Chapter 4 - Evaluation of Earlier Theories

1. Criteria of Evaluation

Having reviewed in a more or less objective way the raw facts of autism, we are now in a position to evaluate the various theories presented earlier. There are several basic criteria by which they must be judged. Most important is whether a theory *agrees with the facts*. Is the description of autism accurate, or has it been distorted, for instance, by treating traits that are not present in all cases as if they were universal? Does the actual course of development agree with that proposed by the theory? Obviously, any theory that is contradicted by the facts is unacceptable.

Beyond that, theories must be evaluated for *completeness*. How much of the phenomenon does the theory explain? Does it offer explanations for all the various traits, or does it address only a portion of them? Incompleteness, however, does not invalidate a theory. A partial theory may be a step toward a more complete one. It may need only to be supplemented or modified slightly. A theory that explains even a single trait or a single correlation between traits may contribute to our understanding of the syndrome. And since there are distinct subgroups within the broad range of autism, there may be theories that are relevant to some but not to others.

However, incompleteness may also be an indication that a theory is not correct at all. If it fails to address significant aspects of the syndrome, especially if those aspects are so radically different from those that it does address that there seems to be no way to modify the explanation to include them, then those unanswered questions might indicate that this explanation is entirely in error. Nonetheless, even theories that are ultimately found to be mistaken may provide valuable insights. So although our goal is a unified theory that explains the entire syndrome and applies to all varieties, partial explanations and incorrect theories may still prove valuable.

Theories must also be examined for *uniqueness*. If a particular trait is claimed to cause autism, whether as the primary cause or as one of several links in the chain that produces the syndrome, it must be investigated to determine whether that trait is found in any who are not autistic. If so, the failure to produce autism in those cases must be explained. Conversely, if autism is claimed to necessarily entail a certain trait, that trait must be universal in autism. It may or may not also occur in those who are not autistic, but in autism it must be present without exception.

As we have seen, there are many traits that tend to occur in autism but that are not universal. Since these constitute a large part of the syndrome, it is very important that they be addressed. This is one of the more difficult requirements of a complete theory, because it must account for both correlation and independence.

It is not our purpose to absolutely disprove any of these explanations, although some of them are clearly wrong and none of them are sufficient. It is, rather, to see both the positive and negative in each, and what each can contribute to our understanding of autism.

2. Psychogenic Theories

The psychogenic approach was popular during the first two decades after the discovery of autism, but as the syndrome became better understood numerous facts were found that contradicted it, and it was eventually completely rejected. From the start there were some obvious problems.

2.1 Lack of Correlation between Conditions Claimed to Cause Autism and its Actual Occurrence

Were parental treatment the cause, the number of autistic siblings would be expected to be high. Parents who are cold and unresponsive are generally so to all their children, not just to one. Siblings raised by the same parents in more or less the same way would be expected to develop similarly in those aspects that are derived from the environment. This is clearly not the case. The percentage, while slightly greater than in the general population, is still very low, making it unlikely that early environment, in particular treatment by parents, is the cause. Actual observation of home environment and children's early experiences in most cases of autism did not support the psychogenic explanation either. Many homes were normal, treatment did not differ from that of other children, and there were no traumatic separations.

Proponents of the psychogenic approach were, of course, aware of this problem, and offered various solutions. Since in families in which there were other children the autistic child was often the oldest, some attributed the difference in development to new parents' lack of experience. This, however, could not explain the many autistic children in other positions among siblings. Furthermore, it did not seem reasonable that the difference was one of skill or experience, because even with practice, parents' basic personalities do not change radically in the short time between one child and another.

Conversely, there are many children that grow up in homes in which they are subject to extreme neglect, yet do not become autistic. Indeed, the supposed mistreatment of autistic children is benign by comparison to some of these. In ways, this objection is even stronger than the first. There are documented cases of the sort of neglect that, according to psychogenic theories, would engender feelings of abandonment, helplessness, and threat to life, yet those children, while emotionally damaged in various ways, do not develop the characteristic syndrome of autism.

