Towards a Balanced Account of Autism Etiology

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Drash and Tudor describe six sets of reinforcement contingencies which may be present in the environments of some children eventually diagnosed with autism and suggest that these contingencies account for the etiology of "autistic" behaviors. Nevertheless, merely observing such contingencies in the environments of these children is insufficient to establish a positive correlation between the contingencies and "autistic" behaviors, let alone a causal relationship. To demonstrate a positive correlation, it is necessary to present evidence that the relevant contingencies are present more often in the environments of children exhibiting these behaviors than in the environments of children not exhibiting these behaviors. This condition has not been met, since no evidence to the effect that such contingencies are absent in the environments of typical children or children with disabilities other than autism has been presented. In fact, the opposite appears to be true, as is argued in the present commentary. It appears that Drash and Tudor's account of autism etiology is incomplete in that it neglects the role of unlearned differences between children and their possible interactions with the social environment in shaping "autistic" behaviors. Despite the misconception held by some that behavior analysis is a radically environmental approach, unlearned differences may be discussed within a behavioral framework. A "completely behavioral" account may discuss such differences in terms of susceptibility to reinforcement or punishment, speed of conditioning, or other unlearned characteristics which are potentially testable.

Drash and Tudor propose six sets of reinforcement contingencies which purportedly account for the etiology of behaviors which may lead to an autism diagnosis. In contrast to the behavioral theories of autism etiology proposed by Lovaas & Smith (1989), Koegel et al. (1994), Spradlin & Brady (1999), and Bijou & Ghezzi (1999), the authors argue that their account is "completely behavioral" in that it explains the etiology of autism without appealing to hypothetical neurobiological variables. They suggest that autism may be viewed as a contingency-shaped disorder of verbal behavior rather than a neurobiological disability or disease entity. According to the authors, this purely environmental perspective may promote earlier intervention: Identifying and correcting defective reinforcement contingencies in effect from birth to 18 to 24 months may prevent behavior patterns from developing that may eventually lead to a diagnosis of autism. In this way, it may be possible to "prevent autism."

The existence of the six sets of contingencies described by Drash & Tudor is quite plausible. Behavioral consultants often evaluate these and other contingencies when children

and adults with developmental disabilities are referred for services due to their behavioral excesses or deficits. After the contingencies which shape and maintain challenging behaviors and fail to support adaptive behaviors have been identified via functional analysis methodology, behavioral interventions are developed and implemented, and more socially acceptable and functional repertoires are often established. Nevertheless, the mere presence of one or more sets of contingencies described by Drash and Tudor seems insufficient to demonstrate a positive correlation between these contingencies and "autistic" behaviors, let alone a causal relationship.

To establish a positive correlation, it is necessary to show that the relevant sets of contingencies are often present in the environments of individuals exhibiting these behaviors and not in the environments of individuals not exhibiting these behaviors. Drash and Tudor assert that many of the children who were evaluated in their autism clinic were exposed to these sets of contingencies, but fail to address the issue of whether such contingencies might occur with other populations of individuals.

It appears that all of the sets of contingencies described by the authors may also occur with individuals who have not been diagnosed with autism. For instance, *reinforcement for aversive vocal manding such as crying or screaming, or other avoidance behaviors that*

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may be incompatible with acquiring age-appropriate verbal behavior occurs with virtually all infants; in fact, crying/screaming becomes the child's first form of verbal behavior, as noted by the authors. Such behavior may function to obtain items that currently function as reinforcement or to escape or avoid aversive events. Unless an infant (or young child) who cannot yet speak is taught to mand via manual signs, gestures, or PECS, crying/screaming may be the only verbal response form available. The authors claim that a strong repertoire of aversive vocal manding may be established if caregivers continue to reinforce such behavior without beginning to prompt and/or shape age-appropriate vocal mands. Failing to explicitly teach initial verbal behavior appears to be the rule rather than the exception, however. In their longitudinal study of early language acquisition, Hart and Risley (1995) noted:

