Familial Mental Retardation: A Continuing Dilemma

A controversy exists between the general developmental and defect approaches to the problem of etiology.

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The past decade has witnessed renewed interest in the problem of mental retardation. The interest has resulted in vigorous research activity and the construction of a number of theories which attempt an explanation of attenuated intellectual functioning. However, much of the research and many of the theoretical efforts in the area appear to be hampered by a variety of conceptual ambiguities. Much of this ambiguity is due to the very heterogeneity of phenomena included within the rubric of intellectual retardation. A portion of this ambiguity also appears to be the product of many workers' general conceptual orientation to the area of mental retardation.

The typical textbook pictures the distribution of intelligence as normal or Gaussian in nature, with approximately the lowest 3 percent of the distribution encompassing the mentally retarded (see Fig. 1a). A homogeneous class of persons is thus constructed, a class defined by intelligence-test performance which results in a score between 0 and 70. This schema has misled many laymen and students, and has subtly influenced the approach of experienced workers in the area. For if one fails to appreciate the arbitrary nature of the 70-I.Q. cutoff point, it is but a short step to the formulation that all persons falling below this point compose a homogeneous class of "subnormals," qualitatively different from persons having a higher I.Q. The view that mental retardates comprise a homogeneous group is seen in numerous research studies in which comparisons are made between retardates and normal individuals with the two groups defined solely on the basis of an I.Q. classification.

This practice gives rise to a "difference," or "defect," orientation to mental retardation. Such an approach historically included the notion of moral defect and had many origins, ranging from the belief that retardates were possessed by a variety of devils to the empirical evidence of the higher incidence among them of socially unacceptable behaviors, such as crime and illegitimacy. More recently, the notion of defect has referred to defects in either physical or cognitive structures. This defect approach has one unquestionably valid component. There is a sizable group of retardates who suffer from any of a variety of known physical defects. For example, mental retardation may be due to a dominant gene, as in epiloia; to a single recessive gene, as in gargoylism, phenylketonuria, and amaurotic idiocy; to infections, such as congenital syphilis, encephalitis, or rubella in the mother; to chromosomal defects, as in mongolism; to toxic agents, as in retardation caused by radiation in utero, lead poisoning, or Rh incompatibility; and to cerebral trauma.

The diverse etiologies noted above have one factor in common; in every instance, examination reveals an abnormal physiological process. Persons who are retarded as a result of an abnormal physiological process *are* abnormal in the orthodox sense, since they suffer from a known physiological defect. However, in addition to this group, which forms a minority of all retardates, there is the group labeled "familial"—or, more recently, "cultural-familial"—which compromises approximately 75 percent of all retardates. This group presents the greatest mystery and has been the object of the most heated disputes in the area of mental retardation. The diagnosis of familial retardation is made when an examination reveals none of the physiological manifestations noted above, and when retardation of this same type exists among parents, siblings, or other relatives. Several writers have extended the defect notion to this type of retardate as well, although they differ as to what they propose as the specific nature of the defect. On the basis of differences in performance between retardates and normals on some experimental task, rather than on the basis of physiological evidence, they have advanced the view that all retardates suffer from some specifiable defect over and above their general intellectual retardation.

Some order can be brought to the area of mental retardation if a distinction is maintained between physiologically defective retardates, with retardation of known etiology, and familial retardates, with retardation of unknown etiology. For the most part, work with physiologically defective retardates involves investigation into the exact nature of the underlying physiological processes, with prevention or amelioration of the physical and intellectual symptoms as the goal. Jervis (*1*) has suggested that such "pathological" mental deficiency is primarily in the domain of the medical sciences, whereas familial retardation represents a problem to be solved by behavioral scientists, including educators and behavioral geneticists. Diagnostic and incidence studies of these two types of retardates have disclosed certain striking differences. The retardate having an extremely low I.Q. (below 40) is almost invariably of the physiologically defective type. Familial retardates, on the other hand, are almost invariably mildly retarded, usually with I.Q.'s above 50. This difference in the general intellectual level of the two groups of retardates is an important empirical phenomenon that supports the two-group approach to mental retardation, the approach supported in this article.

