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A set of male monozygotic triplets with schizophrenic psychoses: nature or nurture?

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Abstract A set of schizophrenic male monozygotic triplets is described. At age 20 years, within 8 months the three men independently developed acute fulminant schizophrenic disorders (DSM-III-R) with auditory hallucinations, bizarre delusions, and thought disturbances. There were also great similarities between the triplets with regard to the chronic intermittent course of the disorder, impairment of social adjustment, and loss of working ability. The psychoses responded rapidly to conventional neuroleptic treatment. Neuropsychological assessment demonstrated similar marked reductions of attentional, mnestic, and executive functions. Magnetic resonance imaging (MRI) showed similar borderline ventricular enlargement and widened subarachnoid spaces over frontoparietal and basal regions as well as around the pituitary gland (empty sella). All the boys also had a right-sided hearing defect with a marked reduction of the ossicular bones on the right side. Possible clues as to etiological mechanisms were the lack of reported family history for the disorder and a possible influenza infection in the mother during the first trimester. It is suggested that a DNA aberration being present or occurring at conception initiated a precise timeprogrammed series of events that produced the very similar schizophrenic phenotypes. Such an aberration might have been induced by an external agent, occurred spontaneously, or been inherited by a recessive mechanism. It seems possible that the psychoses, the reductions of neuropsychological functions, the morphological MRI changes, and the right-sided ossicular reductions may all be related to such a DNA alteration.

Key words Schizophrenic \cdot Monozygotic triplet \cdot DNA \cdot MRI \cdot Malleolus

Introduction

It is well known that there is a higher risk for schizophrenia in the relatives of schizophrenic patients than in the general population (Kendler 1988). Twin and adoption studies indicate that genetic factors play a major role in the familial transmission of the disorder, but also that environmental factors are involved (Kety et al. 1975; Onstad et al. 1991). However, the relative importance of genetic vs environmental factors and the likelihood of heterogeneity in the pathogenesis of schizophrenia remains unclear.

Similarities and differences in phenocopies of genetically identical siblings may give important information as to possible etiological and pathophysiological mechanisms for schizophrenia. Only a few reports of more than two genetically identical siblings among schizophrenic patients have been published previously. The Genain quadruplets were investigated in the late 1950s and again in 1981 (Mirsky and Quinn 1988). The quadruplets obtained different diagnoses, but all were within the schizophrenia spectrum of disorders. A genetically identical set of triplets with discordant major Kraepelinian types of psychoses was described by McGuffin et al. (1982). Two of these siblings received the diagnosis of schizophrenia, whereas the third was considered to be manic-depressive. We are unaware of other reports of schizophrenic illness in triplets. We have examined a set of male psychotic triplets, born in 1958, since 1979. Here we report their case histories including prenatal and family-history data. Results of DNA and cytogenetic screening, psychometric tests, and magnetic resonance imaging (MRI) are presented in order to explore possible genetic and environmental mechanisms involved in the pathogenesis of their psychotic disorders.

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Table 1 Weight, height, head circumference, and width of the ventricular system (Evans Index) for a set of monozygotic schizophrenic male triplets (S, T, and M)

	S	T	M
At birth		•	
Weight (g)	1520	1220	1380
Height (cm)	39.5	38	39
Head circumference (cm)	29.5	27	27.5
Time of birth	6.23 a.m.	6.30 a.m.	6.38 a.m.
At age 34 years			
Weight (kg)	107	95.6	99
Height (cm)	166	163	168
Head circumference (cm)	60	59	59
Evans Index	0.28	0.30	0.30

Case report

The triplets were born in 1958 (Table 1). They were brought up by their biological mother. She separated from the father before giving birth to the boys. None of the boys have ever had contact with the father. In 1979, at age 20 years, each one of the three brothers (called S, T, and M) were independently admitted to a psychiatric clinic, because of the acute onset of psychotic signs and symptoms.

