jects with DSMIII-R schizophrenia and normal premorbid I.Q. (randomly matched from the Lothian Psychiatric Case Register, N = 20), and subjects with mild learning disability alone (N = 16). The Quick I.Q. Test and The Positive and Negative Symptom Scale (PANSS) were administered to all 57 participants. A National Adult Reading Test (NART) was also performed on all schizophrenic control subjects to confirm a premorbid I.Q. within the normal range.

A one way ANOVA with Bonferroni Test for multiple comparisons was performed on symptom clusters (Positive, Negative and General), obtained from the PANSS. Dual diagnosis subjects showed significantly more negative symptoms (at p = 0.05) than either the schizophrenic group or the group of subjects with learning disability alone. Whereas the schizophrenic patients, without premorbid learning disability, showed significantly more positive symptoms than either the dual diagnosis or learning disability groups. Furthermore, regression analysis indicated a significant negative correlation between Quick I.Q. and negative symptomatology in all schizophrenic subjects.

This study confirms that preschizophrenic subjects with a low I.Q. develop a form of psychosis characterised by predominantly negative symptomatology.

THE LONG-TERM COURSE OF CHILDHOOD-ONSET SCHIZOPHRENIA. A SECOND FOLLOWUP OF 44 PATIENTS 27 YEARS AFTER THE FIRST FOLLOWUP AND 42 YEARS AFTER THE INITIAL PSYCHOTIC EPISODE: A FIRST REPORT

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First results of a long-term followup (M = 41.9 years, SD = 8.2 years) of 44 patients (19 males, 25 females) with Childhood-onset Schizophrenia are presented. Age at onset ranged from 7 to 14 years (M = 11.8 y., SD = 2.0 y.). Patients and/or their first-degree relatives were interviewed personally in 1994 with the Present-State-Examination (PSE) and the Disability-Assessment-Schedule (WHO-DAS) — about 27 years after the first followup. Clinical records were analyzed with the Instrument for the retrospective assessment of onset of Schizophrenia (WHO-IRAOS) and with sections of the PSE. The cases were re-diagnosed with DSM-III-R based on longitudinal data obtained between onset and first the first hospital admission. *Main results:* The cumulative prevalence of illness-onset with age is flatter in boys than in girls. An acute (vs. insidious) onset was significantly more frequent after 12 years of age. There was a negative correlation between age of onset and the social disability scores (WHO-DAS). 25% showed complete, 25% partial, and 50% very bad recovery at followup. None of the chronically psychotic patients showed an acute onset. The results are discussed with respect to epidemiology, gender differences, and etiological hypotheses of Childhood Schizophrenia.

DERMATOGLYPHICS OF SCHIZOPHRENIA

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Objectives: Dermatoglyphics elicit the genetic aetiology of many diseases. The genetically determined dermatoglyphic features include the total finger ridge count (TFRC), the A-B ridge and the ATD angle.

The pattern of the palmar flexion creases and the white lines were studied in addition to the genetic traits.

Methods: 80 schizophrenics (patients group) were compared to 100 psychiatrically free subjects (control group) using the inked method, results were compared with our findings in indiopathic epilepsy.

Summary of results: 1- Changes in the dermatoglyphic genetic traits were similar to those changes found in patients with idiopathic epilepsy.

2- Schizophrenics showed characteristic dermatoglyphic features of finger tips and palms which represent quantitative varying polygenic traits.

3- The pattern of the palmarflexion creases and the white lines showed different varieties that also indicate the polygenic nature of the disease.

Conclusions: 1- Schizophrenia is genetically determined and has a common aetiological relationship with idiopathic epilepsy.

2- The mode of genetic transmission in schizophrenia is polygenic.

SUSTAINED 5HT$_{2A}$ RECEPTOR OCCUPANCY OF ZIPRASIDONE USING PET LIGAND ^{18}F SETOPERONE IN HEALTHY VOLUNTEERS

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Ziprasidone is a novel antipsychotic in late clinical development. The time course of its D$_2$ receptor occupancy has been previously demonstrated in healthy volunteers [1] and ziprasidone is associated with a low incidence of extrapyramidal side-effects (EPS). This study aimed to determine 5HT$_{2A}$ receptor occupancy, and whether high occupancy may account for the low incidence of EPS. Eight healthy volunteers were each scanned on two separate occasions approximately 1 week apart. Nanomolar doses of ^{18}F-setoperone (7 mCi) were used as the 5HT$_{2A}$ receptor ligand [2]. The first scan provided baseline binding for each individual. At pre-determined time points prior to the second scan, after at least 4 hours fasting, they received 40 mg ziprasidone orally, so that two volunteers were scanned at each time point post dose. Three-compartment modelling of setoperone pharmacokinetics was performed. The mean 5HT$_{2A}$ receptor occupancy by ziprasidone is shown below:

	Ziprasidone receptor occupancy at various time points (hr)			
	4	8	12	18
5HT2A (%)	95.4	92.0	78.4	46.7
D2 (%)	79.4	68.2	52.8	32.2

Means of two individuals are shown, but differences between subjects were very small. Data on D$_2$ occupancy [1] obtained following the same dose of ziprasidone in a separate study are listed for comparison. Plasma levels of ziprasidone are being determined to confirm that exposure was similar in the two studies. *Conclusions:* 5HT$_{2A}$ receptor occupancy in this study substantially exceeds the known D$_2$ occupancy at all time points. This may explain the low incidence of EPS with ziprasidone.

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THE ANATOMY OF THE FUNCTIONAL PSYCHOSES

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The idea that the 'functional' psychoses have their origins in brain as opposed to psychological dysfunction has a long history. Advances in imaging procedures have facilitated the study of brain architecture in psychiatric syndromes, and a number of correlates have emerged.

