fig.87: Photomicrograph of brain of normal mouse revealed normal tissue with clear Purkinje fiber without synaptic loss. (H &E, 40X).

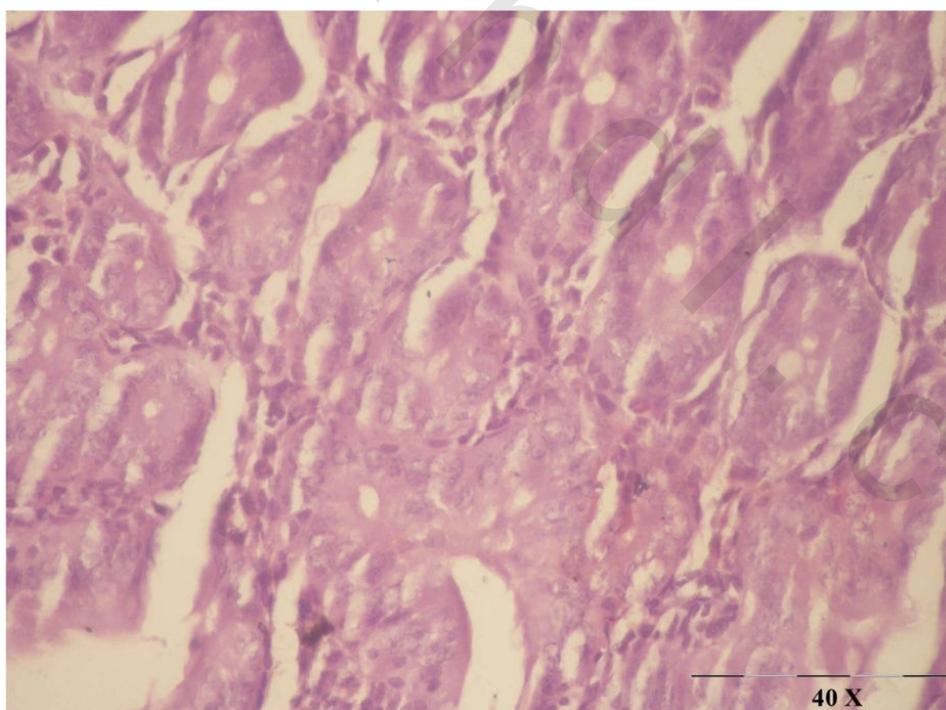


Fig.88: Photomicrograph of small intestine of normal mouse revealed normal villi, normal crypt, without any signs of inflammation. (H &E, 40X).

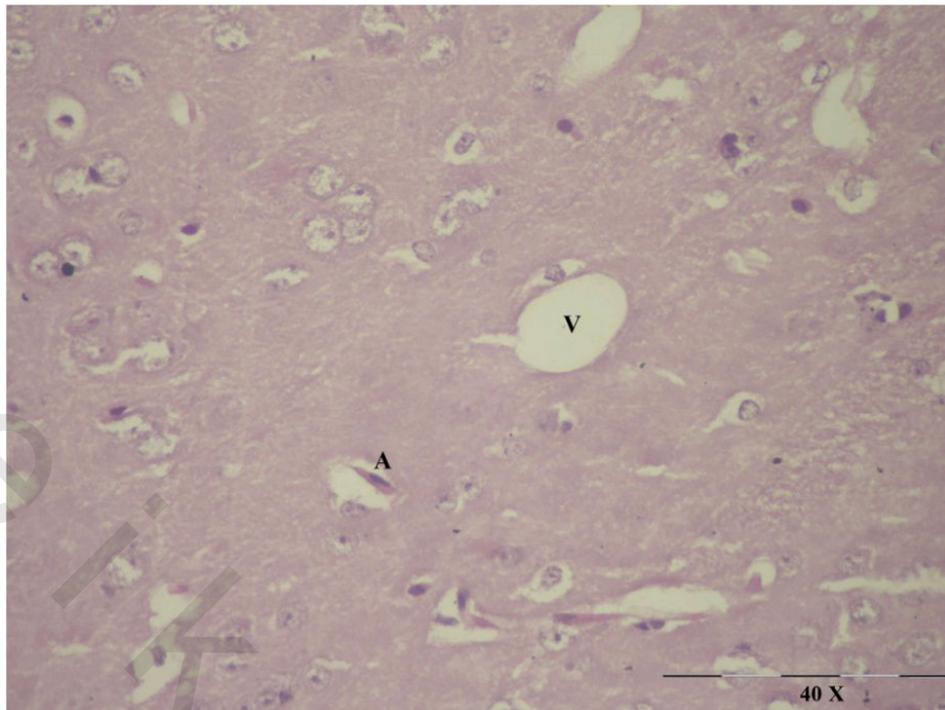


Fig.89: Photomicrograph of brain of mouse exposed to acute factory noise (100 dB, 12 hrs) (G1a) showing some chromatolysis change, vacuolation and mild astrocytosis. (H &E, 40X).

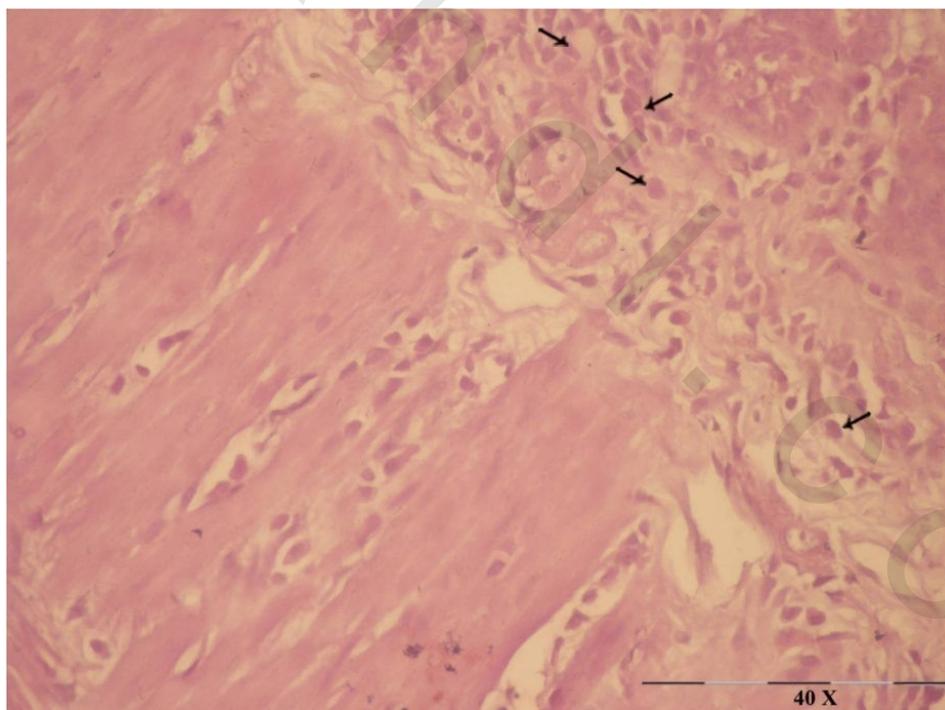


Fig.90: Photomicrograph of small intestine of mouse exposed to acute factory noise (100 dB, 12 hrs) (G1a) showing moderate inflammatory changes, mild edema in the mucosa and musculosa. (H &E, 40X).

