**Results**

**DRUG EFFECTS ON EPILEPTIC RATS**

I. **Seizure frequency after Carbamazepine, *Bacopa monnieri* and Bacoside A, in post-treated epileptic rats.**

Seizure frequency per 4 hours over 72 hours video recording period showed a significant increase in epileptic group. Treatment with Carbamazepine, *Bacopa monnieri* and Bacoside A significantly ($p<0.01$) reduced the seizure frequency when compared to epileptic group (Table-1; Figure- 1-4).

II. **Magnitude of Drug Effect**

Mean difference of seizure frequency showed a significant decrease ($p<0.01$) in the Carbamazepine, *Bacopa monnieri* and Bacoside A, post-treatment when compared to epileptic group (Figure- 5).

III. **Seizure onset latency after Carbamazepine, *Bacopa monnieri* and Bacoside A, pre-treatment.**

Seizure onset latency showed a significant decrease ($p<0.01$) in the epileptic group when compared to Carbamazepine, *Bacopa monnieri* and Bacoside A, pre-treated groups (Table- 2, Figure- 6).
IV. Duration of *Status epilepticus* after Carbamazepine, *Bacopa monnieri* and Bacoside A, pre-treatment.

Duration of *Status epilepticus* significantly increased (p<0.01) in epileptic group when compared to Carbamazepine, *Bacopa monnieri* and Bacoside A, pre-treated groups (Table-3, Figure-7).

V. Effect of different dosage of Carbamazepine, *Bacopa monnieri* and Bacoside A, in post-treated epileptic rats

Carbamazepine and Bacoside A treatment in 150 and 300 mg/kg/day showed a significant decrease (p<0.01) in the seizure frequency when compared to epileptic group (Table-4a, Figure-8). *Bacopa monnieri* treatment in 300 and 500 mg/kg/day showed a significant decrease (p<0.01) in the seizure frequency when compared to epileptic group (Table-4b, Figure-8).

**ACETYLCHOLINE ESTERASE ACTIVITY IN THE BRAIN REGIONS OF EXPERIMENTAL RATS**

**Hippocampus**

Acetylcholine esterase kinetic studies showed that $V_{\text{max}}$ significantly increased (p<0.001) in the hippocampus of epileptic group with no significant difference in the $K_{\text{m}}$ when compared to control. Carbamazepine and extract of *Bacopa monnieri* treatment significantly reversed the $V_{\text{max}}$ (p<0.001) to near control when compared to epileptic group (Table-5, Figure-9). Bacoside A treatment significantly reversed the $V_{\text{max}}$ (p<0.001) to near control when compared to epileptic group (Table-6, Figure-10). $K_{\text{m}}$ showed no significant change in all the treated groups.
**Brainstem**

$V_{\text{max}}$ of acetylcholine esterase significantly increased ($p<0.01$) in the brainstem of epileptic rats with no significant change in $K_m$ when compared to control. Carbamazepine and *Bacopa monnieri* treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-7, Figure-11). Bacoside A treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-8, Figure-12).

**GLUTAMATE DEHYDROGENASE ACTIVITY IN THE BRAIN REGIONS OF EXPERIMENTAL RATS**

**Hippocampus**

Glutamate dehydrogenase kinetics studies showed that $V_{\text{max}}$ significantly increased ($p<0.01$) in the hippocampus of epileptic group with no significant change in $K_m$. Extract of *Bacopa monnieri* treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-9, Figure-13).

**Cerebellum**

Glutamate dehydrogenase kinetics studies showed that $V_{\text{max}}$ significantly increased ($p<0.01$) in the cerebellum of epileptic group with no significant change in $K_m$. Extract of *Bacopa monnieri* treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-10, Figure-14).