A Two-Group Approach

Hirsch (*2*) has asserted that we will not make much headway in understanding individual differences in intelligence, and in many other traits, unless we recognize that, to a large degree, such differences reflect the inherent bi-

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ological properties of man. We can all agree that no genotype spells itself out in a vacuum, and that the phenotypic expression is finally the result of environment interacting with the genotype. However, an appreciation of the importance of genetic differences allows us to bring considerable order to the area of mental retardation.

We need simply to accept the generally recognized fact that the gene pool of any population is such that there will always be variations in the behavioral or phenotypic expression of virtually every measurable trait or characteristic of man. From the polygenic model advanced by geneticists, we deduce that the distribution of intelligence is characterized by a bisymmetrical bell-shaped curve, which is characteristic of such a large number of distributions that we have come to refer to it as the normal curve. With the qualification noted below, this theoretical distribution is a fairly good approximation of the observed distribution of intelligence. In the polygenic model of intelligence (see *2–4*), the genetic foundation of intelligence is not viewed as dependent upon a single gene. Rather, intelligence is viewed as the result of a number of discrete genetic units. (This is not to assert, however, that single gene effects are never encountered in mental retardation. As noted above, certain relatively rare types of mental retardation are the product of such simple genetic effects.)

Various specific polygenic models have been advanced which generate theoretical distributions of intelligence that are in keeping with observed distributions (*3, 5, 6*). An aspect of polygenic models of special importance for the two-group approach is the fact that they generate I.Q. distributions of approximately 50 to 150. Since an I.Q. of approximately 50 appears to be the lower limit for familial retardates, it has been concluded (*4, 5, 7*) that the etiology of this form of retardation reflects the same factors that determine "normal" intelligence. With this approach, the familial retardate may be viewed as normal, where "normal" is defined as meaning an integral part of the distribution of intelligence that we would expect from the normal manifestations of the genetic pool in our population. Within such a framework it is possible to refer to the familial retardate as less intelligent than other normal manifestations of the genetic pool, but he is just as integral a part of the normal distribution as are the 3 percent of the population whom we view as superior, or the more numerous group of individuals whom we consider to be average (*8*).

The two-group approach to mental retardation calls attention to the fact that the second group of retardates, those who have known physiological defects, represents a distribution of intelligence with a mean which is considerably lower than that of the familial retardates. Such children, for the most part, fall outside the range of normal intelligence—that is, below I.Q. of 50—although there are certain exceptions. Considerable clarity could be brought to the area of mental retardation through doing away with the practice of conceptualizing the intelligence distribution as a single, continuous, normal curve. Perhaps a more appropriate representation of the empirical distribution of intelligence would involve two curves, as Fig. 1b illustrates. The intelligence of the bulk of the population, including the familial retardate, would be depicted as a normal distribution having a mean of 100, with lower and upper limits of approximately 50 and 150, respectively. Superimposed on this curve would be a second, somewhat normal distribution having a mean of approximately 35 and a range from 0 to 70. (That the population encompassed by the second curve in Fig. 1b extends beyond the 70-I.Q. cutoff point is due to the fact that a very small number of individuals with known defects—for example, brain damage—may be found throughout the I.Q. continuum.) The first curve would represent the polygenic distribution of intelligence; the second would represent all those individuals whose intellectual functioning reflects factors other than the normal polygenic expression—that is, those retardates having an identifiable physiological defect. This two-group approach to the problem of mental retardation has been supported by Penrose (*4*), Roberts (*9*), and Lewis (*10*). The very nature of the observed distribution of I.Q.'s below the mean, especially in the range 0 to 50 (see Fig. 1c), seems to demand such an approach. This distribution, in which we find an overabundance of individuals at the very low I.Q. levels, is exactly what we would expect if we combined the two distributions discussed above, as is the general practice.

