LETTER TO THE EDITOR

ANTICONVULSANT ACTIVITY OF THE MIXED FATTY ACIDS OF *ELAEOCARPUS GANITRUS* ROXB. (RUDRAKSH)*

Sir,

(Received on April 19, 1984)

The ethanolic extract of the fruits of Rudraksh has been reported to have significant anticonvulsant effect against maximum electroshock seizures (MES) but not against metrazol-induced convulsions (1). Total ethanolic extract of the fruits may contain material besides the principle active against M.E.S. We observed that Rudraksh ethanolic extract contained a mixture of fatty acids in large quantities. Sodium valproate, a branched-chain fatty acid, is a well established anticonvulsant agent. It was, therefore, thought worthwhile to study the anti-convulsant activity of mixed fatty acid of Rudraksh against leptazol and electroshock induced convulsions and compare it with sodium valproate.

Albino mice (CDRI strain) of either sex (15–40 g) were divided in groups (4 to 6 animals per group). They were maintained on pellet diet (Hindustan Lever): all experiments were conducted in winter (ambient room temperature, from 14 to 16°C). Before the tests food was withheld for 24 hrs but water was given *ad lib*.

Seeds obtained from the fruits of *Elaeocarpus ganitrus* (Rudraksh) were extracted by cold maceration in petroleum ether (40–60°C). The extract was saponified to obtain the mixed fatty-acids (yield 26.012 *mg/ml*.) The aqueous solution of the mixed fatty acids was administered orally to mice (400 to 1200 *mg/kg*) prior to convulsion tests. Leptazol 10% I.P. (Bengal Immunity, Calcutta) was used after dilution. Sodium valproate (Reckitt and Coleman, Bombay) tablets were crushed and the powder was suspended in 1% methyl cellulose made in syrup base.

Leptazol test was carried out by injecting the drug (80 *mg/kg*, sc) in groups of mice pretreated either with sodium valproate or mixed fatty acids of Rudraksh. Protection against convulsions was taken as end point and was recorded in quantal assays. ED_{50} was determined by probit analysis, MES test included induction of seizures by electroshocks (45 mA for 1 sec) given through ear electrodes. Abolition of tonic convulsion was considered evidence of protection and was recorded in quantal assays. The assays were performed

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using animals pretreated with either sodium valproate or Rudraksh fatty acids. Toxicity (loss of sense of position, righting reflex, gait and stance, muscle tone and equilibrium) occurring after administration of Rudraksh fatty acid was also watched for.

Using Rudraksh fatty acid oral pretreatment (1200 *mg/kg*) and injection of leptazol in the scraff of the neck the time of peak protective effect was found to be between 4 and 5 hrs. In case of sodium valproate (400 *mg/kg* the peak activity was found to be between 30 min and 1 hr. The ED_{50} of mixed fatty acids was found to be 866 *mg/kg* (with 1137 and 659.4 *mg/kg* as 95% fiducial limits). Dosing the mice with mixed fatty acids at approximately 5 times the ED_{50} did not lead to any neurotoxicity.

Oral administration of sodium valproate (400 *mg/kg*, 30 min to 1 hr before) afforded a total protection against MES. Fatty acids obtained from Rudraksh failed to give any protection to mice against MES even at the dose of 1200 *mg/kg*. Our findings are, thus, different from those of Bhattacharya *et al.* (1) who reported that total ethanolic extract failed to protect rats against metrazol induced convulsions. The mixed fatty acids were relatively nontoxic in mice and hence these fatty acids (and their derivatives) need a more elaborate study as potential anticonvulsants, particularly as anti-petitmal compounds.

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