**Brainstem**

Glutamate dehydrogenase kinetics studies showed that $V_{\text{max}}$ significantly increased ($p<0.001$) in the brainstem of epileptic group with no significant change in $K_m$. Extract of *Bacopa monnieri* treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-7, Figure-11). Bacoside A treatment significantly reversed the $V_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group (Table-8, Figure-12).
K\textsubscript{m}. Extract of *Bacopa monnieri* treatment significantly reversed the V\textsubscript{max} (p<0.001) to near control when compared to epileptic group (Table- 11, Figure- 15).

CENTRAL MUSCARINIC RECEPTOR ALTERATIONS IN THE BRAIN REGIONS OF EXPERIMENTAL RATS

**Hippocampus**

I) Total Muscarinic receptor analysis in post-treated epileptic rats

a) *Scatchard analysis of \[^{3}H\] QNB binding against atropine in the hippocampus of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacoside A, post-treated group rats*

The total muscarinic receptor status was assayed using the specific ligand, \[^{3}H\]QNB and muscarinic general antagonist, atropine. Scatchard analysis showed that the B\textsubscript{max} increased significantly (p<0.001) in epileptic rats with a significant increase (p<0.01) in the K\textsubscript{d} when compared to control. In Carbamazepine treated epileptic rats, B\textsubscript{max} significantly (p<0.001) reversed to near control when compared to epileptic group. K\textsubscript{d} also significantly (p<0.05) reversed to near control when compared to epileptic group. Extract of *Bacopa monnieri* treatment significantly reversed the B\textsubscript{max} (p<0.001) and K\textsubscript{d} (p<0.01) to near control when compared to epileptic group (Table-12 & Figure- 16). Bacoside A treatment significantly reversed the B\textsubscript{max} (p<0.001) and K\textsubscript{d} (p<0.01) to near control when compared to epileptic group (Table- 14 & Figure- 18).
b) **Displacement analysis of \( ^3H \) QNB against atropine**

The competition curve for atropine against \( ^3H \) QNB fitted for one site model in all groups. The \( \log (EC_{50}) \) and \( K_i \) increased in epileptic condition and reversed to near control in Carbamazepine and Bacoside A treated epileptic rats (Table- 13,15 & Figure- 17,19).

II) **Muscarinic M1 receptor analysis in post-treated epileptic rats**

a) **Scatchard analysis of \( ^3H \) QNB binding against pirenzepine in the Hippocampus of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacoside A post- treated Epileptic rats**

Binding analysis of muscarinic M1 receptors was done using \( ^3H \)QNB and M1 subtype specific antagonist pirenzepine. The \( B_{\text{max}} \) increased significantly (p<0.001) in epileptic group when compared to control group. The \( K_d \) also increased significantly when compared to control group (p<0.001). In Carbamazepine treated epileptic rats \( B_{\text{max}} \) significantly (p<0.001) reversed back to near control when compared to epileptic group. \( K_d \) also significantly (p<0.001) reversed back to near control when compared to epileptic group. Extract of *Bacopa monnieri* treatment significantly reversed the \( B_{\text{max}} \) (p<0.001) and \( K_d \) (p<0.01) to near control when compared to epileptic group. (Table- 16 & Figure- 20). Bacoside A treatment significantly reversed the \( B_{\text{max}} \) (p<0.001) and \( K_d \) (p<0.001) to near control when compared to epileptic group (Table- 18 & Figure- 22).
b) *Displacement analysis of* $[^3H]$ QNB *using pirenzepine*

The competition curve for pirenzepine against $[^3H]$QNB fitted for one site model in all groups. The log (EC$_{50}$) increased in epileptic group and reduced during Carbamazepine and *Bacopa monnieri* treatment. The $K_i$ increased in epileptic condition and reversed to near control in Bacoside A treated epileptic rats (Table- 17, 19 & Figure- 21, 23).

III) *Real Time-PCR analysis of Muscarinic M1 receptor mRNA in post-treated epileptic rats*

Real Time-PCR analysis showed that the muscarinic M1 receptor mRNA increased significantly ($p<0.001$) in epileptic condition. It was reversed to near control in Carbamazepine ($p<0.001$), *Bacopa monnieri* ($p<0.01$) and Bacoside A ($p<0.01$) treated epileptic rats (Table-20, 21 & Figure- 24, 25).