Victor's theory that autism-producing treatment is the result of a specific relationship between the mother and this particular child due to conditions in the mother's life at the time of birth avoids this problem entirely. Others modified the original theory by attributed autism to a particularly unfortunate combination of accidents in the child's development. Ultimately, the claim that autism was the result of enduring forces had to be abandoned and replaced by explanations involving accidents and other unusual circumstances. Such claims, however, were always eventually reduced to unsupportable speculations. Furthermore, whatever experience was claimed to have produced autism in those cases could be found in other cases in which autism did not occur.

The strong correlation between autism and mental handicap also contradicted psychogenic theories, which were based on the premise of that the autistic child had potentially normal intelligence but was unable to actualise it due to psychological problems. The recognition of autism in severely mentally handicapped children who showed no evidence of normal intelligence in any area made this untenable. These children did not have the cognitive ability necessary for the kind of reasoning upon which psychogenic explanations were based. In such cases, on the contrary, it was clear that there was a

neurological problem, and that autistic behaviour was related to overall mental impairment.

The failure of therapy to cure autism indicates biological, not environmental origin. Were there even isolated instances of cure this would not absolutely disprove psychological origin, since early psychological damage can sometimes be so deep that it becomes practically irreversible. However, consistent failure to completely eradicate the syndrome even where, thanks to superior intelligence, essentially normal behaviour has been achieved, indicates that there is an underlying condition that cannot be changed. On the other hand, the success of therapy to achieve partial improvement in many cases indicates that it is the kind of biological condition that can be modified by experience. *It can therefore be concluded that autism is a complex syndrome of mixed origin. At root there is a biological condition, but development tends to proceed in certain ways, producing traits not directly caused by the root condition, and which are therefore amenable to treatment.*

2.2 The Flaw of Comparing the Child to an Adult

One serious flaw in reasoning to which psychogenic and certain other theories have been prone is the comparison of psychological conditions of children to those of adults. Bettelheim compared the trauma that he believed caused autism and the response that it evoked to those suffered by adults in concentration camps. Victor compared them to those that cause depression. Behaviour of children, however, cannot be compared to that of adults. In an adult, cognition and personality have already developed. There is already a complex system of habits and other patterns of behaviour that has been formed over the course of many years of growth and experience. There is also a conscious world-view and a complex of ideas and expectations. Whatever the adult experiences is evaluated and processed within the context of this great body of firmly established cognitive, emotional, and behavioural mechanisms. Nothing comparable exists in the developing child. The infant has no concepts, no way of structuring experience, no world-view. Most experiences have no particular meaning for the infant and he has no specific responses to them. Those that he does have are in a state of flux, continually being refined, modified and expanded. The distress of an infant upon being put down and left alone is not a concern about the future, a fear the caregiver will not return and that he will be abandoned, for these are concepts that he does not yet have. He has no concept of the future, let alone expectations of what his own future will be like. He has not yet developed a sense of self and others. His crying is a response only to the present, to his current experience, the cessation of the pleasant feelings of being held and fed. An adult can feel alienated from others, but a small child cannot. To be alienated, one must once have been together with them. The child does not achieve an appreciation of these concepts until he develops a sense of self and other. Similarly, deficits in Executive Functions in autism do not resemble those of adults who have suffered brain damage. Even when the same activities are affected, they are not affect in the same way.

2.3 Positive Aspects of the Psychogenic Approach

There are, nonetheless, valuable insights that can be gained from psychogenic explanations, especially involving questions of cognitive and emotional development. Whatever the underlying biological causes of autism are, the autistic state is the product of a long course of development. The autistic child passes through various stages, in some ways similar to those of normal children and in some ways different.

Experience, especially through interaction with other human beings around him, plays an important role in how that development proceeds. Biological explanations have, for the most part, failed to address these factors, disregarding both the developmental process and the current dynamics of the autistic mind at any stage of its development. They have adopted the approach of treating autism as if it were a physical trait like red hair, produced directly by internal processes and in which experience plays little or no role. Typically, they are of the form, "An autistic child has biological condition X, which causes him to think and behave in way A" (the latter being the autistic syndrome as conceived by that theory), rather than a more complex but more appropriate form such as, "An autistic child has biological condition X, which, together with experiences F, G, H causes him in infancy to think and behave in way A; in that state his biological condition X together with further experiences I, J, K causes him to proceed to state B" and so forth. When an autistic child of ten behaves a certain way, it is not because his genes or genetically determined neural structures are directly causing this behaviour, but because they have influenced his development in various ways of which this is the product.