Prenatal history

According to the obstetric record the mother of the triplets had her last menstruation before the pregnancy in February 1958, and during the first or second month of the pregnancy she had symptoms of "Asian" influenza and cystitis. Upon direct questioning today, however, the mother strongly denies having had any influenza-like condition during the pregnancy, but admits to having had the cystitis. The children were born in September 1958 in an obstetric clinic in Stockholm after a pregnancy of 7 months. The delivery started spontaneously.

Premorbid development

The combined weight of the triplets at birth was 4120 g (Table 1). After the delivery the children were placed in incubators for 1 h because M was pale. According to the mother all the boys developed satisfactorily during early childhood. However, there was a constant pattern between them during childhood development, i.e. T being the brightest followed by M. During the first 2 years of school (age 7–9 years) the triplets were placed in a special class for pupils with difficulties in reading. After that their results in school improved and they were placed in an ordinary class. In 1970, at age 12 years, right-sided hearing difficulties were found in all the triplets during a routine check by the school nurse. The mother had not noticed hearing

problems before that age. The follow-up investigations in an audiology clinic revealed hearing conduction failure of the same type in all the boys. Otological inspection indicated reduction of the shaft of the malleolus on the right side in all the boys. The left side was normal. When the triplets were 15 years of age their school marks were at the average level of the class. The triplets continued their studies for another 2 years at a secondary-school music line. Their grades deteriorated markedly during those years (15–17 years), except for the results in music, where they all obtained top grades during all of their school years. It is reported that all the boys have always lived relatively isolated lives with few friends and social contacts. None of them have ever had a romantic partner. Before falling ill all the triplets were employed.

Triplet S

S was admitted to a Stockholm psychiatric clinic in 1979 (ages 20 years, 5 months). He had called for the fire brigade without any rational reason. On being asked about the fire S pointed toward his chest and gave odd explanations, which made the police ask his mother to take him to the hospital. There the patient admitted to hearing internal voices and showed peculiar behavior. He often sat staring out of the window receiving messages from the forest on the horizon. He was treated with chlorpromazine 400 mg daily, gradually improved, and the positive psychotic symptoms disappeared. After 8 weeks his state was satisfactory. After discharge he tried to work as an assistant in a hospital archive. After a few months, however, he left the job and has not had regular work since. He has remained in contact with psychiatric services during subsequent years, because of auditory hallucinations and a tendency toward delusions and thought disturbances. During the first 7 years of illness he was hospitalized for seven periods, due to increasing psychotic symptoms and anxiety in relation to drug withdrawal. In the past 6 years, however, he has spent only 4 days in the hospital. Presently, he is treated daily with sulpiride 600 mg and fluphenazine 5 mg. For the past few years he has lived by himself in his own room in a hostel for mentally ill individuals. He is now a friendly, rather inactive man, preferentially spending a lot of time in bed. His main interests are music and esoteric philosophy.

Triplet T

At about the same time as S was admitted to the hospital (March 1979), T began to feel "sick" in a way he could not explain. In September 1979 he became overtly psychotic with vivid auditory hallucinations and delusions. He had the conviction that he could predict the life of others 3 years forward. Then he started to teach students at the Royal Academy of Music to play the piano, although he had no obligation to do so and no one had asked for his help. He was subsequently admitted to a psychiatric

clinic, where he exhibited auditory hallucinations and thought disturbances. He was treated with chlorpromazine and haloperidol. After 6 weeks he had markedly improved. After 10 weeks he had no remaining psychotic symptoms and was discharged. As with his brothers T has kept in contact with a psychiatric outpatient clinic continuously since then. He has had just one more period of hospitalization, because of marked deterioration including auditory hallucinations and disorganized behavior. When in a deteriorated state T exhibits marked anxiety and inner tension. Ideas of reference and persecution also appear. He has been able to work most of the time. For many years he was employed as a porter in the library of an academic institution. Two years ago, however, he lost his job when the staff was reduced because of financial restrictions. He deteriorated markedly during the months before and after the loss of his employment. He left his room at the hostel, where he had lived for 2 years, and returned to his mother. He improved after a few weeks and is now living in his own apartment. He is interested in music, and spends a lot of time playing the piano skillfully.