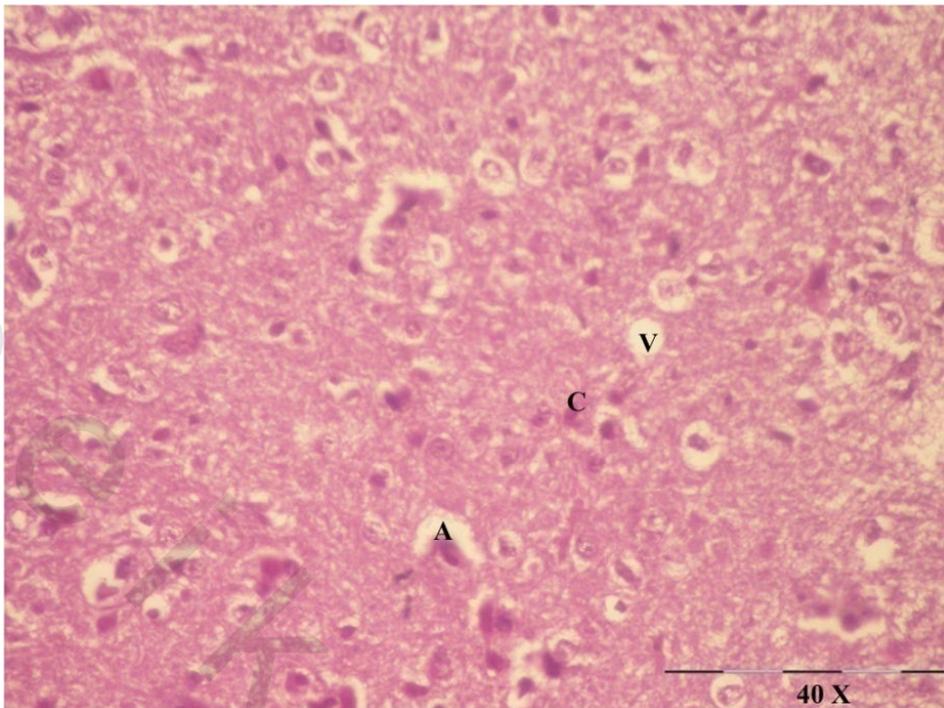


Fig. 91: Photomicrograph of brain of mouse exposed to acute traffic noise (100 dB, 12 hrs) (GIIa) showing mild to moderate vacuolation, moderate astrocytosis with condensed chromatin. (H &E, 40X).

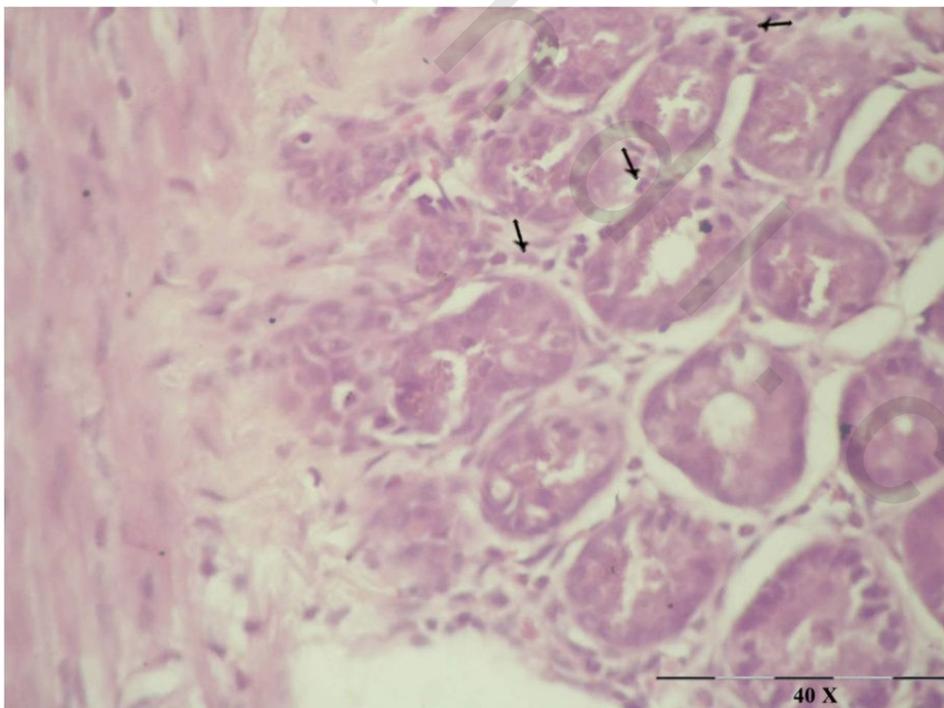


Fig.92: Photomicrograph of small intestine of mouse exposed to acute traffic noise (100 dB, 12 hrs) (GIIa) showing mild to moderate inflammatory cells, note the edema between muscle bundle. (H &E, 40X).

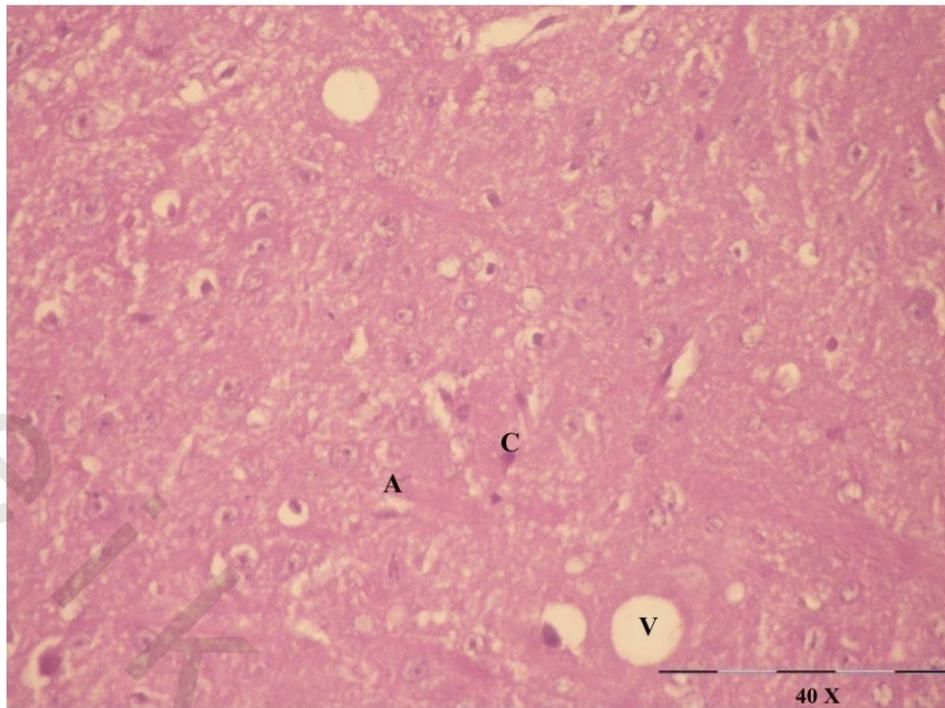


Fig.93: Photomicrograph of brain of mouse exposed to acute furance noise (100 dB, 12 hrs) (GIIIa) showing mild to mooderate vacuolation, astrocytosis, with condensed chromatin. (H &E, 40X).

