Editorial

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How many varieties of depression are there? This seems a strange and perhaps inconsequential question to ask but the answer is quite a few. We know that depressions differ in their temporal associations (e.g. antenatal, postnatal, brief reactive depression, etc.), but there has been some sort of tacit consensus that all depressions are symptomatically similar, if not identical, and all respond to antidepressants. In this issue, Lisa Lloyd and colleagues systematically review data relating to bipolar depression and conclude that it is importantly different from unipolar depression. For example, people with bipolar depression have more frequent episodes which are relatively more resistant to treatment and which are sometimes characterized by atypical symptoms such as hypersomnia and hyperphagia. Perhaps most importantly, the effect of antidepressants in bipolar depression is questionable, at best. This makes early and accurate diagnosis all the more crucial so that appropriate and effective treatment can be given. However, as Lloyd and colleagues point out, many people with bipolar depression wait many years for an accurate diagnosis and many remain undiagnosed and are assumed to have recurrent unipolar depression.

Antidepressants are undoubtedly effective in unipolar or major depression and have a wide range of benefits on mood and cognition. The study in this issue by Ula Knorr and colleagues shows that

beneficial effects of cognition are very probably only seen in depressed individuals. In their study of nondepressed first-degree relatives of patients with depression, escitalopram had no effect on cognitive function or mood. This absence of any effect in nondepressed people is intriguing and seems to indicate a specific biological mechanism in treating depression. It also renders ridiculous the popular press's puerile labelling of antidepressants as 'happy pills': antidepressants only make depressed people less depressed.

In the third paper in this issue, Johan Lataster and coworkers examine the effects of the use of aripiprazole on emotional well being. Dopamine is the key neurotransmitter in the mammalian reward system and blocking dopamine transmission (with standard antipsychotics) is known to lead to dysphoria and to activities which suggest inhibition of reward mechanisms (increased eating and smoking, for example). Aripiprazole is a partial agonist which both blocks and, to a lesser extent, stimulates dopamine receptors. It was conjectured that patients switched from pure dopamine antagonists to aripiprazole might show improved emotional well being. In fact, subjects tended to show an increase in emotional dampening when switched to aripiprazole. An unexpected but potentially fruitful observation insofar as what this indicates about the action of partial agonists.

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