IV) *Muscarinic M1 receptor analysis in pre-treated epileptic rats*

a) *Scatchard analysis of* $[^3H]$ QNB *binding against pirenzepine in the Hippocampus of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacopside A pre-treated Epileptic rats*

Binding analysis of Muscarinic M1 receptors was done using $[^3H]$QNB and M1 subtype specific antagonist pirenzepine. $B_{\text{max}}$ decreased significantly ($p<0.001$) in epileptic group when compared to control group. $K_d$ also decreased significantly when compared to control group ($p<0.01$). In Carbamazepine treated epileptic rats $B_{\text{max}}$ ($p<0.001$) and $K_d$ ($p<0.01$) reversed significantly to near control when compared to epileptic group. Extract of *Bacopa monnieri* treatment significantly reversed the $B_{\text{max}}$. 73
(p<0.001) and $K_d$ (p<0.01) to near control when compared to epileptic group (Table-22 & Figure-26). Bacoside A treatment significantly reversed the $B_{\text{max}}$ (p<0.001) and $K_d$ (p<0.01) to near control when compared to epileptic group (Table-24 & Figure-28).

**b) Displacement analysis of $[^3\text{H}]QNB$ using pirenzepine**

The competition curve for pirenzepine against $[^3\text{H}]QNB$ fitted for one site model in all groups. The log (EC$_{50}$) decreased in epileptic group and reversed to near control in Carbamazepine, *Bacopa monnieri* and Bacoside A treated epileptic rats. $K_i$ increased in epileptic group and reversed to near control in Carbamazepine, *Bacopa monnieri* and Bacoside A treated epileptic rats (Table-23, 25 & Figure-27, 29).

**V) Real Time-PCR analysis of Muscarinic M1 receptor mRNA in pre-treated epileptic rats**

Real Time-PCR analysis showed that the muscarinic M1 receptor mRNA significantly (p<0.001) decreased in epileptic condition and it reversed to near control in Carbamazepine (p<0.001) *Bacopa monnieri* (p<0.001) and Bacoside A (p<0.001) treated epileptic rats (Table-26, 27 & Figure-30, 31).
Cerebellum

I) Total Muscarinic receptor analysis in post-treated epileptic rats.

a) Scatchard analysis of [3H] QNB binding against atropine in the Cerebellum of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacoside A post- treated Epileptic rats

Scatchard analysis of cerebellar total muscarinic receptors status showed that the $B_{\text{max}}$ decreased significantly ($p<0.01$) in epileptic condition when compared to control group. The $K_d$ showed no significant change in the epileptic group compared to control. In Carbamazepine treated epileptic condition $B_{\text{max}}$ significantly ($p<0.01$) reversed to near control when compared to epileptic group. Extract of Bacopa monnieri treatment significantly reversed the $B_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group without any change in $K_d$ (Table- 28 & Figure- 32). Bacoside A treatment also significantly reversed the $B_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group with out any change in $K_d$ (Table-30 & Figure-34).

b) Displacement analysis of $[^3\text{H}]\text{QNB}$ against Atropine

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The $K_i$ and $EC_{50}$ decreased in epileptic condition and reversed to near control in Carbamazepine, Bacopa monnieri and Bacoside A treated epileptic rats (Table- 29, 31 & Figure- 33, 35).
II) Muscarinic M1 receptor analysis in post-treated epileptic rats

a) Scatchard analysis of $^3$HJ QNB binding against pirenzepine in the Cerebellum of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacoside A, post-treated Epileptic rats

Scatchard analysis of muscarinic M1 receptors showed that there was a significant decrease in $B_{\text{max}}$ ($p<0.001$) and $K_d$ ($p<0.001$) in epileptic rats compared to control group. In Carbamazepine treated epileptic group $B_{\text{max}}$ significantly ($p<0.001$) reversed to near control when compared to epileptic group. The $K_d$ also reversed to the near control level. Extract of Bacopa monnieri treatment significantly reversed the $B_{\text{max}}$ ($p<0.001$) and $K_d$ ($p<0.01$) to near control when compared to epileptic group (Table-32 & Figure-36). Bacoside A treatment significantly reversed the $B_{\text{max}}$ ($p<0.001$) and $K_d$ ($p<0.01$) to near control when compared to epileptic group (Table-34 & Figure-38).

