Is Autism a Preventable Disorder of Verbal Behavior? A Response to Five Commentaries

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This paper presents a response to five commentaries on our article *An Analysis of Autism as a Contingency-Shaped Disorder of Verbal Behavior* (Drash & Tudor, 2004). One of the principal objectives of that article is to provide the behavior analysis community and the autism community with a conceptual basis for analyzing autism as a behavioral disorder rather than a neurological disorder. This analysis provides a logical and testable behavioral answer to the question of the etiology of autism, a question that has baffled scientists and researchers for more than half a century. Elements of the original article with which reviewers expressed concern include: need for more data, need for greater emphasis on neurological and epidemiological factors in autism, the relative importance of verbal behavior as a core deficit of autism, and disruptive and avoidant behavior as a primary variable in the etiology of autism. We provide a behavioral response to each of these concerns. We also show how our analysis will provide a conceptual foundation for behavior analysis to begin developing urgently needed programs for prevention and earlier intervention in autism.

A THEORETICAL ANALYSIS OF ETIOLOGICAL FACTORS IN AUTISM

In our article "An Analysis of Autism as a Contingency-Shaped Disorder of Verbal Behavior" (Drash & Tudor, 2004) we presented a theoretical analysis that demonstrated how contingencies of reinforcement operating between caregiver and infant during the first year to 18 months of *any typical infant's life* might shape a repertoire of behaviors that could later result in a diagnosis of autism. We also presented six specific reinforcement paradigms that could theoretically prevent the acquisition of verbal behavior and other social behaviors and shape the spectrum of behaviors that might later result in a diagnosis of autism.

One of the major objectives of our article is to provide the behavior analysis community with a conceptual basis for viewing autism primarily as a behavioral disorder rather than a neurobiological or genetic disorder. This analysis provides a logical and testable behavioral

answer to the question of the etiology of autism, a question that has baffled scientists and researchers for more than half a century. Such a conceptualization will, we believe, provide a foundation for behavior analysis to begin developing an experimental analysis of the acquisition of verbal behavior during the first year of life and to move forward with urgently needed programs for prevention and earlier intervention in autism.

RESPONSE TO FIVE COMMENTARIES

Malott (2004) has provided a very insightful and highly supportive commentary in which he indicates substantial agreement with our analysis. We have referenced a number of his comments in our response. Carr and LeBlanc (2004) agree that a behavioral analysis of autistic behavior might lead to technological advances such as behavioral prevention efforts, nationwide screening networks, and earlier treatment delivery that would not be possible based on a purely neurological explanation of the etiology of autism. They also agree with one of our major premises, that autistic behavior is shaped and maintained by its consequences. However, they and the other reviewers, Hall (2004), Hixson (2004), and Romanczyk and Gillis (2004), express concern with specific aspects of our analysis that we discuss below.

Lack of sufficient experimental evidence to

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support our theory. The majority of our reviewers commented that more experimental data is needed to support our theoretical analysis (Carr & LeBlanc, 2004; Hall, 2004; Hixson, 2004; Romanczyk and Gillis, 2004). We agree that having more experimental data would be desirable. However, we concur with Skinner (1957, 1974) that speculation and analysis that go well beyond currently established facts may be necessary to direct attention to new areas of research and investigation. Skinner clearly recognized and supported the need for speculation and theory in science. In *About Behaviorism* (1974) he stated:

Much of the argument goes well beyond the established facts. I am concerned with interpretation rather than prediction and control. Every scientific field has a boundary beyond which discussion, though necessary, cannot be as precise as one would wish.... Speculation is necessary, in fact, to devise methods that will bring a subject matter under better control. (p. 19)

Thus, we believe that we have a strong scientific precedent for presenting our theory with limited data, particularly in view of the fact that all of its elements are based on behaviors that are highly observable, measurable, and modifiable.

It is also our judgment that both the field of behavior analysis and the autism community will benefit from having available a complete behavioral analysis of the etiology of autism. It is our belief that the benefits to the autism community and the field of behavior analysis in the areas of prevention and earlier intervention will greatly outweigh the fact that the theory will initially be distressing to some parents and perhaps to some behavior analysts. It is our judgment that, despite the absence of definitive experimental support, having a theory that is known and available to the behavioral community will lead to a sharper focus on its strengths and weaknesses that may lead directly to further behavioral research in prevention, earlier intervention, and the initial acquisition of verbal behavior.

