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On Innate Releasing Mechanisms Dysfunction Theory of Early Childhood Autism

Osamu Nakayama

Abstract

This article is a theoretical discussion about the etiology of early childhood autism. First, the author criticizes both the hypothesis that autism is a basic disorder of cognition and language and Tinbergen's ethological model of autism. And the author proposes 'innate releasing mechanisms dysfunction theory' derived from Lorenz's ethological theory. This hypothesis is that early childhood autism is caused by the dysfunction of innate releasing mechanisms which results from biological deficiencies. In other words, the author assumes that both, a severe failure to develop social relationships and a specific language impairment, are concurrently and interdependently developed by the dysfunction of innate releasing mechanisms in early infancy.

This paper deals with the etiology of early childhood autism from the viewpoint of ethology. Firstly the author criticizes two theories: (a) Tinbergen's ethological model of autism, and (b) the widely discussed hypothesis that autism is a central disorder of cognition and language. And the author proposes a hypothesis that early childhood autism is caused by dysfunction of innate releasing mechanisms resulting from biological deficiencies. In other words, the author assumes that both, a severe failure to develop social relationships and a cognitive and language deficit, are concurrently and interdependently developed by the dysfunction of the innate releasing mechanisms in early infancy. As a result of an interaction of these two factors, an idiosyncratic imprinting-analogous behavior pattern appears, especially in the social behavior and language of an autistic infant. The types of the dysfunction of innate releasing mechanisms and implications for educational treatment are also discussed.

In this study, the author's etiology of early childhood autism is theoretically discussed on the basis of Lorenz's ethology and Eibl-Eibesfeldt's human ethology. Although Tinbergen previously presented an ethological model of autism (Tinbergen & Tinbergen, 1972), the author's ethological model is entirely different from it. It is because Tinbergen and Lorenz differed from each other in the methodologies of ethology they have developed, and the author's view agrees with that of Lorenz. Therefore the author thinks tnat his developmental model proposed here concerning early childhood autism might be unique one based mainly upon Lorenz's ideas.

A Criticism of Tinbergen's Ethological Model of Early Childhood Autism

The review and criticism of Tinbergen's

model of early childhood autism was conducted by Wing (1976). Her criticism, making use of a huge amount of accumulated data on autism is much to the point. From the viewpoint of Lorenz's ethology as well as Wing's view, Tinbergen's ethological model of autism is a false lead.

Originally, there was a fundamental difference of opinions between Lorenz and Tinbergen concerning a methodology of ethology. The point is whether the dichotomy of behavior into innate and learned components carries validity or not. According to Lorenz (1965),

> ...many modern ethologists, particularly those publishing in English, contend that the term innate is not only useless, but heuristically harmful. They assume that phylogenetic adaptation and adaptative modification can be added to and mixed with each other in any behavior mechanism, however minute and elementary. For this reason, they regard it as hopeless and even dangerous to try separating, in experiment of thought, innate and learned elements of behavior. (p. 102)

Lorenz called Tinbergen's view the mixed hypothesis, in line with this criticism.

The author thinks Tinbergen's ethological model of autism is also based upon the above criticism of Lorenz against Tinbergen. Tinbergen's model seems to neglect the presence of innate species-specific action and response patterns in human beings. Namely Tinbergen's model of autism has only put the vulnerable child in an abnormal environment hypothesis proposed by Kanner (1949) and others in terms of ethology. Because, from the viewpoint of the mixed hypothesis about phylogenetic adaptation and adaptive modification, any behavior disorders including autism should inevitably be caused from both innate abnormalities and environmental ones, however minute and elementary.

Now, in contrast with Tinbergen, Lorenz (1965) thought the concepts of the innate and the learned represent the following:

> Both are defined by the provenience of the information which is the prerequisite of behavior being adapted to enviornment. There are but two ways in which this information can be fed into the organic system. The first is the interaction of the species with its environment during evolution. The second is the interaction with the environment in which the individual acquires information. (p. 103)

Every case of adaptive modifiability (the function of learning) presupposes its own, phylogenetically adapted mechanism.

To sum up, Lorenz insisted that nobody had to dream of abandoning the concepts on the innate and the learned in behavior research as well as in morphogeny, in spite of the fact that both processes are usually superimposed, one upon the other, in one phenomenon (Lorenz, 1965).

The above discussion leads the author to argue first whether autism is caused from abnormalities of the psychological environment or not. There has so far been an extensive discussion about the possibility that autism might be a psychogenic disorder. However, when systematic investigations were carried out, the results were largely negative. The parents of autistic children varied considerably in personality; environmental stresses were no more common than with any other group of subnormal children; and no particular pattern of family interaction was found (Rutter, 1974).

On the whole, well-controlled studies have failed to show any specific abnormalities of personality of child-rearing practices among groups of parents (Wing, 1976). Thus environmental theories are not yet supported by any firm experimental evidence.

