we are currently attempting to replicate the findings in more detail and under blind conditions.

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Quinine Pychosis

Sir: We report a case in which a psychotic illness may have been precipitated by exposure to quinine.

Case report: A 36-year-old female was admitted under Section 2 of the Mental Health Act with a three-day history of persecutory delusions and increasingly disturbed behaviour. She had no previous psychiatric history. She had not taken any medication but admitted to the possibility that she may have taken homeopathic doses of quinine.

On admission she was carrying a crucifix and praying aloud. She appeared agitated and suspicious, keeping her distance, and behaving aggressively if approached. She refused to answer questions, and her talk was rapid and rambling with outbursts of shouting. She expressed delusions of reference, and claimed that she had uncovered a drugs ring involving her employer. She did not appear to have any disorder of perception and there was no clouding of consciousness. A provisional diagnosis was made of acute psychosis – cause unknown: she was treated with parenteral chlorpromazine (100 mg) and diazepam (17 mg). She received no other medication, and within 36 hours of admission mental state examination had become essentially normal. Her relatives confirmed that she was by then her usual self.

Physical examination was normal. Investigations that were reported as normal included full blood count, plasma urea and electrolytes, and tests of liver, renal, and thyroid function. Blood glucose was 3.40 $mmol^{-1}$. However, a routine drug screen of urine revealed the presence of quinine and its metabolites: quantitative assay was not possible.

At follow-up 3 months later she remained well and the final diagnosis was therefore acute psychosis probably secondary to quinine ingestion.

While it is difficult to establish direct cause and effect in these conditions, the clinical picture, time course, and rapid resolution with very little treatment in this case were typical of the 'symptomatic' psychoses (Lishman, 1987; Granville-Grossman, 1971) and no other psychological or physical precipitants could be identified.

Quinine is the optical isomer of quinidine, which has itself recently been reported as causing a transient psychotic state in two cases (Deleu & Schmedding, 1987). Quinine is used mainly as an anti-malarial drug and quinidine in the treatment of cardiac arrythmias, but both are apparently also used in homeopathic medicine. The main side-effects of both drugs are described as 'cinchonism' and include nausea, tinnitus, and blurred vision; these are usually dose-related, but hypersensitivity reactions to smaller doses do occur (Dukes, 1984).

The mechanism for any possible effect is unclear. Deleu & Schmedding (1987) suggested that it may be an idiosyncratic reaction, but this does not really explain any relationship. In view of their two cases and our findings we suggest that quinine and quinidine should be added to the list of possible precipitants of symptomatic psychoses and that they should be considered in the differential diagnosis of acute psychotic reactions. The urine assay is relatively simple and may be carried out as part of a routine drug screen.

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Bulimia Nervosa in an Atypical Setting: Case Report from Nigeria

Sir: Eating disorders (bulimia nervosa and anorexia nervosa) have been proposed as culture-bound syndromes occurring in Western and rapidly westernising cultures (Swartz, 1985; Prince & Tchengh-Laroche, 1987; Selvini Palazzoli, 1985). Until recently, the typical profile of sufferers was of young, middle class, female Caucasians. Selvini Palazzoli has