Need for a greater stress on possible neurological origins of autism. Carr and LeBlanc (2004) and Romanczyk and Gillis (2004) suggest that greater emphasis should be placed on neurological or genetic theories of etiology. As we stated in our original article, and as con-

firmed by Hixson (2004), many years of well-funded neurobiological research by hundreds of investigators nationwide have failed to discover a neurological basis for autism. Moreover, should such a basis be discovered in the future, it is highly probable that the behavioral approach toward earlier intervention and treatment would not change. In support of our position, Hixson (2004) states, "... even if autism was found to be highly heritable, this does not mitigate the necessity for behavioral interventions." Our research with infants with Down syndrome as young as two months of age and their parents is quite informative (Drash, 1982; Drash & Tudor, 1990). This research demonstrated that a combined parent-infant verbal behavior training program could produce significant improvements in language and intelligence, even in children with Down syndrome who are clearly limited by their genetic disorder. The verbal behavior program used with these infants and their parents was essentially the same as that used to successfully increase the verbal behavior and cognitive skills of typical infants ranging in age initially from 3 to 6 months (Drash & Tudor, 1990; Storf, 1990). Thus, within the behavior analysis community we already have the technology necessary to proceed with programs of prevention and earlier intervention in autism (Bijou, 1983; Drash, High, & Tudor, 1999; Drash & Tudor, 1991). As Malott (2004) stated, "We do not want to be a western version of the Pacific-island cargo cults waiting generation after generation, for the ship laden with the gene-fixing, autism-curing pills that will so easily take care of all of our problems." We completely agree.

*Epidemiological phenomena.*¹ Three of our reviewers (Carr & LeBlanc, 2004; Hixson, 2004; Romanczyk & Gillis, 2004) commented that concordance data for twin studies and non-twin siblings appear to present a strong case for a genetic influence in the etiology of autism. They point out that a 60% concordance for monozygotic twins versus a 4.5% concordance for dizygotic twins, which is the same as for non-twin siblings, suggests a powerful hereditary factor in autism. The conventional

¹ Dick Malott has generously shared his views on the concordance data with us. We are pleased to include his views, with which we completely concur, in this section.

interpretation of such data is to automatically and uncritically regard high concordance rates as evidence for a genetic influence.

However, there is an alternative interpretation that should be much more acceptable to most behavior analysts. Malott (personal communication, February 9, 2004) stated the issue very articulately:

The question is, what are the underlying behavioral mechanisms or processes that account for this high concordance for monozygotic twins? We should not automatically assume that this high concordance indicates a direct genetic etiology for autistic behavior and values, especially because that behavior and those values are so complex and variable.... I know of no unambiguous example of genetic programming of any complex behavioral repertoire.

Therefore, I find it hard to accept the notion that there is a genetic program that causes some preschool children to have high rates of counter-controlling tantrums and aggression, low rates of verbal behavior, and high rates of self-stimulatory behavior, especially all in one child. The problem is that this repertoire and these values seem far too complex for genetic programming. And given the dubious history of claims for genetic programming of other complex repertoires, like intellectual skills (IQ) and criminality, I think we should always be cautious about accepting similar claims for genetic programming of complex autistic repertoires and values.

Given the difficulty of going from monozygotic-twin data to the behavioral processes necessary for the acquisition of complex autistic repertoires and values, I think it may be up to those advocating a genetic basis for autism to provide the missing steps rather than for those advocating against such a genetic basis.

Hixson (2004) provided further evidence that calls into question the uncritical acceptance of concordance data. He stated, "... heritability estimates are based on common or current environmental arrangements; if the environments are changed substantially, the heritability estimate may change." Intensive ABA treatment represents one such substantial change in a child's environment. Hall (2004) suggests that

there are predisposing genetic factors that function to make a child with autism more or less responsive to certain reinforcement contingencies. Even if this could be shown, it would not alter the fact that the role of behavior analysts remains essentially the same, that is, to identify and modify specific functional relationships that initially establish repertoires of autistic behaviors.

Carr and LeBlanc (2004) and Hall (2004) also discuss several other factors that, in our opinion, have only marginal, if any, relation to possible genetic factors. The issue of why or how a parent might have one typical child and one with autism is, we believe, primarily an environmental question. Catastrophic events such as the death of a spouse, long term-chronic illness of the primary caretaker, or divorce frequently cause major and easily observed changes in the environmental contingencies in effect for a child born before the event as opposed to contingencies in effect for a child born after the event. Carr and LeBlanc (2004) also state that the male to female ratio in autism (4-5 to 1) indicates a genetic influence. However, this ratio is consistent with the fact that similar or higher ratios of males to females exist for many other behavioral disorders such as attention deficit hyperactivity disorder, oppositional defiant disorder, noncompliance, and sociopathic behavior. While the specific contingencies that might create this general male to female disparity have yet to be discovered and analyzed, the general consensus appears to be that these behaviors are shaped by a combination of parental and cultural factors. Hixson (2004), in this issue, has provided a detailed description of how antisocial behavior is an acquired repertoire that is shaped by contingencies of reinforcement.

