

Causes of Infantile Autism: Some Considerations from Recent Research¹

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Experimental, clinical, and longitudinal studies of infantile autism are reviewed and the evidence with respect to different views on the causation of the condition is considered. Several independent investigations have shown the presence of a severe, extensive defect in language comprehension, in control functions associated with language, and with the processing of symbolic or sequenced information. Circumstantial evidence suggests that this cognitive defect constitutes the primary handicap in autism, the social and behavioral abnormalities arising as secondary consequences. It remains uncertain whether the cognitive/language defect is a sufficient cause for autism or whether some interaction with particular personality attributes or family environment is necessary. The basic cause of the cognitive defect is unknown, although the high rate of fits in intellectually retarded autistic children suggests the presence of some kind of organic brain disorder. Among the intelligent autistic children, there is less evidence for structural brain pathology; it remains possible that in some cases autism may arise on the basis of some maturational disorder or genetically determined condition.

“Since 1938 there have come to our attention a number of children whose condition differs markedly and uniquely from anything reported so far.” So began Kanner’s epoch-making first account of the condition now known as infantile autism. After a brilliant clinical description—which could scarcely be bettered today—he went on to end the paper by stating that “we must, then,

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assume that these children have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps” (Kanner, 1943).

Since then, from many different centers in all parts of the world, there have come numerous other reports of similar children (Rimland, 1964; Bender, 1969). There is no doubt now that such a condition can be recognized and that in their behavior and development, children with infantile autism do differ from children with other psychiatric disorders. However, there has been dispute on the question of whether autistic children differ qualitatively, or merely quantitatively, from the normal. There has also been a proliferation of hypotheses on the nature of autism and on its causation. The basic cause of infantile autism still remains unknown; but experimental, clinical and longitudinal studies carried out over the last 10 years have gone a long way toward the elucidation of the mechanisms involved. This paper briefly surveys the findings of this research in order to arrive at some appraisal of what is known about causes.

DIAGNOSIS OF AUTISM

Before appraising causes, it is necessary to define the condition we are to consider. As the name “autism” implies, one of the key features consists of an autistic-type abnormality in interpersonal relationships. This may be shown in early childhood by a relative lack of eye-to-eye gaze, limited emotional attachments to parents, little variation in facial expression, and an appearance of aloofness and distance together with an apparent lack of interest in people. Autistic children often fail to cuddle and do not come to parents for comfort when hurt or upset. They do not join in group play activities or form friendships; they show little emotion, little sympathy or empathy for other people. However, these social abnormalities are not (and were not in Kanner’s first description) the only features of the syndrome. In the present state of knowledge it appears that the diagnosis of infantile autism should be restricted to children who show three groups of abnormalities: autism (as already described), delays in speech and language development, and ritualistic and compulsive phenomena (Rutter, 1970a). Speech and language delay is usually accompanied by at least some reduction in response to sounds; and when language develops it is usually abnormal in quality—pronominal reversal and echolalia being the most striking features. The ritualistic and compulsive phenomena may take any of four forms: a morbid attachment to unusual objects, peculiar preoccupations, a resistance to change or quasi-obsessive rituals.

Hand and finger mannerisms and complex whole body movements of a stereotyped kind are also common but should not be included as diagnostic criteria in view of their frequency in the (nonautistic) inmates of long-stay institutions, in mentally subnormal children, and in blind children. It should be emphasized that the clinical picture changes somewhat as they grow older; some of these diagnostic features may not be apparent in the older child.

One further diagnostic criterion is necessary and that is onset in infancy—or at least by the age of 30 months. The majority of autistic children have shown abnormalities from the first year of life; but in a minority of cases—probably about a fifth—there does seem to have been a period of normal development before the onset of autism. Whether these cases differ in pathogenesis from the others is not yet known.

