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FEMALE RATS INDUCE BEHAVIOUR IMPAIRMENT & OXIDATIVE DAMAGE IN MOTOR CORTEX AFTER ALCOHOL CONSUMPTION AND ALTERED BY HIGH FAT DIET

Archita Srivastava[*A], Dr. Sunil Kumar Singh [B], Prof. Dr. Alok Mukerjee [B], Dr. Abhishek Tripathi [B], Ashvani Tripathi [A]

[a] Department of pharmacology, United Institute of Pharmacy, Prayagraj 211010, Uttar Pradesh, India
[b] Department of pharmacology, United Institute of Pharmacy, Prayagraj 211010, Uttar Pradesh, India

ABSTRACT-
The display think about was conduct to look at the impression of a binge-feeding within the frame of high-fat slim down (HFD) having impacts on liquor utilization, behavior action and level of engine cortex oxidative harm. To do this, female wistar rats gotten rat chow and Discontinuous or unremitting HFD for 6 weeks. Rat chow and water were accessible ad-libitum to all bunches all through the try. Taking after 6 weeks of HFD cycling, 20.0% ethanol or water admissions was assessed from last 3 weeks by the assistance of Discontinuous get to 2 bottle choice worldview in dark phase. In expansion, anxiety-like behavior was measured employing a light-dark demonstrate and lifted additionally labyrinth, and learning and locomotor action by Morris water labyrinth or Rota bar demonstrate was moreover performed after which the estimation of oxidative push was too taken note. Strikingly, irregular access HFD rats shown lower liquor utilization with more admissions of HFD instead of rat chow. Rats uncovered to HFD went through more time within the ligh/tand maintain the balance on Rota rod and easily found the hidden platform on water maze. It represents that binge-feeding has anxiolytic effects. Further investigation for oxidative stress marks that MDA returns near to the normal value in those groups of animals which were exposed to HFD in chronic or intermittent way as compared to positive control who only consumed alcohol without HFD exposure, similarly there is no alteration in SOD level. The study revealed that animals which were treated with HFD in chronic and intermittent manner has consumed less alcohol as compared to non HFD groups, and also found that behavior alteration was minor with HFD. Intermittent exposure group showed more significant reduction in alcohol consumption, anxiolytic activity, as well learning and motor balance activity and oxidative stress range as compared to chronic HFD.

KEYWORD – Behavior activity, HFD, Ethanol**,** oxidative stress, female wistar rat, Motor cortex.

1. INTRODUCTION-

In developing countries, in terms of individual health, alcohol (EtOH) is one of the most demanded beverages around the world, [1]. According to the World Health organization (WHO) data from 2010 up to 2017, the consumption of alcohol in India is hiked by 38%. It increased to 5.7 liter per adult per year in 2016[2]. Within the population EtOH abuse is a chronic relapsing disorder occurs by regular intake, as it became preferred choices over other needs. The heritability of alcoholism is estimated to be as high as 50 to 60% [3].

In general, administration of EtOH causes unhealthy body profile like lowers the body weight and less fat content as well as abnormal nutrients value[4]. In healthy human causes some serious issues like cardiovascular disease, renal disease and also changes in brain neuro circuit [5]. That has undesirable impact upon body energy balance and reduces hunger activity. Interestingly highly alcohol addicted person expresses high intake of palatable food in early stage of recovery for management of AUD. But pattern of nutritional effect on management of AUD is unclear[6]. Binge feeding is a type of food consumption pattern in short period of time or lack of sense for a balance diet. it could be easily understood, that neuroendocrine peptide regulates reinforcing property for alcohol and eating in brain reward circuit[7].

By this set of combination various researches revealed that highly palatable foodstuff having sugar and fat involves the same mechanism that causes craving for EtOHconsumption[7,8]. Another study appraises decreases alcohol consumption in different sex of mice[9] (Corwin and Babbs, 2012). On the whole, it was assumed that intermittent and chronic access HFD would lower the drinking and palatability, but not diet which itself triggers reduction in alcohol drinking following our intermittent PD access paradigm[7]. To elucidate the role of caloric overload vs. palatability, we assessed that if intermittent and chronic exposure will impact alcohol drinking in a similar manner [6,8]. In intermittent and chronic access paradigm, rats received HFD for six-weeks and alcohol drinking was evaluated to examine the contribution of palatability with reduced alcohol drinking. It was predicted that intermittent and chronic HFD access would reduce alcohol drinking[10].

Various researches had been performed to investigate the correlation between fat diet and binge alcohol drinking[11] in male rats, since the addiction phenomenon is depending upon sex, so in this research we try to evaluate and hypothesis mechanism behind HFD[5,6,7,8] intake effect on EtOH drinking in female rat[11].