Using the Lothian Psychiatric Case Register, all patients discharged from in-patient care at the Royal Edinburgh Hospital during the years 1993 and 1994 with ICD-9 codes corresponding to the 'functional' psychoses (295, 296, 297 and 298) and aged between

18 and 55 were selected. From their number, around 1000, 250 patients were selected by a method which allowed an adequate spread of diagnostic categories. The OPCRIT programme (McGuffin et al, 1991) was applied to the case notes, each patient having a minimum of 2 admissions, and admissions being assessed to a maximum of 5. Diagnoses were generated according to a variety of operational criteria, and 5 patient groups emerged — namely schizophrenic, manic depressive, depressive, atypical, and inconsistent. The schizophrenic group was comprised of patients who on at least two admissions were given a schizophrenic diagnosis by at least four out of five sets of OPCRIT criteria. The manic depressive group was comprised of patients who on at least two admissions were given a manic diagnosis by at least three out of four sets of OPCRIT criteria, or who on at least one admission were given a manic diagnosis and on at least one other admission were given a depressive diagnosis by at least three out of four sets of OPCRIT criteria. The depressive group was comprised of patients who received a depressive diagnosis on at least two admissions by at least three out of four sets of OPCRIT criteria, and the atypical group was comprised of patients who were not given a diagnosis of schizophrenia, mania or depression on as many as four out of five OPCRIT criteria, on more than one admission. The inconsistent group was comprised of patients whose diagnoses showed no consistency and who fitted none of the other categories.

A number of these patients will be scanned using a 1-Tesla MRI machine, and correlates of the diagnostic categories and of the symptom spread will be determined.

BRAIN MAPPING IN THE STUDY OF DEPRESSION IN ALCOHOLISM

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Introduction: The possible links between dependence of alcohol and depression patterns have been dealt with in many specialized publications. However, a final and direct relationship between them has not been proved. How is know, brain mapping is a study computerized of the electric cerebral activity and its topographical distribution. Our objective has been to try to determine a different neurophysiology pattern in patients with alcoholism, in function or not of the presence of depressive symptomatology.

Material and method: Subjects: Three groups of study were made in a sample of 50 alcoholic patients who followed the ICD-10 criteria for alcoholism and they were hospitalized for dexintoxication program. These groups were alcoholics without depressive symptomatology (ND), alcoholics with depressive symptomatology i(CD) and alcoholics with false depression (FD). The Hamilton Scale for depression and the Beck inventory for the depression were used 3 and 10 days after the admission. Likewise the Hamilton Anxiety Scale was used.

Methodology: Brain mapping was performed to every patient eight days after admission. Maps of the means for each group were obtained and also an analysis of variance with T punctuations in order to compare the different groups and establish the statistical significance.

Results: When comparing neurophysiological behavior of each group, in both studied situations (closed and open eyes), an appropriate alpha activity blockade was verified at the ocular opening in the three groups and a significant reduction of the quick activity upon closing the eyes, except for the FD group that showed minimum changes in the values of relative power in this band. On the other hand, we obtained significant differences on power value in CD group in relation to the other groups. Likewise differences in interhemispheric synchronization were verified between them.

Discussion: The data obtained in our study point out significant differences between the three groups, suggesting that the presence or not of depressive symptomatology in alcoholic patients is correlated with a different neurophysiological behavior. Brain mapping is an useful technique for their study. Presence of anxiety provided that symptomatology could associate to a different mapping pattern which characterized FD group. Nonetheless our data have to be considered with the precaution due to such a preliminary study with little samples. Moreover it has to be taken into account that the study has been made in patients under psychopharmacological treatment.

Conclusions: Our data point out that the brain mapping pattern in alcoholic patients is significantly different in the three studied groups. At the same time, this study suggests that symptomatology of false depression could correspond to a state of anxiety. The repercussions of these discoveries in the prognosis and treatment of this problem could be very important.

EVIDENCE FOR MEDIAL TEMPORAL LOBE CHANGES IN SCHIZOPHRENIA: A QUANTITATIVE MRI STUDY

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Background: Temporal limbic changes were described in numerous post mortem and neuroimaging studies. However, the questions if these changes refer to a generalised process affecting all parts of the brain at a line or do reflect local changes remained unsolved.

Aim: To investigate the relation of temporal limbic changes with respect to the subcortical and frontal changes.

Method: Up to now, 10 healthy controls and 10 DSM IV schizophrenics participated in our ongoing trial. We used a 3D MPRAGE sequence for the T-1-weighted images and a 3D PSIF sequence for the T-2-weighted images on a 1.5T Siemens Magnetom. This sequences provide 128 contiguous slices through the head with a thickness/gap of 1.4 mm/0 mm, TR/TE: 10.0 ms/4 ms, xf0v: 230 mm/230 mm in about 9 min. Total intracranial volume (TIV), volumes of the frontal and temporal lobe, the amygdalahippocampus complex (ABC), the hippocampus, the parahippocampal gyrus, the caudatum and the putamen-pallidum were assessed using the newly developed software NMRWin. Measurements were performed by two independent raters (interrater-reliability: $r = 0.95$–0.96, $p < 0.0001$) on a conventional PC. The volumetric data were corrected for head size by dividing the absolute values by the TIV.

Results: All, but the right AHC and the parahippocampal gyrus, temporal limbic structures differed significantly ($p < 0.05$) between patients. Within the schizophrenics the right AHC was negatively correlated with the right hippocampus ($p < 0.01$) and the left AHC ($p < 0.001$). None of the temporal limbic structures were significantly correlated with the volumes of the frontal lobe or any of the subcortical measures.

Conclusion: Our preliminary findings confirm earlier reports demonstrating temporal limbic changes in schizophrenia and suggest that these changes do not correspond to generalised brain changes.

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