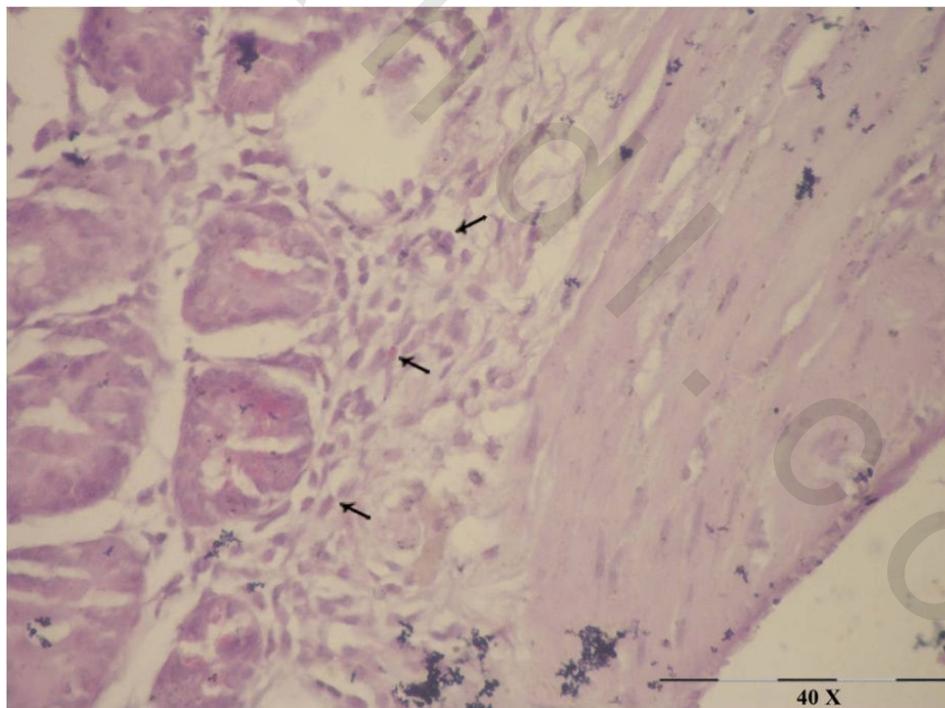


Fig.94: Photomicrograph of small intestine of mouse exposed to acute furance noise (100 dB, 12 hrs) (GIIIa) showing normal villi with mild distortion, mild inflammatory changes, separation of muscle fiber by edema. (H &E, 40X).

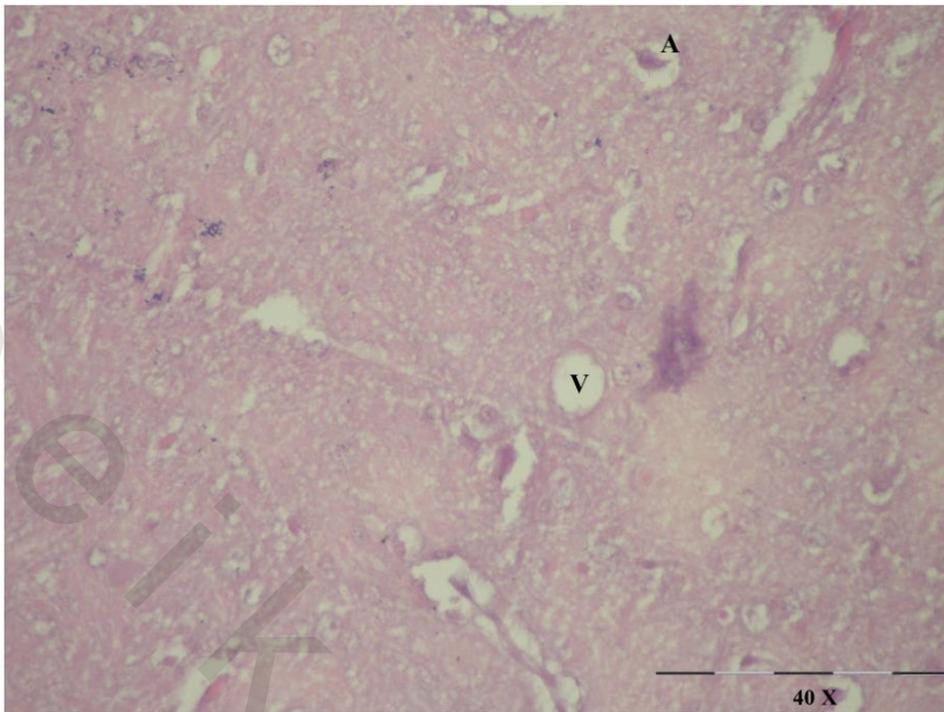


Fig.95: Photomicrograph of brain of mouse exposed to acute factory predominant frequency (100 dB, 12 hrs) (GI_{af}) showing mild vacuolation, mild astrocytosis. (H &E, 40X).

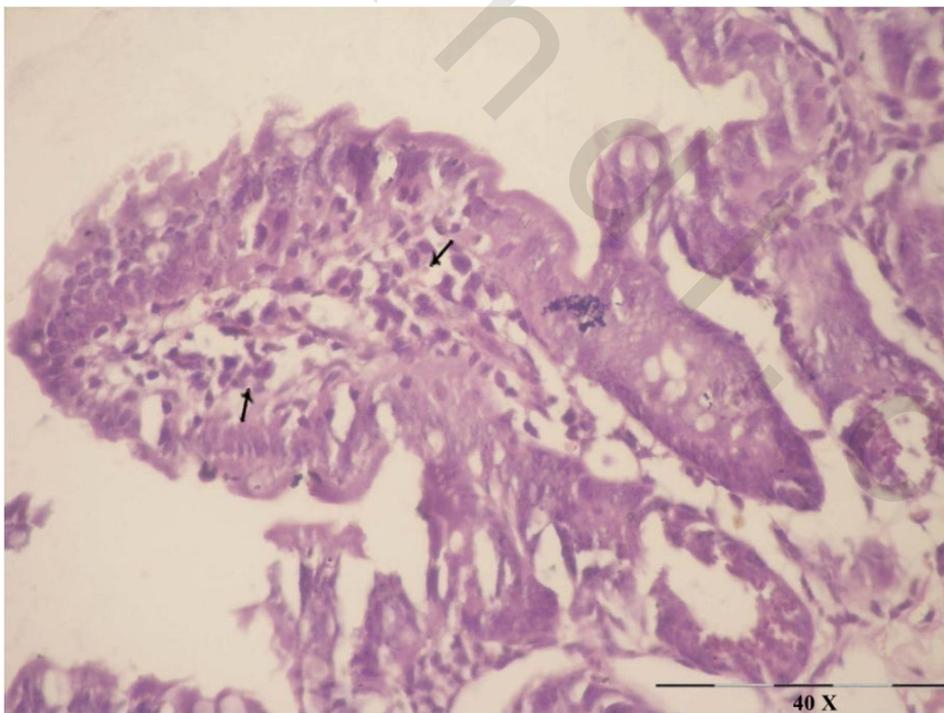


Fig.96: Photomicrograph of small intestine of mouse exposed to acute factory predominant frequency (100 dB, 12 hrs) (GI_{af}) showing normal intestinal villi, normal crypt with mild inflammatory changes, mild edema. (H &E, 40X).