b) Displacement analysis of $^3$HJ QNB against pirenzepine

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and $K_i$ showed a decrease in all the epileptic group. Treatment with Carbamazepine, Bacopa monnieri and Bacoside A reversed the log (EC$_{50}$) and $K_i$ to near control when compared to epileptic group (Table-33, 35 & Figure-37, 39).
III) Real Time-PCR analysis of Muscarinic M1 receptor mRNA in post-treated epileptic rats

Real Time -PCR analysis showed that the muscarinic M1 receptor mRNA significantly decreased (p<0.001) in epileptic condition and it reversed to control level in Carbamazepine (p<0.01), Bacopa monnieri (p<0.001) and Bacoside A (p<0.001) treated epileptic rats (Table- 36, 37 & Figure- 40, 41).

IV) Muscarinic M1 receptor analysis in pre-treated epileptic rats

a) Scatchard analysis of [3H] QNB binding against pirenzepine in the Cerebellum of Control, Epileptic, Epileptic+Carbamazepine, Epileptic+Bacopa monnieri and Epileptic+Bacoside A, pre- treated Epileptic rats

Scatchard analysis of Muscarinic M1 receptors showed that there was a significant increase in $B_{\text{max}}$ (p<0.001) in epileptic rats when compared to control group. $K_d$ showed no significant change. In Carbamazepine treated epileptic group $B_{\text{max}}$ significantly (p<0.01) reversed to near control when compared to epileptic group. Extract of Bacopa monnieri treatment significantly reversed the $B_{\text{max}}$ (p<0.01) to near control when compared to epileptic group (Table- 38 & Figure- 42). Bacoside A treatment also significantly reversed the $B_{\text{max}}$ (p<0.01) to near control when compared to epileptic group with out any significant change in $K_d$ (Table-40 & Figure- 44).
b) Displacement analysis of $[^3]$H QNB against pirenzepine

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and $K_i$ showed no significant change in the epileptic group compared to control. Carbamazepine, Bacopa monnieri and Bacoside A treatment decreased the log (EC$_{50}$) and $K_i$ (Table-39, 41 & Figure-43, 45).

**Brainstem**

1) Total Muscarinic receptor analysis in post-treated epileptic rats.


Scatchard analysis showed that the $B_{max}$ decreased significantly ($p<0.001$) in the brainstem of epileptic rats with a significant decrease ($p<0.01$) in the $K_d$ when compared to control group. In Carbamazepine treated epileptic rats $B_{max}$ significantly ($p<0.001$) reversed back to near control when compared to epileptic group. The $K_d$ also reversed to near control when compared to epileptic group. Bacopa monnieri treatment significantly reverse the $B_{max}$ ($p<0.001$) to near control when compared to epileptic group with a significant increase in $K_d$ (Table- 42 & Figure- 46). Bacoside A treatment significantly reversed the $B_{max}$ ($p<0.01$) and $K_d$ ($p<0.01$) to near control when compared to epileptic group (Table-44 & Figure- 48).

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and K$_i$ showed no change in all the experimental groups. (Table- 43, 45 & Figure- 47, 49).

II) Muscarinic M1 receptor analysis in post-treated epileptic rats.


Scatchard analysis showed that the B$_{max}$ decreased significantly (p<0.001) in epileptic condition when compared to control group. K$_d$ also decreased significantly when compared to control group (p<0.01). In Carbamazepine treated epileptic condition B$_{max}$ (p<0.001) and K$_d$ (p<0.05) were significantly reversed to near control when compared to epileptic group. Bacopa monnieri extract treatment significantly reversed the B$_{max}$ (p<0.001) to near control when compared to epileptic group. K$_d$ also reversed to near control when compared to epileptic group (p<0.01) (Table- 46 & Figure- 50). Bacoside A treatment also significantly reversed the B$_{max}$ (p<0.001) and K$_d$ (p<0.01) to near control when compared to epileptic group (Table- 48 & Figure- 52).


