as in the normal elderly population. Antipsychiotics and most antidepressant medications impair sexual function even further, leading to non-compliance and relapse. The prevalence of sexual dysfunction in schizophrenics treated with typical and atypical antipsychotics is 30–60%. Attempts to treat sexual dysfunction using dopaminergic drugs such as L-dopa, apomorphine, amantadine and L-deprenyl were disappointing. Sildenafil citrate (Viagra) seems to be an effective treatment in less deteriorated male patients who are capable of maintaining a reasonable relationship with their partners. A variety of strategies have been used in the management of SSRI-induced sexual dysfunction: waiting for spontaneous resolution, dosage reduction, drug holidays, adjunctive pharmacotherapy or switching antidepressants. Adjunctive agents are SHT_2 antagonists (cyproheptadine, mianserin, mirtazapine), dopamine receptor agonists (psychostimulants, bupropion) and Viagra. Substitute antidepressants are bupropion, nefazodone, mirtazapine and reboxetine. In an open-label study with 10 male SSRI-treated PTSD patients who complained of sexual dysfunction, the use of sildenafil (50 mg) significantly improved their erectile function and intercourse satisfaction. Sildenafil (25–50 mg) was efficacious also for antidepressant-induced erectile dysfunction in elderly male depressed patients (n=11, age 70–81, mean age 73 yrs). In 8 out of 11 patients, erectile function returned to a normal level. Side effects were noted in two patients (headache). It appears that sildenafil co-administration is efficacious, safe, and well-tolerated in special populations.

S09. Is schizophrenia really just a neurodevelopmental disorder?

Chairs: E. Johnstone (GB), S.M. Lawrie (GB)

S09.1

Recent evidence on the neurodevelopmental model of schizophrenia

J. Parnas. *Denmark*

No abstract was available at the time of printing.

S09.2

Clinical and cognitive markers of the development of schizophrenia

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Neuropsychological impairments have been reported in patients with schizophrenia, in the adult relatives of such patients, and in children at high genetic risk for the disorder. In the Edinburgh High Risk for Schizophrenia Study we examined the relationship between neuro-psychological impairments and risk for schizophrenia, and the development of psychotic symptoms in subjects at enhanced genetic risk for schizophrenia. The results from a battery of neuro-psychological assessments were compared among 157 high-risk subjects, and 34 normal controls. Findings were related to a quantitative measure of genetic risk, calculated for the high-risk group according to the number of ill and well relatives in the family and their relationship to the subject, and to development of psychotic symptoms. Neuropsychological differences were identified in many areas of function and were not accounted for by the presence of psychotic symptoms in some subjects. The quantitative measure of genetic liability was not associated with either neuropsychological function or with the development of psychotic symptoms. These results suggest that what is inherited is not the disorder itself, but a state of vulnerability manifested by neuropsychological impairment occurring in many more individuals than are predicted to develop the disorder.

S09.3

Structural and functional MRI in the Edinburgh High Risk Study

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MRI studies of the brain in schizophrenia have demonstrated structural abnormalities, particularly of the temporal lobes, and disruptions of fronto-temporal functional connectivity. We conducted sMRI scans in 150 high risk subjects aged 16–25 at baseline and 66 of them after approximately 2 years, and have now conducted sMRI and fMRI scans in almost 100 after a further 2–3 years. Healthy age-matched controls have also been scanned.

We have found associations between pre-frontal and basal ganglia volumes with genetic liability, and reductions in medial temporal lobe and thalamus volumes in the high risk group compared to controls, at baseline. Those with psychotic symptoms had relatively large brains at baseline as well as reductions in temporal lobe volumes over two years. More detailed analyses of temporal lobe abnormalities and fronto-temporal dysconnectivity are in progress.

Overall, the results suggest that some abnormalities of the brain in high risk subjects are genetically mediated and developmental, that others may only become apparent in late adolescence for unclear reasons, and that psychotic symptoms are associated with further structural changes.

S09.4

An MRI study of subjects in the prodromal phase of psychosis

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Introduction: Recent prospective neuroimaging studies have suggested that there are progressive volumetric changes in grey matter over the course of psychotic disorders. We sought to investigate this issue using magnetic resonance imaging (MRI) to examine brain structure in subjects before and after the first episode of psychosis.

Methods: a) *Cross-sectional comparison*: Subjects identified as being at ultra high-risk (UHR) of developing psychosis were scanned using MRI; at 12 month follow-up 31% had developed a psychosis and 69% had not. The MRI data from these 2 subgroups at baseline were compared by ANCOVA, controlling for age. b) *Longitudinal comparison*: Subjects were scanned at baseline and again, either after the onset of psychosis, or at least 12 months

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