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COMMENTARY

It has always been assumed that parents of children who migrate away leaving their elderly parents behind are more at risk of becoming depressed. In Pakistani culture this applies as much to migration abroad as rural-urban migration. The first study by Abbas et al shows a very interesting, almost counter-intuitive result that outmigration of children was actually associated with less parental depression, possibly due to economic benefits of migration.

It is universally accepted now that schizophrenia is a neurodevelopmental disorder rather than a 'functional' disorder as was thought previously. However, we are still far from finding a definitive etiological factor for schizophrenia or schizophrenia for that matter. The second study by Ghose et al adds some further clues in the search for genetic factors implicated in the development and treatment of schizophrenia.

There has been an exponential increase worldwide in the last one to two decades in the number of children who have been diagnosed autistic spectrum disorders. There has been a lot of discussion whether this represents a true increase in prevalence of these disorders or are more children with autism being recognized now as a result of increased awareness among parents and doctors. If this is a true increase in prevalence no one seems to know what is causing it. The systemic review above by Gardener et al reports that while there is insufficient evidence to implicate any one prenatal factor, there is some evidence to suggest that exposure to pregnancy complications may be associated with an increased risk.

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RURAL-URBAN MIGRATION AND DEPRESSION IN AGEING FAMILY MEMBERS LEFT BEHIND

BACKGROUND: It has been suggested that rural-urban migration will have adverse consequences for older parents left behind. AIMS: To describe correlates of outmigration and to estimate any association between outmigration of children and depression in rural-dwelling older parents. METHOD: Population-based survey of 1147 parents aged 60 and over in rural Thailand. We randomly oversampled parents living without children. We defined an outmigrant child as living outside their parent's district, and measured depression as a continuous outcome with a Thai version of the EURO-D. RESULTS: Outmigration of all children, compared with outmigration of some or no children, was independently associated with less depression in parents. This association remained after taking account of social support, parent characteristics, health and wealth. Parents with all children outmigrated received more economic remittances and they perceived support to be as good as that of those with children close by. CONCLUSIONS: Outmigration of children was not associated with greater depression in older parents and, after taking account of a range of possible covariables, was actually associated with less parental depression. This could be explained by pre-existing advantages in families sending more migrants and by the economic benefits of migration.

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DIFFERENTIAL EXPRESSION OF METABOTROPIC GLUTAMATE RECEPTORS 2 AND 3 IN SCHIZOPHRENIA: A MECHANISM FOR ANTIPSYCHOTIC DRUG ACTION?

OBJECTIVE: Preclinical and clinical data implicate the group II metabotropic glutamate receptors mGluR2 and mGluR3 in the pathophysiology of schizophrenia. Moreover, a recent phase II clinical trial demonstrated the antipsychotic efficacy of a mGluR2/mGluR3 agonist. The purpose of the present study was to distinguish the expression of mGluR2 and mGluR3 receptor proteins in schizophrenia and to quantify glutamate carboxypeptidase II (GCP II) in order to explore a role for the metabotropic receptors in schizophrenia therapeutics. GCP II is an enzyme that metabolizes N-acetyl-aspartyl-glutamate (NAAG), which is the only known specific endogenous agonist of mGluR3 in the mammalian brain. METHOD: The normal expression levels of mGluR2, mGluR3, and GCP II were determined for 10 regions of the postmortem human brain using specific antibodies. Differences in expression levels of each protein were examined in the dorsolateral prefrontal cortex, temporal cortex, and motor cortex in 15 postmortem schizophrenia subjects and 15 postmortem matched normal comparison subjects. Chronic antipsychotic treatment in rodents was conducted to examine the potential effect of antipsychotic drugs on expression of the three proteins. RESULTS: Findings revealed a significant increase in GCP II protein and a reduction in mGluR3 protein in the dorsolateral prefrontal cortex in schizophrenia subjects, with mGluR2 protein levels unchanged. Chronic antipsychotic treatment in rodents did not influence GCP II or mGluR3 levels. CONCLUSIONS: Increased GCP II expression and low mGluR3 expression in the dorsolateral prefrontal cortex suggest that NAAG-mediated signaling is impaired in this brain region in schizophrenia. Further, these data implicate the mGluR3 receptor in the antipsychotic action of mGluR2/mGluR3 agonists.

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PRENATAL RISK FACTORS FOR AUTISM: COMPREHENSIVE META-ANALYSIS

BACKGROUND: The aetiology of autism is unknown, although prenatal exposures have been the focus of epidemiological research for over 40 years. AIMS: To provide the first quantitative review and meta-analysis of the association between maternal pregnancy complications and pregnancy-related factors and risk of autism. METHOD: PubMed, Embase and PsycINFO databases were searched for epidemiological studies that examined the association between pregnancy-related factors and autism. Forty studies were eligible for inclusion in the meta-analysis. Summary effect estimates were calculated for factors examined in multiple studies. RESULTS: Over 50 prenatal factors have been examined. The factors associated with autism risk in the meta-analysis were advanced parental age at birth, maternal prenatal medication use, bleeding, gestational diabetes, being first born v. third or later, and having a mother born abroad. The factors with the strongest evidence against a role in autism risk included previous fetal loss and maternal hypertension, proteinuria, pre-eclampsia and swelling. CONCLUSIONS: There is insufficient evidence to implicate any one prenatal factor in autism aetiology, although there is some evidence to suggest that exposure to pregnancy complications may increase the risk.