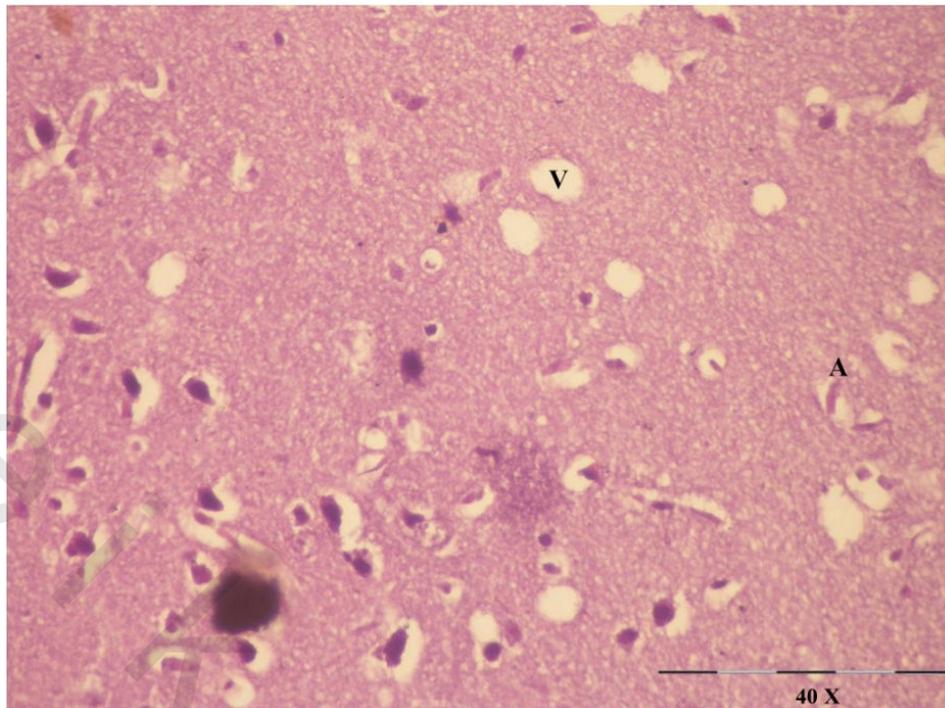


Fig.97: Photomicrograph of brain of mouse exposed to acute traffic predominant frequency (100 dB, 12 hrs) (GII_{af}) showing modulate vacuolation, moderate astrocytosis. (H &E, 40X).

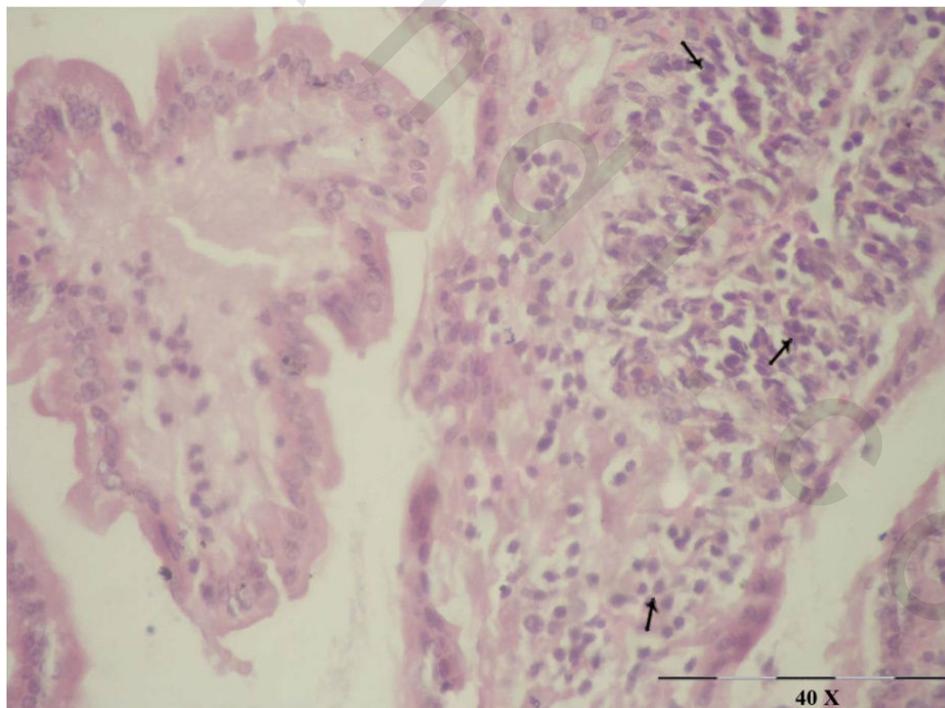


Fig.98: Photomicrograph of small intestine of mouse exposed to acute traffic predominant frequency (100 dB, 12 hrs) (GII_{af}) showing mild distortion in intestinal villi, with mild to moderate inflamatory changes, mild edema. (H &E, 40X).

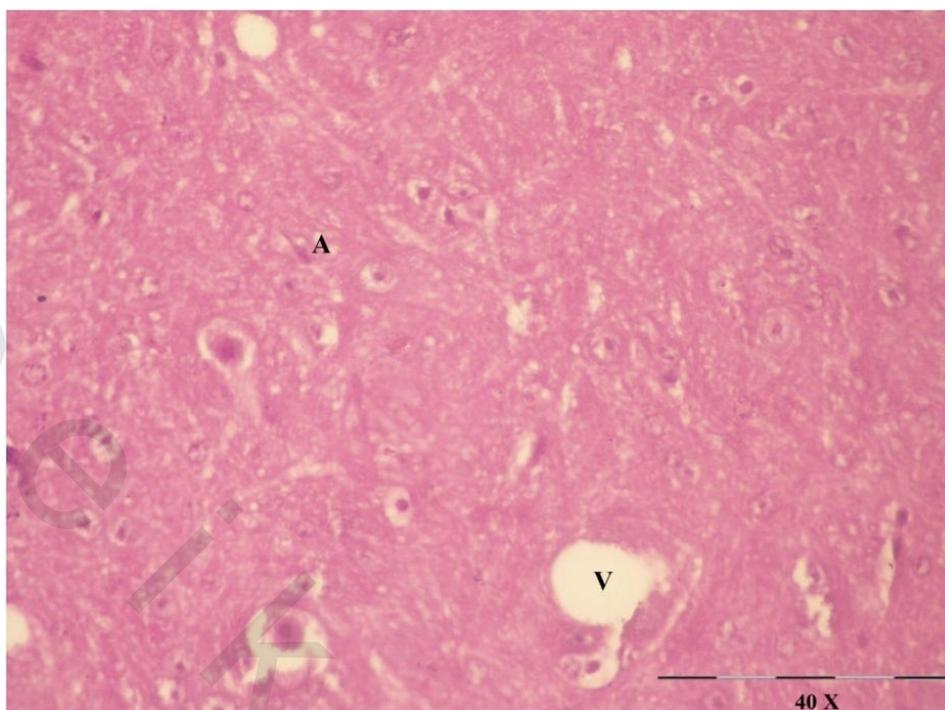


Fig. 99: Photomicrograph of brain of mouse exposed to acute furance predominant frequency (100 dB, 12 hrs) (GIII_{af}) showing mild vaculation, mild astrocytosis. (H &E, 40X).