Understanding the dynamics of autism and the factors that affect autistic development has profound implications for therapy. Among the most important questions in designing a therapeutic approach is which aspects of autism are due to heredity and which to environmental influences. This is not simply a matter of accepting certain aspects as innate and unchangeable. It is more a question of "how" than of "what". Understanding the developmental process contributes to determining what kinds of intervention might be effective and to developing appropriate intervention techniques. All of this tends to be neglected by biological theories, but was addressed, though not always correctly, by psychogenic ones.

2.4 Resilience of the Human Personality

Indirectly, the psychogenic theory has drawn our attention to a very important feature of the human organism. By its failure, the psychogenic argument reveals the amazing resilience of the human personality. Contrary to the predictions of the psychogenic theory, infants raised in conditions of neglect and emotional deprivation do not become autistic. The social aspect of the human personality is so sturdy that it persists in spite of experiences that teach it to distrust and withdraw from human beings. On the other hand, for those born with the predisposition for autism, even a warm and responsive environment is not sufficient to engender normal development. So although the human personality is indeed profoundly affected by experience, the basic social nature of the human organism cannot easily be negated. The psychogenic approach committed the error of assuming that the human organism was so fragile that environmental conditions alone could radically alter the normal course of development in this way.

Affirming environmental elements in autistic development should not, however, be taken as placing blame on parents, as was done in some psychogenic theories. An unusual child needs special care. Raised in the normal way it will not develop properly. Without appropriate guidance and training, even the most conscientious of parents rarely know the right thing to do for such children. Even raised in the most ideal environment, the underlying biological factor is always there, so the child will be autistic however he is raised. The question is how that autism expresses itself. Affirming environmental factors means recognising that the way the autistic child and eventually the autistic adult thinks and behaves can be significantly modified by the way he is educated, guided, and treated by others.

3. Early Biological Theories

Unlike the psychogenic theories, which at first seemed plausible, the early biological theories of Rimland, Hermelin and O'Connor, and Delacato immediately presented problems. Though they seemed appropriate for cases of severe or moderate mental handicap, they could not explain cases in which intelligence was normal. Even cases of isolated abilities were difficult to explain. If a child is unable to derive information from the environment, to structure experience or to connect current sensations to memory of past ones, how can he play the piano or assemble jigsaw puzzles? This objection applies to any explanation that attributes autism to neurological deficits that have global effects on behaviour and cognition. It is a particularly serious objection to those theories that suggest deficits that have not themselves actually been observed, but are merely hypotheses offered to explain observed traits.

Such theories could be defended only by limiting the definition of autism to exclude those individuals who were not deficient in these areas. With respect to early biological theories, it meant excluding all those of normal intelligence. But that meant ignoring the fundamental similarities between those whose intelligence was normal and those whose intelligence was not. Given the deep similarities of autistic aloneness and other traits belonging to the three main categories, such an artificial distinction is difficult to justify. More serious, however, is that this division results in the creation of a new condition, similar to autism in all essential ways except for intelligence. We are then left facing the very same problem explaining this new condition that we originally had with respect to autism in general.

4. Theory of Mind

After the psychogenic explanations, the explanation that seemed most promising was that the autistic child suffers from an innate deficit in Theory of Mind. Since this is a specifically social deficit, it is consistent with the centrality of social abnormalities. However, upon closer examination it is found to fail most of the criteria of an acceptable explanation. The first is *completeness*. There are important traits which a deficit in Theory of Mind cannot explain. Though it seems plausible that such a deficit would cause many of the traits in the areas of language and social interaction, it offers no explanation for resistance to change, nor for many of the traits that are not in the three main categories, including sensory abnormalities, weak Central Coherence and deficits in Executive Functions. A primary deficit in Theory of Mind is therefore not adequate to explain the entire syndrome. If, indeed, there is such an innate deficit, it must be accompanied by other innate factors that cause these other traits.

