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Trazodone: A Review of its Pharmacological Properties and Therapeutic Use in Depression and Anxiety

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Authors **Authors and affiliations**

R. N. Brogden, R. C. Heel, T. M. Speight, G. S. Avery

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Summary

Synopsis

Trazodone[1] is a triazolopyridine derivative with antidepressant activity, that is chemically unrelated to other currently available antidepressants. Its pharmacological properties differ from those of most other antidepressants. Trazodone exhibits antiserotonin activity in animal studies, but its mechanism of action in depressive illness in humans is not clear. It has an overall therapeutic efficacy comparable with imipramine and amitriptyline in depressive illness but, at dosages which have achieved a similar overall clinical improvement, trazodone causes fewer anticholinergic side effects than the tricyclic antidepressants. Trazodone appears also to have activity against the concomitant anxiety in depressed patients and in limited studies was comparable with diazepam and chlordiazepoxide in anxiety neurosis. Trazodone has been reported to be of value in tremors and chronic alcoholism. Studies in animals, limited human studies and the low incidence of cardiovascular side effects in controlled therapeutic trials, suggest that trazodone is less likely than imipramine to cause cardiotoxicity at therapeutic doses, but the effects of overdosage are not known at

present. Trazodone appears to be well tolerated by the elderly, seldom aggravates psychotic symptoms and does not produce neurological side effects.

Pharmacology

Trazodone is a triazolopyridine derivative which differs chemically from other currently available antidepressants. Although trazodone bears some resemblance to the benzodiazepines, phenothiazines and tricyclic antidepressants, its pharmacological profile differs from each of these classes of drugs. The basic idea for the development of trazodone was the hypothesis that depression involves an imbalance of the mechanism responsible for the emotional integration of unpleasant experiences. Consequently, new animal models of depression consisting of responses to unpleasant or noxious stimuli, instead of the current tests related to the aminergic theory of depression, were used in studying the drug. Trazodone inhibits serotonin uptake into rat brain synaptosomes and by rat platelets, at relatively high concentrations and inhibits brain uptake of noradrenaline *in vitro* only at very high concentrations. It possesses antiserotonin and α-adrenergic blocking effects. The anticholinergic activity of trazodone is less than that of the tricyclic antidepressants in animal studies and this has been confirmed in therapeutic trials in depressed patients.

The electroencephalographic profile of trazodone in humans is distinct from that of the tricyclic antidepressants or the benzodiazepines, although bearing some resemblance to these agents in its effect on certain wavebands. Limited studies of the cardiovascular effects of trazodone in humans and His-bundle and surface electrocardiograms in dogs suggest that trazodone is less liable than imipramine to cause important adverse effects on the heart, but further studies and reports on the effect of overdosage are necessary to determine its safety relative to that of mianserin, nomifensine or zimelidine.

Pharmacokinetics

Trazodone is well absorbed after oral administration. Absorption is delayed and somewhat enhanced by food. The area under the plasma concentration-time curve is directly proportional to dosage after oral administration of 25 to 100mg. Trazodone is extensively metabolised, less than 1% of an oral dose being excreted unchanged in the urine. The main route of elimination is via the kidneys with 70 to 75% of an oral dose being recovered in the urine within the first 72 hours of ingestion. The elimination half-life for unchanged drug has been reported as 6.3 hours and for total drug and metabolites 13 hours.

Therapeutic Trials

Open studies in patients with depressive illness have reported some degree of improvement in about 90% of patients with about 50% showing considerable

improvement, but such studies are of no value in demonstratinng the therapeutic efficacy of the drug. A wide range of dosages have been used, but there is little clear indication of the optimum dosage for the various diagnoses. Favourable results have also been reported in geriatric patients who have generally tolerated the drug well.

In double-blind controlled trials trazodone has been compared with placebo, imipramine, amitriptyline, and desipramine in depressive illness, with amitriptyline in depression with associated anxiety and with the benzodiazepines in anxiety with or without depressive symptomatology. In endogenous depression trazodone is efficacious in doses approximately twice that of imipramine. In a series of generally fairly well designed placebo-controlled studies in homogenous groups of patients, there was no significant difference between trazodone and imipramine with respect to overall efficacy, but there were some differences with respect to their effect on individual symptom clusters on the Hamilton rating scale for depression. However, trazodone caused fewer and less severe anticholinergic side effects than imipramine. No significant difference between trazodone 150 to 400mg and amitriptyline 75 to 200mg was detected in a well designed and conducted study but, not surprisingly, the antianxiety activity of trazodone was greater than that of desipramine. There was no consistent difference between the drugs with respect to the rapidity of onset of their antidepressant activity.

In geriatric patients as in younger patients, trazodone was comparable in therapeutic efficacy with imipramine but was better tolerated than the tricyclic antidepressant. Trazodone 75 to 150mg daily was more effective than placebo and comparable with 50 to 150mg of thioridazine in depressed geriatric patients.

In unselected groups of schizophrenic patients trazodone alone was less effective than chlorpromazine, but trazodone improves depression in patients with schizophrenia and secondary depression. As suggested in open trials, trazodone seldom appears to worsen psychotic symptoms or to cause extrapyramidal side effects.

The response to trazodone 75mg daily of patients with anxiety neurosis or predominantly anxious symptoms, was comparable with that of diazepam 15mg in limited studies. Similarly, trazodone 75 to 150mg was comparable in efficacy and tolerability to chlordiazepoxide 30 to 60mg, but further studies are needed to determine the role of trazodone in such patients.

In the studies conducted to date, the lack of statistically significant differences between trazodone and the standard comparative drugs may indicate that they are therapeutically equivalent, but as the number of patients in each treatment group has generally been small and the duration of observation rather short, it is also likely that too few patients were studied to detect significant differences between drugs expected to give similar results.

Oral and intravenous trazodone has been successfully used to treat tremor as well as anxiety and depression associated with alcoholism, and drug-induced Parkinsonism, dyskinesia and tardive dyskinesias.

Side Effects

Trazodone has generally been well tolerated, the most frequently reported adverse effects being drowsiness, dizziness and gastrointestinal upset. The low incidence of cardiovascular side effects along with findings in studies in dogs and rats and limited studies in humans indicate that important adverse effects on the heart may be less likely with trazodone than with the tricyclic antidepressants. Anticholinergic-like side effects occur less frequently with trazodone than with imipramine and amitriptyline.

Dosage

The initial dosage is 50 to 150mg daily in divided doses and should be increased gradually according to response. The usual adult therapeutic dose is 150 to 400mg daily for outpatients and up to 600mg daily in divided doses for inpatients or more severely depressed patients. Daytime drowsiness can be minimised by giving most of a daily dose at bedtime.

Various sections of the manuscript reviewed by: *F.J. Ayd*, Baltimore, Maryland, USA; *J.I. Escobar*, Veterans Administration Medical Center, Los Angeles USA; *S. Garattini*, Istituto Di Ricerche Farmacologiche 'Mario Negri', Milan, Italy; *R.H. Gerner*, Neuropsychiatric Institute, University of California, Los Angeles, USA; *M.H. Lader*, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London; *K. Rickels*, Psychopharmacology Research Unit, University of Pennsylvania, Philadelphia, USA; *G. Tognoni*, Istituto Di Ricerche Farmacologiche 'Mario Negri', Milan, Italy; *D. Wheatley*, The General Practitioner Research Group, Twickenham, England.

'Desyrel' (Mead Johnson); 'Molipaxin' (Roussel); 'Trittico' (Angelini); 'Thombran' (Thomae).

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