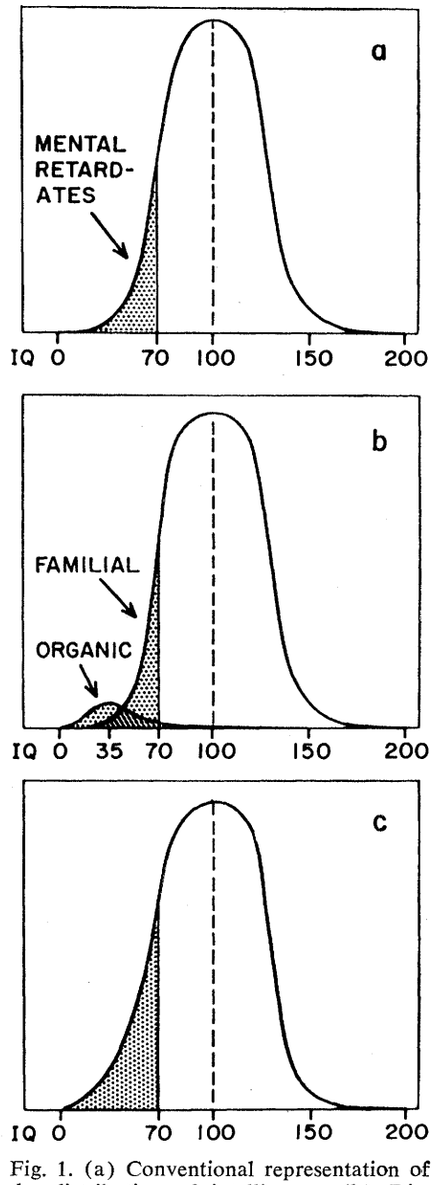

Fig. 1. (a) Conventional representation of the distribution of intelligence. (b) Distribution of intelligence as represented in the two-group approach. (c) Actual distribution of intelligence. [After Penrose (*4*)]

Limitations of space prevent consideration here of the controversy concerning the role of environmental factors in the etiology of familial retardation. Although such factors cannot be ignored by the serious student of mental retardation, the general dispute, discussed below, between adherents of the defect theory and of the general developmental theory can be examined somewhat independently of the environmental issue. That there will always be a distribution of a particular shape is a conclusion inherent in the polygenic argument, but the absolute amounts of intelligence represented by the various points on the distribution would still depend in large part on environmental factors.

Developmental versus Defect Orientation

Once one adopts the position that the familial mental retardate is not defective or pathological but is essentially a normal individual of low intelligence, then the familial retardate no longer represents a mystery but, rather, is viewed as a particular manifestation of the general developmental process. According to this approach, the familial retardate's cognitive development differs from that of the normal individual only in respect to its rate and the upper limit achieved. Such a view generates the expectation that, when rate of development is controlled, as is grossly the case when groups of retardates and normals are matched with respect to mental age, there should be no difference in formal cognitive processes related to I.Q. Stated somewhat differently, this means that the familial retardate with a chronological age of 10, an I.Q. of 70, and thus a mental age of 7, would be viewed as being at the same developmental level intellectually as a child with a chronological age of 7 and an I.Q. of 100.

In contrast, according to the defect orientation, all retardates suffer from a specific physiological or cognitive defect over and above their slower general rate of cognitive development. This view generates the expectation that, even when the rate of cognitive development is controlled, as in the situation where mental ages are matched, differences in intellectual functioning which are related to I.Q. will be found. On their face, the repeated findings of differences in performance between groups of normals and retardates matched as to mental age have lent credence to the defect theory and have cast doubt on the validity of the developmental theory.

The developmental theorist's response to these frequently reported differences has been to point out that performance on any experimental task is not inexorably the product of the subject's cognitive structure alone but reflects a variety of emotional and motivational factors as well. To the developmentalist, then, it seems more reasonable to attribute differences in performance between normals and retardates of the same mental age to motivational differences which do not inhere in mental retardation but are, rather, the result of the particular histories of the retarded subjects.

It should be noted that most theories in the area of mental retardation are basically defect theories. These differ among themselves, however. A major difference involves the theoretician's effort to relate the postulated defect to some specific physiological structure. The theoretical language of some defect theoreticians is explicitly physiological, that of others is nonphysiological, while that of others remains vague. Particular defects that have been attributed to the retarded include the following: relative impermeability of the boundaries between regions in the cognitive structure (*11, 12*); primary and secondary rigidity caused by subcortical and cortical malformations, respectively (*13*); inadequate neural satiation related to brain modifiability or cortical conductivity (*14*); malfunctioning disinhibitory mechanisms (*15*); improper development of the verbal system, resulting in a dissociation between verbal and motor systems (*16, 17*); relative brevity in the persistence of the stimulus trace (*18*); and impaired attention-directing mechanisms (*19*).