Triplet M

In November 1979, approximately 2 months after T and 8 months after S, M was admitted to the same psychiatric clinic after the abrupt onset of severe psychotic symptoms. He experienced auditory and visual hallucinations, thought disturbances, and severe inner tension. He felt as if his mother and his two brothers were penetrating his skin and going into his body - all three together. He improved gradually when treated with chlorpromazine 400 mg daily. After 10 weeks he could leave the hospital; however, vague auditory hallucinations have remained continuously. As with his brothers he has remained in contact with psychiatry, mostly as an outpatient. In the period leading up to 1986 he was hospitalized seven times usually in relation to drug withdrawal. Even when in a relatively satisfactory state of mind, he often complains of anxiety and pain in different parts of the body. He feels uneasy and afraid in ordinary situations. At the beginning of the illness we tried to get him to continue with his job at a post office. As with S, however, he soon stopped working. He tends to be more active during the day than his brothers, visiting museums and a day center for psychiatric patients. He is a talented artist, and, as in the case of T, spends a lot of time playing the piano. Two years ago he left his parental home and is now living by himself in an apartment.

Psychiatric family history

The father of the triplets, a Caucasian born in 1928, arrived in Sweden in 1956 as a refugee after leaving his wife and daughter in his native eastern European country. He met the Swedish mother of the triplets and had a short-lived relationship with her in the early phase of the preg-

nancy. Approximately 15 years after the birth of the triplets the father remarried another woman, but had no further children. One year ago he again separated. The father has been employed as a typographer in Sweden. In 1988 he had to retire from work, because of back pains after an accident. He denies any type of psychiatric problem in himself and his relatives, and has never been hospitalized in Sweden according to case registers. He has one sister who has never been hospitalized and is reported to be healthy.

The mother of the triplets, a Caucasian born in 1925, is of Swedish origin. She was mentally healthy until approximately 1 year after the onset of the psychoses in the triplets. Then she had a period of mild depressive symptoms and was treated as an outpatient. She has worked as a clerk. At age 62 years she retired from work, because of back pains. She has remained single and lived in the same house with her parents for as long as they lived, and later with her three boys. The mother described her childhood as happy. She had no siblings and no children besides the triplets.

Both sets of grandparents and their siblings were reported to have been mentally healthy. However, the paternal grandfather died for unknown reasons when the father was only 8 months of age. Psychiatric disturbances among other relatives are not known to either of the parents.

Materials and methods

All three boys, the mother, and the father were interviewed and examined by two of us (C.H. and E.J.) with regard to their mental and physical health on several occasions between 1992 and 1994. When the present psychiatric and psychological investigations were made all the triplets were given neuroleptic treatment. Triplet S received daily doses of sulpiride 600 mg and fluphenazine 5 mg. Triplets T and M were treated with zuclopenthixol 200 and 300 mg, respectively.

Psychiatric diagnosis

The Structured Clinical Interview for DSM-III-R (SCID) (Spitzer et al. 1986) was performed on the triplets by two psychiatrists (C.H. and E.J.) independently. A similar interview with the mother including assessment of personality disorders (Spitzer and Williams 1985) was performed by C.H., and a partial SCID was performed in the father by E.J.

Genetic methods

The genetic relationship between the triplets was examined by "minisatellite" DNA regions with the probe MZ1.3 (Jeffreys et al. 1985). Cytogenetic analyses, based on leukocytes cultivated in folate-depleted media, were performed in 50 cells from each of the triplets according to Nielsen and Tommerup (1984).

Neuropsychological assessment

A comprehensive neuropsychological test battery was used to examine the triplets. The battery included all 11 subtests of the WAIS-R (Wechsler 1981), Claeson-Dahl's verbal learning and retention test (Claeson and Dahl 1971), the Rey-Osterrieth Complex Figure (copy and delayed retention scores), verbal fluency (FAS), the Trail Making Test, and the Wisconsin Card Sorting Test (Lezak 1983). Standardized administration and scoring was used,

and the results were compared with normative data presented in the references cited.