The numbers of studies had been designed to associate a correlation between AUD and Binge feeding and it is recommended that binge feeding and alcohol intake somewhere connect with behavior materialization[6.7.8,11]. However, the exact mechanism is unknown for reduce alcohol intake.

On the other side, self-administered EtOH[11] may readily passed through the esophageal and stomach mucous membranes since it is soluble in both water and is found in both urine and expired air after it has been absorbed[12]. It is not kept in the body since everything that is consumed is oxidised. It is completely metabolised in the liver. The metabolism of ethanol is connected to alcohol-induced oxidative damage[11,12,13]. In the human body, three EtOH metabolic routes have been identified, involving the enzymes alcohol dehydrogenase, microsomal ethanol oxidation system (MEOS), and catalase. Each of these processes has the potential to generate free radicals that harm the antioxidant system. A traditional EtOH metabolism route, which is catalysed by alcohol dehydrogenase to produce acetaldehyde, produces free radicals. NADH levels and NADH/NAD+ redox ratios both fluctuate at the same time. The majority of EtOH is processed by alcohol dehydrogenase in the liver when ingested in modest levels[12,13,14,15].

Although this topic has been debated in research contexts, tests have been conducted to investigate the drinking patterns of certain groups of animals and to evaluate the blood alcohol content of persons at regular intervals. The rat DID (Drinking in Dark) paradigm [16] will be adequate to induce higher initial intake and increased consumption/hour for EtOH, despite the shorter access time (3 weeks). Once the rats remained hooked to alcohol, we withdrew the alcohol and observed the physical and behavioral changes that occurred during abstinence. Because it is undistinguishable how HFD may control or aid in the reduction of addiction signals in animals that lead to alcohol misuse.

2. MATERIALS AND METHOD

2.1 Animal Profile and Housing

Female Wistar rats (180-200grams) were collected from M/S Chakraborty Enterprise Kolkata and housed in the animal facility of United Institute of Pharmacy, Allahabad. The animals were kept in polypropylene cages in standard environmental conditions, 12 h light and 12 h dark cycle at 25±2 °C. Before and during the experiments, the rats were fed with standard laboratory pellet diet and water ad libitum. Animals were acclimatized to the laboratory condition for at least 5 days prior to the experiment and were maintained in a well-ventilated animal house. The experimental protocol was approved by the Institutional Animal Ethical Committee (IAEC) with approval number (REG. No: **(UIP/IAEC/Nov.-2020/02)**. The care of animals and the laboratory was carried out as per the CPCSEA regulation.

2.2 Diet

According to the protocol, two different diets were taken, High Fat Diet (40%fat) and rodent chow (2.5% fat). The HFD was given on Tuesday and Thursday in intermittent and daily in chronic group for six weeks.

S.No	Ingredients	Gm/100gm
1.	Crushed rat food	68.0
2.	Corn oil	6.0
3.	Vegetable Ghee	6.0
4.	Nestle powder	20.0

List of ingredients used to make HFD

2.3 General Procedure

Female wistar rat (n=24), 100-150gm were used throughout the experiment. At 1 week prior to experiment, similar diet and water intake animals were separated into four respective groups. Following 6 week of study, all rats received rodent chow daily simultaneously, chronic group fed with HFD (7D) and intermittent group was fed with HFD on Tuesday and Thursday (2D). Food intake was measured daily whereas body weight of the animals was taken weekly.

An intermittent access two bottle choice (IA2BC) paradigm was used for alcohol drinking evaluation in every session. Rats of either diet was received one bottle of EtOH (20%v/v) on Monday, Wednesday, and Friday for last 3 weeks and one water bottle. After the completion of alcohol drinking studies, behavioral activity was checked by Light and Dark Model, Elevated Plus Maze, Rota Rod and Morris Water Maze methods.

2.4 Alcohol Drinking Evaluation

After 3-weeks of diet exposure, EtOH drinking evaluation was carried. All the rats were given non sweetened EtOH (20% v/v) and water using a two-bottle choice model[16]. The position of EtOH and water bottles were swapped every day to account for conditioning effects on EtOH intake. EtOHconsumption was calculated by weighing EtOHand water bottles before and after drinking tests. Preference was calculated as the volume of EtOH and water consumed in 24hr divided by total intake (EtOH+ water) consumed on serving days.

2.5 Behavior Parameters

2.5.1 Anxiolytic activity - Light and dark or Elevated plus Maze models was performed for detection of anxiolytic activity by HFD exposure upon EtOH.

A. Light and Dark evaluation- Rats of each group were gently introduced in the light side, facing dark side of the box and allowed to freely explore between compartments for 5 min. The total number of entries and time spent in both compartments were counted.[6,7.17]

B. Elevated Plus Maze evaluation- Rats was individually placed in the center of the maze, and the measures are scored by observer- The number of entries into open vs. closed arms; and the time spend in the open vs. closed arms[17]. Video recording was also done by fixing camera on ceiling of the room.