Moreover, autistic children even if they were given information for adaptation through concentrated operant conditioning, showed both the very limited gains and a lack of generalization to other environments (Lovaas, 1973). By contrast, excellent progress of adaptation was made by children who had been kept in conditions of extreme physical and mental deprivation for long periods (Wing, 1976). Furthermore, autistic children regressed without special circumstances of specific-contingencies of reinforcement (Lovaas,

1973; Nakayama & Nakayama, 1977).

These findings, from Lorenz's ethological viewpoint, suggest that autism is not caused by distortion in the information itself which is needed for adaptation and which is obtained from the environment, but it is caused by innate defects which make autistic children unable to accept or use effectively such information. In other words, the author's ethological model of autism to be developed from Lorenz's theory is completely different from that of Tinbergen because the author's model denies abnormalities of the psychological environment and suggests instead that the innate deficiencies exist which result from various biological deficiencies of hereditary ones and organic disorders of the brain.

A Criticism of the Hypothesis that Autism Is A Central Disorder of Cognition and Language

Recently the hypothesis has prevailed that autistic children have a specific cognitive deficit (Rutter, 1971, 1974; Ricks & Wing, 1976; Wing, 1976). Although this hypothesis varies slightly from researcher to researcher, its common feature lies in the view that an impairment in the linguistic functions is central (Rutter, 1971, 1974) or that cognitive problems which affect the comprehension and use of language are the underlying impairments (Wing, 1976). In other words, this hypothesis puts an emphasis on linguistic impairments affected by abnormal cognitive skills. Accordingly a profound and general failure to develop social relationships such as apparent aloofness and indifference to other people is regarded to be only one of the secondary behavior problems resulting from this deficit, in contrast with Kanner's opinion.

Nevertheless, it is clear that most children with handicaps such as mental retardation, cerebral palsy, and developmental aphasia, who have a varied degree of impairment of cognition and language, are not autistic. Consequently the view that linguistic functions and cognitive skills are affected to give rise to autism is not established yet.

Moreover, the author thinks that the hypothesis ignores phylogenetic adaptation, it consequently confuses the relation between cause and effect. From the standpoint of phylogenetic adaptation, social relationships are peculiar not only to human beings but also to gregarious animals. Similarities among species have phylogenetically common origins. Especially social behavior of many higher mammals resembles that of human beings to the extent that comparison is provoked (Lorenz, 1971; Eibl-Eibesfeldt, 1970, 1973).

On the other hand, language behavior is a species-specific behavior pattern unique to human beings not to be found in other species (Lenneberg, 1967). Therefore, from the viewpoint of phylogenetic adaptation, social behavior and language are considered as different mechanisms, although both are closely related. And, Lorenz also supported the view that certain fundamental structures in language and thought were innate and to be found in identical form in only human beings and were the product of the selection pressure, not of communication, but of logical thought (Lorenz, 1977).

Accordingly, the author rules out the possibility that a profound and general failure to develop social relationships is directly caused by a severe impairment in linguistic functions, though admitting that the latter disturbs the normal communication patterns of a child. In other words, the author believes both, a severe failure to develop social relationships and a cognitive and language deficit, are concurrently and interdependently developed in autism. And this view is supported by the author's clinical experience.

An Innate Releasing Mechanisms Dysfunction Theory

It is generally believed that latent abilities in organisms are brought out under an external stimulus in the environment. However, this view is only partially correct in that the presence of innate mechanisms intervening between latent abilities and the environment is found. Namely it is now obvious that only the intervening mechanisms bear direct relation with the environment and bring out

latent abilities.

At the level of molecular biology, intervening mechanisms were found by Monod (1971). In animal behavior, they are known as innate releasing mechanisms. According to Lorenz (1977), innate releasing mechanisms (IRM) is a physiological apparatus that filters the stimuli, in other words, "allows only those [stimuli] to impinge which are reasonably likely to define the particular environment in which the elicited behavior pattern can have its proper effect" (p. 53).

And endogenous-automatic stimulus production of animal species are combined with innate releasing mechanisms and as a result endogenous-automatic motor patterns are produced. Innate releasing mechanisms tell innate action and response norms at what moment and in what circumstances the relevant behavior pattern is likely to ensure survival throught it. And Lorenz (1977) believes:

> ...in higher animals with well-developed sense organs and central nervous system, and with a wide choice of behavior patterns, more exacting demands are made on the powers of selectivity of the innate releasing mechanisms, particularly if a variety of combinations of stimuli issuing from one single sense organ must elicit different responses. (p. 54)

Thus imprinting which is defined as many behavior patterns, especially social patterns, becoming irreversibly fixed on an object at certain early, sensitive phases of their development is also learned within the framework of innate releasing mechanisms.