MRI of the brain

The MRI technique was performed using a 1.5 Tesla system (Sigma, GE medical systems). All patients had the head fixed to the table using a plastic-helmet system (Greitz et al. 1980). This helmet was shaped to fit the head of one of the triplets. Because all three individuals could use the same helmet, almost identical transaxial slices could be obtained (see Fig. 2); T1-weighted repetition time ([TR]/echotime [TE] = 500/17 ms) 4-mm mid-sagittal images were obtained; T2-weighted (4000/30/120) transaxial images were obtained using a 4-mm slice thickness and a 2.5-mm gap. The head was also examined with a 3D gradient echo (GE) sequence using 128 transaxial partitions of 1.3 mm thickness. Herniation of the suprasellar cistern was considered to be present when an obvious bulge of the suprasellar cisterns into the sella turcica was seen on sagittal MR images (Brismar et al. 1978). This was estimated to correspond to a herniation of 3-5 mm. The distance between the lateral walls of the frontal horns was divided by the largest inner-skull diameter and used as a brain ventricular index (VIX; Hansson et al. 1975). The ventricular system was estimated using the Evans Index (EI), which was obtained by dividing the maximum width of the frontal horns with the maximum width of the inner skull (Hanson et al. 1975).

Results

Although all the triplets were markedly overweight and were of short stature (Table 1), they had a normal male physique, and the physical and neurological examination did not reveal any deviations. The same was true for routine blood and urine analyses.

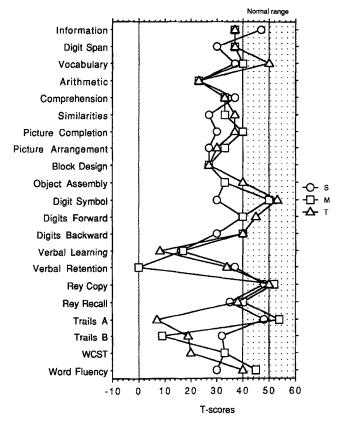


Fig. 1 Neuropsychological performance profiles of the triplets (S, M, and T). All raw scores were transformed into standard T-scores according to published results from normal subjects. WCST Wisconsin Card Sorting Test

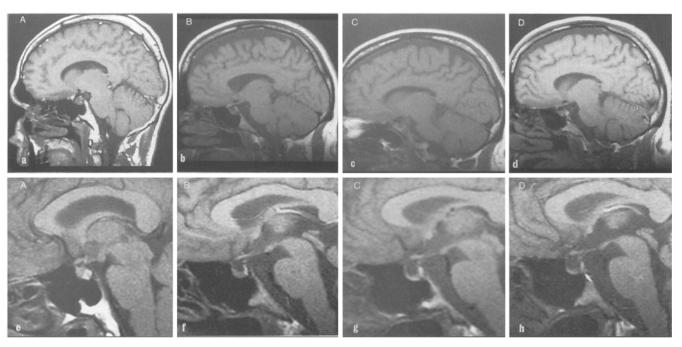


Fig. 2a-h Sagittal T1-weighted magnetic resonance (MR)-images of four people (placed vertically) taken in the midline a-d and 1 cm lateral to the midline e-h. Person A a, e is a healthy man, age 37 years. In triplet B b, f, C c, g and D d, h the morphological changes are similar. The suprasellar cisterns are enlarged and her-

niate into the sella turcica f-h, the so-called empty sella. The sub-arachnoid space is widened in the frontoparietal region b-d, and there is an interposition of the cerebrospinal fluid between the brain and the vault as compared to the normal condition e