2.5.2 Motor Balance activity- Locomotor activity was performed for estimation of EtOH effect on motor balance in differentgroups of rat. Rat of each groups was placed to the Rota rod apparatus at 8rpm and the time duration was recorded for 180sec record the time duration in which rat was move on the rod for 5min.[11,17]

2.5.3 Learning and memory function test: Morris Water Maze apparatus used for evaluation of learning and memory activity of animals. One fourth of the tank was filled with water and the rats were given a maximum time of 120sec to find the hidden platform in the tank.[17]

2.6 Biochemical estimation

2.6.1 Sample – followed the completion of behavior activity and diet or liquid exposure, rats were sacrificed by cervical dislocation for further study. The blood is collected by cardiac puncture and stored in vacationer, simultaneously brain was removed and placed in formalin solution.

2.6.2 Lipid profile estimation- Blood serum were carried out to identify the serum TG, Tch, HDL, LDL level using assay kit (Span Diagnostic kit).[11,12]

2.6.3 Estimation of Oxidative stress by tissue-

1. Quantification of Lipid Peroxidation- monoaldehyde (MDA) is a highly reactive oxygen species present in brain due to higher intake of alcohol. The level of MDA is detected by appearance of pink color under spectrophotometer when reacted with TBA. It is a kind of quantitative estimation [11, 12.14]

2. Quantification of SOD- Determination of superoxide dismutase activity by tissue was performed according to the Luanna[11] . To measure the antioxidant enzyme concentration in brain, homogenized supernatant was taken and mixed with tris buffer or EDTA (pH 8.0). The total percentage conversion of O^{2-} to H_2O_2 and O_2 was evaluate under UV at 550 nm.

2.6.4 Histopathology of Brain Cortex- After removing the brain, brain tissues were fixed in 40% buffered formalin, embedded in paraffin, sectioned (5 μm thick), and stained eosin All sections were stained and surveyed on a light microscope (Nikon Eclipse E200). Illustrative images from all experimental groups were obtained using a digital camera attached to the microscope (Nikon Eclipse 50i), using the software Moticam 2500 for qualitative analysis.

