

mild depressive disorders, also be associated with continuation of smoking in some smokers in the community?

**Aims:** 1. Whether psychiatric morbidity 12 months ago is associated with increased smoking 2. Whether worsening psychiatric morbidity during a 12 month period is associated with increased smoking.

**Method:** the design was a prospective panel cohort study of a community sample aged 16–75 years. A secondary analysis of the British Household Panel Data which has five annual waves of data with 45 341 records on 12057 individuals. Repeated observations on number of cigarettes smoked and psychiatric morbidity, as measured by the 12-General Health Questionnaire (cut off score = 3), were used to measure changes in smoking and psychiatric status. The associations between increased smoking from the previous year (by 5 or more cigarettes/day) with a history of psychiatric morbidity 12 months ago and with worsening psychiatric morbidity were tested using classical, regression and clustering methods. Confounding by age, sex, socio-economic status, educational level, marital status and health problems were accounted for.

**Results:** Increased smoking was weakly associated with previous psychiatric morbidity (adjusted OR 1.12, 95% CI 1.00 to 1.25,  $p = 0.046$ ). Current psychiatric morbidity had a stronger association with increased smoking (adjusted OR 1.34, 95% CI 1.20 to 1.50,  $p < 0.0001$ ). Worsening psychiatric morbidity over one year was associated with increased smoking (adjusted OR 1.25, 95% CI 1.09 to 1.42,  $p = 0.001$ ).

**Conclusions:** Psychiatric morbidity, particularly current, makes a small contribution to increased smoking in the general population. Repeated observations on the same individual is a useful epidemiological approach to understanding the mechanisms between smoking and psychiatric morbidity.

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## S45. Bipolar disorders

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### S45-1

#### GENETIC ASPECTS OF BIPOLAR DISORDERS

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The recent advances in molecular genetic techniques, applied to linkage and association methodologies and the use of candidate genes and polymorphic markers have contributed to underline some promising DNA regions of susceptibility in the etiology of affective disorders. These include markers on chromosomes X, 18, 5, 4, 21, 11 and 17. Some of these DNA regions contain candidate genes implicated in central nervous system neurotransmission. The lack of replication between studies has often been attributed to the genetic heterogeneity which is now well recognized in behavioral disorders. Molecular genetic studies in affective disorders have recently extended beyond the field of classical DNA markers by studying dynamic mutations such as trinucleotide repeat expansion, which may play a role in phenomenon of anticipation clinically observed in some families of patients with affective disorders. These new molecular studies will be discussed in relation to gene-environment interactions and therapeutic implications.

### S45-2

#### ASSORTATIVE MATING IN BIPOLAR DISORDER

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**Objective:** It has long been observed that persons do not select their mates at random within the population but rather choose persons physically and psychologically similar to themselves. Studies on the occurrence of assortative mating with respect to psychiatric disorders and personality have reported contradictory findings, most likely as a result of differences in study methodology. In the context of an ongoing family study of treated bipolars, we compared the rates of lifetime psychiatric diagnoses in spouses of bipolar probands and normal controls.

**Method:** Both proband and spouse DSM-IV Axis I diagnoses (including childhood diagnoses) were derived from the semi-structured Diagnostic Interview for Genetic Studies or, in the case of spouses who refused to be directly interviewed, from a modified version of the Family History-Research Diagnostic Criteria.

**Results:** Spouses of male bipolar probands had significantly higher rates of affective and overall psychiatric diagnoses.

**Conclusion:** Mating type is an important element to consider both in treatment settings and family study research on bipolar disorder.

### S45-3

#### EPIDEMIOLOGY, VALIDITY AND COMORBIDITY OF MILDER BIPOLAR SUBGROUPS

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The scope of most epidemiological studies on bipolar illness has been limited to bipolar I disorder, with the largest studies having found a lifetime prevalence rate of 1.2 to 1.6%. However, less is known about the prevalence of bipolar II disorder (0.5 to 1.0% or more) and cyclothymia (1.5 to 2.8%). A Hungarian study and our Zurich Study found DSM-mania/hypomania in 5% of the population, while, in addition, the Zurich Study identified brief hypomania in a further 2.2%.

All subgroups of hypomania and mania can be regarded as constituting a spectrum subdivided artificially by operational criteria (severity, length, frequency). In line with the spectrum concept, the Zurich Study data showed that the milder subgroups had very similar symptom profiles and comparable validity. Hypomania and brief hypomania were shown to overlap to a very great extent with diagnoses of depression (especially atypical depression), a history of suicide attempts, with panic disorder, GAD, obsessive-compulsive syndromes and with substance (including tobacco and cannabis) abuse. Hypomania was also associated with binge eating, menstrual, gastrointestinal, respiratory and cardiovascular symptoms and, paradoxically, with neurasthenia, but not with backache or migraine. Hypomania showed a family load of mood disorders, elevated levels of self-induced stressful life events, high divorce rates and diminished quality of life. The personality features found were: cyclothymic (30 to 40%) or anxious personality traits and elevated neuroticism scores and, on the other hand, sociopathic features, with truancy in childhood/adolescence, elevated aggression scores and a risk of delinquency in adult life.