We were astonished to see all the parents simply wait for their children to say words. They prompted, "Say 'bye'," or, "Say 'milk'," but they never coached or held out for a word; a parent waved a child's arm or gave a child a cup without seeming to expect words before the child began to say one or two words spontaneously. As if learning to talk was much the same as learning to walk, the parents just let their children practice babbling, apparently sure that words, like pulling themselves up, would appear in due time. When the children began to say words, we were delighted rather than surprised at how enthusiastically parents took up repeating their children's words and prompting the children to say more." (p. 55)

These findings support the view that carefully designed interventions are generally not needed for typical children to begin to acquire verbal behavior. Less than ideal reinforcement contingencies and informal teaching conditions are often in effect, yet most children develop normally. In contrast, it is well-documented that children diagnosed with autism fail to learn from everyday environments and require specialized behavioral instruction to acquire verbal, social, and other adaptive skills (Green, 1996, p. 30; Lovaas, 2003, pp. 7–11). Although early intensive behavioral intervention is the treatment of choice in remediating "autistic" behaviors, it does not follow that the absence of this intervention caused the behaviors. If it

did, the percentage of children displaying such behaviors would be very high, since most children do not receive this intervention.

A second set of contingencies, *extinction of verbal behavior* (i.e., failing to actively prompt, respond to, and reinforce a child's vocal utterances) is also said to account for the verbal delays characteristic of autism. The authors cite Hart and Risley's (1995) longitudinal research which reported major differences in children's verbal repertoires at the age of three which were positively correlated with the amount and complexity of verbal interactions to which they were exposed. Nevertheless, all of the children studied by Hart and Risley acquired functional verbal repertoires and none were diagnosed with autism or any other developmental disability, although the children of welfare parents received much less verbal interaction and their language acquisition was delayed, compared with the other children in the study. As explained by Hart and Risley:

We saw differences in how much parents talked to their children associated with differences in all the other relative advantages of SES.... Yet, despite these differences, all 42 children learned to talk. All the parents had apparently provided whatever amount of experience was necessary for the children to become effective users of the language. The family experience that gave rise to differences in rate of vocabulary growth must lie beyond whatever was essential to basic competencies. (p. 71)

A third set of contingencies, *non-suppression of disruptive behavior and failure to establish early verbal instructional control and compliance*, appears to be a common problem, whether or not a child is diagnosed with autism. Forehand and McMahon (1981) discuss the prevalence of noncompliance:

Numerous examples from the psychological literature on both "normal" and clinic-referred populations can be cited to illustrate the prevalence of noncompliance as a childhood behavior problem. For example, in extensive naturalistic observations in the homes of 33 nonclinical "normal" children, Johnson, Wahl, Martin, and Johansson (1973) found that among 13 "deviant" child behaviors, noncompliance was the most frequent response and oc-

curred among more children than any other deviant behavior. Among children referred to clinics for treatment of behavior problems, noncompliance is the more frequent presenting complaint of parents...." (p. 2)

The remaining sets of contingencies proposed by the authors, *reinforcement for gestural manding and other nonvocal forms of manding, anticipating the child's needs and thus reinforcing a repertoire of nonresponding that prevents both vocal and nonvocal mands, and interaction between organic or presumed organic factors and behavioral factors*, may also occur with many children and adults, whether or not they have been diagnosed with autism. Individuals who have not acquired conventional mands may emit idiosyncratic mands, which are reinforced by caregivers who have not yet received specialized behavioral training on how to teach conventional mands. Anticipating the child's needs and thus reinforcing nonresponding may occur with any child, disabled or otherwise. Failing to prompt verbal behavior due to concerns about a possible medical condition also may occur whenever a child is believed to have a medical condition.