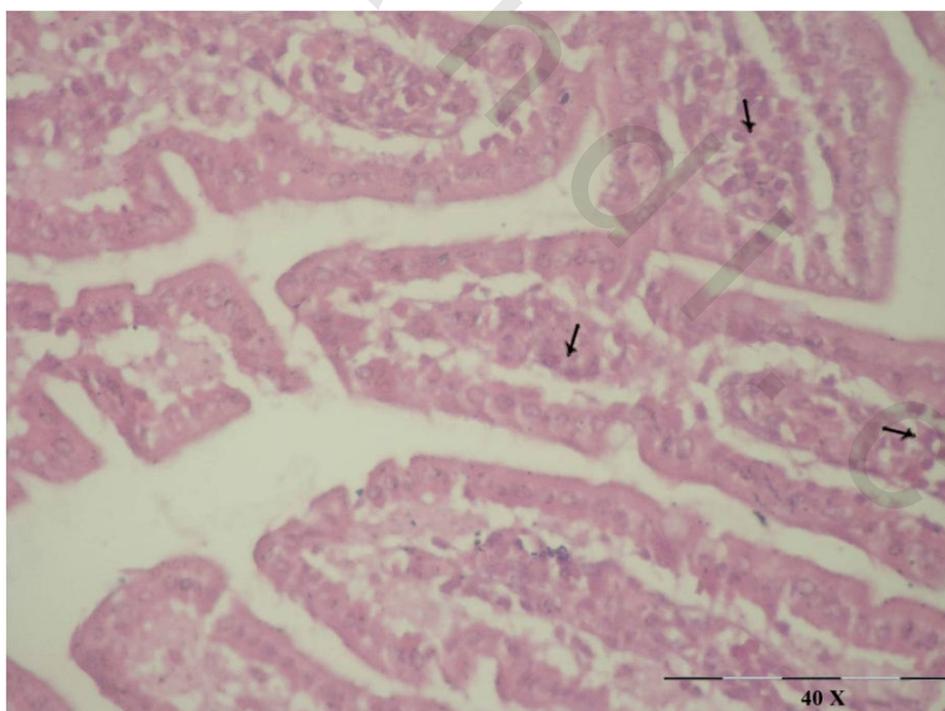


Fig.100: Photomicrograph of small intestine of mouse exposed to acute furance predominant frequency (100 dB, 12 hrs) (GIII_{af}) showing mild distortion in intestinal villi, with mild inflamatory changes, mild edema. (H &E, 40X).

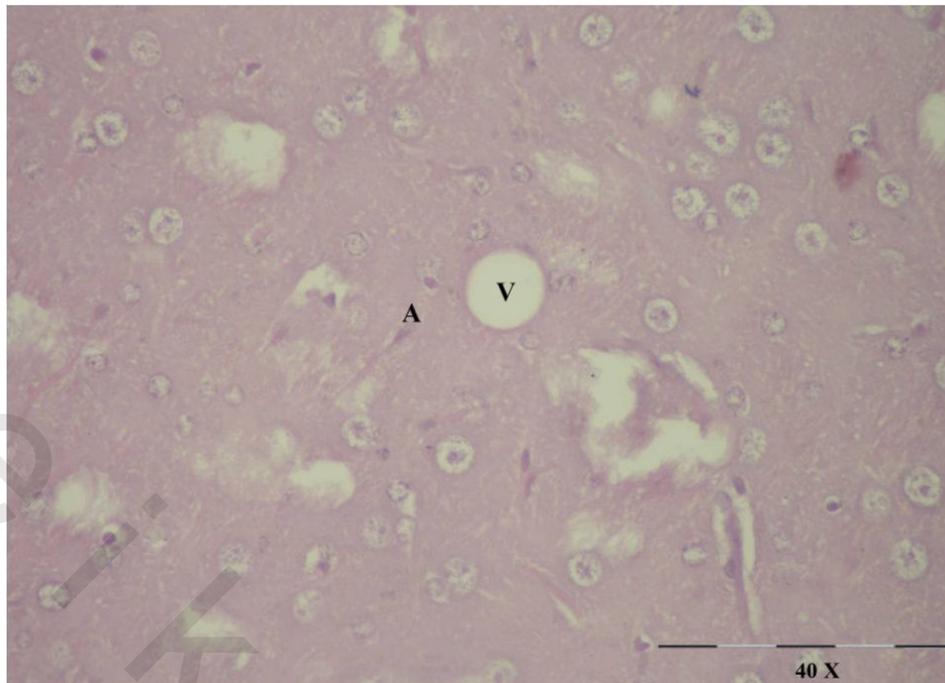


Fig.101: Photomicrograph of brain of mouse exposed to chronic factory noise (100 dB, 8 hrs/21d) (GI_c) showing mild vacuolation, moderate astrocytosis. (H &E, 40X).

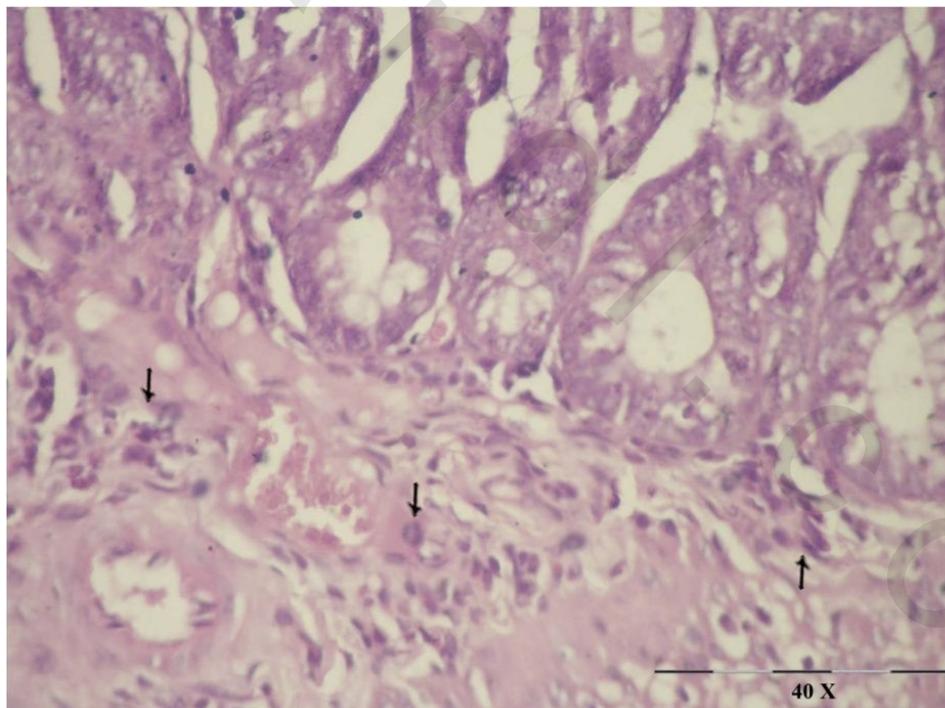


Fig.102: Photomicrograph of small intestine of mouse exposed to chronic traffic noise (100 dB, 8 hrs/21 d) (GI_c) showing mild distortion in intestinal villi, with mild to moderate inflammatory changes, mild edema. (H &E, 40X).

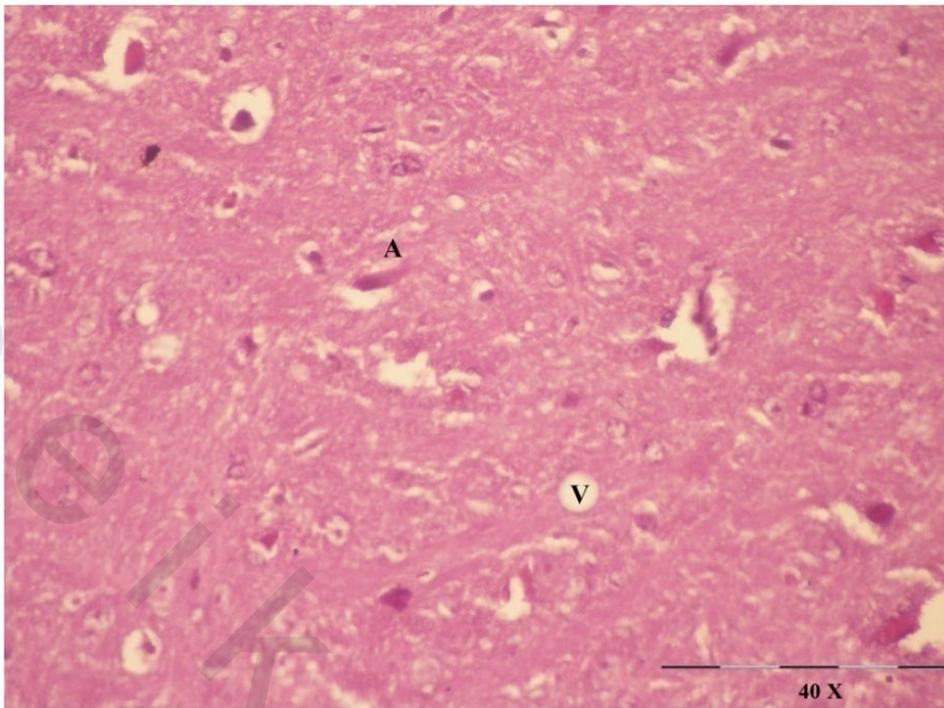


Fig. 103: Photomicrograph of brain of mouse exposed to chronic traffic noise (100 dB, 8 hrs/21d) (GII_c) showing modulate vaculation, moderate astrocytosis, and condensed chromatin. (H &E, 40X).