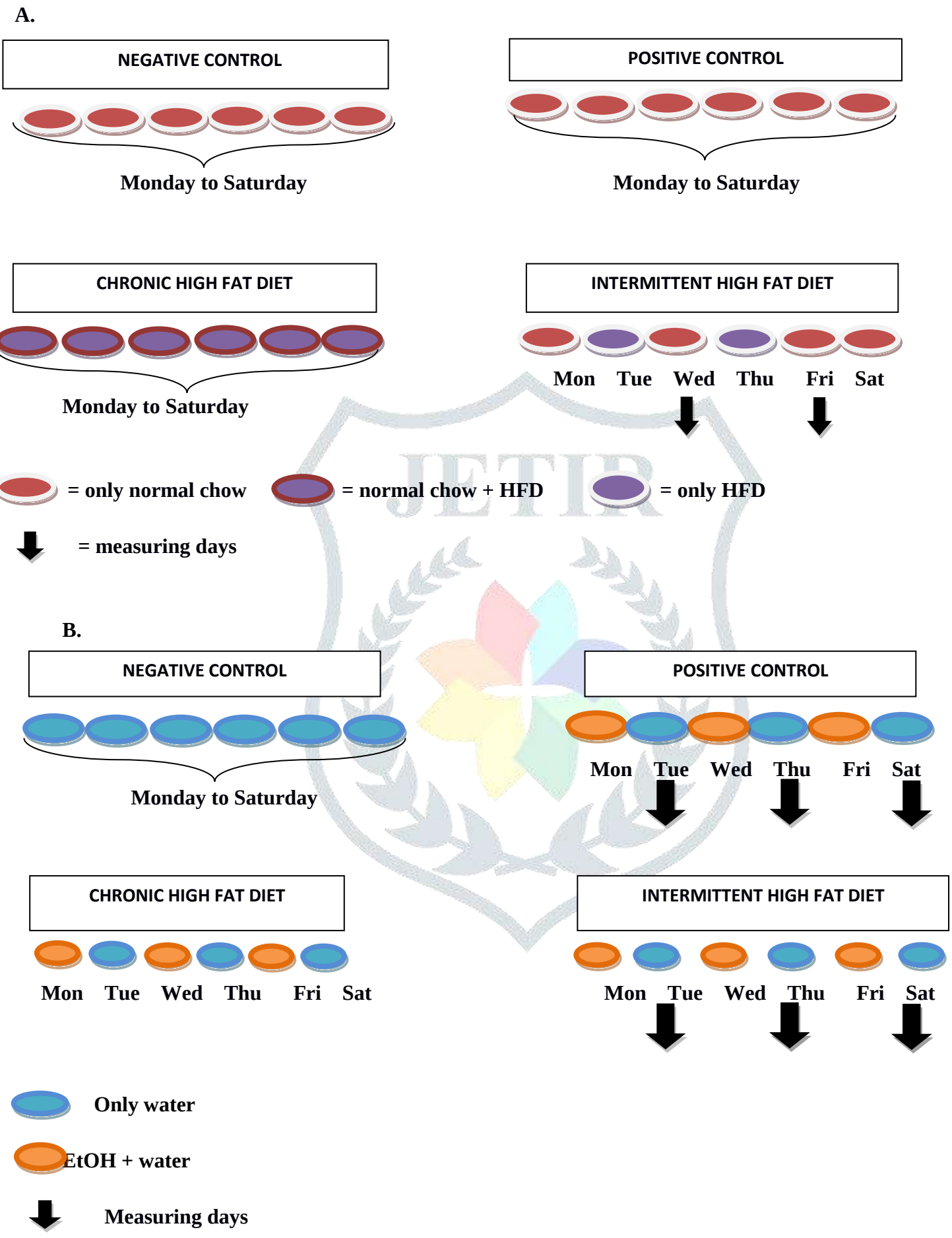

Figure 1 – Flow diagram (A) the pattern of feeding with respective of HFD and its measurement days and (B) the pattern of drinking of water and EtOH by IA2BC paradigm.

2.7 Statistical Analysis-Data was expressed as mean±SD and statistical analysis was carried out by using GraphPad Prism 9.0.1 software. Food intake, EtOH intake, body weight, behavior models light and dark and elevated plus maze values were analyzed by mixed-model two- way ANOVA. The within-subject variable was time intervals (time of measurement) and the between group variables, was exposure (chow and HFD) by student t-test method.

3. RESULT

3.1 Six weeks of Intermittent and Chronic HFD exposure:

Body weight-Oral EtOH exposure of different experimental groups showed significant reduction on body weight in all groups as compared to negative control. The overall reduction in positive control was found to be 30.85%, the body weight is significantly increased in Intermittent and chronic group as compared to positive control as shown in Figure 1.

Table 3.1- Effect of Oral EtOH administration and HFD on animal Body weight.

EXPERIMENTAL GROUPS	INTIAL WEIGHT	FINAL WEIGHT
Negative Control	112.16±2.31	175.17± 4.94
Positive Control	121.50±3.10	121.33±2.39
Chronic HFD	131.00±2.68	138.33±5.10
Intermittent HFD	127.83±2.78	144.83±2.99

Data compare mean±SD

3.2 Six week of Intermittent and Chronic HFD exposure:

Alcohol drinking-Oral EtOH exposure to different experimental groups showed significant increase in positive control. The consumption of EtOH in chronic HFD treated rats was significantly reduced by 30.28%. Similarly, in Intermittent group consumption of EtOH reduced significantly by 39.59% $p<0.001$ as compared to positive control group. HFD groups & Intermitted group consumed less EtOH as compared to chronic group as represented in figure 2.

Table 3.2 Consumption of total EtOHin different groups.

EXPERIMENTAL GROUPS	1st week of EtOH exposure	2nd week of EtOH exposure	3rd week of EtOH exposure
Negative Control	-	-	-
Positive Control	11.63 ± 3.21^{z}	11.833 ± 3.57^{z}	11.9 ± 2.57^{z}
Chronic HFD	11.85±1.70	9.53 ± 2.57^{b}	8.31 ± 1.32^{c}
Intermittent HFD	8.15 ± 1.45^{c}	7.60 ± 1.22^{c}	7.20 ± 2.20^{c}

Data comparesmean±SD on weekly Oral EtOH administration for all groups. $^{b}p < 0.01$, $^{c}p < 0.001$ compared to positive control group,$^{z}p<0.001$.

3.3 Six week of Intermittent and Chronic HFD exposure: Induce anxiolytic like effect by Light and dark-Normally, animals spent more time in light area, time lag decreased in positive control as compared to negative control while time lag increased significantly (..) as compared to positive control in Intermittent and chronic-HFD group as shown in Figure 3.

Table 3.3 Effect of Oral EtOH exposure with HFD for evaluation of anxiety by light and dark paradigm

EXPERIMENTAL GROUPS	Time spent in light chamber (sec)	Time spent in dark chamber (sec)
Negative Control	218.33± 1.32	82.67±2.20
Positive Control	100.05±1.38	200.02±2.0
Chronic HFD	185.5±1.05	175.5±1.76
Intermittent HFD	201±1.04	86±1.38

Data expressed as Mean±SD for 6 rat per group. [b]p<0.01 and [c] p <0.001 compared to positive control group,[z]p<0.001 as compared to negative control group.