For the reasons described above, a positive correlation between the sets of contingencies proposed by the authors and the presence of "autistic" behaviors does not appear to be present. The fact that most children diagnosed with autism have typical siblings also fails to support such a correlation. If parents and/or other caregivers engage in child-rearing practices which shape these behaviors in one child, one might expect the other children in the family to also display these behaviors, if all the children in the family learn in similar ways. There is little reason to expect that parents would engage in drastically different childrearing patterns with children diagnosed with autism unless there is something initially different about those children which shapes different interactional patterns with their parents. As explained by Barkley (2000), research conducted with children diagnosed with ADHD has demonstrated that child differences may strongly affect caregiver behaviors:

Research on the interactions between children with ADHD and other adults and children outside the family, such as teachers and peers, shows that when the chil-

dren with ADHD are placed in a classroom, teachers, like mothers, are likely to increase their commands to, reprimands of, and discipline of the children.... Studies have shown that when children with ADHD are placed on stimulant medication, the use by mothers, teachers, and peers of commands, disapproval, and general control diminishes to that seen with children who do not have ADHD, and the interactions become generally more positive. If the parents of children with ADHD were the major cause of the conflict, medicating their children should produce little change in the parents' behavior or little decline in the conflicts. (p. 110)

Without a positive correlation between the hypothesized contingencies and the presence of "autistic" behaviors, one cannot claim that these contingencies account for the etiology of such behaviors. As Bijou and Ghezzi (1999) point out:

A number of studies on children with autism have focused on their atavisms—tantrums, self-injurious behavior, protests, whining, shrieking, screaming, aggression, non-compliance, and oppositional behavior. These are, in the main, socially reinforced operant behaviors that occur in normally developing children as well as children with other handicapping conditions. From this point of view, they should be considered as behaviors that can accompany autism (Koegel, Valdez-Menchaca, & Koegel, 1994). (p. 40)

Rather than attributing "autistic" behaviors solely to existing patterns of reinforcement contingencies, it may be more productive to identify and characterize unlearned differences in these children which allow such patterns of contingencies to evolve. It appears that children later diagnosed with autism interact differently with their social environments from an early age, compared with their typical peers, and that these different patterns of interactions shape "autistic" behaviors. For instance, Bijou & Ghezzi (1999) suggest how an unlearned tendency to escape and avoid tactile and auditory stimuli may interact with the environment in such a way that secondary reinforcers are not conditioned, other stimulus functions necessary for further learning are not established, and social and verbal behaviors are not shaped.

Early child differences which interact in a

unique way with the environment are typically viewed as biological in origin, although specific biological factors associated with certain "autistic" behaviors have not yet been identified. Many potential biological causes for autism have been studied, including genetic influences, preconception and prenatal factors, brain biochemistry, endocrinological and metabolic abnormalities, immunological factors, neurophysiology, structural brain abnormalities, and infectious diseases (Gillberg & Coleman, 1992). The heterogeneity of behaviors included in an autism diagnosis may impede the search for such causes, however, as explained by Lovaas (2003):

Demeyer et al. (1981) and Rutter (1978) wrote excellent reviews of the difficulty posed by individual differences and behavioral overlap. In their reviews, they suggested that the diagnosis of autism may represent a multiplicity of behavior problems with a multiplicity of etiologies. Consequently, it is not surprising that efforts to identify causes or effective treatments with traditional approaches have thus far been largely unsuccessful. In essence, the problem is that the existence of an entity called autism is a hypothesis (Rutter, 1978). (p. 7)

Lovaas further suggests:

... the possibility is left open that each behavioral deviation may have its own neurobiological entity, and the delay in the development of a complex behavior such as language may be the product of many and diverse causes. This implies that remedying different language deviations may require separate and distinct interventions. (p. 8)