Carr and LeBlanc (2004) also suggest that developmental regression in autism may indicate a genetic factor. For example, they state that up to 50% of parents indicate that their children were developing typically until a regression occurred between the ages of 18 and 24 months. This figure contrasts markedly with the much smaller regression rate of 25% cited by Lord & Paul (1997). Even the lower rate could be subject to question, as memory for historical events is often inaccurate. Moreover, in our own experience, this "regression" frequently consists of little more than the loss of a few single words that could just as easily, or

perhaps more easily, be accounted for by environmental factors such as those reported by Fowler (1990) or Ferster (1961) in our original article. Thus, the statement that our model does not account for up to 50% of the cases is clearly questionable.

Engaging in endless debates regarding whether or not there are predisposing genetic or neurological factors in autism appears relatively unproductive. We believe it would be much more useful to devote the unique skills of behavior analysts to increasing the effectiveness of existing ABA early intervention programs.

Verbal behavior or social behavior as the core deficit of autism? In their commentary Carr and LeBlanc (2004) state that, "Drash and Tudor (2004) propose that verbal behavior deficits are the foundation of all deficits associated with autism." While we do place great emphasis on the importance of verbal behavior, this quote represents a partial misunderstanding of our analysis of the etiology of autistic behaviors. In our original article we stated, "... the social-emotional deficits of children with autism can be causally linked ... to the deficiencies in verbal behavior and the presence of disruptive avoidance behaviors."

Our analysis thus states that the caregiver-provided contingencies of reinforcement initially produce a repertoire of avoidance and disruptive behaviors that in turn prevent or delay the development of *both verbal behavior and social behavior*. It is only after a substantial repertoire of avoidance behaviors has been established and verbal behavior is absent or significantly delayed, usually during the second year, that the inability to communicate with or to understand others prevents or further inhibits social development.

There have been differing opinions in the literature over the years regarding the relative theoretical importance of verbal behavior as contrasted with social behavior in autism. Churchill (1978) stated, "Language disorder is proposed as a central explanatory concept to account for those behavioral features which autistic children have in common: profound impairment of speech and gestures for communication, nonfunctional use of objects, and severe impairment of interpersonal relations" (p. 71). Conversely, Volkmar, Klin, and Cohen (1997) stated, "The usual pattern of social deficit described by Kanner remains the central defining core of the condition" (p. 10).

From a practical standpoint, however, there can be no doubt that lack of verbal behavior is by far the most important and devastating aspect of autism. The literature shows that a delay in spoken language is the primary reason that parents initially become concerned about their children's development (Lord & Paul, 1997), and that a delay in spoken language is often the principal cause of referral for diagnosis and treatment. After entering treatment, the overwhelming concern of the parents of a nonverbal child with autism is that their child acquire functional language. Autistic children without speech by age five have a very poor prognosis for achieving independent functioning (Lord & Paul, 1997). Without functional language, recovery is, in most cases, essentially not possible.

Carr and LeBlanc (2004) also discuss the fact that many behaviors used to identify children at risk for autism as early as 8 months are primarily social or social-communicative rather than verbal. We reemphasize that contingencies of reinforcement operating during the first year strengthen repertoires of avoidance and disruptive behaviors that may in turn prevent or inhibit the development of *both language and social behaviors*. Since the typical child does not begin to produce words before about 12 months, lack of spoken language obviously could not be used as a predictive criterion for children 12 months or younger. Conversely, if a repertoire of avoidance behaviors exists during the first 12 months, the most observable negative impact of such behaviors would be upon social-interactive behaviors. Moreover, many of the social and social-communicative behaviors are considered by some authorities to be "pre-speech" behaviors (Rutter, 1978).

Extinction of verbal behavior. As we stated in our original article (Drash & Tudor, 2004), extinction of verbal behavior may potentially occur whenever any typical infant is placed in an environment in which caregivers do not actively prompt, respond to, and reinforce the infant's or child's vocal utterances. We presented several examples of how and where such extinction might occur, including care at home by a nanny, relatives, a baby sitter, some in-home infant-care placements, and some day-care settings. Carr and LeBlanc (2004) suggested that extinction in such settings is quite improbable. They cite an article by Scarr (1997) in support of their position that day-care set-

tings have no long-term impact on children's development. While this may be true in most cases, it is also quite possible that in very rare instances, events may coalesce to produce very subtle extinction contingencies (Malott, 2004) that may go largely unrecognized by non-behaviorally trained observers.

