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RESEARCH ARTICLE ON TO CARRY OUT PHARMACOLOGICAL EVALUATION OF KAEMPFEROL ON STRYCHNINE INDUCED CONVULSION IN LABORATORY MICE

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ABSTRACT
The naturally occurring flavonoid kaempferol is well-known for its many pharmacological characteristics, which include neuroprotective, anti-inflammatory, and antioxidant actions. This study uses a murine model of strychnine-induced convulsions to assess kaempferol's anticonvulsant potential. Strychnine, a glycine receptor antagonist that causes seizures, was administered to laboratory mice after they had received graded dosages of kaempferol. The latency to seizure onset, length of convulsions, and survival rate were recorded and compared to control groups. Kaempferol's potential as an anticonvulsant agent was shown by the results, which showed that it greatly delayed the start of seizures, decreased the intensity of convulsions, and increased survival rates in a dose-dependent way. The study sheds light on kaempferol's potential as a treatment for convulsive disorders.

KEYWORDS: Kaempferol, anticonvulsant, strychnine, convulsions, laboratory mice, glycine receptor, seizure latency, neuroprotection.

Historical background of epilepsy
The word epilepsy is derived from Greek word Epilambane in and means to seizure upon or to taking hold of or to take over.

Epilepsy is a chronic neurological disorder, with a prevalence of about 1%, which is characterized by the recurrent appearance of spontaneous seizures due to neuronal hyperactivity in the brain (Dell;1986)

Definition of epilepsy
Epilepsy is a chronic neurological disorder, with a prevalence of about 1%, which is characterized by the recurrent appearance of spontaneous seizures due to neuronal hyperactivity in the brain

In 2005, a Task Force of the International League against Epilepsy (ILAE) formulated conceptual and operational definitions of “seizure” and “epilepsy”

Causes of epilepsy
The diagnosis of epilepsy usually requires that the seizures occur spontaneously. Nevertheless, certain epilepsy syndromes require particular precipitants or triggers for seizures to occur. These are termed reflex epilepsy. For example, patients with primary reading epilepsy have seizures triggered by reading. Photosensitive epilepsy can be limited to seizures triggered by flashing lights. Other precipitants can trigger an epileptic seizure in patients who otherwise would be susceptible to spontaneous seizures. For example, children with childhood absence epilepsy may be susceptible to hyperventilation. In fact, flashing lights and hyperventilation are activating procedures used in clinical EEG to help trigger seizures to aid diagnosis.

Finally, other precipitants can facilitate, rather than obligately trigger, seizures in susceptible individuals. Emotional stress, sleep deprivation, sleep itself, and febrile illness are examples of precipitants cited by patients with epilepsy. Notably, the influence of various precipitants varies with the epilepsy syndrome. Likewise, the menstrual cycle in women with epilepsy can influence pattern of seizure recurrence. Catamenial epilepsy is the term denoting seizures linked to the menstrual cycle (Fruchtet al; 2000).

Epidemiology of epilepsy
Epilepsy is clinically similar in developing and developedcountries, but the extent to which patients

withepilepsy are recognized, investigated, and managed isdifferent. Epidemiology, etiology, sociocultural, andeconomic factors all contribute to these differences. Indiais a country with diverse socioeconomic groupings.

Indian cities is as advanced as that anywhere in theworld, yet there remains a large rural population of patientswith epilepsy whose illness is unrecognized anduntreated by medical personnel

Classification of epilepsy
Classification on the basis of etiology
The classification (database) of etiologies of the epilepsiesis divided into four main categories
a) Idiopathic epilepsy—defined here as an epilepsy of predominately genetic or presumed genetic origin and inwhich there is no gross neuroanatomic or neuropathologicabnormality. Included here are epilepsies of presumedmultigenic or complex inheritance, but for which currentlythe genetic basis has not been elucidated.

b) Symptomatic epilepsy—defined here as epilepsy ofan acquired or genetic cause, associated with gross anatomicor pathologic abnormalities, and/or clinical features, indicative of underlying disease or condition. We thus include in this category developmental and congenital disorders where these are associated with cerebral pathologic changes, whether genetic or acquired (or indeed cryptogenic) in origin. Also included are single gene and other genetic disorders in which epilepsy is only one feature of a broader phenotype with other cerebral or systemic effects.

c) Provoked epilepsy—defined here as an epilepsy in whicha specific systemic or environmental factor is the predominantcause of the seizures and in which there are no gross causative neuroanatomic or neuropathologic changes. Some ‘‘provoked epilepsies’’ will have a genetic basis and some an acquired basis, but in many no inherent cause can be identified. The reflex epilepsies are included in this category (which are usually genetic) as well as the epilepsies with a marked seizure precipitant.

d) Cryptogenic epilepsy—defined here as an epilepsy of presumed symptomatic nature in which the cause has not been identified. The number of such cases is diminishing, but currently this is still an important category, accountingfor at least 40% of adult-onset cases of epilepsy. It must be emphasized that there are obviously cases for which categorization is difficult and to a significant extent arbitrary.