AUTISM AS SOCIAL WITHDRAWAL

Let us now turn to the first major issue: does autism differ *qualitatively* from normality? That some features of autism may occur in other conditions or even, as transient features, in normal children is generally accepted. However, some writers have gone further. O'Gorman (1970) observed that dissociation and social withdrawal are normal phenomena. All of us show this when concentrating on work or a book. He suggests that autistic children withdraw from auditory stimuli in infancy and continue to do so until it becomes a habit. Thus, he sees autism as a defense mechanism and the syndrome as merely an exaggeration of normal phenomena. The crucial feature in this view is that the social withdrawal in autism is similar in kind to that shown in normal people. That autistic children are seriously impaired in their social relationships in early childhood is obviously true, but O'Gorman's suggestion that this is the same type of selective withdrawal found at times in normal people has no empirical support. The hypothesis fails to account for the other features of the syndrome, it does not explain why autism is four times as common in boys as in girls, and it by-passes the evidence of organic impairment in autistic children.

Selective social withdrawal and avoidance of speech does, of course, occur in the condition known as "elective mutism." However, the clinical features and outcome are quite different from autism and, unlike autism, elective mutism is found in girls as often as in boys. On the available evidence to date, the hypothesis that autism is due to selective social withdrawal may be rejected. Others have suggested that although the social withdrawal may differ in kind from that in the general population, nevertheless, such withdrawal, however caused, is the basic handicap. On the face of it, this seems more plausible in that

impaired relationships are certainly a cardinal feature of autism. However, social withdrawal is one of the first symptoms to improve in autism, and some autistic children cease to show the symptom in adult life (Rutter, Greenfield, & Lockyer, 1967). The finding that even after they cease to be withdrawn, they may still remain retarded in language, have a low I.Q., and show obsessive-like symptoms argues strongly against social withdrawal (of any type) being the cause of the other handicaps (Rutter, 1968).

AUTISM AS EXTREME INTROVERSION

Ounsted and the Huttts at Oxford, noting the importance of gaze aversion, have suggested that autism consists of a genetically determined extreme degree of introversion associated with high arousal in social situations (Hutt & Ounsted, 1966; Hutt, C., & Hutt, S. J., 1970). This hypothesis requires serious attention. Ounsted (1970) has marshalled the arguments in a recent review of the subject and ends by listing 11 predictions stemming from his theory. Some of these have received support and some have still to be tested. However, one crucial prediction has failed the test. Ounsted (1970) argued that if his view was correct, the parents of autistic children should be markedly more introverted than normal. Kolvin (1971) has recently shown that this is not so. Mothers of autistic children are actually slightly (but not significantly) more extroverted than the general population. Another facet of Ounsted's theory—that of high arousal (Hutt, S.J., Hutt, C., Lee, & Ounsted, 1965)—has also been called in question by recent work. Hermelin and O'Connor (1968; 1970) found that in the resting state autistic children do not differ from normal or subnormal controls in their cortical aroused state, at least as measured by alpha blocking. Complex results were obtained with regard to arousal in conditions of sensory stimulation. In intermittent (but not continuous) light stimulation, autistic children became less aroused than normal people, whereas in continuous (but not intermittent) sound stimulation, they were more highly aroused. Arousal is a multifaceted concept, and different measures of arousal do not always agree with one another. Exactly what is the correct explanation of the two sets of findings on arousal is uncertain. It may be that in some circumstances autistic children are overaroused as a response to certain types of stimulation but clearly they cannot be regarded as generally showing high arousal as a basic handicap. ?

The Oxford group found that autistic children's stereotypes increased in complex environments which, they argued, suggested high arousal (Hutt, C., & Hutt, S.J., 1965; 1966); but preliminary findings from Ornitz, Brown, Sorosky, Ritvo, and Dietrich (1970) in Los Angeles failed to confirm this. Finally, the

introversion theory cannot account for the heavy male preponderance among autistic children. In the general population, there is no evidence that introversion is more common to either sex (Eysenck, H.J., & Eysenck, S.B.C., 1969). In view of these recent findings, one must conclude that Ounsted's theory will not stand without serious modification. It still poses some important research questions but it cannot account for the causation of autism.