Where the hypothesized defect is an explicitly physiological one, it would appear to be a simple matter to obtain direct evidence that the defect does or does not exist. Such evidence would come from biochemical and physiological analyses as well as from pathological studies of familial retardates. A number of such studies have, of course, been carried out. Although there is an occasional report of some physical anomaly, the bulk of the evidence has indicated that the familial retardate does not suffer from any gross physiological defects. Indeed, if such evidence were readily available the defect theorist would cease relying on the more ambiguous data provided by studies of molar behavior. Failure to find direct evidence of a physiological defect in familial retardates has not deterred, and should not deter theorists from postulating such defects.

In spite of the negative physiological evidence, workers such as Spitz (*14*) maintain that all retardates, including familial retardates, are physically defective, and that our failure to discover defects in familial retardates is due to the relatively primitive nature of our diagnostic techniques. This view is bolstered by Masland (*20*), who has also noted the inadequacies of such techniques. It is perfectly legitimate for the defect theorist to assert that, although not at present observable, the physical defect that causes familial retardates to behave differently from normals of the same mental age will someday be seen. These theorists operate very much as the physicists of a not-too-distant era did when they asserted that the electron existed even though it was not directly observable. Analogously, defect theorists in the area of mental retardation undertake to validate the existence of a defect by first asserting that it should manifest itself in particular phenomena—that is, in particular behaviors of the retarded—and then devising experiments in which, if the predicted behavior is observed, the existence of the hypothesized defect is confirmed. Not only is this approach legitimate but, as noted above, it has become increasingly popular as well. A relatively comprehensive review of the literature emanating from the general defect position is now available (*21*). In the following paragraphs I briefly summarize the major defect positions.

An influential defect position is that of the Russian investigator A. R. Luria (*16*), whose work has now also influenced investigators in England and the United States. In the Soviet Union no distinction is made between retardates having known organic impairment and that larger group whose retardation is of unknown etiology, nor are genetic or cultural factors considered to be determinants of mental retardation. All grades of mental retardation are attributed to central-nervous-system damage believed to have occurred initially during the intrauterine period or during early childhood. Thus the diagnosis of mental retardation necessarily involves specification of a defect in some neurophysiological system; in fact, in the Soviet Union, professionals who work with the mentally retarded are called "defectologists."

Luria's interest in defective functioning appears to be an outgrowth of his more basic concern with the development of the higher cognitive processes in man. The influence of both Vygotsky and Pavlov may be seen in his work, which has been primarily concerned with the highly intricate development of the role of speech and language in regulating the child's behavior. In his comparisons between normal and retarded children, Luria has demonstrated that the behavior of retardates resembles that of chrono-

logically younger normal children in that verbal instructions do not result in smooth regulation of motor behavior. Luria has found that retarded subjects have considerable difficulty with tasks requiring verbal mediation. Thus, Luria has inferred that the major defect in the retarded child involves an underdevelopment or a general "inertness" of the verbal system, and a dissociation of this system from the motor or action system. This dissociation is vaguely conceptualized as resulting from a disturbance in normal cortical activity.

The view that the behavior of a retardate resembles that of a chronologically younger child is, of course, consistent with the general developmental position. However, several English and American investigators (see, for example, *17* and *22*) have demonstrated that, even with mental age level controlled, retardates have more difficulty on tasks requiring verbal mediation than normal subjects have. On the other hand, other such investigations have failed to provide support for Luria's position (*23*). To date, findings related to this position can best be described as equivocal.

Another major defect position is that of Herman Spitz (*14*), who has extended the Köhler-Wallach (*24*) cortical satiation theory to the area of mental retardation. According to Spitz, all retardates suffer from inadequate neural or cortical functioning; the inadequacy is best characterized by a certain sluggishness, or less-than-normal modifiability, in the functioning of cortical cells. Thus, Spitz believes that in retardates it takes longer to induce temporary, as well as permanent, electrical, chemical, and physical changes in stimulated cortical cells, and furthermore, that once such a change is produced, it is less readily modified than in the case of normal persons.

