INTRODUCTION
Ayurvedic formulations are generally multicomponent and are based on the concept that such combinations provide synergistic therapeutic effects and eliminate any adverse effects (Charaka). Mentat is an herbal psychotropic preparation containing the following indigenous ingredients, reputed in the ancient system of Ayurvedic medicine to be of value in the management of nervous disorders: Brahmi (*Hydrocotyl asiatica*), Ashvagandha (*Withania somnifera*), Vacha (*Acorus calamus*), Shatavari (*Asparagus racemosus*), Amla (*Emblica officinalis*), Shankhapushpi (*Evolvulus alsinoides*) and Triphala. The majority of the ingredients are plant derived nerve tonics and some putative general tonics and vitalisers.

EFFECTS ON LEARNING AND MEMORY
Mentat was extensively investigated for its psychopharmacological actions employing different animal models. The studies of Kulkarni and Verma demonstrated that Mentat improved memory functions. It reduced scopolamine-induced delay in transfer latency in mice when tested on elevated plus maze and also attenuated acute and chronic retrograde amnesia induced by electroconvulsive shocks (ECS) in rats tested for a passive avoidance paradigm. Mice treated with Mentat when tested for their learning task in the passive avoidance paradigm, showed decreased latency to reach the shock-free zone (acquisition) and exhibited lesser number of mistakes (descents). Compound also reversed scopolamine-induced delay in retention latency and reduced the number of mistakes. While its effects were comparable with aniracetam, a known nootropic, a combination of Mentat and a low dose of aniracetam had a more superior action in reducing the number of mistakes (descents) in the passive avoidance task. These workers used Mentat both acutely and chronically.

Using a different task performance, other studies have confirmed the protective effect of Mentat against ECS-induced anterograde and retrograde amnesia in the complex and T-mazes. In an animal model of Alzheimer’s disease, colchicine and ibotenic acid (intracerebroventricularly) produced marked deficits of the learned active avoidance task and Mentat reversed this effect in a dose-dependent fashion. Furthermore, compound also augmented learning acquisition and retention of learning in normal rats, as well as in states of cognitive deficits induced by prenatal undernutrition, postnatal environmental impoverishment and sodium nitrite hypoxia.

CLINICAL STUDIES
Clinical studies with Mentat have shown that it improves memory quotient in normal subjects of different age groups, increases memory span and attenuates fluctuations of attention in normal adults and improves learning ability in children with behavioural problems or minimal brain damage.

DEADDICTION PROFILE
In a series of experiments conducted in rats and mice, Kulkarni and co-workers tested the deaddiction profile of Mentat for its usefulness in preventing development of tolerance and addiction to opiates, alcohol and benzodiazepines.

Chronic administration of morphine produced rapid development of tolerance to analgesic response and the animals exhibited naloxone-precipitated withdrawal jumps on the 10th day of testing. Although Mentat did not exhibit any analgesic response *per se*, it did prevent the development of tolerance to morphine.
tolerance to the analgesic response to morphine. Mentat also suppressed the precipitated withdrawal jumps due to naloxone in a dose-dependent manner\textsuperscript{12}.

Mice made alcohol-dependent by chronic administration, on deprival of alcohol showed increased anxiety as they preferred to stay on the closed arm of the elevated plus maze. They also showed greater percent preference to closed arms. However, mice receiving chronic Mentat followed by alcohol failed to show any withdrawal-induced anxiety\textsuperscript{13}.

In another classical paradigm where threshold to pentylenetetrazole (PTZ) convulsion was studied in alcohol withdrawn rats, Mentat treatment offered protection against PTZ-induced convulsions. The protective effect was more pronounced when Mentat was given chronically\textsuperscript{13}. Similarly, abrupt cessation of chronic treatment of diazepam (20 mg/kg/day for 21 days) produced withdrawal reactions. Animals showed anxiety and hyperlocomotion. Concomitant administration of Mentat reversed the acute as well as chronic diazepam-induced hyperlocomotion and behavioural excitation\textsuperscript{14}.

In another study with triazolam, a short-acting benzodiazepine, Mentat also prevented the development of withdrawal-induced anxiety response. Treatment with compound also prevented the amnesic effect of the benzodiazepines\textsuperscript{15}.

OTHER EFFECTS
The effects of Mentat on animal behaviour have been reported using battery of test procedures\textsuperscript{16-18}. Mentat exhibited a dose-dependent response on open-field test, elevated plus maze, social interaction and Vogel’s conflict tests. It also exhibited antiaggressive and antidepressant effects. In the chronic model of epilepsy, Mentat offered significant protection against PTZ-induced chemical kindling in mice. The protective effect was comparable to the protective effect of classical antiepileptic agents\textsuperscript{19}. Mentat did not interfere with $\alpha_2$ or dopamine autoreceptors but did enhance postsynaptic dopamine receptor activity\textsuperscript{20}.

SAFETY STUDIES
Since times immemorial, man has made use of plants to treat diseases. The history of use of plants dates back to the Rig Veda (circa 2500 BC). Although the principles of preparation of Ayurvedic drugs takes into consideration their safety, modern testing procedures were also used to assess the safety of Mentat preparation. Both acute and subacute toxicity studies in mice revealed that the preparation was safe up to 2400 mg/kg p.o. The preliminary reports on teratogenic studies indicated no foetal abnormalities. Chronic administration of Mentat (3 mg/kg) for 90 days did not cause any change in biochemical or haematological parameters\textsuperscript{21}.

MANUFACTURER: The Himalaya Drug Co.

REFERENCES
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