To turn to human beings, the presence of pre-programmed learning abilities which were named 'innate learning dispositions' by Eibl-Eibesfeldt (1970) was confirmed in the spheres of social behavior and language. According to Eibl-Eibesfeldt (1970, 1973), these are also incorporated into innate releasing mechanisms which permit man to recognize specific releasing stimulus situations before he has an experience and are activated through them. Innate releasing mechanisms play a major role in the functioning of imprinting-analogous behavior patterns in early development of human behavior, especially social behavior and language.

For example, in social behavior, aggression and bond-forming in expressive movements have been studied with comparative methods. As a result of these comparative studies, it has become evident that there is an innate disposition to establish personal bonds and that it has sensitive periods in which basic social relationships become fixated as in imprinting (Eibl-Eibesfeldt, 1970, 1973). That is, man is especially responsive to specific environmental influences at an early particular phase of his ontogenetic development.

These facts also apply to language behavior. Comparative linguistics has taught us that man possesses his innate disposition for learning language and is likewise talented for learning in quite special ways (Lenneberg, 1967).

From the viewpoint of phylogenetic adaptation, innate releasing mechanisms of social behavior are mechanisms different from those of language. However, both hold sensory receptors in common and are closely and reciprocally related. For example, visual perception is available not only to social orientation but also to imitation of speech. Therefore, the fact that innate releasing mechanisms have common properties makes the identification of basic disturbances underlying autistic behavior ambiguous and it worked as a factor causing a disarray between cause and effect.

From now, the author will state his own etiology of early childhood autism from the above developmental viewpoint of human ethology. The author thinks that early childhood autism is caused by dysfunction of innate releasing mechanisms resulting from biological deficiencies. Consequently, the author assumes that both, a severe failure to develop social relationships and a specific linguistic impairment, are concurrently and interdependently developed by dysfunction of innate releasing mechanisms from early infancy, accompanied by various cognitive abnormalities such as gaze avoidance and abnormal responses to sensory experiences.

To put it in other way, idiosyncratic imprinting-analogous behavior patterns occur especially in the domain of social behavior and language of an infant. As innate releasing mechanisms as a sort of innate recognistion play a role of activating innate learning dispositions for these behaviors, the distortion of the basic developmental structures is developed and it affects these behaviors of an infant as in imprinting.

However, the model of dysfunction of innate releasing mechanisms, the author believes, is differentiated as follows: (a) dysfunction of innate releasing mechanisms is a central disorder, (b) impairment of innate learning dispositions is central and it additionally causes dysfunction of innate releasing mechanisms because the selectivitiy of innate releasing mechanisms largely depends upon the maturity of innate learning dispositions, (c) both innate releasing mechanisms and innate learning dispositions are impaired. These three types of dysfunction produce a large variety of autism, combined with a different degree of dysfunctions. These would especially affect the types of abnormal imprinting-analogous behavior patterns and the degree of mental retardation of autistic children.

Take for an example, in the former, these would produce the differences ranging from some children continually persisting mute and severe social withdrawal to other children losing their social withdrawal quite early in life. As to the latter, these would produce the differences ranging from severely retarded children to normal IQ children.

From the viewpoint of educational treatment, the individual differences are so great that some children cannot be entirely modified through operant conditioning and other children can easily progress up to approximately the level of a normal child. A peculiar imbalance of IQ-profile of autism could be caused by the relation between the abnormal imprinting-analogous behavior patterns and the degree of intelligence.

Incidentally, there are some autistic children who recover from their social withdrawal quite early in life but still retain the other features of abnormal behavior (Wing, 1976). From the author's experience, the degree of improvement in social withdrawal and linguistic impairment differed in that improvement in social withdrawal progresses better than improvement in linguistic impairment. The author thinks that it is because social behavior is more primary and phylogenetically older than language.

It is the author's opinion that his hypothesis can be verified through the experiment of a destruction of innate releasing mechanisms in social animals, as the presence and function of innate releasing mechanisms in social animals is identified. The destruction should be of two kinds: the destruction of environmental factors through deprivation experiments, and the destruction of biological factors through the use of drugs, mutation and domestication through breeding. Then the behavior differences between social animals whose function of innate releasing mechanisms are destroyed, and early childhood autism must be compared in detail.

Implications for Educational Treatment

Various educational treatments have been conducted for autistic children in recent years. The author would like to suggest that one has to make use of the method of 'aptitude-treatment interaction: ATI' (Cronbach, 1975; Namike & Hayashi, 1977), because the syndrome of autism has a variety of differences among individuals. A way of thinking like Lorenz's theory about the roots of conceptual thought which, on the basis of phylogenetic adaptation, suggests the significance of gestalt perception and voluntary movement can be a productive theoretical base for the educational treatment of autistic children.

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