Pathophysiology of epilepsy
Epileptic seizures arise from an excessively synchronous and sustained discharge of a group of neurons. The single feature of all epileptic syndromes is a persistent increase of neuronal excitability. Abnormal cellular discharges may be associated with a variety of causative factors such as trauma, oxygen deprivation, tumors, infection, and metabolic derangements. However, no specific causative factors are found in about half of the patients suffering from epilepsy.

Underlying causes and pathophysiological mechanisms are (partially) understood for some forms of epilepsy, e.g. epilepsies caused by disorders of neuronal migration and monogenic epilepsies.

Synaptic Mechanisms
Synaptic pathophysiology of epilepsy and epileptic disorders primarily involves reduced GABA ergic inhibitionor enhanced glutamatergic excitation.

GABA
GABA levels have been shown to be reduced in the cerebrospinal fluid (CSF) of patients with certain kinds ofepilepsy, such as infantile spasms and untreated generalized tonic-clonic seizures, and in excised epileptictissue from patients with drug-resistant epilepsy, suggesting that these patients have decreased inhibition.

Dogs with epilepsy have been shown to have low CSF levels of GABA, and mice genetically susceptibleto audiogenic seizures have a lower number of GABA receptors than non-seizure prone animals. Reduced[3H]-GABA binding to GABA receptors has been reported in human brain tissue, and low glutamic aciddecarboxylase levels have been shown in indled rats and in excised human epileptic tissue, suggestive ofdecreased GABAergic inhibition.

Glutamate
Hippocampal recordings from conscious human brains have shown sustained increases in the levels ofextracellular glutamate levels during and preceding seizures. GABA levels remain low in the epileptogenichippocampus, but during seizures, GABA concentrations increase, although mostly in the non-epileptogenichippocampus. This leads to a toxic increase in extracellular glutamate due to reduced inhibition in theepileptogenic areas.

In human hippocampal epilepsy, densities of glutamate AMPA receptor subunits correlated with the locationsof the densest aberrant mossy fibers. Increases in AMPA receptors inKA receptors have also been shown to beinvolved in ongoing glutamatergic transmission in granulecells of chronic epileptic animals. Thus, while the role of NMDA receptors in epilepsy have been known for sometime, there is now growing evidence of the role of AMPAand KA receptors in epilepsy (During and Spencer; 1993).

Enhancement of synaptic inhibition
Many of the existing AEDs aim to enhance GABAergic inhibition by interacting with fast ionotropic GABAA receptors or by modifying the activity of enzymes and transporters involved in GABA synthesisor reuptake (Meldrum and Rogawski; 2007).

Suppression of synaptic excitation
Glutamatergic excitation may be influenced through action on NMDA, AMPA, or KA receptors.However, AMPA receptors are the most abundant ionotropic glutamate receptors that mediate synapticsignaling (Rogawski; 2011).

Table No. 1: Present treatment of epilepsy.

Seizure Type (epilepsy syndrome)	First choice AED	Other AEDs that are used
Infantile spasms (Westsyndrome)	Vigabatrin	Corticosteroids Nitrazepam Sodium valproate
Focal (Partial seizure)	Carbamazapine	Lamotrigine Levetiracetam Clobazam Topiramate
Generalised Tonic- clonic	Sodium valproate	Carbamazepine Lamotrigine Levetiracetam Topiramate
Myoclonic	Sodium valproate	Levetiracetam Clobazam/clonazepam Topiramate

Role of Neurotransmitters In Epilepsy
Excitatory & Inhibitory Neurotransmitters involved

These play a role in the development of seizure discharge. Glutamate & aspartate are the major excitatory neurotransmitters found in the mammalian brain. Focal application of glutamate to hippocampal slices induces a calcium ion current & depolarizes the neuron. Another excitatory neurotransmitter system, the cholinergic system, has been successfully manipulated to produce experimental limbic seizures.