3.4 Six weeks of Intermittent and Chronic HFD exposure:

Induce anxiolytic like effect by Elevated Plus Maze– we observed that the value of positive control is significantly reduced p<0.001 as compared to negative control. While the value of chronic and an intermittent were improved significantly p<0.001, p<0.01 as compare to positive control group is shown in figure 4

Table 3.4 Assessment of anxiety by EPM model

EXPERIMENTAL GROUPS	Total Time spent in open area (sec)
Negative Control	207.00±2.60
Positive Control	102.16±2.31[z]
Chronic HFD	164.83±2.99[c]
Intermittent HFD	178.83±3.31[b]

Data expressed as Mean±SD for 6 rat per group. [b]p<0.01 and [c] p <0.001 compared to positive control group,[z]p<0.001 as compared to negative control group.

3.5 Six week of Intermittent and Chronic HFD exposure:

Enhance Learning and memory activity- Oral EtOH exposure of different experimental group showed changes in their spatial learning memory in term to reach on the hidden platform in the tank. The different values were reported in different group. Latency period of positive control was significantly (p<0.001) raised as compared to negative control whereas latency period of chronic was significantly reduced by 41.09% (p<0.01), and for Intermittent group the reduction was found to be 46.80% (p<0.01) as compared to positive control shown in Figure 5.

Table3.5 Evaluation of memory-based examination by MWM.

EXPERIMENTAL GROUPS	Latency time A (sec)	Latency time B (sec)	Latency time C (sec)
Negative Control	70.33±14.7873	64.66±12.1764	37.33±11.3431
Positive Control	77.83±11.8223[z]	87.16±7.3869[z]	73.00±9.4445[z]
Chronic HFD	66.16±16.1668[a]	44.83±10.6473[b]	43.00±8.3904[b]
Intermittent HFD	59.66±15.9582[b]	47.33±6.8605[c]	38.83±8.4478[c]

Result expressed as Mean±SD for 6 rat per group [b] p<0.01,[a] p<0.05 and [c] p <0.001 compared to positive control group,[z] p<0.001 as compared to negative control group.

3.6 Six week of Intermittent and Chronic HFD exposure:

Maintain Motor balance activity–Different experimental groups showed variation in mean of motor coordination by EtOH exposure with HFD. Data suggested that positive control significantly ($p<0.001$) spent less time on Rota rod as compared to negative control, motor balance is higher in Intermittent and Chronic as compared to positive control ($p<0.01$) represented in Figure 6.

Table.3.6 Assessment of locomotor activity by Rota rod apparatus

EXPERIMENTAL GROUPS	Duration of time spent on rotating rod (Sec) 1st trial	Duration of time spent on rotating rod (Sec) 2nd trial
Negative Control	20.83±2.31	17.0. ±2.36
Positive Control	10.5±1.56^{z}	11.6±2.58^{z}
Chronic HFD	13.5±2.07^{b}	15.16±1.16^{b}
Intermittent HFD	17.0±3.09^{c}	16.33±1.96^{b}

Data expressed as Mean±SD for 6 rat per group. $^{b}p<0.01$ and ^{c} $p<0.001$ compared to positive control group,$^{z}p<0.001$ as compared to negative control group.

3.7 Six week of Intermittent and Chronic HFD exposure:

Reverse Plasma Lipid profile- Oral EtOH exposure to different experimental groups showed significant increase in serum triglycerides, total cholesterol, LDL in positive control ($p<0.001$) compared to negative control. Data indicates that there was significant Reduction in triglycerides total cholesterol and LDL in intermittent or chronic HFD groups ($p<0.01$; $p<0.05$) compared to positive control represented in Figure 7

Table 3.7 Effect of Oral EtOH exposure with HFD on the lipid profile level in blood serum.

EXPERIMENTAL GROUPS	Lipid profile			
	TCh	TG	HDL	LDL
Negative Control	74.16±3.31	70.0±1.78	25.16±2.31	35.83±2.63
Positive Control	153.37±5.5^{z}	113.16±5.3^{z}	13.5±2.44^{z}	74.66±2.94^{z}
Chronic HFD	86.50±3.83^{b}	85.15±3.81^{a}	20.83±2.6^{b}	42.66±2.13^{b}
Intermittent HFD	81.00±4.14^{b}	73.66±4.50^{b}	18.50±1.87^{b}	49.33±2.73^{b}

Data represents the mean ±SD for six rats per group.$^{b}p<0.01$ and $^{a}p<0.05$ compared to positive control group,$^{z}p<0.001$ as compared to negative control group.