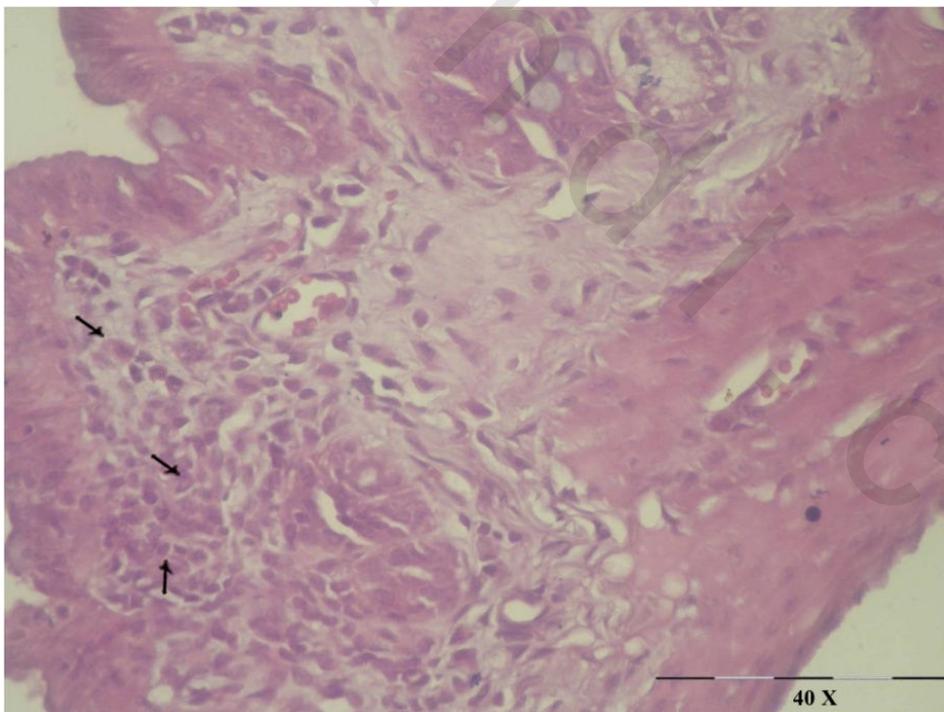


Fig.104: Photomicrograph of small intestine of mouse exposed to chronic traffic noise (100 dB, 8 hrs/21 d) (GII_c) showing mild distortion in intestinal villi, with moderate inflammatory changes. (H &E, 40X).

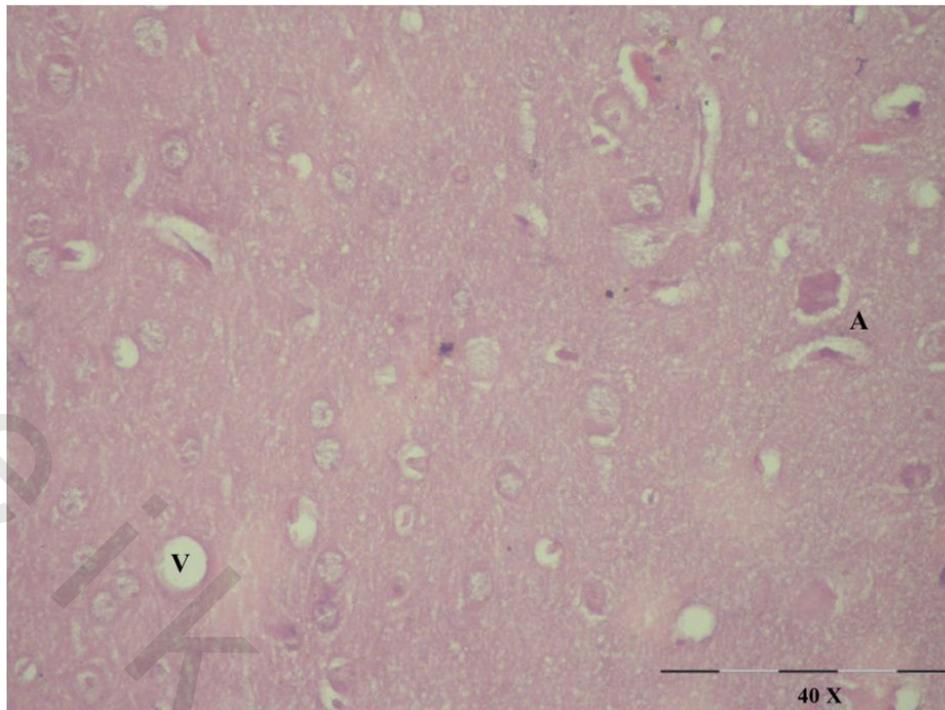


Fig.105: Photomicrograph of brain of mouse exposed to chronic furance noise (100 dB, 8 hrs/21d) (GIII_c) showing modulate vaculation, moderate astrocytosis. (H &E, 40X).

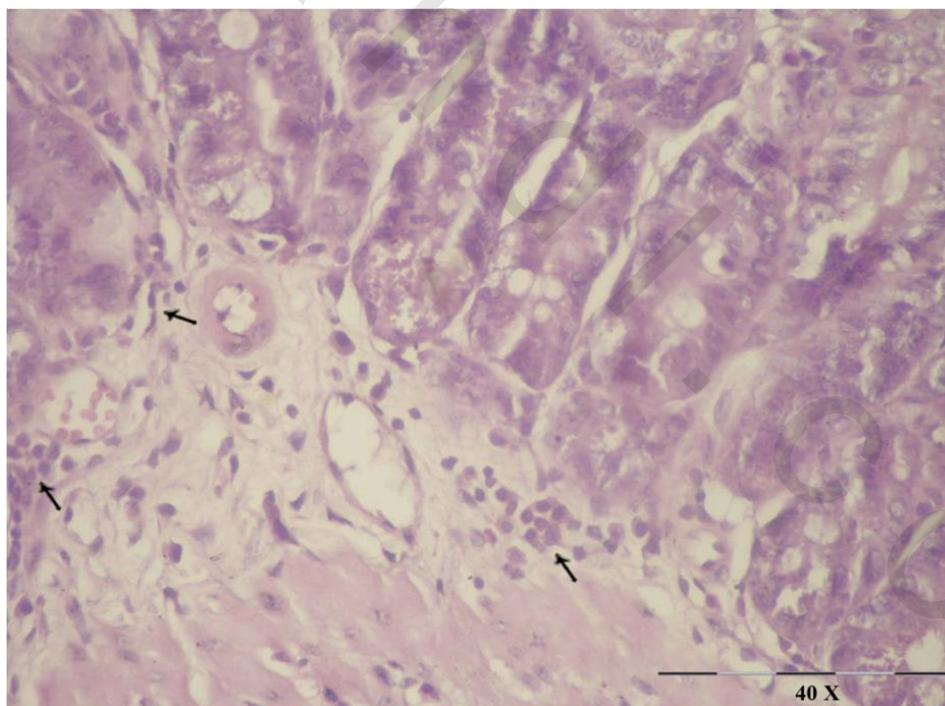


Fig.106: Photomicrograph of small intestine of mouse exposed to chronic traffic noise (100 dB, 8 hrs/21 d) (GIII_c) showing mild distortion in intestinal villi, with moderate inflamatory changes, moderate edema. (H &E, 40X).