Fortunately, it appears that unlearned child differences important in understanding the etiology of "autistic" behaviors may be addressed within a behavioral framework. Although radical behaviorism is often characterized as purely environmentalistic, this is a pervasive misrepresentation, as explained by Todd & Morris (1992). Radical behaviorism does not ignore or deny the contribution of genetic endowment. Behavioral explanations of behavior may incorporate the organism's genetic makeup, past conditioning history, and current circumstances, although each of these elements is not

equally accessible for analysis. In response to claims made by Herrnstein that Skinner minimized the role of phylogenetic behavior and that operant conditioners neglected genetic contributions to behavior, Skinner (1977) clarified his position:

I have always regarded myself as on the middle ground. "Skinnerianism" is not "dedicated to environmentalism as opposed to nativism."... I have commented on the four ways in which a culture or social environment can deal with innate susceptibilities to reinforcement by salt, sugar, sexual contact, and signs of aggressive damage ... and have written at length on the relation between the phylogeny and ontogeny of behavior ..., pointing to many similarities between natural selection and operant conditioning. (p. 1007)

Catania (1998) also addressed this issue:

To what extent does behavior depend on evolutionary history and to what extent on learning? Heated controversy often results when such questions are addressed to socially significant issues ..., especially when the alternatives are presented as oppositions (e.g., nature versus nurture, heredity versus environment).... Our primary interest ... is in behavior that is learned, but we must always entertain the possibility that the behavior we study has phylogenetic sources. (p. 39)

One unlearned variable mentioned by Skinner (1969, 1977) is susceptibility to reinforcement. Humans may be innately susceptible to primary reinforcers such as food, water, salt, sugar, sexual contact, reduction or removal of painful or noxious stimulation, and signs of aggressive damage. Other susceptibilities to reinforcement may be characteristic of different species. Skinner (1977) discussed a particular type of susceptibility to reinforcement in ducklings that seems to occur in imprinting:

Innate susceptibility to reinforcement is different from innate behavior, as Peterson (1960) showed in his study of imprinting. The young duckling does not necessarily approach or follow its mother or an imprinted object because it inherits the behavior of doing so. It also inherits, possibly as a redundant mechanism, a susceptibility to reinforcement from getting close

to mother or object. Approaching an imprinted object is the simplest natural way to increase proximity, but Peterson showed that a quite arbitrary response such as pecking a key could also be reinforced by bringing the object closer to the duckling. (p. 1011)

For as yet undetermined reasons, children diagnosed with autism seem to show different susceptibilities to reinforcement (and punishment), compared with typical children. These different susceptibilities may partially explain why "autistic" behaviors are shaped in these children, but not in their typical peers. Bijou and Ghezzi (1999) suggest that children diagnosed with autism show an unlearned tendency to escape from and to avoid tactile and mild auditory stimuli. Such a tendency may be viewed as a susceptibility to the removal of tactile and auditory stimuli as a form of negative reinforcement or, conversely, a susceptibility to the presentation of tactile and auditory stimuli as forms of punishment. According to Bijou and Ghezzi, the tendency to escape or avoid tactile or auditory stimuli interferes with the initial development of conditional positive social stimuli and their subsequent functions as conditioned reinforcers, discriminative stimuli, and generalized reinforcers. These stimulus functions are established with typical children according to the following general process:

Stimuli from the mother—her presence, proximity, odor, movements, holding, talking, and touching—acquire conditional social properties by virtue of their association with the presentation of positive unconditional stimuli ... and the removal of aversive stimuli.... These conditional social stimuli begin to acquire a reinforcing function because they occur consequent to the infant's behavior. They also acquire a discriminative function since they will soon serve as signals that the caretaker is present and will probably provide the infant with positive unconditional stimuli, or will remove an aversive stimulus. Further, certain stimuli from the caretaker, notably her attention, approval, and disapproval which will occur in more than one deprivational state will acquire generalized reinforcing functions.... (pp. 35–36)