3.8 Six weeks of Intermittent and Chronic HFD exposure:

Reverse oxidative stress alteration- Oral administration of EtOH with HFD showed the changes in level of ROS in brain region, the Oxidative stress monoaldehyde (MDA) was significantly increased($p<0.001$) in positive control as compared to negative control and in HFD fed group Int. and Chronic was significantly lowered the MDA concentration $p< 0.01$ or $p<0.05$ as compared to positive control. In addition, our result showed no significant changes in antioxidant enzyme (SOD) after HFD consumption upon EtOH ($p< 0.01$) as represented in figure – 8

Table 3.8 Evaluation of oxidative stress of LPO & SOD by Oral EtOH exposure and HFD

EXPERIMENTAL GROUPS	Concentration of MDA	Concentration of SOD
Negative Control	92.5±4.23	103.16±3.06
Positive Control	132.66±3.73^{z}	96.16±2.22^{z}
Chronic HFD	100.66±2.99^{a}	99.16±1.99^{a}
Intermittent HFD	95.5±3.61^{b}	101.66±2.50^{a}

Data expressed as Mean±SD for 6 rat per group. $^{b}p<0.01$ and ^{c} $p<0.001$ compared to positive control group,$^{z}p<0.001$ as compared to negative control group.

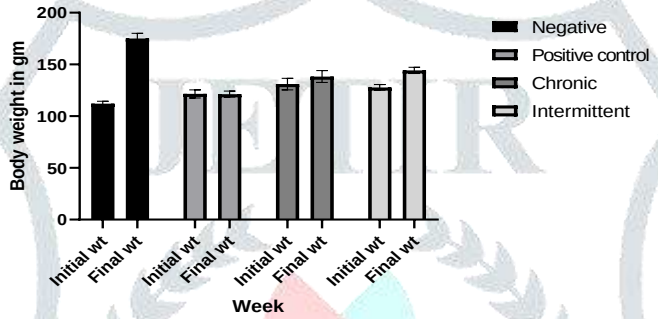

Figure 1– Systematic representation of animal's body weight by using one way ANOVA.

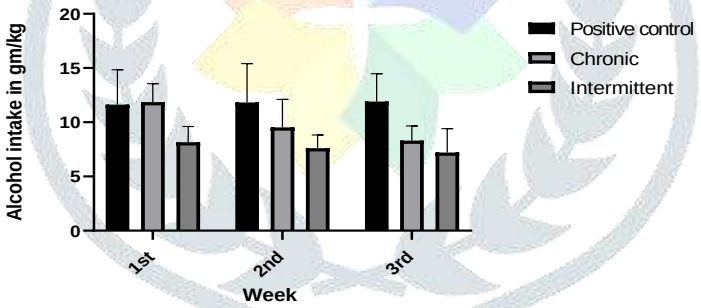

Figure 2 – Systematic representation of total Alcohol intake by experimental groups Data comparesmean±SD on weekly Oral EtOH administration for all groups. $^{b}p < 0.01$, $^{c}p < 0.001$ compared to positive control group,$^{z}p<0.001$.

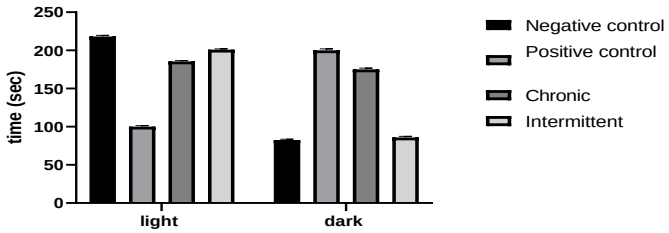

Figure 3- Systematic representation of light and dark model. Data expressed as Mean±SD for 6 rat per group. $^{b}p<0.01$ and ^{c} $p<0.001$ compared to positive control group,$^{z}p<0.001$ as compared to negative control group.

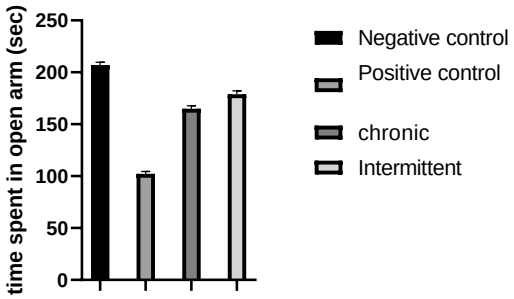

Figure 4- Systematic representation of EPM. Data expressed as Mean±SD for 6 rat per group. [b]p<0.01 and [c] p <0.001 compared to positive control group,[z]p<0.001 as compared to negative control group.