GABA
Gamma-aminobutyric acid (GABA) has been shown to have inhibitory post-synaptic activity & is one of the principal inhibitory neurotransmitters in the mammalian brain . The GABA receptor has been found in all areas of the brain. This receptor is coupled to the chloride channel (Chloride ionophore), so that GABA binding to its receptor results in a rapid opening of the chloride channel, with an ensuing increase in the post synaptic membrane conductance to chloride. Increased chloride ion permeability stabilizes the cell near its resting membrane potential & reduces its response to excitatory in puts. Modulation of GABA reception chloride ionophore complex mediates the actions of benzodiazepines, & barbiturates, as well as the convulsant effect of picrotoxin& its analogues (Olsen; 1987).

Acetylcholine
The cholinergic system plays a crucial role in modulating cortical and inparticular hippocampal functions including processes such as learning and memory. Cholinergic actions are involved in thephysiopathogenesis of epileptic discharges as suggested by the ability of somecholinergic agents to induce limbic seizures and histopathological changes resemblingthose seen in patients with temporal lobe epilepsy.

Review of literature
BIOLOGICAL ACTIVITIES
de Almeida, R. N., de Sousa *et al*., 2008
Though the etiology of epilepsy is poorly understood, it is thought that an imbalance between glutamate-mediated excitatory neurotransmission and gamma-aminobutyric acid's (GABA) inhibitory influence on neurotransmission causes crises to arise. Furthermore, epileptic seizures can be classified as either generalized seizures, which entail cell malfunction in both hemispheres of the brain, or focal seizures, which impact only a subset of neurons in a specific area of the brain.
Dallas et al., 2006; Potschka and Brodie, 2012, Ferreira et al. (2018.

The transmembrane glycoprotein P (gp-P), which is encoded by the MDR1 gene, serves to shield cells against xenotoxins. Neurones, endothelial cells, and astrocytes in the brain express gp-P. Since it limits the drugs' ability to enter the brain tissue, the overexpression of this protein in the epileptogenic tissue is linked to resistance to antiepileptic medications. suggested assessing the potential of a number of flavonoids, including KPF, to counteract gp-mediated multidrug resistance.

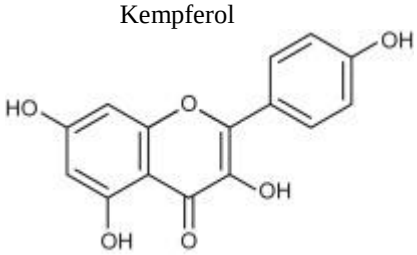
Vezzani et al., 2011; Zhang et al., 2013; Trovato Salinaro et al., 2018
In addition, studies have showed a relationship between neuroinflammation and neurodegenerative diseases such as AD, PD, and epileptogenesis or the development of recurrent epileptic seizures (). Thus, an *in vitro* study with a tiliroside, a KPF derivative 3-O-[(E)-(2-oxo-4- (*p*-tolyl) but-3-en-1-yl] (2.5, 5, and 10 µM), using cell culture BV-2 mouse microglia evidenced an important inhibitory action in the production of proinflammatory mediators such as TNFα, IL-6, PGE2, and nitrite and reduced the levels of proinflammatory proteins COX-2 and iNOS.

Wound healing activity(Mohammed et al., 2012) studied wound healing capacity of Basella alba, in male albino rats. They created burn wounds on theback of rats and treated them with Basella alba leaf extract in glycerin for about 20 days. Their resahs concluded that, rats treated with aqueous leaf extracts showed a maximum wound healing capacity with significant wound ckssure and indicated wound healing capacity of Basella alba.

Antiviral activity (Verma *et al*., 1995) reviewed antiviral activity of many plant tissues. This property is due to the presence of ribosome inactivating proteins (RIP's) present in the extracts of plant tissue (Barbieri *et al*, 1993). All RIPs are with single chain (type 1) or two chains (type 11). (Bolognesi *et al*., 1997) isolated single chain (type 1) ribosome inactivating proteins from the seeds of Basella rubra and tested them for antiviral

activity and inhibited infection of Nicotiana benthamiana by AMVC (Bolognesi *et al*., 1997, Liu et al. 2006).

Taxonomical information of Kaempferol

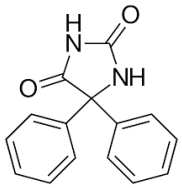
Iupac Name	(3,4,5,7-tetrahydroxyflavone) & 3,5,7-trihydroxy-2-(4-hydroxyphenyl)-4H-chromen-4-one
Chemical Name Structure	Kempferol 
Molecular Formula	C15H10O6
Drug class	Anti-inflammatory, Antiulcer, Antiepileptic
Melting point	277^0C
Form	Solid
Colour	Yellow needles

Name of second Test Drug

Phenytoin

The first antiepileptic drug (AED) to be produced using systematic, scientifically based molecular screening as opposed to random discovery was phenytoin.