The competition curve for pirenzepine against $[^3]H$QNB fitted for one site model in all groups. The K$_i$ showed an increase in epileptic group which reversed to
control in the *Bacopa monnieri* and Bacoside A treated rat groups (Table- 47, 49 & Figure- 51, 53). Carbamazepine did not reverse the increased $K_i$ in epileptic rats.

III) Real Time-PCR analysis of Muscarinic M1 receptor mRNA in post-treated epileptic rats

Real Time-PCR analysis showed that the muscarinic M1 receptor mRNA significantly decreased ($p<0.001$) in epileptic condition and it reversed to near control in Carbamazepine treated ($p<0.001$), *Bacopa monnieri* ($p<0.001$) and Bacoside A ($p<0.001$) treated epileptic rats (Table-50, 51 & Figure- 54, 55).

GLUTAMATE RECEPTOR ALTERATIONS DURING EPILEPSY AND AFTER THE TREATMENT WITH *Bacopa monnieri* EXTRACT

**Hippocampus**

I) Total Glutamate receptor analysis in post-treated epileptic rats.

a) *Scatchard analysis of $[^3H] Go glutamate binding against glutamate in the Hippocampus of Control, Epileptic and Epileptic+Bacopa monnieri treated Epileptic rats*

Scatchard analysis showed that the $B_{\text{max}}$ decreased significantly ($p<0.01$) in the hippocampus of epileptic rats without a significant change in $K_d$. Extract of *Bacopa monnieri* treatment significantly reversed the $B_{\text{max}}$ ($p<0.01$) to near control when compared to epileptic group without any change in $K_d$ (Table-52 & Figure-56).
b) Displacement analysis of $[^3]$H Glutamate against glutamate

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and $K_i$ showed a significant decrease in the epileptic group which reversed to near control by Bacopa monnieri treatment (Table- 53 & Figure- 57).

II) Real Time-PCR analysis of NMDA R1 receptor in post-treated epileptic rats.

Real Time-PCR analysis showed that the NMDA R1 receptor mRNA significantly decreased (p<0.01) in epileptic group and it reversed to control in Bacopa monnieri treated (p<0.05) epileptic rats (Table- 54 & Figure- 58).

Cerebellum

I) Total Glutamate receptor analysis in post-treated epileptic rats.

a) Scatchard analysis of $[^3]$H Glutamate binding against glutamate in the Cerebellum of Control, Epileptic and Epileptic+Bacopa monnieri post- treated Epileptic rats

Scatchard analysis showed that the $B_{max}$ decreased significantly (p<0.001) in the cerebellum of epileptic rats with out a significant change in $K_d$. Bacopa monnieri treatment significantly reversed the $B_{max}$ (p<0.001) to near control when compared to epileptic group with out a change in $K_d$ (Table- 55 & Figure- 59).

b) Displacement analysis of $[^3]$H Glutamate against glutamate

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and $K_i$ showed a
decrease in the epileptic group. Treatment with *Bacopa monnieri* reversed the $K_i$ to near control. *Bacopa monnieri* treatment also increased the $EC_{50}$ compared to the epileptic group (Table-56 & Figure-60).

II) Real Time-PCR analysis of NMDA R1 and metabotrophic glutamate 8 receptor in post-treated epileptic rats.

Real Time-PCR analysis showed that the NMDA R1 receptor mRNA significantly decreased ($p<0.001$) in epileptic condition and it reversed to near control in *Bacopa monnieri* ($p<0.01$) treated epileptic rats (Table- 57 & Figure- 61). Real Time-PCR analysis showed that the metabotrophic glutamate 8 receptor mRNA significantly decreased ($p<0.01$) in epileptic condition and it reversed to near control in *Bacopa monnieri* ($p<0.05$) treated epileptic rats (Table- 58 & Figure- 62).