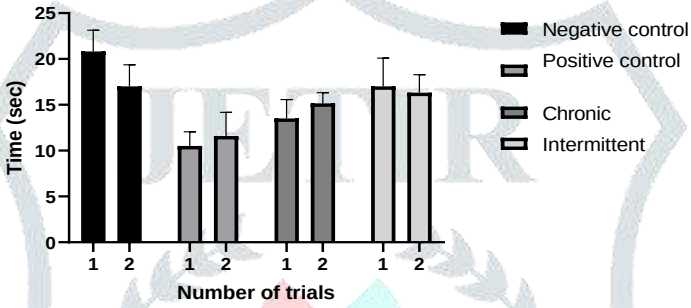

Figure 5 – Systematic representation of Motor balance activity. Result expressed as Mean±SD for 6 rat per group [b] p<0.01,[a] p<0.05 and [c] p <0.001 compared to positive control group,[z] p<0.001 as compared to negative control group.

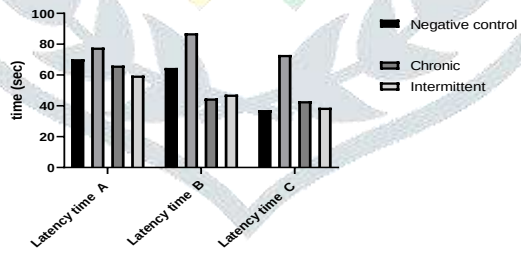

Figure 6- Systematic representation of learning-based study by Morris Water Maze. Data expressed as Mean±SD for 6 rat per group. [b]p<0.01 and [c] p <0.001 compared to positive control group,[z]p<0.001 as compared to negative control group.

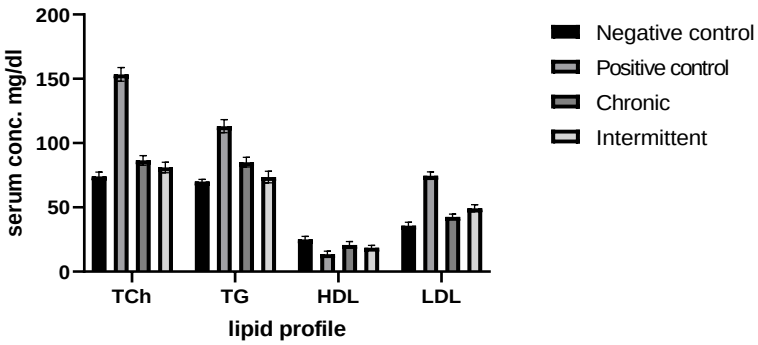

Figure 7- Systematic representation of lipid profile. Data represents the mean ±SD for six rats per group.[b]p<0.01 and [a]p <0.05 compared to positive control group,[z]p<0.001 as compared to negative control group.

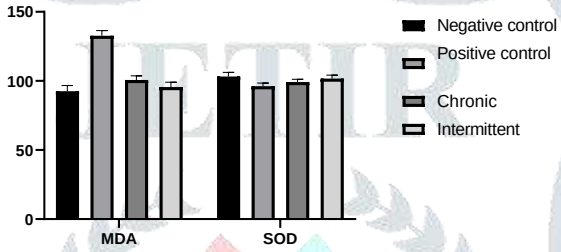

Figure 8- Systematic representation of oxidative stress estimation. Data expressed as Mean±SD for 6 rat per group. [b]p<0.01 and [c] p <0.001 compared to positive control group, [z]p<0.001 as compared to negative control group.

3.9 Histopathology of Brain Cortex-

Histopathological studies were carried out to show a relationship with biochemical alteration and structural changes in different brain regions following administration of EtOH with respect of HFD in term of oxidative damage of Cerebral cortex of different groups. The histopathology study proven that excessive Oral EtOH administration may damage the brain tissue by the production of free radical. Since the positive group had no choice between the food and drink, hence this group intake more and more EtOH as compare to chronic HFD and Intermittent HFD group.

A. B. C. D.

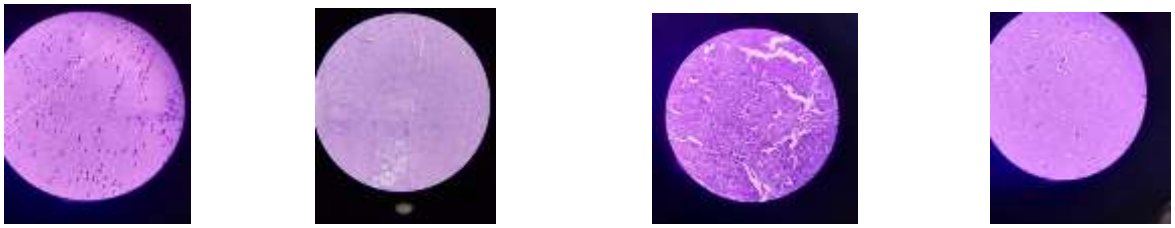

Figure 10. A. motor cortex of negative control is estimated as normal brain with no treatment no disease. **B**. considure as positive control which shows the necrosis and cell damage in cortex reason due to oxidative stress or formation of MDA **C**. Treated with HFD in regular way so this shows less damage in cell due to less consumption of EtOH as compare to positive control. **D** is considure as more effective then chronic and positive due to less EtOH intake**.**