Some proponents of the Theory of Mind explanation minimised this problem by adopting the position that resistance to change was only a peripheral trait, while considering deficits in pretence, which they referred to as "imagination", to be central. In this way, even though there were still traits that could not be explained, they were no longer among the essential parts of the syndrome. This, however, is a deviation from the facts to fit the theory, because autism always involves some kind of resistance to change or repetitive behaviour. Moreover, pretence is not consistently deficient. Otherwise intelligent autistic children do learn to pretend, though later than normal ones, and many pretend not only when prompted but spontaneously as well, though perhaps less than other children. Imagining things to be other than they are is an ability that may be weaker than normal in many autistic children, but is not entirely lacking. Lack of Theory of Mind is therefore at best a partial explanation. There are, however, two more

failures that invalidate it completely.

The second criterion failed is that deficits in Theory of Mind are not *unique* to autism. They are also found, to some extent, in children who are blind or deaf, and are almost always found in those who are mentally handicapped. Deficits in Theory of Mind cannot therefore, in themselves be the cause of autism, since children can have deficits in Theory of Mind without being autistic. Moreover, Theory of Mind is initially lacking in normal children too. Already in infancy autistic children have begun to behave differently from normal ones, even though at that time neither has yet acquired Theory of Mind. Indeed, the kind of social interaction that is lacking in autism is already a central part of the behaviour of normal children before they attain these concepts, so it cannot be dependent upon them. Since at least the early stages of normal behaviour do not require Theory of Mind, early autistic behaviour must have a different cause.

But the most serious challenge to this explanation is that Theory of Mind is not consistently lacking in autism. There are children who are definitely autistic by all accepted criteria, but do not lack Theory of Mind. Numerous experiments have been performed to study Theory of Mind in autism, and all show that for every test of Theory of Mind there is a minority of autistic children that pass. Although statistically Theory of Mind is weaker in autism, and most autistic children do tend to lag behind normal ones in its acquisition, as long as intelligence is in the normal range it is eventually attained.

4.1 Implications of the Failure of the Theory of Mind Explanation

The failure of the Theory of Mind explanation has disturbing implications for the entire modularist paradigm. Social interaction is a central part of human behaviour and cognition, so according to the modularist paradigm there should be a specific module devoted to it, probably a rather large and complex one. Before this deficit was suggested as the cause of autism, proponents of modularism had begun to hypothesise about such a module. In keeping with the premise that abstract concepts cannot be inferred from experience but must be innately granted, innate concepts of emotions, thoughts and volition, together with the ability to interpret observed events in terms of them, were considered to necessarily be part of that module. Evidence of these concepts in the behaviour of apes as well as humans was seen as support of their innateness, since the reasoning power of apes was considered to be insufficient to infer them. But to establish their innateness in humans, the strongest proof would be the existence of individuals in whom they were missing, especially if these were individuals whose cognitive abilities were otherwise intact. The identification of autism as just such a condition was therefore welcomed enthusiastically by the modularist school.

But as great a support as its success would be, its failure would be even more serious a challenge. If the lack of these concepts in autistic children at the age at which they normally appear is taken as evidence of an innate deficit, then later acquisition would indicate that it was possible for a human being who did not have them innately to somehow develop them on his own. If so, such development must be within human capability, in contradiction of the modularist assumption. The question of whether Theory of Mind can be attained in autism is therefore a pivotal question for modularism. Much more is at stake than understanding autism itself.

When, contrary to original expectations, autistic children did not consistently fail tests of Theory of Mind, some explanation had to be found. One approach was to maintain that in those autistic children

who passed the tests, the module was not completely inoperative but only partially defective, that defect being sufficient to cause autism. To support this claim, further experiments were conducted to show that even in the "talented minority" of autistic children who passed, understanding of thoughts of others was inferior to that of normal children. These experiments tested grasp of more advanced mental concepts such as the ability to conceive not only of the thoughts of others about the physical world, but of their thoughts about the thoughts of a third person. Subjects were told complex stories in which one character makes a mistake about the thoughts of another character who himself is making a mistake about reality.