Spitz's evidence in support of his theory has come primarily from comparisons of the performance of retardates and normals of the same chronological age on a variety of perceptual tasks—for example, figural aftereffects and Necker-cube reversals. The heuristic value of Spitz's position may be seen in his recent efforts to extend his postulates beyond the visual perception area and employ them to generate specific predictions concerning the phenomena of learning, transposition, generalization, and problem solving. The evidence in favor of Spitz's position is far from clear-cut, however. Spivack (*25*) has pointed out that Spitz's findings are in marked contrast to those of other investigators. The very nature of many of Spitz's measures—for example, a verbal report—raises the troublesome issue of how well they reflect the perceptual responses being investigated. It should be noted that, in respect to this point as well as to other criticisms, Spitz himself has become one of the most cogent critics of his own efforts.

Many of Spitz's findings could be encompassed by the general developmental position. The developmental theorist would argue that it is not surprising that one gets different results for normals and for retardates matched with respect to chronological age, since such groups are at different developmental levels (as defined by mental age). One would be tempted to say that Spitz's work has little relevance to the issue of whether familial retardates suffer from a defect over and above their slower and more limited rate of cognitive development. However, Spitz has been quite explicit in his views that the differences he obtains are not developmental phenomena but reflect a physical deficit that should manifest itself even in comparisons with normal subjects matched in mental age to the retardates.

Ellis (*18*) has also advanced the view that the retardate is basically different from the normal individual and that this difference is a result of central-nervous-system pathology from which all retardates suffer. Ellis views this central-nervous-system pathology as producing a short-term memory deficit which, in turn, underlies the inadequacy of much of the retardate's behavior. The theoretical model presented by Ellis includes two major constructs, stimulus trace and neural integrity.

The stimulus trace, the mechanism underlying short-term memory functions, is conceptualized as a neural event or response which varies with the intensity, duration, and meaning of the stimulus situation confronting the subject. The stimulus-trace construct is thus anchored to stimulus characteristics on the one hand and to the subject's responses to these characteristics on the other. The neural-integrity construct is conceptualized as the determinant of the nature of stimulus-trace activity, and is defined by "measures of behavioral adequacy." The typical measure of neural integrity employed by Ellis is the I.Q. Thus, a person of low I.Q. is said to suffer from a lack of neural integrity. This lack, in turn, delimits or restricts stimulus-trace activity, and such restriction results in a variety of inadequate behaviors.

In support of his theory, Ellis has noted findings from numerous experiments involving short-term retention phenomena. These include studies on serial learning, delayed-reaction tasks, fixed-interval operant behavior, electroencephalographic investigations, reaction time, and factor analyses of the WISC test (the Wechsler Intelligence Scale for Children), as well as several studies of discrimination learning in brain-damaged animals (see *18*). In respect to his own experimental tests, Ellis's reliance on the I.Q. as the measure of neural integrity has produced two types of comparisons: comparison of retardates and normals of the same chronological age and comparison of retardates and normals of the same mental age. In either comparison Ellis's model would predict that the retardates would be inferior on tasks involving short-term retention, due to their lower I.Q. In general, the findings obtained with groups matched as to chronological age have supported Ellis's position, while those obtained with groups matched as to mental age have not.

It should be noted that the demonstration that retardates do less well than normals of the same chronological age on tasks requiring short-term memory is a somewhat circular undertaking. It is circular to the extent that a deficit in short-term memory would influence the I.Q. score itself through its effect on certain of the intelligence subtests—for example, the digit-span test. Again, it should be emphasized that the discovery of a difference between normals and retardates of the same chronological age is just as amenable to a general developmental interpretation as to the view that all retardates suffer from central-nervous-system pathology, since the mental age of such retardates is necessarily lower than that of normal subjects in the control group.

Perhaps the oldest of the more influential defect positions is the Lewin-Kounin (*11, 12*) formulation that familial retardates are inherently more "rigid" than normal individuals of the same mental age. This position differs from the others discussed above in that the defect is conceptualized as inhering in a hypothesized cognitive structure

without reference or reduction to any specific physiological entities. By the term *rigidity*, Lewin and Kounin were referring not to behaviors, as such, but rather to characteristics of the cognitive structure. These theorists felt that the essential defect, in retardation, was the lowered capacity for dynamic rearrangement in the "psychical system." This "stiffness" in cognitive functioning was conceptualized as being due to the relative impermeability of the boundaries between cells or regions of the cognitive structure. *Rigidity*, then, referred primarily to the nature of these boundaries, and to the resulting degree of communication or fluidity between regions.