Table. Taxonomical information of Phenytoin.

Iupac Name	5,5-diphenylimidazolidine-2,4-dione
Chemical Name Structure	Phenytoin 
Molecular Formula	*C15H12N2O2*
Drug class	Anticonvulsants
Melting point	$295\text{-}298^0$C
Form	Solid
Colour	white
Solubility	Insoluble in water

Phenytoin was tested in rats with induced generalized tonic-clonic seizures. The study found that phenytoin significantly reduced both the frequency and severity of seizures. These findings support its effectiveness in treating similar seizure types in humans, highlighting its role in controlling generalized tonic-clonic seizures. The results contribute to the understanding of phenytoin's clinical applications in epilepsy management.

Neuroprotective Effects

Sayeed et al., 2009 Phenytoin has shown neuroprotective properties in various animal models. Research on traumatic brain injury (TBI) in rodents indicates that phenytoin can reduce neuronal loss and improve functional recovery. Additionally, in cerebral ischemia models, phenytoin administration has been associated with decreased infarct size and better neurological outcomes.

To determine the effect of kaempferol (25, 50 and 100 mg/kg, p.o) on strychnine (STR)-induced convulsions in mice by following parameters:

***In-Vivo* parameters**

- Body weight
- Onset of convulsion
- Duration of clonic convulsion
- Duration of tonic convulsion
- Locomotor activity

***Ex-vivo* parameters**

1. Oxidative stress (SOD, GSH, MDA, Nitric oxide and Total Protein) in brain
2. Brain dopamine levels
3. Brain GABA levels
4. Brain 5-HT levels
5. Brain Na-K-ATPase activity

MATERIAL AND METHODS

Animals

Swiss albino mice weighing 18-22 gm were purchased from Global Bioresearch Solutions Private Limited, H No 251 Nhavi, Tal - Bhor, Dist- Pune, Pune. The animals were housed in polypropylene cages and maintained under the environmental condition of temperature 25±1 ºC and relative humidity of 45-55 % under a 12h light: 12 dark cycles. The animals had free access to food pellets (Nav Maharashtra Chakan oil mills Ltd., Pune) and water ad libitum. The Institutional Animal Ethics Committee (IAEC) of Loknete shri dadapatil Pharate College of pharmacy, Mandavgan pharata approved all the experimental protocols under the Committee for the Purpose of Control and Supervision of Experiment on Animals (CPCSEA). The protocol approval number is 2168/PO/Re/S/22/CPCSEA.

For test drug

Substances: Kaempferol
Dose: 25, 50, and 100 mg/kg
Sites: Oral
Volumes: Not more than 1-2ml
Blood withdrawal: No

For test drug

Substances: Phenytoin
Dose: 25mg/kg
Sites: Oral
Volumes: Not more than 1-2ml
Blood withdrawal: No

Experimental procedure

The effects of Kaempferol and Phenytoin shall be evaluatein the following groups

Experimental designs:

The animals were divided randomly into groups with six mice per group as follows:

Group I: Normal group

The mice received only vehicle (Distilled water).

Group II: STR control

The mice receive STR (5 mg/kg, i.p.) and only vehicle (Distilled water, 10 mg/kg)

Group III: Phenytoin (25) group

The mice have received STR (5 mg/kg, i.p.). They were pre-treated with Phenytoin at a dose of 25 mg/kg, p.o., for 7 days.

Group IV: Kaempferol (25) group

The mice have received STR (5 mg/kg, i.p.). They were pre-treated with Kaempferol at a low dose of 25 mg/kg, p.o for 7 days.

Group V: Kaempferol (50) group

The mice have received STR (5 mg/kg, i.p.). They were pre-treated with Kaempferol at a medium dose of 50 mg/kg, p.o for 7 days.