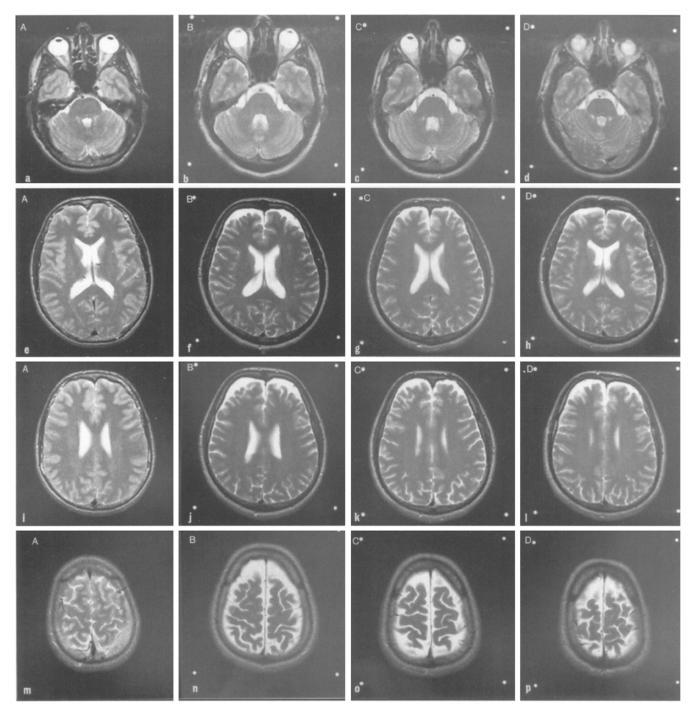


Fig. 3a–p. Transaxial T2-weighted MR images of four people (placed vertically). A 37-year-old healthy male control is presented in the first column (A). The morphological changes are close to identical in all three triplets (B, C, and D). The cerebellopontine cisterns are markedly enlarged **b–d**; so is the subarachnoid space in the frontoparietal region **f–h, j–l, n-p**. The ventricular system is at the upper limit of the normal variation (Evans Index = 0.30 in C and D). The sulci at the convexity are not enlarged

Psychiatric diagnosis

According to the SCID interviews DSM-III-R criteria for schizophrenia, undifferentiated subtype, were fulfilled in

all the triplets. The SCID interview of the mother did not reveal any psychiatric or personality disorder, and the same was true for the partial interview with the father.

Genetics

The DNA investigation (Jeffreys et al. 1985) showed that the triplets were monozygotic (P < 0.0001). There was no evidence of folate-sensitive fragile sites at Xq27 or any other detectable cytogenetic abnormality.

Neuropsychological functions

By assessment of school records and the results in some of the WAIS-R subtests, the premorbid intellectual levels were estimated to have been normal in all the triplets. At the time of the present examination, approximately 14 years after falling ill, and when receiving neuroleptic maintenance treatment, all three patients were found to be clearly impaired in several neuropsychological functions. The individual results are shown in Fig. 1. The patients generally had a global impairment of neuropsychological functions. The differences between the brothers were small, and the rank order differed in a nonsystematic way in different tests. Impairment of attentional, mnestic, and executive functions were prominent in all the subjects.

MRI of the brain

The MRI examinations demonstrated wider subarachnoid spaces in frontoparietal and basal regions in all the triplets, as compared with normal subjects (Fig. 2 and 3). The changes were most pronounced in S. The cortical sulci were generally not widened (less than 5 mm; Gyldensted 1977). The ventricular system in T and M was at the upper border of the normal limit (EI = 0.30). There was also an intrasellar cisternal herniation, "empty sella" (Brismar et al. 1978; Robertson 1957), which was moderate in S and marked in T and M (Fig. 2).

