and delineated distinct biological characteristics of this group of psychoses.

In a twin-study, a very similar concordance rate between monozygotic and dizygotic pairs of probands with cycloid psychoses was found, suggesting a lack of hereditary influence according to the rule of Galton. On the other hand, in mothers of patients with cycloid psychoses infectious diseases in the first trimester of gestation occured significantly more frequently than in patients with other psychoses. These infections were correlated with further obstetric complications and an early onset of the disorder. In agreement with these findings, patients with cycloid psychoses showed an increased rate of non-specific CCT-abnormalities of the brain which most likely resulted from pre- or perinatal brain damage. Neurophysiological investigations also provided specific findings. Studies of event-related potentials revealed characteristic features of amplitude and topography of the P300 in cycloid psychoses which were distinguishable from P300-alterations in schizophrenic psychoses and other psychiatric disorders. Moreover, cortical blood flow during acute phases proved to be significantly elevated in patients with cycloid psychoses, but showed no persistent abnormalities after clinical remission, especially no hypofrontality.

Altogether, these findings point to the fact that somatic influences may play an important role in the aetiology of cycloid psychoses and suggest that cycloid psychoses represent a nosologically independent entity which should be separated from affective and schizophrenic psychoses and is not identical with schizoaffective psychoses.

S70-4

ELECTROPHYSIOLOGICAL EVIDENCE FOR SUBGROUPING OF SCHIZOPHRENIA

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The current international diagnostic systems are developed based on a mixture of rationales including clinical utility and reliability of the diagnostic assessment. Monopolization of this method for the evolution of the psychiatric classification which follows practical clinical goals or even the simplicity of the diagnostic criteria instead of hypothetic ethiopathogenetical mechanisms is suspected to lead to a scientific impasse due to categories representing an amalgam of different natural disease entities. Leonhard's classification is based on clinical cross-sectional and longitudinal observations, ordered according to possible pathophysiological mechanisms, and allows to formulate testable hypothesis. In a series of studies based on 20-channel recordings of cognitive event-related potentials (ERPs), the neurophysiological differences between psychotic subgroups in Leonhard's classification and in the categories of the DSM and ICD were investigated. The P300 component of the ERPs differed between cycloid psychoses and systematic/unsystematic schizophrenia, and between these groups and manic disorders. While topographical alterations indicated deficits of left temporal lobe function in schizophrenia (Strik et al, Psychiat Res: Neuroimaging, 55: 153–166; 1993), increased P300 amplitudes were found in cycloid psychosis as a sign of a generalized increase of arousal (Strik et al, Acta Psychiat Scand, 94: 471–476; 1996). These differences were blurred with loss of statistical significance when the international diagnostic standard categories were applied. In manic patients, on the other hand, no amplitude differences compared to controls, and a topographical difference possibly indicating reduced frontal lobe control were found. The results revealed different neurophysiological mechanisms at the basis of the investigated subgroups and, thus, support the existence of different natural disease entities beyond the classical dichotomy of psychoses.

S70-5

MATERNAL GESTATIONAL INFECTIONS IN THE ETIOLOGY OF SCHIZOPHRENIC PSYCHOSES

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The contemporary neurodevelopmental hypothesis of schizophrenia and affective psychosis has emerged from pathobiological findings of early brain lesions and malformations of fetal brain development. In chronic schizophrenia we found that not the frequency, but the monthly distribution of maternal gestational infections was significantly different compared to controls. Twenty per cent of the mothers of schizophrenics recalled a manifest infection during pregnancy. The incidence of maternal gestational infections was significantly increased in the second trimester, especially during the fifth month of gestation. Respiratory infectious disesases (i.e. influenza and febrile cold) were frequent and accounted for 56% of all infections and of 64% of mid-pregnancy infections. Infections during mid-pregnancy were significantly associated with Leonhard's systematic schizophrenias with low familial aggregation of psychosis and a chronic non-remitting course with severe psychopathology. Furthermore, prenatal infections were significantly associated with the occurrence of further OCs, which are thought to constitute a significant risk factor for the development of schizophrenic psychosis. In our recent study, the cycloid psychoses with low heritability and good long-term prognosis were found to be significantly associated with first trimester respiratory infections (i.e. influenza, febrile cold). Acute respiratory infections explained 56% of all infections and all first trimester infections in cycloid psychosis. Furthermore, maternal infections seem to cause an early onset in cycloids. In manic-depression we failed to identify such associations to maternal gestational infections or other obstetric complications. These findings are suggestive that exogenously induced disturbances of fetal brain maturation during the first trimester of gestation are involved in the etiology of cycloid psychosis and those during the second trimester in systematic schizophrenias.

S71. Genetic epidemiology of mental illness

Chairs: P Munk-Jørgensen (DK), H Ewald (DK)

S71-1

ALZHEIMER'S DISEASE, GENETIC AND ENVIRONMENTAL FACTORS

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Several genetic and environmental factors have been implicated in Alzheimer's disease (AD). In recent years, considerable progress has been made in unraveling the genetic etiology. Three genes have been identified that are predominantly implicated in autosomal dominant forms of early-onset AD, the β-amyloid precursor protein gene and two homologous genes presenilin 1 and 2. Further, the apolipoprotein E gene (APOE) has been shown to be an important genetic risk factor for early- and late-onset AD. Although it is

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