But even on the most difficult tests, there were always some autistic children who passed. Ultimately, it was only those who were also mentally impaired that did not eventually attain normal understanding of thoughts and emotions. Furthermore, success on tests of Theory of Mind was correlated not with severity of autism but with verbal ability. Experimental evidence therefore indicated that in both autistic and normal children, grasp of Theory of Mind increases gradually from infancy, in which it is completely lacking, until complete acquisition in adulthood. The difference between autistic and normal children is that autistic children tend to acquire it more slowly relative to their overall cognitive development. Autism therefore involves a slower rate and perhaps a different course of development, but one that can eventually arrive at the same fundamental concepts, given otherwise adequate reasoning abilities.

The finding that concepts of mind and the ability to interpret the actions of oneself and others in terms of thoughts and feelings increase gradually during the course of childhood is not in itself inconsistent with the modularist paradigm. It need not be interpreted as evidence that they are products of learning and development, since emergence due to gradual maturation of an innate biological module would produce the same pattern. The contradiction is rather in the correlation between Theory of Mind abilities and other areas of intelligence, and, on the other hand, lack of correlation with severity of autistic traits, implying that they are derived from global rather than specifically social abilities. The failure of mentally impaired children who are not autistic to attain the more advanced levels of Theory of Mind, in contrast to the success of intelligent autistic children, implies that these abilities are performed by the same mechanisms that perform other kinds of intelligent behaviour, not by an independent module.

4.2 Responses

Attempts to defend the Theory of Mind explanation deviated from the modularist position in ways reminiscent of the deviations we saw earlier in modifications of psychogenic theories. One was that in the "talented minority" the innate Theory of Mind module, while not deficient, emerges late, and its absence during crucial stages of early development leaves the child permanently impaired. Cognitive and behavioural processes, whose development normally involves mental concepts, develop without them instead, and the product is therefore abnormal. That abnormalcy then becomes fixed, so that even after Theory of Mind emerges, deficits remain. Granting development this crucial role is a radical departure from the modularist position that the essential role of innate mechanisms is constantly being played "on-line", at the time of the performance of the behaviour, not before.

Another approach was to maintain that the Theory of Mind achieved by the talented minority is essentially different from the normal one. While normal children have an innate module that causes them to automatically interpret the behaviour of those around them in terms of mental states, the autistic child does not, so he must construct an artificial simulation. The normal Theory of Mind is automatic and

intuitive, the autistic one analytic and conscious. This explained the observation that intelligent autistic children who succeed under experimental conditions still tend to manifest deficits in Theory of Mind in real life. Their inability to function in high-demand situations the way others do reveals the essential difference between the artificial mechanism and the innate one. Since test situations are standardised and enough time is provided to work the problem out analytically, the artificial mechanism is sufficient, but the split-second decisions that must be made in real-life situations can only be accomplished by the natural one.

To prove this under laboratory conditions, a different sort of experiment was designed, in which subjects were required to infer thoughts and emotions from sparse data. Some involved recognition of emotions on the basis of eye expression alone. Others involved geometric shapes representing human beings interacting with one another. Subjects were required to identify the nature of the interaction on the basis of their motion. It was reasoned that an automatic mechanism would attribute mental states even from minimal evidence (perhaps not sufficient to justify it), while an analytic one, on the contrary, might require more than the minimum logically necessary. The poor performance of intelligent autistic children and adults in these experiments was consistent with the predictions, although here again, performance was correlated with general intelligence and verbal ability, not severity of autism, and here too it improved with age, indicating that even if the mechanism was artificial, it had not reached its limit.