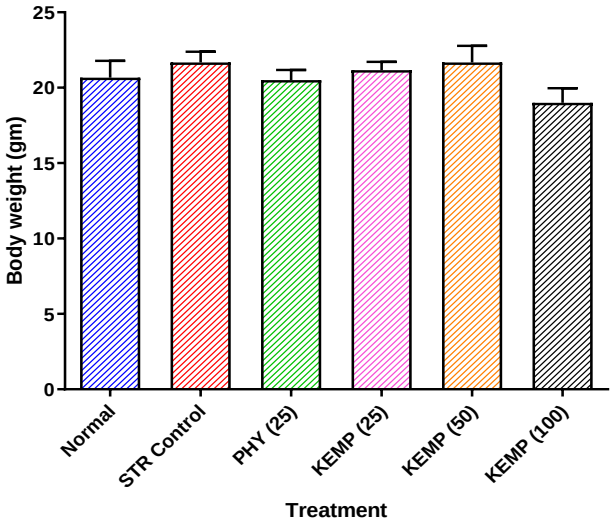
Group VI: Kaempferol (100) group

The mice have received STR (5 mg/kg, i.p.). They were pre-treated with Kaempferol at a high dose of 100 mg/kg, p.o for 7 days.

RESULTS

Effect of kaempferol on body weight

Body weight (gm) Mean ± SEM					
Normal	**STR control**	**Phenytoin (25 mg/kg)**	**Kaempferol (25 mg/kg)**	**Kaempferol (50 mg/kg)**	**Kaempferol (100 mg/kg)**
20.67 ± 1.12	21.67 ± 0.71	20.50 ± 0.67	21.17 ± 0.54	21.67 ± 1.12	19.00 ± 0.97



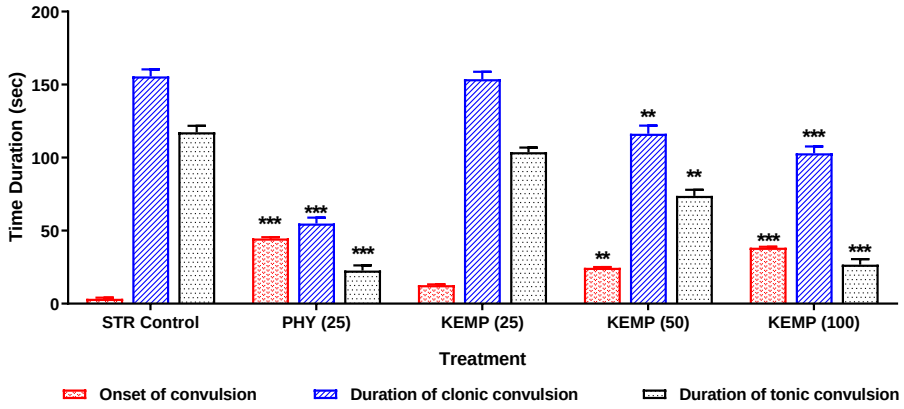
Graphical representation of effect of kaempferol on body weight

Data were analyzed by One-Way ANOVA followed by Dunnett's test.

Body weight did not differ significantly in STR control rats compared to normal rats. There was no significant difference in the body weight of STR control rats post Administration of STR compared with normal rats. Body weight of phenytoin (25 mg/kg, p.o) and kaempferol (25, 50 and 100 mg/kg, p.o.) treated group also did not differ markedly.

Effect of kaempferol on onset and duration of convulsion in STR-induced epilepsy

Parameter	Onset and duration of convulsion Mean ± SEM					
	Normal	STR control	Phenytoin (25 mg/kg)	Kaempferol (25 mg/kg)	Kaempferol (50 mg/kg)	Kaempferol (100 mg/kg)
Onset of convulsion	---	3.33 ± 0.80	44.67 ± 0.80	12.67 ± 0.56	24.50 ± 0.56	38.33 ± 0.67
Duration of clonic	---	155.67 ± 4.75	54.83 ± 4.06	153.67 ± 5.05	116.33 ± 5.52	103.00 ± 4.60
Duration of tonic	---	117.33 ± 4.43	22.67 ± 3.50	103.67 ± 3.22	73.83 ± 4.09	26.67 ± 3.89

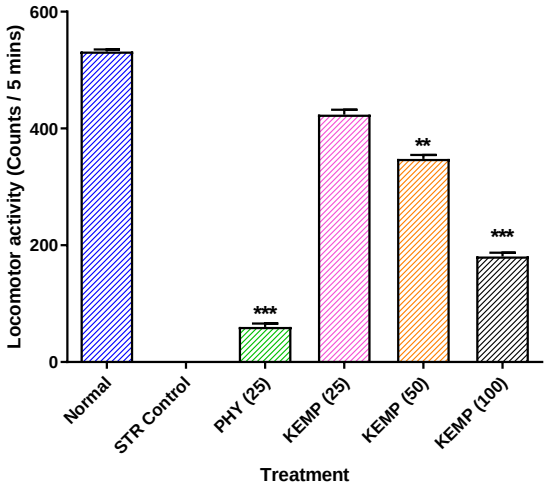


Graphical representation of effect of kaempferol on onset and duration of convulsion in STR-induced epilepsy.