Discussion

In the present paper we have described a male set of monozygotic triplets where the three subjects independently developed acute fulminant schizophrenic disorders fulfilling DSM-III-R criteria within a time period of 8 months in the year they became age 21. The clinical type of the disorder was very similar in the three subjects. Thus, they all fulfilled DSM-III-R criteria for undifferentiated-type schizophrenia with vivid auditory hallucinations, bizarre delusions, and evidence of formal thought disorder. They have all had a chronic course of the disorder with marked impairment of social adjustment, ability to work, and initiative. All three subjects responded typically to conventional neuroleptic treatment with a marked reduction or disappearance of the positive psychotic symptoms within a few weeks after the initiation of treatment. Since their first admission they have all had acute exacerbations of the disorder after drug withdrawal, and have repeatedly shown a rapid response to pharmacological treatment. Neuropsychological assessment demonstrated a similar degree of general reduction of attentional, mnestic, and executive functions in all the subjects. The MRI of the brain demonstrated that they all tended to have a general widening of the ventricular system. Thus, their VIX indices were at the upper range of age-matched controls. Moreover, there was a widening of subarachnoid spaces in frontal, parietal, and basal areas. All three also showed signs of an empty sella, a pathological expansion of the suprasellar cistern into the sella turcica.

There were indeed great similarities in practically all the clinical features of the three subjects, i.e., the time of onset of the psychosis, the symptoms and signs, the chronic intermittent course, the distinct response of positive symptoms to drug treatment, the evidence of cognitive and conative impairment, and the expanded cerebroventricular spaces. All this evidence strongly indicates that each of the triplets has a typical schizophrenic disorder with features of aberrant neuropsychological functions and brain morphology that have been shown to be typical for a substantial group of such patients (Blanchard and Neale 1994; Johnstone et al. 1976).

The great similarities that the schizophrenic phenotypes of the triplets represent stimulated our interest to further analyze the relative influences of nature and nurture, i.e., genetic and environmental mechanisms for the development of these similarities. The detailed clinical exploration of familial, prenatal, and premorbid history of the triplets gave several interesting clues as to the possible influences of genetic and environmental factors in the etiology.

The reported absence of psychiatric disease in the parental and grandparental generations gave no indication for a fully penetrant dominant genetic transmission as an etiological mechanism. However, this does not rule out other ways of genetic transmission. The notation in the mother's obstetric journal concerning an "Asian" influenza infection during the first trimester of the pregnancy might also be considered an etiological factor for schizophrenia in the three men. However, this information was compromised by the fact that the mother retrospectively strongly denied having had influenza during the pregnancy. Furthermore, the reported relationship between influenza infection and schizophrenia indicates that infection in the second, rather than the first, trimester is important (Adams et al. 1993).

The otological investigations performed when the patients were aged 11 years indicated a right-sided hearing defect and a reduction of the shaft of the right malleolus in all the triplets. It seems highly unlikely that an exogenous noxious agent should cause an almost identical ossicular lesion on the same side in three separate individuals even if they are triplets. The most critical phase for auricular bone damage would occur earlier in pregnancy than the critical phase for developing an influenza-induced vulnerability for schizophrenic illness later in life (Adams et al. 1993; Austin 1985). The type of auricular bone malformation in the triplet strongly indicates a genetic mechanism. According to a literature search there appear to be no known chromosomal loci for congenital hearing-bone defects. Similarly, there seem to be no reports of concurrence of schizophrenic psychosis and ear-bone defects.

Triplet pregnancy per se is a very rare condition especially before the introduction of hormone therapy for infertility. Schizophrenia is also a rare condition with a lifetime prevalence of approximately 1% (Jablensky et al. 1992). Congenital defects of the ear are rare, occurring in

approximately 2/10,000 births (Austin 1985). Empty sella is a more common finding, reported to occur in approximately 5% of autopsy materials (Brismar et al. 1978). Thus, the three male triplets all demonstrated four remarkably rare conditions. The risk involved in the general population to acquire all these states independently of each other would be very low from a statistical point of view. It is therefore reasonable to tentatively conclude that genomic mechanisms may be responsible for a substantial part – if not all – of the abnormalities found in the triplets. It therefore seems possible that a DNA aberration that was present before or occurred at conception initiated a precise time-programmed series of events that produced the very similar schizophrenic phenotypes. Such an aberration might have been inherited or induced by an external agent, or might have occurred spontaneously. It seems possible that the psychosis, the reductions of neuropsychological functions, the morphological MRI changes, and the right-sided malleolar reduction may all be related to such DNA alteration. The further search for DNA abberations in the genome of the triplets might help in testing this hypothesis and also give guidelines for genomic localizations of the rare aberrations found in the present triplets.

