of schizophrenia or to provide a biochemical basis. Most of the observations which support the neurodevelopmental hypothesis can be explained on the basis of genetically determined abnormalities in membrane phospholipid metabolism which can be attenuated or exacerbated by various environmental events. The phospholipids provide the main structural basis for all neuronal membranes and influence the behaviour of all membrane-bound and membrane-associated proteins such as receptors, ion channels and ATPases. The phospholipids also modulate the actions of all neuronal cell signalling systems, and the fatty acids they contain provide many of the second messengers which are activated as a result of receptor occupancy. The phospholipids provide a unique site where genes and environment interact: basic phospholipid structure depends on the enzymes involved in their synthesis and breakdown, whereas those enzymes depend on the environment for the supply of the fatty acids which are major components of the phospholipids. The phospholipid hypothesis proposes that in schizophrenia there are reduced rates of incorporation into phospholipids and increased rates of loss from phospholipids of the long chain polyunsaturated fatty acids (LCPS) which make up about 20% of the dry weight of the brain. Because phospholipids are so important in the brain, such defects could lead to abnormal brain morphology and synaptic remodelling, abnormal susceptibility to viral infections and perinatal hypoxia, insults which are known to interfere with phospholipid metabolism, and the onset of overt symptoms around or soon after puberty when phospholipid metabolism is known to change. This concept provides novel proposals for improved treatment of schizophrenia.

Wed-P40

P50 IN DEFICIT AND NON DEFICIT SCHIZOPHRENIA

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Sensory gating impairments have been found in schizophrenia, with prevalence estimates as high as 90%. Schizophrenic patients show a diminished suppression of the auditory - evoked P50 potential to the second (test) of two paired click stimuli as compared with control subjects. The P50 suppression measure was calculed as the ratio of the test amplitude to the conditioning amplitude (C/T ratio). In order to investigate the relationship between P50 suppression and negative symptoms, we have compared P50 measures between 19 deficit and 32 non deficit schizophrenic patients. The 51 patients were all neuroleptic-treated at the time of the testing and the Schedule for the Deficit syndrome (Kirkpatrick et al., 1989; Ribeyre et al. 1994) was used to classify patients as either deficit or non deficit subtypes.

P50 measures were obtained at vertex during a conditioning-testing paradigm.

P50 amplitudes, latencies and C/T ratio were similar for both deficit and non deficit subgroups. These results suggest that sensory gating impairment is not related to the clinical subtype of schizophrenia.

Wed-P41

ACTIVE ^{3}H-DOPAMINE UPTAKE OCCURS IN PLATELETS AND IN ARTIFICIAL NEURONAL CELL LINES OF HUMAN ORIGIN BUT NOT IN LYMPHOCYTES

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As lymphocytes have been proposed as a peripheral model of dopamine reuptake in brain neurones, which might play a role in several psychiatric diseases, kinetic and pharmacological properties of ^{3}H-dopamine uptake by native human lymphocytes were investigated.

Our results suggest that the transport of ^{3}H-dopamine measured with lymphocytes after separation over Ficoll-Paque™ or Percoll™ is mainly generated by platelets which are always part of freshly prepared lymphocyte suspensions.

The investigations were extended to well defined cell lines in order to compare the pharmacological properties of native and artificial cells without any influence of contaminating cells such as platelets in lymphocyte suspensions. The artificial cell lines MOLT-3 and EBV-transformed lymphoblasts were not able to transport ^{3}H-dopamine which is consistent with our hypothesis that native lymphocytes do not exhibit a dopamine uptake. The investigation of the human neuroblastoma cell line IMR-32 demonstrated a GBR-12909 and cocaine-sensitive specific transport of dopamine, whereas dopamine transport in platelets is performed by a imipramine-sensitive serotonin transporter.

Our results do not support the existence of a dopamine transporter in human lymphocytes and demonstrate the possibility of verifying experiments conducted with crude native cells by the use of artificial, but homogeneous cell lines.

Wed-P42

INFLUENCE OF SEVERITY AND DURATION OF PSYCHOTIC STATE ON THE LEVEL OF MIDDLE MOLECULES

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It is known, that various toxic conditions are accompanied by increased level of middle molecules (molecular wheight 500–5000 Dalton) in blood plasma. It is considered, that the increase of middle molecules (MM) concentration in plasma in schizophrenia proves endotoxicosis hypothesis.

It was studied the level of MM in plasma in schizophrenia. There was examined 14 patients: 10 patients with paranoid schizophrenia (F 20.0, ICD-10) and 4 patients with postschizophrenic depression (F 20.4). Patients with organic or somatic pathology were excluded.

In schizophrenic patients with acute episode, characterized by severe paranoid and hallucinosis symptomatology (6–7 CGI score, less than month duration) the MM plasma concentration has exceeded 3–4 time the normal level. In psychotic patients with less severe states (4–5 CGI score) the duration of which was more than month, the MM concentration was less increased and exceeded the normal level by 1.5–2 times.

In all patients with postschizophrenic depression (3–4 CGI score, state duration more than month) the MM level did not exceed norm.

Control MM level was 0.45 ± 0.06 g/l.

It was found the tendency to negative relationship between MM plasma concentration and duration of psychotic symptomatology and positive relationship between MM plasma concentration and severity of psychotic symptomatology.

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