Data were analyzed by one-way ANOVA followed by Dunnett's test $^{**}P < 0.01$ and $^{***}P < 0.001$ compared to the STR control group.

Effect of kaempferol on locomotor activity during STR-induced post-ictal depression

Locomotor activity (Counts / 5 mins) Mean ± SEM					
Normal	STR control	Phenytoin (25 mg/kg)	Kaempferol (25 mg/kg)	Kaempferol (50 mg/kg)	Kaempferol (100 mg/kg)
531.70 ± 3.70	--	60.00 ± 5.83	423.80 ± 8.33	347.80 ± 6.99	180.80 ± 6.33

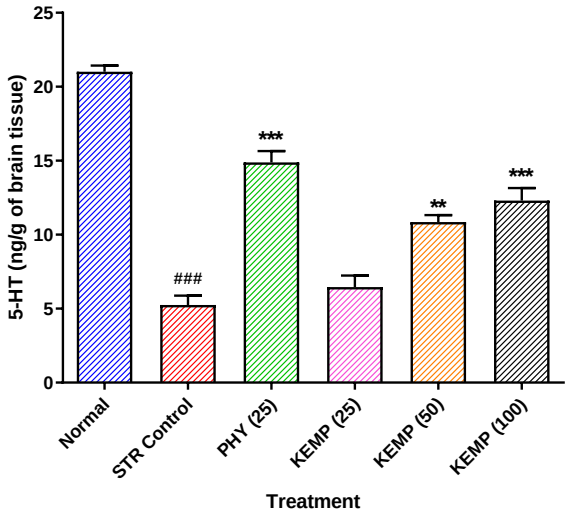


Graphical representation of effect of kaempferol on locomotor activity during STR-induced post-ictal depression

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{**}P < 0.01$ and $^{***}P < 0.001$ compared to with STR control group.

Effect of kaempferol on STR-induced alteration in brain noradrenaline levels

Brain NA (ng/g of brain tissue) Mean ± SEM					
Normal	**STR control**	**Phenytoin (25 mg/kg)**	**Kaempferol (25 mg/kg)**	**Kaempferol (50 mg/kg)**	**Kaempferol (100 mg/kg)**
21.02 ± 0.42	5.25 ± 0.63	14.89 ± 0.77	6.46 ± 0.78	10.85 ± 0.47	12.30 ± 0.86

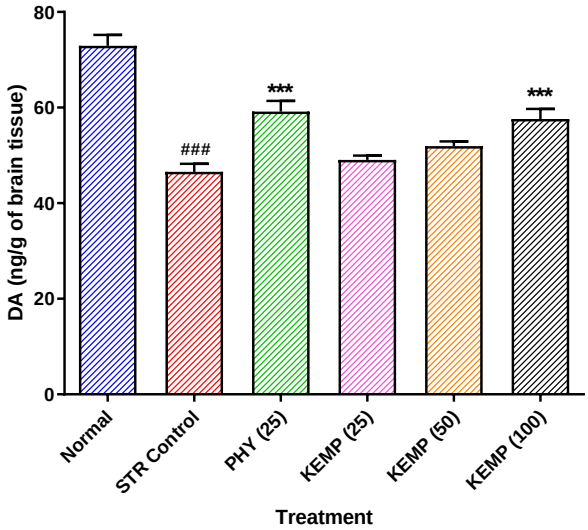


Graphical representation of effect of kaempferol on STR-induced alteration in brain NA levels.

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{###}P < 0.001$ compared to normal group and $^{**}P < 0.01$ and $^{***}P < 0.001$ compared to with STR control group.