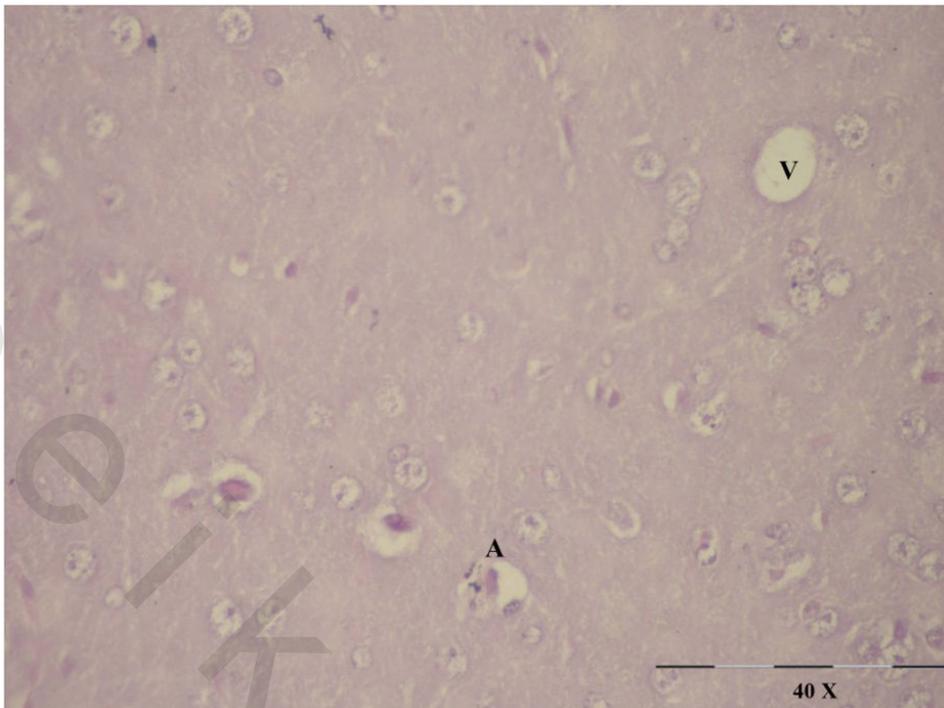


Fig.107: Photomicrograph of brain of mouse exposed to chronic factory predominant frequency (100 dB, 8 hrs/21d) (GI_{cf}) showing modulate vacuolation, moderate astrocytosis. (H &E, 40X).

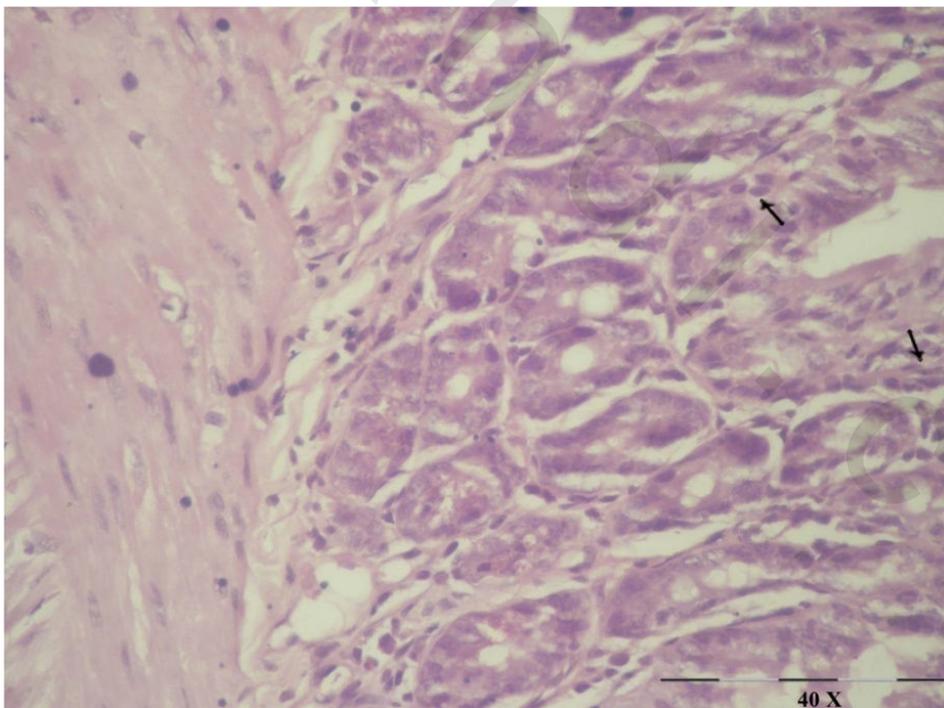


Fig.108: Photomicrograph of small intestine of mouse exposed to chronic factory predominant frequency (100 dB, 8 hrs/21 d) (GI_{cf}) showing mild distortion in intestinal villi, with moderate inflammatory changes, and mild edema. (H &E, 40X).

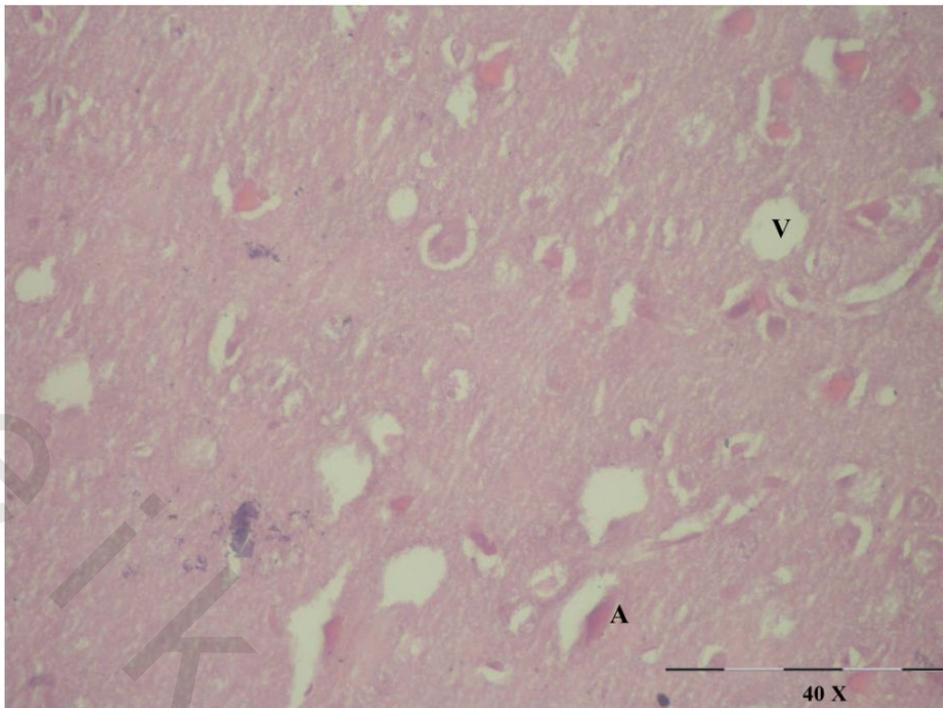


Fig.109: Photomicrograph of brain of mouse exposed to chronic traffic predominant frequency (100 dB, 8 hrs/21d) (GII_{cf}) showing mild to moderate vacuolation, mild astrocytosis. (H &E, 40X).

