Prevalence of obsessive compulsive disorder in first- and multi-episode male patients with schizophrenia-spectrum disorders

To the Editor: Studies suggest that obsessive compulsive disorder (OCD) is a prevalent disorder (7.8 - 31.7%) in patients with schizophrenia and first-episode psychosis.[1,2] Despite the varied study designs that have been employed, calculated co-morbidity rates support the conclusion that this co-morbidity is not likely to be an incidental finding.[2]

However, there has been little study of co-morbid OCD in non-Caucasian patients with schizophrenia-spectrum disorders. Since it has been suggested that certain ethnic groups (e.g. Asians) may have a lower prevalence of OCD,[3] this paper reports on the prevalence of co-morbid OCD in a group of mixed-race male patients with either first-episode psychosis or multiple-episode schizophrenia. This mixed-race group can be traced back to Caucasian, Eastern and African origin and is predominantly Afrikaans-speaking.[4]

Male patients were recruited from inpatient acute admission units at Stikland Hospital (Bellville, Western Cape, South Africa). The two groups (first-episode psychosis and multiple-episode schizophrenia) were recruited independently of one another and each group was assessed by a separate interviewer. The studies were approved by the Ethics Committee of the University of Stellenbosch and all subjects provided written, informed consent.

The first group ($N = 24$) comprised first-episode psychosis patients with a diagnosis of schizophrenia, schizoaffective disorder or schizophreniform disorder. Patients were diagnosed at baseline (before or within 4 weeks of initiation of antipsychotic treatment) according to *Diagnostic and Statistical Manual-IV (DSM-IV)* criteria[5] using the Structured Clinical Interview for *DSM-IV* (SCID-I)[6] and if obsessive compulsive symptoms (or OCD) were present, these were quantified by means of the Yale-Brown Obsessive-Compulsive Checklist and Severity Scale (Y-BOCS).[7]

Participants in the second group ($N = 63$) had a diagnosis of schizophrenia and at least one previous admission for a psychotic episode. Patients were diagnosed according to *DSM-IV* criteria[5] using the Diagnostic Interview for Genetic Studies (DIGS version 2.0)[8] one week after admission.

The diagnosis of OCD was made using the relevant sections of the DIGS or SCID-I. Both these assessment tools are comprehensive clinical interviews designed for assessment of anxiety, psychotic disorders, mood disorders and their spectrum conditions. Ratings were performed by a psychiatrist (PPO and LK). Prevalence rates were calculated using SPSS 10.0 for Windows.

The sample across the two groups comprised 87 men. The first-episode group consisted of 20 men with a mean age of 29.4 (± 9.3) years and a duration of illness of 1 (± 2.07) years. The multiple-episode psychosis group consisted of 63 men with a mean age of 34.3 (± 8.6) years and a duration of illness of 13.27 (± 9.1) years. The prevalence of OCD in the sample as a whole was 1.1%. One patient (4.2%) in the first-episode group (diagnosis of schizophrenia, treatment-naïve) and none in the multiple-episode group fulfilled criteria for OCD.

Therefore, in this group of male patients of mixed ethnicity with first-episode psychosis and multiple-episode schizophrenia, the prevalence of co-morbid OCD was lower than that previously reported in Caucasian samples. In considering the first-episode group separately, the rate (4.2%) is still in the lower range of reported values.[1,2]

A number of limitations are worth considering. First, as this report focuses on male patients no conclusions can be drawn about prevalence rates of OCD in this population of females of mixed ethnicity. However, gender does not appear to impact significantly on rates of OCD in schizophrenia and other psychotic disorders since no consistent gender differences have been reported in prevalence studies to date.[1,2] Second, although structured clinical interviews were used, these (SCID-I, DIGS, Y-BOCS) were not standardised across the groups. Moreover, the Y-BOCS was administered to the first-episode psychosis group only, which did not permit assessment of OC symptom severity in the other group. Third, since this was a cross-sectional interview historical information may have been missed and the different symptom patterns waxing and waning during the course of illness may have impacted on these results. Fourth, assessment instruments (DIGS and SCID-I) were translated orally into Afrikaans during the interviews. Formal translation and cross-cultural adaptation of these instruments would have been preferable. Nevertheless, the patients with co-morbid OCD displayed OCD symptom patterns comparable to those documented in Caucasian and black African patients.[9,10]

In conclusion, further studies using culturally adapted instruments are needed. If low rates of OCD are replicated in other studies of patients (of mixed ethnicity) with schizophrenia, it may suggest that other factors, such as cultural or genetic factors, have an important role to play.