AUTISM AS A TYPE OF SCHIZOPHRENIA

Before turning to what might be the causes of autism it is necessary first to consider a few other hypotheses which have been influential but which now appear mistaken. For many years autism was regarded as a particularly early manifestation of schizophrenia. It is now reasonably certain that this view is wrong. Autism and schizophrenia differ in terms of sex distribution, social background, family history of schizophrenia, intellectual level, cognitive pattern, presence of delusions and hallucinations, and course of disorder (Rutter, 1968). Kolvin's important series of studies comparing infantile and late onset psychosis now provide the most decisive evidence that the two conditions are quite different (Kolvin, 1971).

AUTISM AS A PSYCHOGENIC DISORDER

Until recently some workers have regarded autism as a psychogenically determined disorder. Autism has been thought to represent the child's response to parental detachment, lack of warmth and obsessionality. This theory also now seems most unlikely. A recent study of ours (Rutter, Bartak, & Newman, 1971) has shown that the parents of autistic children do not differ in the characteristics from the parents of children with other language disorders. Kolvin (1971) found that the mothers were not at all unusual in terms of either neuroticism or introversion and that both clinical and questionnaire studies have failed to show any personality stereotype in the parents (Rutter, 1967).

Yet others have suggested that there may be parental pathology which is specifically concerned with the child who becomes autistic, but again supporting evidence is lacking. Furthermore, such parental abnormalities as do occur are at least as likely to result from pathology in the child as the other way around. It is relevant in this connection that Schopler and Loftin (1969) found that parental qualities differ according to the setting in which they are assessed.

It may be concluded that autism almost certainly is not due solely to psychogenic influences, although, as in any other child, environmental circumstances may well be influential in the development of secondary handicaps.

AUTISM AS A DEPRIVATION SYNDROME

In similar fashion it has been suggested that autism develops because of a lack of adequate social stimulation and personal contact. It is well known, of course, that social deprivation can lead to social withdrawal and language retardation; but it probably affects boys and girls equally and the pattern of symptoms is different from that in autistic children. In particular, there is usually little defect in language comprehension (almost always seriously impaired in autistic children) and ritualistic and compulsive activities are not a characteristic feature (Rutter, 1971). In any case, autistic children do not usually have a history of depriving experiences. It may be concluded that it is most unlikely that social deprivation is a cause of autism.

AUTISM AS A GENETIC DISORDER

Genetic studies have been disappointingly limited and inconclusive so far, and there is a need for a well-conducted twin study on adequate numbers (Rutter, 1967; 1968). The rate of autism in siblings (less than 2%) is low, which is rather against a decisive hereditary element. On the other hand, the rate is considerably above that in the general population (4 to 5 per 10,000) and it may be that there is a genetically determined type of autism that constitutes a small subgroup of autistic disorders. Several investigations have shown that autistic children have the normal chromosome complement (Bieseke, Schmid, & Lawlis, 1968; Böök, Nichtern, & Gruenberg, 1963; Judd & Mandell, 1968); but Judd and Mandell reported that some autistic children as well as some fathers had an unusually long arm to the Y chromosome. The significance of this, if any, is quite unknown at present.

AUTISM AS A TYPE OF SUBNORMALITY

In the past it has occasionally been suggested that autism is merely an unusual type of mental subnormality, but there are two main reasons why this is an inadequate explanation.

1. Even on standard intelligence tests, about a quarter of autistic children can be shown to have an I.Q. in the normal range (Rutter & Lockyer, 1967).
2. To some extent the autistic child's poor level of intellectual function appears to be related to specific defects in language rather than to a global deficiency of intelligence (Lockyer & Rutter, 1970).

Yet on the other hand, there can be little doubt that intellectual retardation is an important aspect of autism. The majority of autistic children obtain scores

in the mental subnormal range, and the IQ scores in early childhood have been found to be reasonably stable and a quite good predictor of intellectual functioning later (Lockyer & Rutter, 1969). Moreover, the IQ level remains much the same even in those autistic children who recover or greatly improve (Rutter, 1966). Many autistic children are mentally subnormal as well as being autistic, and the mental subnormality is just as "real" as in any other child who is mentally retarded.