Brainstem

I) Total Glutamate receptor analysis in post-treated epileptic rats.

a) Scatchard analysis of $[^3H] $Glutamate binding against glutamate in the brainstem of Control, Epileptic and Epileptic+Bacopa monnieri treated Epileptic rats

Scatchard analysis showed that the $B_{max}$ decreased significantly ($p<0.01$) in the Brainstem of epileptic rats with out a significant change in $K_d$. *Bacopa monnieri* treatment significantly reversed the $B_{max}$ ($p<0.01$) to near control when compared to epileptic group with out a change in $K_d$ (Table- 59 & Figure- 63).
b) Displacement analysis of $[^3H]$ Glutamate against glutamate

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log (EC$_{50}$) and $K_i$ showed no significant change in experimental groups. (Table- 60 & Figure- 64).

II) Real Time-PCR analysis of NMDA R1 and metabotrophic glutamate 8 receptor in post-treated epileptic rats.

Real Time-PCR analysis showed that the NMDA R1 receptor mRNA significantly decreased (p<0.001) in epileptic condition and it reversed to near control in *Bacopa monnieri* (p<0.01) treated epileptic rats (Table- 61 & Figure- 65). Real Time-PCR analysis showed that the metabotrophic glutamate 8 receptor mRNA significantly decreased (p<0.001) in epileptic condition and it reversed to near control in *Bacopa monnieri* (p<0.05) treated epileptic rats (Table- 58 & Figure- 62)

**Cerebral Cortex**

I) Total Glutamate receptor analysis in post-treated epileptic rats.

a) Scatchard analysis of $[^3H]$ Glutamate binding against glutamate in the Cerebral Cortex of Control, Epileptic and Epileptic+Bacopa monnieri treated Epileptic rats

Scatchard analysis showed that the $B_{\text{max}}$ decreased significantly (p<0.01) in the cerebral cortex of epileptic rats with out any significant change in $K_d$. *Bacopa monnieri* treatment significantly reversed the $B_{\text{max}}$ (p<0.01) to near control when compared to epileptic group with out a change in $K_d$ (Table- 63 & Figure- 67).

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b) Displacement analysis of [3H] Glutamate against glutamate

In the displacement analysis, the competitive curve fitted to a one-site model in all groups with Hill slope values near to unity. The log \( \text{EC}_{50} \) and \( K_i \) showed no significant change in experimental groups. (Table- 64 & Figure- 68).

**NEO-TIMM STAINING IN THE HIPPOCAMPUS IN POST-TREATED EPILEPTIC RATS**

Neo-Timm silver staining in the Hippocampus showed that densely stained CA1 region of the epileptic rats compared to control which confirms mossy fibre sprouting. Treatment with Carbamazepine and *Bacopa monnieri* did not show reversal to the control status (Figure- 69 a-d).

**ELECTROENCEPHALOGRAM ANALYSIS IN PRE- AND POST-TREATED EPILEPTIC RATS.**

Electroencephalogram analysis showed that there is a change in the brain activity of temporal areas of epileptic rats when compared to control. Treatment with Carbamazepine, *Bacopa monnieri* and Bacoside A decreased the change in the brain activity to near control range in both pre-treated and post-treated groups (Figure- 70 a-e, 71 a-e).
MORRIS WATER MAZE EXPERIMENT IN THE POST-TREATED EPILEPTIC RATS

Morris water maze experiment showed a significant increase in the escape latency of epileptic group when compared to control. *Bacopa monnieri* treatment significantly (*p* < 0.001) reversed the escape latency to near control (Table- 65, Figure-72).

Time spent in the platform quadrant of the epileptic rats showed a significant decrease (*p* < 0.01) when compared to control. *Bacopa monnieri* post-treatment (*p* < 0.01) reversed the time spent in the platform quadrant to near control (Table- 66, Figure-73).