Principal support for this position was contained in a series of experiments conducted by Kounin (*11*), in which he found differences between familial retardates and normals of the same mental age on a variety of tasks involving transfer phenomena, sorting, and concept-switching. Although the Lewin-Kounin position continues to receive some support (*26*), a fairly sizable amount of work (*27, 28*) now indicates that the differences discovered by Kounin between retardates and normals of the same mental age were due to differences in motivational variables rather than to an inherent cognitive rigidity of the retardate.

Lewin and Kounin appear to be the only defect theorists who have dealt adequately with the problem of etiology, which becomes a crucial issue in the controversy over the two theories. Their formulation was limited to familial retardates, and only such retardates were employed in Kounin's experiments. The other defect theorists have tended to argue that the distinction between familial and organic retardates is misleading, and, as a result, they have used groups of retardates of both types in their experiments. This presents an almost insurmountable problem when one attempts to evaluate the degree to which any uncovered differences in behavior support the major theoretical premise which underlies most defect approaches. This premise, clearly seen in the work of Luria, Spitz, and Ellis, is that all retardates, familials and organics alike, suffer from some specifiable defect. However, until the etiological issue is attended to in the research design, there is no way of assessing how much of the revealed difference between normals and retardates of the same mental age is a product of the gross organic pathology known to exist in the organic retardates included in the retarded group and how much is a product of the defect thought by the defect theorists to exist in all retardates.

The general developmental approach is applicable only to the familial retardate, and this approach does not speak to the issue of differences discovered between normal children and organic retardates. The developmental theorist also believes that, even when a difference in behavior is found between normals and familial retardates of the same mental age, it need not be attributed to any defect which inheres in familial mental retardation. Such differences are viewed as the possible outcome of differences in a variety of motivational factors which exist between the two groups. A sampling of the literature which lends credence to this view follows.

Motivational and Emotional Factors

The view of those of us who believe that many of the reported differences between retardates and normals of the same mental age are a result of motivational and emotional differences which reflect differences in environmental histories does not imply that we ignore the importance of the lower intelligence per se. In some instances the personality characteristics of the retarded individual will reflect environmental factors that have little or nothing to do with intellectual endowment. For example, many of the effects of institutionalization may be constant, regardless of the person's intelligence level. In other instances we must think in terms of an interaction; that is, a person with low intellectual ability will have certain experiences and develop certain behavior patterns differing from those of a person with greater intellectual endowment. An obvious example of this is the greater amount of failure which the retardate typically experiences. What must be emphasized is the fact that the behavior pattern developed by the retardate as a result of such a history of failure may not differ in kind or ontogenesis from patterns developed by an individual of normal intellect who, because of some environmental circumstance, also experiences an inordinate amount of failure. By the same token, if the retardate can somehow be guaranteed a history of greater success, we would expect his behavior to be more normal, regardless of his intellectual level. Within this framework, I now discuss several of the personality factors which have been known to influence the performance of the retarded.

It has become increasingly clear that our understanding of the performance of the institutionalized familial retardate will be enhanced if we consider the inordinate amount of social deprivation these individuals have experienced before being placed in institutions (*29, 30*). A series of recent studies (*30–34*) has indicated that one result of such early deprivation is a heightened motivation to interact with a supportive adult. These studies suggest that, given this heightened motivation, retardates exhibit considerable compliance with instructions when the effect of such compliance is to increase or maintain the social interaction with the adult. These findings would appear to be consistent with the often-made observation that the retarded seek attention and desire affection (*35, 36*).

Recent findings suggest that the perseveration so frequently noted in the behavior of the retarded is primarily a function of these motivational factors rather than a result of inherent cognitive rigidity, as suggested by Lewin (*12*) and Kounin (*11*). Evidence is now available indicating (i) that the degree of perseveration is directly related to the degree of deprivation the individual experienced before being institutionalized (*30*), and (ii) that institutionalized children of normal intellect are just as perseverative as institutionalized retardates, while noninstitutionalized retardates are no more perseverative than noninstitutionalized children of normal intellect (*31, 32*).