So far our conclusions on etiological factors in autism have been largely negative, so let us now turn to more positive findings.

AUTISM AS A COGNITIVE/LANGUAGE DISORDER

In a review of concepts of autism (Rutter, 1968) it was concluded that autistic children had a central disorder of language involving both the comprehension of language and the utilization of language or conceptual skills in thinking. It was suggested that this disorder of language constituted the basic handicap to which the other symptoms of autism were secondary. Work since then, particularly the systematic experimental studies of Hermelin, O'Connor and Frith (Frith, 1971; Hermelin, 1971; Hermelin & O'Connor, 1970; O'Connor, 1971) have largely confirmed this view, but have also shown that the cognitive defect is rather wider than suggested in 1968.

The evidence in favor of this hypothesis may be summarized as follows. Retardation of speech and language is an almost invariable manifestation in infantile autism, and a lack of response to sounds is frequently the first symptom to be noted. Apart from the level of IQ, language is also the most important prognostic factor (Rutter, 1967). The pattern of cognitive abilities in autism shows that the children are deficient in language skills even on tests that do not require speech (Lockyer & Rutter, 1970). These results show that the failure to speak results from a basic impairment in language function not just from an absence of motivation to speak. Not only is the autistic child retarded in language, but also his *pattern* of linguistic abilities is significantly different from that exhibited by either normal or mentally retarded children (Tubbs, 1966). The autistic child is particularly poor in his transfer of information from one sensory modality to another, in his understanding of the meaning of the spoken word, and in his use of gesture. The last feature also differentiates the autistic child from the child with so-called developmental aphasia (Rutter, Bartak, & Newman, 1971). Pronominal reversal and echolalia which probably reflect defects in comprehension, are also much more common in autistic than "aphasic" children. Thus, autistic children differ from aphasic children in having both a more severe and a more widespread language handicap

The experimental studies of Hermelin, O'Connor, and Frith carry the issue much further. They have shown that autistic children are particularly unresponsive to verbal stimuli and that they make little use of concepts in memorizing. The verbal recall of autistic children is relatively independent of the meaning of what they hear. More recent experiments have shown that, compared with normal and mentally subnormal children of the same mental age, autistic children not only are impaired in their appreciation of the grammatically structured aspects of language but also lack the ability to associate words semantically. However, this defect is not confined to auditory material. Autistic children also have difficulties in perceiving temporal patterns in visually presented stimuli, suggesting a central defect in the processing of any sort of coded, meaningful, or temporally patterned stimuli. Such a deficit is likely to impair severely the infant's and young child's interaction with the environment. It is suggested as a working hypothesis that these specific cognitive defects are the cause of the social and behavior differences in most cases of autism (Rutter, Bartak, & Newman, 1971).

Other workers, while agreeing that autism is based on some form of central cognitive disorder, have laid most emphasis on slightly different central functions (DeMyer, 1971). For example, Ornitz (1971) has suggested that there is a disorder of sensory motor integration; Martin (1971), a central sensory disorder; and Wing (1971), a disorder involving multiple language, cognitive and perceptual handicaps. The fact of the matter is that autistic children usually exhibit multiple central defects. At the present time it is uncertain which of these are *necessary* and *sufficient* causes of the disorder. We have suggested that a central defect in the processing of symbolic or sequence information is likely to prove the basic defect but the evidence is not yet available to decide conclusively between the different types of cognitive or sensory disorders which have been *postulated* for autism.

NEUROLOGICAL ABNORMALITIES IN AUTISM

The cause of the language/cognitive defect is still to be considered. Among young autistic children it is usual to find no abnormalities on neurological examination other than immaturities in developmental functions. In addition, metabolic investigations generally show no specific abnormality (Rutter, 1967; Sankar, 1970). These negative findings have led to the assumption that there is no organic brain dysfunction in autism. However, long term followup studies have now shown that this assumption is frequently wrong. Autistic children first seen at the Maudsley Hospital in early childhood have now been followed