This approach deviates from another of the fundamental principles of modularism, that it is essentially impossible to achieve concepts such as that of Theory of Mind by reasoning, so it is absolutely necessary that they be innately provided. Conceding that even a minority of autistic children are able to construct these concepts undermines the claim that experience never provides an individual with enough information to construct them and therefore the need for an innate mechanism. If they can do it, normal children can too. Furthermore, if the only observable difference between these mechanisms is quantitative, that one functions more quickly than the other, there is no basis for the claim that they are essentially different. Lesser efficiency does not imply essential difference. One automobile may be less efficient than another even though both operate in essentially the same way. The superiority of normal children to autistic ones may mean only that they have constructed better mechanisms for understanding thoughts and emotions, not that theirs are innate.

4.3 Conscious and Automatic Mechanisms

None of these responses were sufficient to save the Theory of Mind hypothesis. On the contrary, the results of these studies showed that autism can exist in spite of normal understanding of mental states. They are, furthermore, entirely hypothetical, there being no evidence either that the damage caused by slow development is irreparable or that the Theory of Mind of intelligence autistic children is essentially different from that of normal ones. There are nonetheless valuable insights that can be gained from them. The distinction between consciously mediated processes and automatic ones is certainly valid. There are some cognitive processes that are conscious and analytic and others that are not, and the latter tend to be faster and more efficient. However, this distinction itself reveals a strong argument against nativism, for there exist skills that are automatic, fast and efficient, yet are clearly not innate but acquired. Skills such as riding a bicycle or typing on a keyboard that are at first performed consciously, if practised sufficiently eventually become automatic. Not only do they become faster and more efficient, they no longer require conscious control. Moreover, they continue to develop and improve after becoming automatic. Whether

the mental mechanisms that are now performing these skills are derived from the original conscious ones or whether they are entirely new and essentially different, their performance is now similar to that of the mechanisms that the modularist school claims to be innate, such as those for recognising and responding to mental states of others. The existence of automatic mechanisms such as these shows that efficient mechanisms can be products of development and need not be innate, proving that differences in efficiency do not imply essentially different origin.

4.4 Skill and Cognition

Another valuable insight that was revealed by these studies was that the enduring difference between intelligent adults who are autistic and those who are not is not a matter of depth or clarity of the concepts of Theory of Mind, but of *skill* at their application. Like the music critic who lacks the musician's skill though his understanding of music may not be inferior, the intelligent autistic person does not lack the normal mental concepts even though in his social interaction he may not always be able to apply them. It is this distinction between *cognition* and *skill* that the nativist argument confuses. The expression "Theory of Mind" refers to cognition, and the question of whether or not one has a Theory of Mind refers to his understanding of these concepts. Performance in real life situations and even on some tests involves not understanding but skill. It is a matter of proficiency. Intelligent autistic people understand the concepts of thoughts and emotions just as normal ones do, even if their skill at applying them is inferior.

5. Executive Functions and Central Coherence

Like the neurological deficits proposed earlier by Rimland and others, deficits in Central Coherence and Executive Functions are not specifically social, making them unlikely candidates for the primary cause of a largely social syndrome. Even though deficits in Central Coherence and Executive Functions could conceivably interfere with language acquisition and grasp of Theory of Mind, when such deficits are sufficiently severe to disable social development they interfere with other areas of cognition as well.

Another problem with ascribing a primary causal role to deficits in Executive Functions is that they do not appear until relatively late, considerably after many symptoms of autism, including deficits in communication and Theory of Mind. With respect to Executive Functions, young autistic children are similar to normal ones. Planning, putting elements into context, and other such abilities are only beginning to develop when the average child starts school. At this point the autistic child's behaviour has already deviated radically in the areas of language and social interaction. This problem, however, is not essentially insurmountable. Late appearance does not necessarily preclude a causal relationship, because underlying deficits might have been present at a much earlier age that, though they were not yet evident in current behaviour, were already affecting development.

Other problems with deficits in Executive Functions and Central Coherence are similar to those involving Theory of Mind. These, too, are not unique to autism. They are found as well in developmental language disorder and other disorders. Adults who are mentally handicapped but not autistic invariably have deficits in these areas, even with respect to social questions. They may be sociable, but they do not interact or empathise on a deep personal level. They can feel sad because someone else is sad, but cannot

understand that the reason for his sadness is that he feels rejected or unfulfilled. They smile, greet and chat, but cannot grasp the larger context in social situations. If they encounter a problem in social interaction, they cannot plan or use strategies to solve it.