Moreover, the Siller and Sigman (2002) article cited by Carr and LeBlanc (2004) is not germane to the present discussion, since our hypothesis states that the extinction paradigm, like our other five paradigms, initially occurs during the first year to 18 months, typically *before the diagnosis of autism is made*. In the article cited the diagnosis of autism had already been made, and the average age of these children with autism was 4 years.

We believe that the number of settings in which extinction of verbal behavior might result in a diagnosis of autism are quite rare, but they can and do exist. Since the diagnosis of autism occurs in only 1 per 1,000 births (Bryson, 1997), the probability that extinction might be a causal factor in one specific case of autism at one specific location is extremely small. However, the knowledge that such contingencies may exist in unlikely environments is important to those desiring to conduct comprehensive functional analyses to establish highly effective programs of prevention or earlier intervention.

Disruptive and avoidant behavior as etiological factors. There seems to be a general consensus in the literature that avoidant and disruptive behavior is highly characteristic of children with autism. As reported in our original paper, Kanner included disruptive and avoidant behavior as one of the typical characteristics of children with autism (Firth, 1991). Numerous well-known investigators have commented on the presence of avoidance behaviors and "negativism" in children with autism. A number of these include Churchill (1978), Cowan et al. (1965), Rendel-Short (1969), Richter (1978), Rutter (1978), Tinbergen and Tinbergen (1972), and Wing (1976).

Carr and LeBlanc (2004) and Hall (2004) have taken exception to our use of avoidance and disruptive behaviors as a principal explanatory variable in the etiology of autistic behaviors, primarily on the basis that such behaviors are not unique to autism. The fact that disruptive behaviors occur in many typical and atypical children *after they have acquired speech* is

not a relevant issue. Since well established verbal behavior is maintained by multiple reinforcement contingencies, the simultaneous occurrence of disruptive behavior would have minimal impact on the child's existing verbal behavior. Common examples of disruptive behavior after the acquisition of speech include toddlers in the "terrible twos" and "oppositional-defiant" preschoolers.

We believe that a repertoire of disruptive and avoidance behaviors are inadvertently and initially shaped by caretakers during the first year to 18 months of a child's life. *The critical element in our hypothesis is the time during which the shaping occurs.* The first year of life is the critical time for the initial development of social, social-communicative, and pre-speech behaviors. If a strong repertoire of avoidant behaviors is established during the first year, this may prevent or inhibit the acquisition of *both pre-verbal and social behaviors*. While both verbal and social behaviors are important, it is the lack of functional verbal behavior that has the most devastating impact on all areas of a child's future development.

Carr and LeBlanc (2004) also believe that stereotypic behaviors in autism that are reinforced by non-social stimuli may indicate a genetic component. Our experience suggests an alternative explanation. The majority of young children (i.e., 2 to 3 years and younger) that we have treated do not engage in the more extreme forms of stereotypic or self-injurious behaviors that are frequently observed in children 6 to 7 years of age and older. Most behaviors that occur are maintained primarily by social reinforcers. If these young children acquire functional verbal behavior and achieve substantial recovery, the stereotypic behaviors are likely to diminish with minimal intervention and are no longer of concern. Those children who do not achieve substantial recovery are likely to develop more complex and more severe stereotypic behaviors, some of which are clearly maintained by social reinforcers. This view also appears to be shared by Bijou and Ghezzi (1999). If there are other stereotypic behaviors that might be maintained by genetic variables, it would appear to be incumbent upon those who advocate for such a position to demonstrate the necessary functional linkages between genetic variables and complex behavioral repertoires.

Both Hall (2004) and Hixson (2004) appear

to agree that avoidant and disruptive behavior can be an etiological factor preventing the development of verbal and social behavior. If this contingent relationship can be demonstrated experimentally, then perhaps one of the most critical unresolved issues remaining is: How is this repertoire of disruptive and avoidant behavior initially established? Hall (2004) prefers to attribute initial avoidance behaviors to inherited variables such as an unlearned tendency to escape and avoid tactile and auditory stimuli, as described by Bijou and Ghezzi (1999), or to other unlearned tendencies such as, a greater or lesser responsiveness to reinforcement.