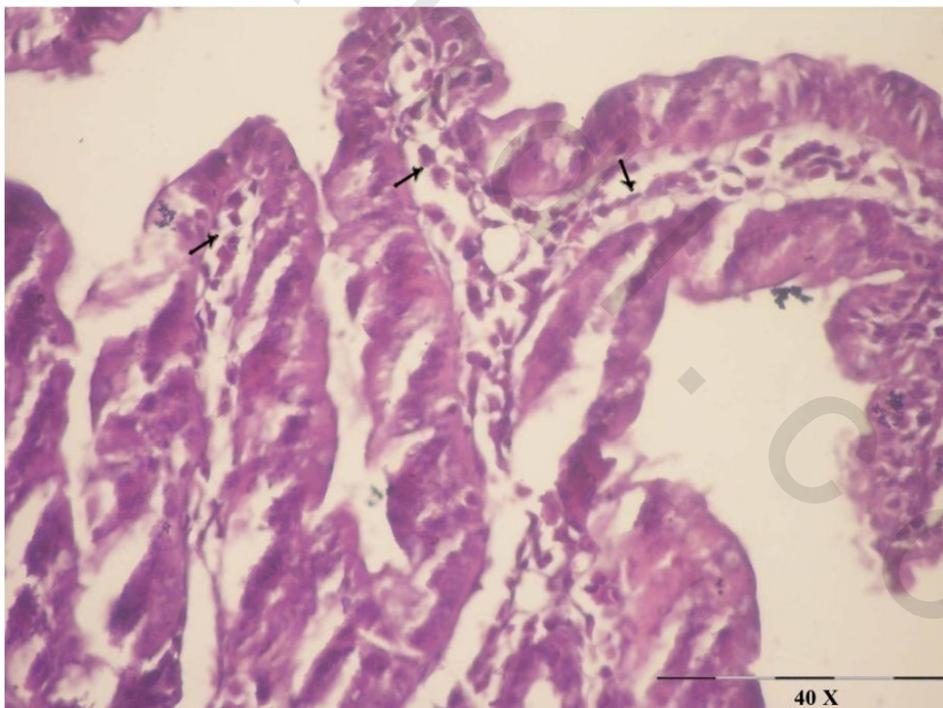


Fig.110: Photomicrograph of small intestine of mouse exposed to chronic traffic predominant frequency (100 dB, 8 hrs/21 d) (GII_{cf}) showing moderate distortion in intestinal villi, with moderate inflammatory changes, and moderate edema. (H &E, 40X).

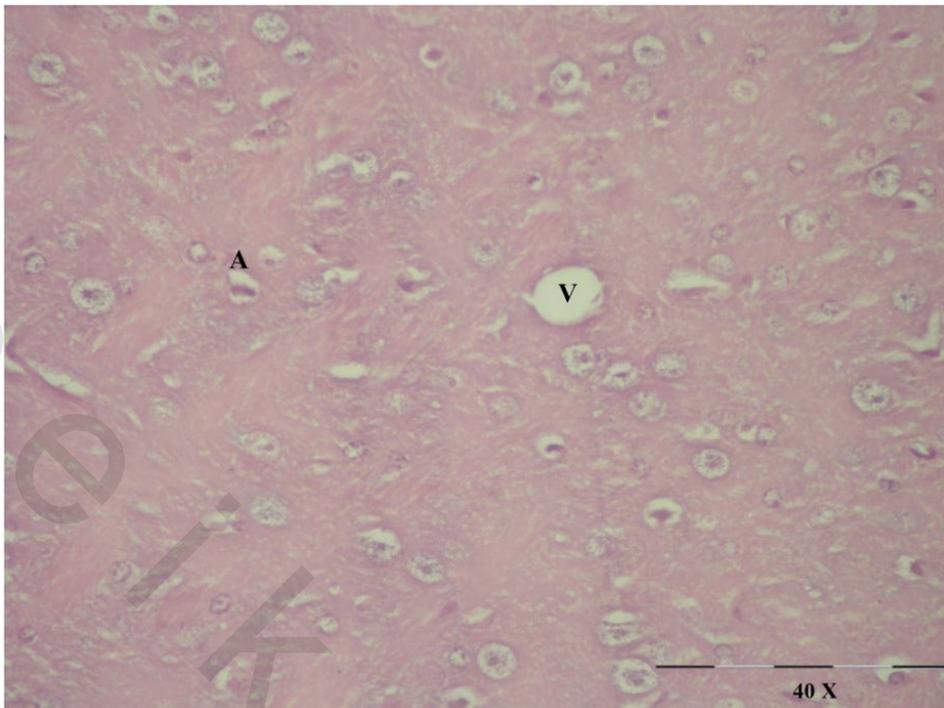


Fig.111: Photomicrograph of brain of mouse exposed to chronic furance predominant frequency (100 dB, 8 hrs/21d) (GIII_{cf}) showing modulate vacuolation, mild astrocytosis. (H &E, 40X).

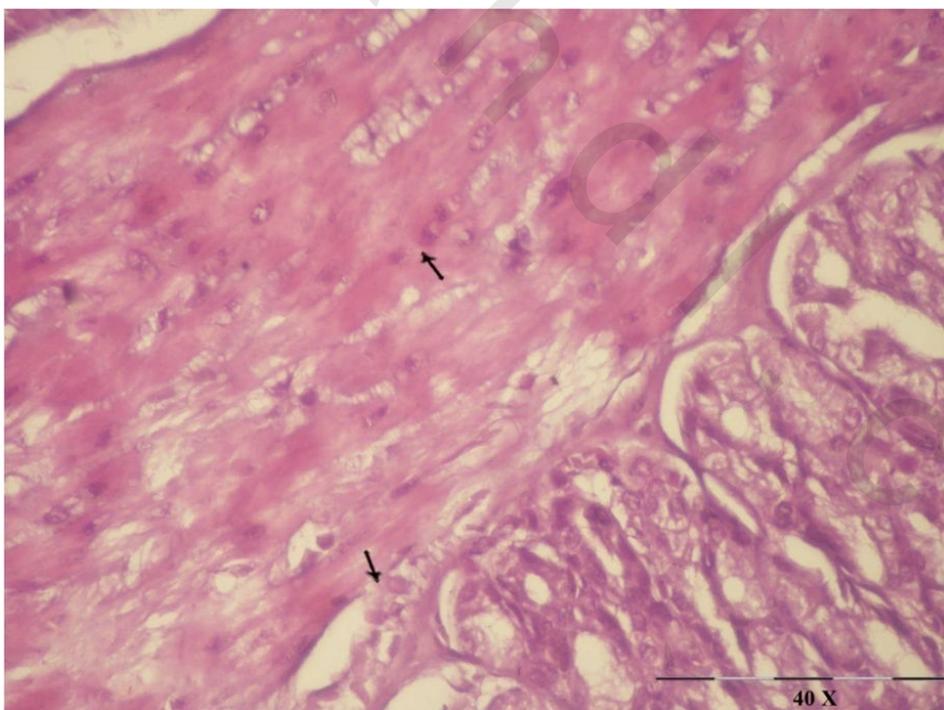


Fig.112: Photomicrograph of small intestine of mouse exposed to chronic furance predominant frequency (100 dB, 8 hrs/21 d) (GIII_{cf}) showing moderate distortion in intestinal villi, with moderate inflammatory changes, and mild edema. (H &E, 40X)