through to adult (a mean age of 22 years) life (Rutter, 1970b). Of these 64 children, no less than 18 (29% of those not epileptic in early childhood) have developed epileptic fits during adolescence. These findings inevitably raise the question of whether autism can be divided into those cases due to organic brain disease and those not due to this disease. The most obvious difference between those who did and those who did not develop fits in adolescence was the initial level of measured intelligence. Of the nine children whose IQ was untestable, and, therefore presumably untestably low (Lockyer & Rutter, 1969), no less than seven have begun to have fits by the 1970 follow-up³. Furthermore, of the 18 children who developed fits, only 2 had an initial IQ of 65 or more. Put the other way around, of the 27 children who were untestable or had an IQ of 50 or less, 15 developed fits in adolescence, another 2 had had fits since early childhood, and a further 4 had indications of possible brain disorder. These findings strongly suggest that most, if not all, autistic children with severe "intellectual retardation" develop their disorder as a result of organic brain dysfunction.

A further pointer to the probability of neurological disorder in some autistic children is provided by the small proportion (7 out of 64 in the follow-up study) who deteriorate in adolescence. In three cases with an IQ below 50, deterioration occurred from an already low level; but in four, where the IQ was in the 59 to 69 range, the decline followed a period of progress and general improvement. The deterioration usually began with a loss of language skills associated with inertia and decreasing activity and was followed by general intellectual decline. In four out of the seven cases the deterioration was accompanied by the development of fits. Investigations of these children are only just beginning; therefore, the cause remains unknown. However, one child not in the followup study, who showed the same clinical picture, was found to have histidinaemia.

The situation is rather different with respect to those autistic children who had normal intelligence. Of the 12 in the followup study with a performance IQ of at least 80 in adolescence, only 4 showed evidence of probable "brain damage".⁴ But unlike the intellectually retarded, the majority showed no clear indication of organic brain dysfunction.

³This includes one who had a few fits in infancy and therefore excluded from the 18 children with developing fits for the first time in adolescence.

⁴One case each of fits developing in adolescence, a persistent, temporal lobe spike focus on the EEG, toxoplasmosis, and congenital lues.

AUTISM AS A DEVELOPMENTAL DISORDER

A further possibility in these children is that the autism developed in relation to a particularly severe developmental language disorder, possibly genetically determined. This is suggested by findings from the comparative study of autistic and developmental "aphasic" children already mentioned (Rutter, Bartak, & Newman, 1971). In the autistic children, 5 out of 14 had a history of language delay in close relatives, as did 5 out of 11 "aphasic" children. Although this study showed important differences between autistic and aphasic children, it had also been found that the two conditions may occur in the same sibship, suggesting a possible link in a subgroup of cases. In these families, autism developed in the intellectually retarded siblings who also had the most severe and global language impairment.

CONCLUSION

Although we are still far from knowing the cause of infantile autism, the nature of the disorder is becoming more clearly understood. Clinical, comparative, and experimental studies of autistic children at all levels of intelligence have demonstrated the presence of a severe and extensive defect in language comprehension and in central functions associated with language and with the processing of symbolic or sequenced information. Circumstantial evidence suggests (but does not prove) that the language and cognitive defect constitutes the primary handicap in autism, the social and behavioral abnormalities arising as secondary consequences.

The cause of this language and cognitive defect remains unknown. The high rate of fits in intellectually retarded autistic children suggests the existence of some kind of organic brain damage or disorder. In the few cases where some specific brain disease has been found, the type of disease has been far from uniform. It seems that the type of physiological dysfunction caused by the disease is more important than the type of tissue pathology. Among the intelligent autistic children the evidence for structural brain pathology is less striking, and it remains possible that few cases arise on the basis of a maturational disorder. The rate of genetic factors remains quite uncertain. This is an area of research requiring further exploration.

It is not yet possible to be sure whether the language/cognitive defect is a *sufficient* cause for autism or whether some interaction with particular personality attributes or family environment is necessary. However, the consistent finding of gross language and cognitive deficits associated with autism

and the consistent failure to find deviant factors in the environmental situation suggest that the language/cognitive impairment is the *main* factor involved in the pathogenesis of autism.

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