We cannot rule out the possibility that at some future date innate tendencies that might contribute to the establishment of autistic behaviors might be discovered. However, this would seem to produce an unnecessary and complicated reliance on hypothetical genetic variables for which there is also no evidence. We prefer the more easily observed and testable behavioral hypothesis that a repertoire of avoidant and disruptive behaviors may be unintentionally shaped by caregiver-provided contingencies of reinforcement during an infant's first year.

A behavioral approach to the prevention of autism. Both Hall (2004) and Hixson (2004) agree that it may be possible to establish programs for the prevention of autism. Moreover, Carr and LeBlanc (2004) recognize that an environmental analysis of autistic behaviors might lead to behavioral prevention efforts, while a neurological approach would not.

If, as we hypothesize, autism is primarily a behavioral disorder rather than a neurobiological disorder, then it should be possible to design preventative and earlier intervention programs in the near future. One model for such a preventative program has been provided by Bijou (1983). Other models are presented in Guralnick (1997) and Odom and Karnes (1988). As discussed in our original article, the goal of preventative behavioral programs would be to identify inappropriate reinforcement contingencies operating during the first year to 18 months and replace them with contingencies that could establish age-appropriate verbal and social behavior. If complete prevention does not occur, preventative programs could merge directly with very early intervention programs before repertoires of avoidance

behaviors are well established, thus enhancing the probability for full recovery.

To have a significant nationwide impact on the reduction of new cases of autism, it will be necessary to establish a national network of easily accessible ABA early intervention screening programs that could routinely screen infants during their first 18 to 24 months. An undertaking of this magnitude will require the commitment of a large segment of the ABA community. It is our hope that this analysis of autism as a contingency-shaped disorder will encourage many in the ABA community to make such a commitment.

Creating a blame-free treatment environment for parents. As Malott (2004) stated, one reason many behavior analysts resist adopting a contingency-shaped approach to the etiology of autism is the fear that by so doing they might be viewed as "insensitive professionals" who blame parents for their children's disabilities.

This represents an unfortunate misunderstanding of our contingency-shaped analysis. *Our analysis in no way attempts to blame parents or hold them responsible.* Instead it simply describes how autistic behaviors are created by early contingencies of reinforcement over which parents have no control. As Malott (2004) observed, all parents are taught by our culture to be warm, loving, and caring and to attend to their child's slightest need, whimper, and whim. Unfortunately, the same contingencies that cause parents to attend to and reinforce their child's every need and desire may also lead some parents into slowly and subtly reinforcing an avoidance repertoire that over time results in behaviors that could hinder, and in some cases prevent, the acquisition of language and social behaviors.

Malott (2004) stated the issue well: "The amazing thing is not that there are so many children who do not talk ... but that so many children do learn to talk and keep their bad alternatives to a minimum. There but for the grace of a few rolls of the dice, go we all."

Is it possible that the acquisition of appropriate verbal and social behavior is simply a matter of sheer luck? Is there no way to insure a better outcome? Fortunately, there is a better way to increase the probability of a more successful outcome and to simultaneously reduce the probability that parents will be trapped by subtle, unrecognized contingencies of reinforcement.

In a previous publication (Drash & Tudor, 1993), we discussed the pitfalls of using a contingency-shaped approach when teaching verbal behavior to infants and young children as opposed to using a rule-governed approach. We stated, "... in contingency-shaped behavior there are numerous times during the shaping process when the immediate contingencies may reinforce behaviors incompatible with the long term goal" (p. 28).

Conversely "rule-governed" parent-infant training programs for increasing language and cognitive development (Drash, 1982; Drash & Tudor, 1990; Guralnick, 1997; Odom & Karnes, 1988; Storfer, 1990) provide parents with specific techniques for identifying and reinforcing appropriate verbal and other behaviors. At the same time these programs teach parents to identify and avoid strengthening inappropriate avoidance behaviors. Such programs are essentially blame-free, since, like other behavioral training programs, they target acquisition of new skills.

Incorporating parent-infant training into prevention and earlier intervention programs would give much needed recognition to the critical importance of these early contingencies in shaping verbal and social behavior during the first year. At the same time it would essentially avoid the issue of parent blame. The overall emphasis of such programs would be on how can we, both parents and professionals, *working together*, increase the probability that autistic repertoires will not be acquired, or if initially acquired, will be identified and treated earlier, thus increasing the probability for substantial recovery from autism. We anticipate the development and implementation of a new generation of even more effective prevention and early intervention programs for children with autism within the foreseeable future.

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