Although there is considerable evidence that social deprivation results in a heightened motivation to interact with a supportive adult, it appears to have other effects as well. The nature of these effects is suggested in observations of fearfulness, wariness, or avoidance of strangers on the part of retardates, or of suspicion and mistrust (*36, 37*). The experimental work done by Zigler and his associates on the behavior of institutionalized retarded individuals has indicated that social deprivation results in both a heightened motivation to interact with supportive adults (a positive-reaction tendency) and a wariness of doing so (a negative-reaction tendency). The construct of a

negative-reaction tendency has been employed to explain certain differences between retardates and normals reported by Kounin, differences that have heretofore been attributed to the greater cognitive rigidity of retarded individuals. For instance, it has been demonstrated (*38*) that, once the institutionalized familial retardate's wariness has been allayed, he becomes much more responsive than the normal individual to social reinforcement. Thus, a motivational rather than a cognitive factor would seem to underlie certain rather mysterious behavioral phenomena frequently observed in familial retardates—for example, a tendency to persist longer on the second of two highly similar tasks than on the first.

Both positive- and negative-reaction tendencies have been recently investigated in a series of studies, with children of normal intellect (*39*), directed at further validation of the "valence position." Stated most simply, this position asserts that the effectiveness of an adult as a reinforcing agent depends upon the valence he has for the particular child whose behavior is being reinforced. (An adult's valence for a child refers to the degree to which that adult is sought or avoided by the child.) This valence is determined by the child's history of positive and negative experiences with adults. The studies noted above have produced considerable evidence that prior positive contacts between the child and the adult increase the adult's effectiveness as a reinforcer, while negative contacts decrease it. If the experimentally manipulated negative encounters in these experiments are viewed as experimental analogs of encounters institutionalized retardates actually have experienced, then the often-reported reluctance of such children to interact with adults and their wariness of such encounters become understandable. Thus it would appear that their relatively high negative-reactive tendency motivates them toward behaviors, such as withdrawal, that reduce the quality of their performance to a level lower than that which one would expect on the basis of their intellectual capacity alone.

Another factor frequently mentioned as a determinant in the performance of the retarded is their high expectancy of failure. This failure expectancy has been viewed as an outgrowth of a lifetime characterized by confrontations with tasks with which they are intellectually ill-equipped to deal. The work of Cromwell and his colleagues (*40*) has lent support to the general proposition that retardates have a higher expectancy of failure than normals have, and that this results in a style of problem-solving in which the retardate is much more highly motivated to avoid failure than to achieve success. However, the results of experimental work with retardates to investigate the success-failure dimension are still somewhat inconsistent, suggesting that even such a relatively simple proposition as this one is in need of further refinement.

Recent studies (*31, 33, 41*) have indicated that the many failures experienced by retardates generate a cognitive style of problem-solving characterized by outer-directedness. That is, the retarded child comes to distrust his own solutions to problems and therefore seeks guides to action in the immediate environment. This outer-directedness may explain the great suggestibility so frequently observed in the retarded child. Evidence has now been presented indicating that, relative to normals of the same mental age, the retarded child is more sensitive to verbal cues from an adult, is more imitative of the behavior of adults and of his peers, and does more visual scanning. Furthermore, certain findings (*31*) suggest that the noninstitutionalized retardate is more outer-directed in his problem solving than the institutionalized retardate is. This makes considerable sense if one remembers that the noninstitutionalized retardate lives in an environment that is not adjusted to his intellectual shortcomings and, therefore, probably experiences more failure than the institutionalized retardate.

Another nonintellective factor important in understanding the behavior of the retarded is the retardate's motivation to obtain various types of reinforcement. The social-deprivation work discussed indicates that retardates have an extremely strong desire for attention, praise, and encouragement. Several investigators (*40, 42*) have suggested that, in normal development, the effectiveness of attention and praise as reinforcers diminishes with maturity and is replaced by the reinforcement inherent in the awareness that one is correct. This latter type of reinforcer appears to serve primarily as a cue for self-reinforcement.

Zigler and his associates (*27, 43, 44*) have argued that various experiences in the lives of the retarded cause them to care less about being correct simply for the sake of correctness than normals of the same mental age. In other words, these investigators have argued that the position of various reinforcers in the reinforcer hierarchies of normal and of retarded children of the same mental age differ.

