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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all **references in Journal style.**

STRESS AND NON-GENETIC SCHIZOPHRENIA

DEAR SIR,

Reveley *et al* (*Journal*, January 1984, **144**, 89–93) show in carefully conducted twin studies that schizophrenics without a family history of major psychiatric illness, other than substance abuse, and therefore presumably of low genetic loading for schizophrenia, when compared with schizophrenics with such a family history, have on the average larger cerebral ventricles and a later age of onset followed by a shorter time in hospital. They find that there is an association between enlarged ventricles and birth complications but that, even when there have been no birth complications, the ventricles are on the average large in the patients with low genetic loading.

The authors postulate that ventricular enlargement may be just one non-specific indicator of past or present pathology necessary to produce a psychotic illness in those with low genetic predisposition.

There is evidence (Parnas *et al*, 1982) that perinatal complications predispose to schizophrenia. Speculation has grown about the role of a virus in its causation (Hare, 1983). Life events cluster in the three weeks before its onset (Birley and Brown, 1970) and sometimes it is thought to be psychogenic (Faergeman, 1963).

Consequently the recognition that stress can bring forward its onset in a person who would necessarily have developed it later on (Brown *et al*, 1973) may need modification in view of the possibility that organic, not necessarily cerebral, or psychological or social trauma can induce it in people with low genetic loading for the illness who would not have developed it without these stresses.

Perhaps we should revive the ideas, reviewed by Lidz (1973), of the schizophrenogenic family. The usual argument against them is that the stress imposed by such a family is more likely to result in neurosis than psychosis but now this reasoning appears to be faulty because just as ventircular enlargement is associated with a variety of clinical manifestations other than schizophrenia (Rieder *et al*, 1983), so family stress could have non-specific effects, among them being schizophrenia.

When the writer of this letter was studying 512 Royal Naval schizophrenics (Wallis, 1965) he often doubted whether many of these young people would ever have become schizophrenic had they not experienced physical and psychological stresses and great unhappiness in the intensely communal and disciplined naval environment. To have said so at the time would have been heretical. Even then, however, if schizophrenia had been preceded by exceptionally severe stress, consisting, for example of service in three or more ships when they were sunk by enemy action, especially if these sinkings were followed by long periods adrift, or of being maimed in Japanese prisoner of war camps, the Ministry of Pensions, as it then was, judged that the schizophrenia was not "constitutional" but aggravated by or even attributable to naval service. The Ministry granted disability awards on these grounds to 45 of the 512 patients.

Moreover when the naval schizophrenics were followed up for five to fifteen years, those who had experienced the most stress fared best. This finding, replicated by many other researchers, accords with the thesis that patients with low genetic loading need the most stress to induce their schizophrenia and have a better outcome.

"Only connect! . . . Live in fragments no longer" (Forster, 1910).

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MENTAL ILLNESS, EPILEPSY AND HYPOTHYROIDISM IN XXYY SYNDROME

DEAR SIR,

I am reporting a case of XXYY syndrome, associated with psychotic episodes, epilepsy and hypothyroidism. The patient is a nineteen year old male with the clinical features of XXYY syndrome, confirmed by cytological investigations.

His height is 194 cms. His intellectual assessment showed him to be in the low average range of intelligence (IQ varied from 72 to 88). There is no familial or prenatal history available, as the patient was adopted at five years of age. There is a history of epilepsy following a febrile illness at six months and a further convulsion at the age of five years following a smallpox vaccination. An EEG confirmed that he suffered from temporal lobe epilepsy with both grand mal and partial attacks.

The patient first came to the attention of psychiatric services when he was ten years old. The reasons for referral were slow learning, marked fantasy flights, behaviour difficulties and a very high degree of anxiety and tension within the family. At the age of fourteen years his problems increased: his behaviour became worse, he developed transvestite fantasies and started stealing ladies underwear. After being admitted to a special school, he started to steal money from other residents, became argumentative, at times depressed, and attempted suicide by trying to cut his wrists.

Since his admission to a mental hospital in 1983 his mental state is gradually improving and he is now placed in a small hostel and attending the hospital occupational therapy department. His epilepsy is fairly well controlled. Psychotic disorder and EEG abnormalities in the XXYY Syndrome have been described by Jancar (1968).

In addition to abnormal mental functioning and epilepsy, it was noted that the patient tended to be sleepy. His pulse rate was 56 per minute, total thyroxine was 64 mol/l and his free thyroxine index was 61 units. Treatment for hypothyroidism started with L-thyroxine 0.1 mg daily to which the patient responded well.

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PSYCHOSIS AND ANTIDIURETIC HORMONE

DEAR SIR,

We were interested in the letter from Emsley and Gledhill (*Journal*, March 1984, **144**, 331–2) and would like to make the following observations.

The possibility of a *common* neurological/hypothalamic disturbance was considered in our paper but discounted in the absence of abnormal drinking behaviour and fluctuation in body weight, with an appropriate ACTH response to hypocortisolaemia. However our patient's prolactin level was normal (370 mU/L n.r. <450 m U/L). This is of interest since in acutely adrenalectomised rats serum prolactin levels are elevated (Ben-David *et al*, 1971) suggesting lack of dopaminergic inhibition. Pituitary dopamine and dopamine metabolite concentrations have, however, been found to be normal in adrenalectomised rats (Leung *et al*, 1980). The mechanism of hyperprolactinaemia is unexplained, but our patient's prolactin level possibly implies an isolated disturbance of hypothalamic function.

While we agree that both present and past phenothiazine therapy may be misleading in interpreting antidiuretic hormone (ADH) levels it seems unnecessary to postulate a drug effect in the presence of such profound hypoadrenalism, with salt and water loss. Moreover Raskind *et al* (1978) whom they cite as evidence for serum ADH elevation in psychosis only discontinued drugs for 4 weeks before the ADH levels were taken.

We would suggest that further studies to investigate the relationship between ADH *per se* and psychosis will need to demonstrate that (a) variations in ADH levels correspond to the course of the psychosis, (b) CSF ADH levels as well as serum ADH levels are raised during psychosis, (c) these disturbances are