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Molecular diagnosis of cystic fibrosis in South African populations

To the Editor: Cystic fibrosis (CF) is present in all South African population groups. In a significant proportion of patients a diagnosis of CF can be confirmed by DNA analysis and the detection of two CF transmembrane conductance regulator (CFTR) mutations, using the panels of mutations developed in this study. The index of suspicion will also be raised in patients with a single CFTR mutation. DNA testing is important, especially in regions without access to reliable sweat tests, and should be considered an aid to diagnosis. In addition to receiving appropriate treatment, patients and their families can receive more accurate genetic counselling, CF carrier testing and prenatal diagnosis.

CF is one of the commonest autosomal recessive disorders among white South Africans, with a prevalence of 1 in 2 000; prevalence in the coloured population is 1 in 12 000.[1] CF was initially thought to be extremely rare in African blacks but a recent study showed a carrier frequency of 1 in 34 and a calculated incidence of 1 in 4 624 births.[2]

CF is characterised by pancreatic insufficiency, chronic pulmonary disease, elevated sweat chloride levels and a number of other features. It can be difficult to diagnose because of the great variability of clinical presentation and severity. The UK CF Foundation Consensus Panel suggests confirmation of diagnosis only after two positive sweat test results on separate occasions in a patient with suggestive clinical features.[3] However, as a diagnostic test the sweat test is not ideal. It requires extreme technical rigour by experienced staff using standardised methods. In large parts of South Africa such services are not readily available.

An alternative method of diagnosis became possible with the cloning of the CFTR gene.[4] CF is caused by mutations in the CFTR gene — patients have two mutations and carriers have one. Over 1 000 mutations have been identified.[5] Patients may have two identical mutations (homozygotes) or two different mutations (compound heterozygotes), but the identification of two CF mutations in a patient confirms the diagnosis of CF.

Given the number of CF-causing mutations and the impracticality of screening the large CFTR gene, testing for mutations that are common in a particular population makes genetic testing useful as a diagnostic tool. The aim of this study was to improve the sensitivity and efficiency of diagnostic genetic testing for CF in South Africa through the development of customised panels of mutations for different South African population groups. A total of 201 white, 43 coloured and 14 black CF patients with confirmed diagnoses were included in this project for CFTR mutation analysis.

White and coloured patients were tested for 24 mutations including $\triangle$F508, 394delTT, Q493X, 3272-26A G, 3120+1G A, R117H, 3659delC, G542X, G551D, R553X, 621+1G T, W1282X, N1303K, 1717-1G A, R1162X, R334W, 3849+10kbC T, A455E, 2183AA G, 1078delT, $\triangle$I507, R347P, S1251N, and E60X. Five coloured patients in whom small amounts of DNA were available from buccal scrapes were only tested for the $\triangle$F508 mutation. Black patients were initially tested for the 3120 +1G A mutation. Those black patients whose mutations were still unidentified were tested for the $\triangle$F508 mutation. Mutation detection assays have been described previously.[6]

Significant differences in the CFTR mutation distribution were found between groups, supporting the notion that population-specific panels of mutations are required when using genetic tests to diagnose CF (Table I). Sixteen mutations were detected in the South African white population, accounting for 91% of all CFTR mutations in this population. In the coloured population, the $\triangle$F508 and 3120+1G A mutations occur at appreciable frequencies and account for 74.4 % (64/86) of mutations. In the black population, 60.7% of mutations (17/28) were identified,