Effect of kaempferol on STR-induced alteration in brain dopamine levels

Brain DA (ng/g of brain tissue) Mean ± SEM					
Normal	**STR control**	**Phenytoin (25 mg/kg)**	**Kaempferol (25 mg/kg)**	**Kaempferol (50 mg/kg)**	**Kaempferol (100 mg/kg)**
72.93 ± 2.28	46.58 ± 1.65	59.16 ± 2.26	49.06 ± 0.91	51.95 ± 0.97	57.63 ± 2.09

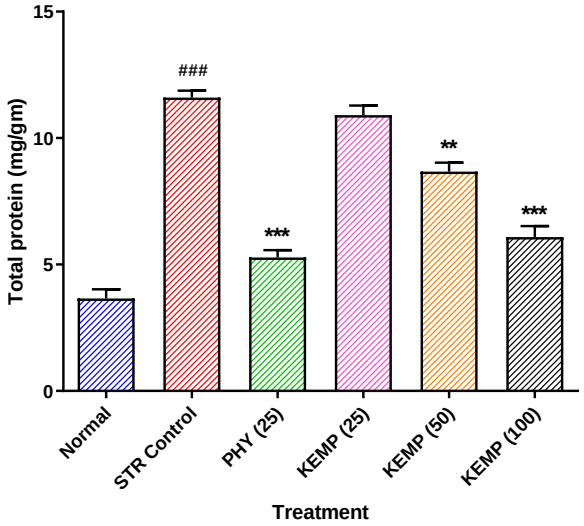


Graphical representation of effect of kaempferol on STR-induced alteration in brain DA levels.

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{###}P < 0.001$ as compared with normal group and $***P < 0.001$ as compared with STR control group.

Effect of kaempferol on STR-induced alteration in brain total protein level

Brain Total protein (mg/gm) Mean ± SEM					
Normal	**STR control**	**Phenytoin (25 mg/kg)**	**Kaempferol (25 mg/kg)**	**Kaempferol (50 mg/kg)**	**Kaempferol (100 mg/kg)**
3.65 ± 0.36	11.60 ± 0.28	5.28 ± 0.29	10.91 ± 0.37	8.67 ± 0.35	6.08 ± 0.44

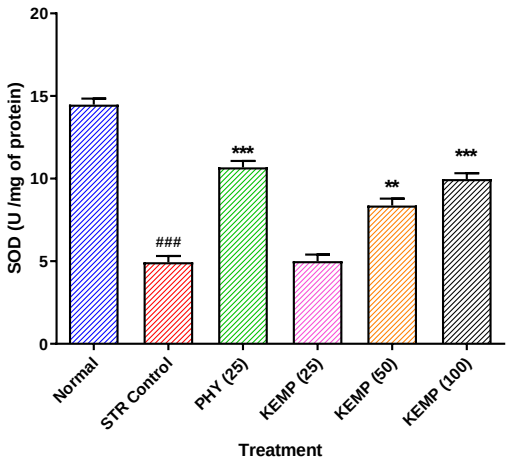


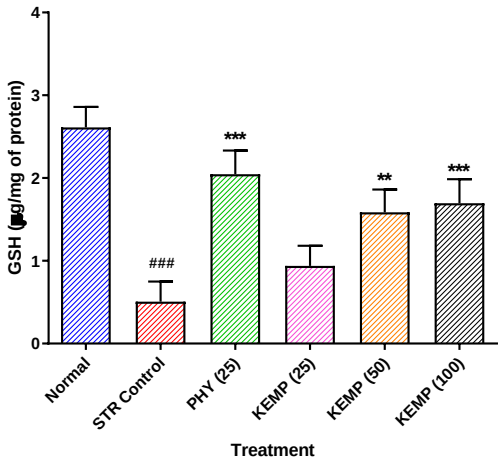
Graphical representation of effect of kaempferol on STR-induced alteration in brain total protein levels.

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{###}P < 0.001$ as compared with normal group and $**P < 0.01$, $***P < 0.001$ as compared with STR control group.

Effect of kaempferol on STR-induced alteration in brain SOD and GSH level

Parameter	Brain SOD (U /mg of protein) and GSH µg/mg of protein) levels Mean ± SEM					
	Normal	**STR control**	**Phenytoin (25 mg/kg)**	**Kaempferol (25 mg/kg)**	**Kaempferol (50 mg/kg)**	**Kaempferol (100 mg/kg)**
SOD (U/mg of protein)	14.48 ± 0.37	4.94 ± 0.38	10.68 ± 0.40	5.00 ± 0.40	8.37 ± 0.41	9.97 ± 0.36
GSH (µg/mg of protein)	2.61 ± 0.25	0.51 ± 0.24	2.05 ± 0.29	0.94 ± 0.24	1.59 ± 0.28	1.70 ± 0.29