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References

- Adams W, Kendell RE, Hare EH, Munk-Jørgensen P (1993) Epidemilogical evidence that maternal influenza contributes to the aetiology of schizophrenia. An analysis of Scottish, English, and Danish data. Br J Psychiatry 163:522–534
- Austin F (1985) Congenital malformation of the ear. In: J. Balinger (ed) Diseases of the Nose, Throat, Ear, Head and Neck. Lea & Febiger, pp 1217–1231
- Blanchard JJ, Neale JM (1994) The neuropsychological signature of schizophrenia generalized or differential deficit. Am J Psychiatry 151:40–48
- Brismar K, Bajraktari X, Goulatia R, Efendíc S (1978) The emty sella syndrome intrasellar herniation in "normal" patients and in patients with communicating hydrocephalus and intracranial tumors. Neuroradiology 17:35–43

- Claeson L-E, Dahl E (1971) Claeson-Dahls inlärningstest för kliniskt bruk. Psykologiförlaget, Stockholm
- Greitz T, Bergström M, Boethius J, Kingsley D, Ribbe T (1980) Head fixation system for integration of radiognostic and therapeutic procedures. Neuroradiology 19:1-6
- Gyldensted C (1977) Measurements of the normal ventricular system and hemispheric sulci of 100 adults with computed tomography. Neuroradiology 14:183–192
- Hansson J, Levander B, Liliequist B (1975) Size of the intracerebral ventricles as measured with computer tomography, encephalography and echoventriculography. Acta Radiol (Suppl) 346:98-106
- Jablensky A, Sartorius N, Ernberg G, Anker M, Korten A, Cooper JE, Day R, Bertelsen A (1992) Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization Ten-Country Study. Psychol Med 1–97
- Jeffreys AJ, Wilson V, Thein SL (1985) Hypervariable "minisatelite" regions in human DNA. Nature 314:67-73
- Johnstone EC, Frith CD, Crow TJ, Husband EJ, Kreel L (1976) Cerebral ventricular size and cognitive impairment in chronic schizophrenia. Lancet 30:924–926
- Kendler KS (1988) The genetics of schizophrenia: an overview. In: M. T. Tsuang and J. C. Simpson (eds) Nosology, epidemiology and genetics of schizophrenia. Elsevier, Amsterdam New York Oxford, pp 437–462
- Kety SS, Rosenthal D, Wender PH, Schlusinger F, Jacobsen B (1975) Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic: a preliminary report based on psychiatric interviews. In: R. Fieve, D. Rosenthal and N. Brill (eds) Genetic Research in Psychiatry. John Hopkins University Press, Baltimore, pp 174–165
- Lezak MD (1983) Neuropsychological assessment (2 ed.). Oxford University Press, New York
- McGuffin P, Reveley A, Holland A (1982) Identical triplets: non-identical psychosis? Br J Psychiatry 140:1-6
- Mirsky AF, Quinn OW (1988) The genain quadruplets. Schizophr Bull 14:595-612
- Nielsen KB, Tommerup N (1984) Cytogenetic investigations in mentally retarded and normal males from 14 families with the fragile site at Xq28. Hum Genet 66:225-229
- Onstad S, Skre I, Torgersen S, Kringlen E (1991) Twin concordance for DSM-III-R schizophrenia. Acta Psychiatr Scand 83: 395–401
- Robertson EG (1957) Pneumoencephalography III. Thomas, Springfield
- Spitzer RL, Williams JBW (1985) Structured Clinical Interview for DSM-III-R Personality Disorders (SCID II). Biometrics Research Department, New York State Psychiatric Institute, New York
- Spitzer RL, Williams JBW, Gibbon M (1986) Structured Clinical Interview for DSM-III-R - Non Patient Version (SCID-NP). Biometrics Research Department, New York State Psychiatric Institute, New York
- Wechsler D (1981) Wechsler Adult Intelligence Scale Revised. The Psychological Corporation, San Antonio