If the child is susceptible to the removal or

tactile or auditory stimuli as a form of negative reinforcement or the presentation of these stimuli as a form of punishment, however, these stimulus functions may not develop. Instead, conditioned punishers, reflexive conditioned establishing operations, and generalized punishers may be established. Since the mother has been paired with tactile and auditory stimuli, which are aversive, her presence may acquire aversive properties. The child may then escape or avoid her presence by looking away (i.e., showing "gaze aversion") or moving away when she approaches. In response to tactile stimuli such as being picked up, the infant may stiffen or arch his/her back and make holding difficult. These child behaviors are likely to punish interactions on the part of the mother and other caregivers. With much fewer interactions and the absence of secondary reinforcers or other stimulus functions, verbal and social behaviors may not be shaped in the child, or shaped to a very limited degree. Without structured behavioral intervention to circumvent the process, reciprocal contingencies between the child and his/her social environment may evolve until the child displays behaviors resulting in an autism diagnosis.

Susceptibility to reinforcement appears to be testable and might potentially identify children at risk for developing "autistic" behaviors at a very early age. In the imprinting example described by Skinner (1977), susceptibility to reinforcement was tested by presenting the putative reinforcer, increased proximity to the imprinted object, contingent on pecking a key. When pecking the key increased, susceptibility to this form of reinforcement was demonstrated. Thus, one method of testing whether an event functions as primary reinforcement is to present that event contingent on a specific behavior and observe whether the behavior increases. A simpler method may involve simply presenting a stimulus and observing the child's response. If the child moves toward the stimulus, looks at it, or engages with it in some way, and does this consistently, the stimulus is likely a form of reinforcement, since it maintains behaviors which achieve contact with the stimulus. If the child repeatedly looks or moves away from the stimulus, however, removal of the stimulus may function as reinforcement. It would seem that children's responses to various tactile and auditory stimuli could be tested in this manner, at a very young age, to provide

preliminary data on whether the child might be at risk for acquiring "autistic" behaviors. If so, a corrective teaching environment (i.e., structured behavioral intervention) might begin very early.

Other possibly innate variables may also be described in behavioral terms. Skinner (1969) provided a behavioral interpretation of "intelligence":

To say that intelligence is inherited is not to say that the specific forms of behavior are inherited. Phylogenic contingencies conceivably responsible for the "selection of intelligence" do not specify responses. What has been selected appears to be a susceptibility to ontogenic contingencies, leading particularly to a greater speed of conditioning and the capacity to maintain a larger repertoire without confusion.

Susceptibility to ontogenic contingencies as evidenced by a greater speed of conditioning is also testable. This variable is informally tested when children diagnosed with autism enter early intensive behavioral intervention programs. From the first weeks of training, children may show quite different rates of progress in these programs. On a smaller scale, it might be possible to assess speed of conditioning by teaching a standard set of simple skills to very young children using appropriate behavioral techniques, and measure how quickly they acquire these skills. Those who fall below a certain criterion might be considered at risk for a diagnosis of mental retardation or autism, and might receive further evaluation and a corrective teaching environment, if necessary.

In conclusion, it seems plausible that the sets of contingencies proposed by the authors occur in the environments of some children diagnosed with autism, but there is no evidence that they are unique to those environments. Such contingencies also occur with individuals diagnosed with other developmental disabilities and with typical children, in some cases. These contingencies alone are insufficient to account for the etiology of "autistic" behaviors. In addition to describing existing contingencies, it is necessary to characterize unlearned differences in children diagnosed with autism which set into motion atypical patterns of interactions with caregivers and shape very different behavioral repertoires from those

of their typical peers. A "completely behavioral" account may address these unlearned child differences, and in fact must do so to adequately explain the behaviors of interest. I share the authors' view that earlier behavioral intervention is desirable, and suggest that testing for various unlearned susceptibilities to reinforcement and possibly speed of conditioning at a very early age might be included in a screening process for children at risk for developing "autistic" behaviors. Other unlearned characteristics that might interact with the social environment and contribute to the shaping of highly deviant behaviors might also be identified and tested, so that remedial steps can be taken as early as possible.

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