On the other hand, like Theory of Mind, Executive Functions and Central Coherence are not entirely lacking in autism. On some tests, autistic children perform as well as normal ones. There is also evidence contradicting the hypothesis that superior ability at tasks involving attention to detail is due to failure to grasp larger patterns. Some who excel at embedded figures and similar tasks have been found to also perform normally on global tasks. It is therefore possible to have these characteristically autistic abilities without lacking the normal ones. And, like Theory of Mind, success at Executive Functions is correlated not with severity of autism but with verbal ability. So although deficits in certain kinds of Executive Functions in autism are well documented, the cause may lie not in the task itself but in other aspects such as social interaction and verbal processing. There has been some experimental evidence to support this hypothesis. When certain experiments are modified so that the social aspect of the test is eliminated by replacing the human examiner by a computer, performance tends to improve.

Unlike early neurological theories, however, tendencies for deficits in Executive Functions and for weak Central Coherence are not speculations but are based on solid experimental data. It is therefore not the fact of deficits in these areas that is in question but their relationship to the core social traits. There is no apparent reason why either one would cause the other. Most of the research has, indeed, avoided this question and addressed the deficits themselves, investigating their exact nature and establishing statistical correlations.

6. Problems with all current theories

Beyond these specific objections, there are others that apply to all current theories.

6.1 Subgroups

As we have seen, while all varieties of autism share certain general traits, there are some ways in which subgroups differ greatly from one another. These differences cannot be overlooked. An adequate theory should explain each of the subgroups, how and why they differ from one another, and how and why they nonetheless bear certain significant similarities. The two questions of *current dynamics*, how various cognitive and behavioural characteristics affect one another, and *aetiology*, what the primary cause is and how it produces the various traits, must be addressed separately for each specific subgroup. Even though we can expect much underlying commonality, there will also necessarily be significant differences. Few of the current theories recognise this need.

6.2 Flaws in Experiments

Neglect of these differences has led to a particularly serious flaw in many experiments. When the group of

autistic subjects is mixed, important differences between them are masked, sometimes producing a false average profile, but more often simply inconclusive results. The fine distinctions that might have been revealed had a narrower group been studied are lost.

Another problem in designing experiments to study autism is finding suitable control groups. To serve as a valid control in any experiment, members of the group must be similar to those being examined except for the factor under consideration. With respect to many questions about autism such a population does not exist, because autistic development does not follow normal patterns. Autistic children cannot be compared to normal children of the same chronological age, since they are behind them in certain ways, nor are they comparable to younger normal children of comparable mental age, because in other ways they are ahead of them. For similar reasons they cannot be compared to mentally handicapped children who are not autistic. The use of control groups consisting of a combination of various types, which has been adopted for want of a better solution, is not truly satisfactory.

Indeed, the course of cognitive development in autism is so radically abnormal that the concepts of IQ and mental age are in themselves problematic. In normal development, various areas of cognition are, by definition, correlated with one another, because the scales are all based on statistical means of normal children. Even though most children do better in some areas than in others, the differences are rarely great, so mental age, which is a composite of a variety of tasks, is a meaningful standard for comparison of individuals. In autism, however, the differences can be vast. Unlike mentally handicapped children who are not autistic, whose scores are uniformly depressed, an autistic child may be considerably above average in some areas and below in others. The composite is representative neither of the child's strengths nor of his weaknesses. It is therefore important to be very careful when using mental age or overall level of development of an autistic child either in comparing him to normal children or in comparing his own abilities in different areas.

7. Conclusion

Although decades of research have increased our understanding of autism considerably, we are still without a comprehensive aetiology. We have found neither the cause nor the relationships between the various traits. It is the failure, after such long and serious effort, to answer these questions, that leads us to suspect that the fundamental theories within which explanations have been attempted are in some way wrong, and that we need to reexamine the foundations of our understanding of human intelligence. Of particular importance is to examine those areas that tend to be impaired in autism and to understand how they function in the normal individual.