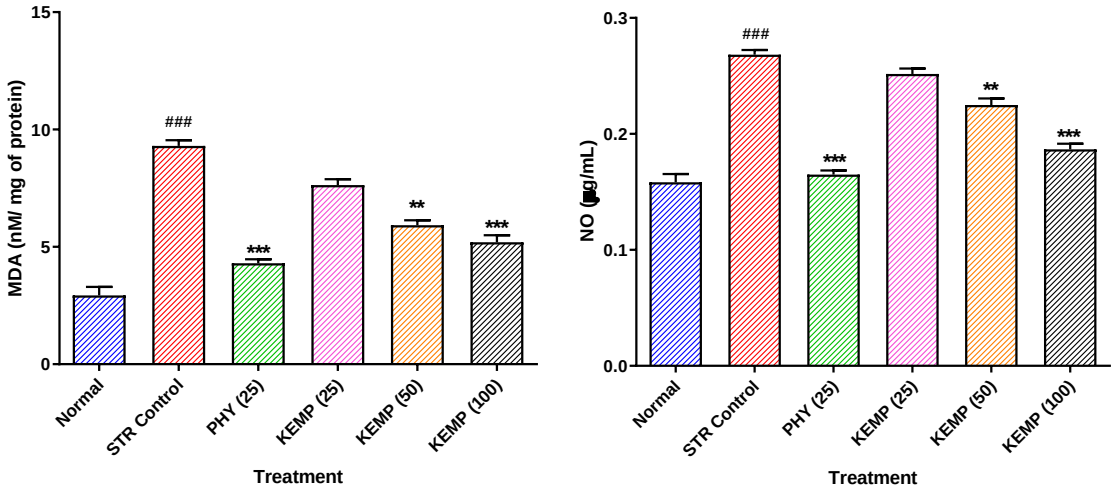


Graphical representation of effect of kaempferol on STR-induced alteration in brain SOD and GSH levels.

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{###}P < 0.001$ as compared with normal group and $^{**}P < 0.01$, $^{***}P < 0.001$ as compared with STR control group.

Effect of kaempferol on STR-induced alteration in brain MDA and nitric oxide level

Parameter	Brain MDA (nM/mg of protein), nitric oxide (µg/mL) Mean ± SEM					
	Normal	STR control	Phenytoin (25 mg/kg)	Kaempferol (25 mg/kg)	Kaempferol (50 mg/kg)	Kaempferol (100 mg/kg)
MDA (nM/mg of protein)	2.93 ± 0.36	9.30 ± 0.24	4.30 ± 0.17	7.63 ± 0.25	5.93 ± 0.21	5.20 ± 0.30
Nitric oxide (µg/mL)	0.158 ± 0.007	0.268 ± 0.004	0.165 ± 0.003	0.252 ± 0.005	0.225 ± 0.006	0.187 ± 0.005



Graphical representation of effect of kaempferol on STR-induced alteration in brain MDA and NO levels.

Data were analyzed by one-way ANOVA followed by Dunnett's test. $^{###}P < 0.001$ as compared with normal group and $^{**}P < 0.01$, $^{***}P < 0.001$ as compared with STR control group.

SUMMARY AND CONCLUSION

Kaempferol treatment demonstrated significant effects on various neurochemical parameters in the study. Notably, brain noradrenaline (NA) levels increased significantly, while dopamine (DA) levels also showed a significant rise at higher doses. Additionally, kaempferol enhanced brain antioxidant activity, with substantial increases in superoxide dismutase (SOD) and glutathione (GSH) levels. Furthermore, these treatments effectively reduced the onset and duration of convulsions induced by STR, as well as significantly decreasing brain total protein, malondialdehyde (MDA), and nitric oxide levels. Overall, kaempferol exhibited promising neuroprotective and anticonvulsant properties, highlighting its potential therapeutic benefits.

Kaempferol exhibits promising anticonvulsant and neuroprotective effects in the context of STR-induced epilepsy. Its ability to modulate neurotransmitter levels, reduce seizure severity, alleviate oxidative stress, and restore enzymatic activity highlights its therapeutic potential. Further research is essential to elucidate the specific mechanisms of action and to assess the safety and efficacy of kaempferol in clinical settings for epilepsy treatment. This study opens avenues for the development of kaempferol as an adjunctive therapy in epilepsy management, particularly for patients who may not respond adequately to conventional antiepileptic drugs.

The study indicates that kaempferol doses of 50 mg/kg and 100 mg/kg showed significant benefits in delaying the onset and reducing the duration of convulsions, as well as improving neurochemical and antioxidant parameters. Thus, these higher doses are considered more effective for its anticonvulsant and neuroprotective effects in the context of STR-induced epilepsy.

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