Clearest support for the view that the retardate cares much less about being correct than the middle-class child of normal intellect does is contained in a study by Zigler and deLabry (*43*). These investigators found, as Kounin (*11*) did, that when the only reinforcement was the information that the child was correct, retardates were poorer on a concept-switching task than middle-class normal children of the same mental age. However, when Zigler and deLabry added another condition, reward with a toy of the child's choice for concept-switching, they found that the retardates performed as well as the middle-class normal children. Since the satisfaction of giving the correct response is the incentive typically used in experimental studies, one wonders how many of the differences in performance found between retardates and normals are actually attributable to differences in capacity rather than to differences in the values such incentives may have for the two types of subjects.

Much of this work on motivational and emotional factors in the performance of the retarded is very recent. The research on several of the factors discussed is more suggestive than definitive. It is clear, however, that these factors are extremely important in determining the retardate's level of functioning. This is not to assert that these motivational factors cause familial mental retardation but to say, rather, that they lead to the retardate's behaving in a manner less effective than that dictated by his intellectual capacity. An increase in knowledge concerning motivational and emotional factors and their ontogenesis and manipulation would hold considerable promise for alleviating much of the social ineffectiveness displayed by that rather sizable group of persons who must function at a relatively low intellectual level.

Summary

The heterogeneous nature of mental retardation, as well as certain common practices of workers in the area, has resulted in a variety of conceptual am-

biguities. Considerable order could be brought to the area if, instead of viewing all retardates as a homogeneous group arbitrarily defined by some I.Q. score, workers would clearly distinguish between the group of retardates known to suffer from some organic defect and the larger group of retardates referred to as familial retardates. It is the etiology of familial retardation that currently constitutes the greatest mystery.

A number of authorities have emphasized the need for employing recent polygenic models of inheritance in an effort to understand the familial retardate. While appreciating the importance of environment in affecting the distribution determined by genetic inheritance, these workers have argued that familial retardates are not essentially different from individuals of greater intellect, but represent, rather, the lower portion of the intellectual curve which reflects normal intellectual variability. As emphasized by the two-group approach, retardates with known physiological or organic defect are viewed as presenting a quite different etiological problem. The familial retardate, on the other hand, is seen as a perfectly normal expression of the population gene pool, of slower and more limited intellectual development than the individual of average intellect.

This view generates the proposition that retardates and normals at the same general cognitive level—that is, of the same mental age—are similar in respect to their cognitive functioning. However, such a proposition runs headlong into findings that retardates and normals of the same mental age often differ in performance. Such findings have bolstered what is currently the most popular theoretical approach to retarded functioning—namely, the view that all retardates suffer from some specific defect which inheres in mental retardation and thus makes the retardate immutably "different" from normals, even when the general level of intellectual development is controlled. While these defect or difference approaches, as exemplified in the work of Luria, Spitz, Ellis, and Lewin and Kounin, dominate the area of mental retardation, the indirect, and therefore equivocal, nature of the evidence of these workers has generated considerable controversy.

In contrast to this approach, the general developmental position has emphasized systematic evaluation of the role of experiential, motivational, and personality factors. As a central thesis, this position asserts that performance on experimental and real-life tasks is never the single inexorable product of the retardate's cognitive structure but, rather, reflects a wide variety of relatively nonintellective factors which greatly influence the general adequacy of performance. Thus, many of the reported behavioral differences between normals and retardates of the same mental age are seen as products of motivational and experiential differences between these groups, rather than as the result of any inherent cognitive deficiency in the retardates. Factors thought to be of particular importance in the behavior of the retardate are social deprivation and the positive- and negative-reaction tendencies to which such deprivation gives rise; the high number of failure experiences and the particular approach to problem-solving which they generate; and atypical reinforcer hierarchies.

There is little question that we are witnessing a productive, exciting, and perhaps inevitably chaotic period in the history of man's concern with the problem of mental retardation. Even the disagreements that presently exist must be considered rather healthy phenomena. These disagreements will unquestionably generate new knowledge which, in the hands of practitioners, may become the vehicle through which the performance of children, regardless of intellectual level, may be improved.

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