behaviors; their social life was relatively less impaired. Globally, both groups were equally depressed, but, for recent cases, depressive symptoms varied according to weight control strategies.

Fifty-eight per cent of the subjects with early onset BN could be reassessed two years after initial contact: 32% still had a DSM-IV diagnosis of BN, 28% had some, but not all, features of the disorder, and 40% were symptom-free. The specific clinical characteristics of the group were maintained.

In conclusion, risk factors for early onset BN are consistent with etiopathogenic factors for BN in general. Although the disorder can last for years, often untreated, BN does not appear more severe when it starts early during adolescence.

NEW DEVELOPMENTS IN THE STUDY OF AFFECTIVE DISORDERS IN YOUNG PEOPLE

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Major depressive disorders are relatively common in school-age children and adolescents. Epidemiological studies have delineated the six month prevalence rate of approximately 5%. New incidents occur with greatest frequency in middle adolescence with a slightly greater preponderance of females to males. In addition the clinical characteristics of major depression appear to vary with age. Studies on clinical populations suggest that as many as 45% of patients with major depressive disorder have alterations in selected adrenal steroid function. Evening cortisol hypersecretion and morning DHEA hyposecretion have both been described in this population. DHEA is a developmental steroid with circulating levels increasing markedly between the ages of 6 and 8 and again in mid adolescence. The implications of the developmental changes in steroid environment and their alterations during episodes of depression remain unclear. By contrast there is now considerable evidence that social adversities predict an increase in depressive symptoms in adolescence. There remains however no clear evidence that social adversities specifically provoke depressive episodes in this age range. Recent findings suggest that genetic factors contribute both to the risk for exposure to life events and difficulties and to the onset of depression, at least in adults. The role of genetic factors in the onset of depressive disorders in adolescence is less certain. Unlike adult studies however, child and adolescent psychopathologists have noted the high levels of comorbidity in depressive disorders in young people. Recent findings suggest that depressive conduct disorder may represent a specific and different sub-type from depression without conduct disorder. There is a need for interdisciplinary research to bring together these different strands of information on depression in young people. Study designs for the future should include family genetic designs so that the relative contributions of genetics, shared and non-shared environmental effects on some types of depressive disorders in this age range can be elucidated. The mechanisms and processes that lead to onset, relapse and recurrence represent the goals for future research. Short term longitudinal studies will enhance current longitudinal prospectives by a more systematic investigation of mechanisms and processes involved in the onset and cessation of episodes of disorder. A developmental approach should be maintained so that continuities and discontinuities between normal development and depressive disorders can be determined.

CONTROLLED TRIAL OF A BRIEF COGNITIVE-BEHAVIOURAL INTERVENTION IN ADOLESCENTS PARENTS WITH DEPRESSIVE DISORDERS

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Fifty-three child and adolescent psychiatric patients with depressive disorders were randomly allocated to brief cognitive-behaviour therapy (CBT) or to a control treatment, relaxation training. 48 patients completed the treatment phase of the trial, which comprised 5–8 treatment sessions. Post-treatment assessments showed a clear advantage of CBT over relaxation on measures of both depression and overall outcome. However, there were no significant differences between the treatments on comorbid anxiety and conduct symptoms. At follow-up, the differences between the groups were reduced, partly because of a high relapse rate in the DTP group and partly because subjects in the relaxation group continued to recover.

S6. A united Europe in psychiatry, too?

Chairmen: J Furedi, E Fombonne

Abstracts not received.

S7. Positive and negative symptoms in schizophrenia

Chairmen: Y Lecrubier, J Waddington

NEGATIVE AND DEPRESSIVE SYMPTOMS IN ACUTE SCHIZOPHRENIC EPISODES- DO THEY IMPROVE UNDER NEUROLEPTIC TREATMENT?

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There has been a continuing debate on the prevalence, association, specifity and development of negative and depressive symptoms in acute schizophrenia with productive symptoms and treatment outcome under antipsychotic drugs. In prospective investigations on the the concomittant occurrence and 5 years' course of negative and depressive symptomatology in schizophrenic and affective disorders we found that — apart from a substantial overlap of the symptomatologies — primary enduring negative symptoms are non-specific and were present in both diagnostic groups. Even in the longitudinal course of schizophrenia, this symptomatology was not more frequent than in affective disorders, and was observed in about 15% of both diagnostic groups.

In order to evaluate the efficacy of the mixed 5-HT_2-/D_2-like receptor antagonist risperidone vs. haloperidol and amitriptyline in a functionally defined combined psychotic and depressive syndrome, 123 patients suffering from either major depression with synthymic or mood-incongruent psychotic features, a depressive

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