4. Discussion

The goal of the present study was to determine the effect of EtOH drinking which changed the behavior consequence in female rat by intermittent and chronic pattern feeding HFD. As Alcohol use disorder (AUD) is one of the most endemic psychiatric disorders. Alcohol has a significant source of calories (7Kcal) so, drinking of alcohol may cause energy imbalance, and Nutrient deficiency and decrease the tendency of food intake which could be alter behavior or induce more chances for alcohol intoxication. Someearlier research and clinical data suggests that HFD containing feed may alter alcohol drinking behavior but the exact mechanism is not clearly known till date.

In general, binge EtOH intake may cause several abnormalities in human as well as in rodents. In this course of study, a numbers of significant values wereachieved;the chronic or Intermittent HFD intake caused significant changes on animal body weight and food intake preference between the rodent chow and HFD. Overall, the data indicates that there were no significant difference was found between the groups during HFD and Chow consumption but later there was a slight decrease in body weight in all groups after EtOH consumption but intermittent and chronic fed groups showed recovery thereafter, but positive group did not.

The observation also suggested that the intermittent and chronic HFD intake reduce self-administration of alcohol intake in specific groups in dark phase voluntary limited access procedure in female rat.It's likely that hedonic feeding[6.7.8] (an alternative feeding) regulates the rewarding effect of diet and drink, which is possible with an intermittent pattern, hence intermittent HFD demonstrates lower alcohol consumption.

Behavior consequences was also seen by self-administration EtOH in all the particular groups, as alcohol causes major changes in behavior activity in term of anxiety, learning and memory or motor balance function, our findings, support that intermittent and chronic HFD groups induce anxiolytic behavior, out of them intermittent group showed more reduction in time lag in dark chamber in light and dark model. Similarly the total time spent in open area on Elevated plus maze is hiked that showed anxiolytic effect of HFD[6.7,17] On the other hand the observations made inMorris tank suggested that intermittent HFD group improve the learning and memory activity over alcohol consumption. The behavior activity of motor balance function by Rota rod[11] suggests that animals who fed with HFD has maintained body balance on rotating rod compared to positive control group.

The plasma testing of lipid profile on different animal groups showed variation in biochemical estimation of blood. Administration of HFD (intermittent and chronic) may alter the blood cholesterol level of TG, Tch, HDL and LDL due to intake of alcohol. Our findings suggest that the intermittently exposed group saw a reversal of the altered range, bringing it closer to normal.

Surprisingly, our research revealed that binge drinking can cause oxidative stress in the brain by increasing the amount of reactive oxygen species (ROS), such as Monoaldehyde (MDA)[14,15], but there was no significant difference between the groups when it came to antioxidant enzyme (SOD), implying that HFD exposure has no effect.Alcohol metabolism generates free radicals in the peripheral or central nervous systems of the body, and the study found that those who consumed the least amount of alcohol produced the least amount of ROS.

5. CONCLUSION

The study indicated that animals treated with HFD in a chronic and intermittent way consumed less alcohol than non-HFD groups, and that the behavioral changes caused by HFD were minimal.Our findings revealed that the positive group, which is not on any diet, has a higher concentration of MDA than the negative group, but the two HFD groups, which are intermittent& chronic, have a lower concentration of MDA than the positive and chronic groups.

In terms of behavioral and oxidative stress consequences, the designed work concludes that the intermittent HFD consumption group had a low importance.

LIST OF ABBREVIATIONS

Full name	Symbols
High Fat Diet	**HFD**
Alcohol (Ethyl alcohol)	**EtOH**
Drinking In Dark	**DID**
Volume /volume	**v/v**
Percentage	**%**
Second	**Sec**
Alcohol used disorder	**AUD**
Palatable diet	**PD**
Seven days	**7D**
Two days	**2D**
Microsomal ethanol oxidation system	**MEOS**
Elevated Plus Maze	**EPM**
Morris water maze	**MWM**
Institutional Animal Ethical Committee	**IAEC**
Superoxide dismutase	**SOD**
Monoaldehyde	**MDA**
Thiobutayric acid	**TBA**
Reactive Oxygen species	**ROS**
Analysis of variables	**ANOVA**
Intermittent	**Int.**
Low density lipoprotein	**LDL**
High density lipoprotein	**HDL**
Triglyceride	**TG**
Total cholesterol	**TCh**

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