Editorial

David Taylor

Ther Adv Psychopharmacol

[2012] 2(1) 3

DOI: 10.1177/

2045125312437191

© The Author(s), 2012.

Reprints and permissions:
[http://www.sagepub.co.uk/
journalsPermissions.nav](http://www.sagepub.co.uk/journalsPermissions.nav)

It is somewhat humbling to reflect upon how little we know about intricacies of human psychopharmacology and on how many questions remain unanswered. In this issue we report on one of the mysteries of prescribing in schizophrenia – how does antipsychotic polypharmacy start and why does it continue. Patrice Grech examined records of 38 patients who had been receiving antipsychotic polypharmacy (in regimens that did not include clozapine) for more than six months. Most often, polypharmacy was deliberately introduced to try to improve efficacy or reduce adverse effects. A good proportion of patients were not obviously treatment-resistant. Improvement in symptoms of psychosis as a result of polypharmacy was noted only in a minority but regimens were maintained because of patients' refusal to change or because of prescribers' fears of symptoms returning. Most prescribers had considered abandoning polypharmacy but continued largely for the reasons already mentioned.

One of the results of antipsychotic polypharmacy is that patients end up on a combined high dose of antipsychotics. It may well be that it is this dose increase that prescribers are seeking and patients holding on to. In long term treatment, it is highly probable that dopamine supersensitivity develops allowing the emergence of tardive dyskinesia. Paul Fallon and colleagues take this observation

further by examining the possibility that this presumed supersensitivity leads not only to tardive dyskinesia but also to psychotic relapse. They found that relapse regularly occurred in patients who were compliant with medication, non substance misusers, and who had had no clear precipitating life event. The presence of abnormal involuntary movements was related in several ways to severity and type of symptoms and to recovery, so strongly associating these outcomes with dopamine supersensitivity.

In a related paper, Hidenobu Suzuki and Keishi Gei report on the effect of formulation (and so pharmacokinetic profile and drug exposure) on response to risperidone. Subjects switched from oral risperidone to equivalent doses of depot risperidone were observed to show improvements in cognitive functioning. The authors aver that this arose because of the smaller peak to trough variation of risperidone injection which allowed lower doses of anticholinergic drugs to be used, so affording improvements in processing and attention.

Antipsychotic dose, polypharmacy and formulation each then have marked effects on patient outcome. The findings of the three full papers in this issue could be said to call for the prescribing of low-dose depot antipsychotics in simple regimens. Advice on which most of us might agree.

Correspondence to:

David Taylor
Maudsley hospital
Denmark Hill,
London United Kingdom
David.Taylor@slam.nhs.uk

Visit SAGE journals online
<http://tpp.sagepub.com>

SAGE JOURNALS
Online