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Introduction

PETER TYRER

Serotonin, or 5-hydroxytryptamine (5-HT), is rapidly becoming as important to biological psychiatry as DNA is to molecular biology. Time after time, investigations into the mechanisms lying behind psychiatric disorders lead back to the 5-HT receptor. This is both encouraging and puzzling. It is encouraging because we now have many drugs available that act on 5-HT receptors, both as agonists and antagonists, and so it seems likely that through these, we will be able to improve our treatment of many types of psychiatric disorder. It is puzzling because the imagination is stretched to believe that the 5-HT receptor is at the heart of psychopathology; some of the changes found are likely to be secondary phenomena and the prime target for investigation likely to lie elsewhere. Unravelling which are the primary serotonergic events is therefore a major task, and we have only just made a start on it.

In this supplement, Dr Cowen maps out the territory for the explorer entering the serotonin jungle and outlines the most encouraging trails. Professors Kellner & Uhlenhuth do the same for anxiety, illustrating the difficulty that investigators have in judging whether any treatment is acting primarily as an anti-anxiety agent or whether it is reducing anxiety by other means. This difficulty in separating anxiety from other disorders, particularly depressive ones, is explored further by Hans-Ulrich Wittchen and his colleagues from Munich, who demonstrate the value of allowing several diagnoses simultaneously in the schema of neurotic disorders, rather than attempting to force them into a hierarchy.

The remaining three papers in the supplement concentrate on the 5-HT_{1A} receptor and in particular on the effects of the drug buspirone. It is an unfortunate fact that almost all anti-anxiety drugs carry with them the risk of dependence. This may be because the underlying mechanism of action of alcohol, barbiturates, chloral hydrate, and the benzodiazepines is fundamentally the same – the facilitation of the inhibitory neurotransmitter, gamma-aminobutyric acid (GABA). The discovery that buspirone and subsequently other drugs act on 5-HT receptors and that their anti-anxiety effects are quite independent of stimulation of GABA is therefore of considerable interest. Dr Tollefson and Professor Lader write articles that neatly dovetail in explaining how buspirone not only lacks the abuse potential of other anti-anxiety drugs, but may be of value in treating alcohol dependence. Drs Napoliello & Domantay survey the literature on buspirone and provide pointers to its use in clinical practice. They return to the anxiety/depression diagnostic debate and suggest that this drug may be useful in a spectrum of disorders which at present are not satisfactorily defined clinically. They also refer to one of the major difficulties with buspirone in clinical practice: the delayed onset of efficacy and absence of sedation and euphoria. Both practitioners and patients have become used to anti-anxiety drugs acting quickly, and the authors note that some education for patients is necessary if good compliance is to be achieved. Education of doctors seems to be an essential preliminary to this, however.

This supplement will be of interest to psychiatric nosologists, epidemiologists, pharmacologists, and clinicians. It is likely to be one of many publications linking serotonin to anxiety in the coming years, and illustrates that understanding of the subject requires appreciation of many disciplines. The future serotonologist needs to be a super-specialist, and a polymath to boot.

Peter Tyrer, MD, MRCP, FRCPsych, *Consultant Psychiatrist, St Charles' Hospital, London W10 6DZ*

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