Autistic Behavior, Behavior Analysis, and the Gene—Part II

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This article reviews the negative behavior-analytic commentary on Drash and Tudor's behavior-analytic analysis of the etiology of autistic repertoires and values. This article also asks that, in our effort to scrub it clean, we not drown Drash and Tudor's beautiful, but fragile, new-born, behavior-analytic baby in hyper-methodological, hyper-scholarly bathwater.

Key words: autism, behavior analysis, gene, etiology of autism, medical model, contingency, verbal behavior.

In a special issue of *The Analysis of Verbal Behavior*, several behavior-analytic colleagues commented on Drash and Tudor's (2004a) theoretical analysis of autism as a contingency-shaped disorder (Carr & LeBlanc, 2004; Drash & Tudor, 2004b; Hall, 2004; Hixson, 2004; Malott, 2004; Romanczyk & Gillis, 2004). Most of the behavior analysts were quite critical of the Drash and Tudor theory and not in a sympathetic way. This might in part be because we have been trained in grad school to review articles as if we were critiquing a *Psych. Review* article resulting from a long series of million dollar NIMH grants, rather than the speculations of two colleagues who have been logging in quite a few years working with children labeled as *autistic*. And I fear this heavy-duty, scholarly criticism may have the unintended effect of killing this nascent behavior-analytic theory in its infancy. Instead, I would encourage our behavior-analytic community to help Drash and Tudor get their theory ready for prime time. Instead of listing all the reasons why their theory is wrong and should be disregarded, I think we would do better to list all the issues the theory still needs to address and then help Drash and Tudor address those issues. In other words, we need a concerted, empirical research effort into the role of behavioral history in the etiology of autism. And such an effort might

produce the most important contribution to our field since the early Lovaas work (e.g., 1987). So, I recommend that these behavior-analytic autism experts and the readers of this essay read Drash's (2004) brave, new research proposal, "Preventing Autism Now: A Possible Next Step for Behavior Analysis"; this is a proposal to empirically exam the role of the child's early behavioral history in the acquisition of autistic behavior and values and might be an excellent starting place for such research. However, in the meantime, the present essay will address some of the negative, behavior-analytic commentary on the Drash and Tudor theory, because I can't resist the immediate reinforcers of criticizing the critics.

TERMINOLOGY

I am skeptical of the very terms *autism* and *disorder* that convention may almost be forcing Drash and Tudor to use, because, guard against it though we might, talking about an *autism disorder* quickly leads to a reification of autistic behavior, resulting in autism as a thing that causes autistic behavior, and we end up on board the medical-model, biological-deterministic band wagon with its circular trip to simplistic analyses and easy answers to hard questions.

For example, even when outstanding behavior analysts acknowledge that autism may represent a multiplicity of behavior problems with a multiplicity of etiologies, they then go on to suggest that each of those behavior problems may have its own neurobiological cause. However, a more behavior-analytic approach might be to look at the contingencies responsible for each problem.

I wrote this article as an assignment in Linda Leblanc's Psychology 697 class. Autism: Etiology, Assessment, and Behavioral Intervention, fall 2004; and I would like to thank Linda for her thoughtful, helpful suggestions.

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As a parallel, when considering a person who has a multiplicity of behavior successes, such as speaking English, riding a bicycle, playing the piano, and voting democrat, we wouldn't say each component of this particular repertoire of successes has its own neurobiological cause. However, if we were to label this more-or-less random multiplicity of successes as the *generation-w syndrome*, then researchers might be seduced into the search for the set of neurobiological causes, just as the label *autistic disorder* may be seducing the best of behavior analysts away from doing a thoughtful behavior-analytic interpretation of the various ways early infant contingencies might wreak profound havoc.

Another parallel is the use of the term *intelligence* to describe a multiplicity of behavior successes and problems. The reifying potential of this word could be what leads even a world-class behavior analyst to suggest that intelligence may be inherited in the form of speed of conditioning and the capacity to maintain a large repertoire without confusion. But a more productive approach for behavior analysts might be to do a careful analysis of the details of behaving intelligently and the contingencies that facilitate the acquisition and maintenance of intelligent behavior. Even concepts like *speed of conditioning* may address such a multiplicity of conditions as to almost be a misleading reification.

EXPERIMENTAL ANALYSIS VS. INFORMAL SPECULATION

Yes, Drash and Tudor have not formally demonstrated a statistical correlation, let alone a causal relation, between autistic behaviors and pathological behavioral contingencies. What they have done is offer plausible suggestions, based on their informal, though extensive, observations of autistic behavior imbedded in pathological contingencies.

It is true that Drash and Tudor are extrapolating well beyond their data set consisting of a small number of single-subject research studies. But their extrapolation is much less extravagant than that of Skinner when he extrapolated from humble, little experiments with only three rats to an analysis of all human nature.

And, it is true that Drash and Tudor's analysis will have much more impact if it is supported with hard experimental and field-study

data; but their analysis is more empirical and less speculative than was that of Skinner (1957) when he wrote *Verbal Behavior*. And it is true that Skinner's analysis did lie dormant, until Jack Michael finally convinced a handful of graduate students that *Verbal Behavior* was profoundly correct. Then those students started a major new approach to working with the language deficits of children classified as autistic (Sundberg & Michael, 2001). In other words, Drash and Tudor's contingency analysis may be a great start, even though it is speculative.

We behavior analysts have long chanted the mantra of caring only about interventions, about what works, not about etiology. And we have been so traumatized by our mentalistic and psychodynamic heritage of the quagmire of speculative psychology that we are afraid to even consider speculative behavior-analyses of etiology. We even enforce compliance with this anti-theory mantra in our professional journals. So appreciating the value of Drash and Tudor's effort may require a shift in our world view, our paradigm for appropriate scholarship.

MEANINGFUL, BUT SUBTLE, DIFFERENCES IN EVERYDAY INFANCY

Drash and Tudor describe a set of pathological behavioral contingencies that might result in autistic behavior. But others criticize their theory because most of those pathological contingencies are present in the infancy of all children, whether or not they acquire serious autistic repertoires; for example, all parents pay attention to their screaming baby. But that just suggests to me that we should be amazed that all children don't acquire serious behavioral problems. It's like any behavior analyst who compares appropriate classroom practices with actual classroom practices should be amazed that any children learn to read, write, or count—a major grade-school miracle, as far as I'm concerned. In other words, we need a more fine-grained analysis of the differences in the contingencies and the interactions of those contingencies that do and do not result in serious autistic repertoires.

To put it another way, none of us is impressed when an untrained teacher says, "I tried that behavior modification stuff with my students, and it didn't work." We know enough about behavior modification to suspect that the contingencies the untrained teacher tried were not

close enough to the contingencies we would use to achieve the desired results. Similarly, we know enough about behavioral contingencies, in general, to suspect that the contingencies involved in the early life of children with normal repertoires must not have been close enough to those of children with serious autistic repertoires to have the undesired results, even when comparing the behavioral histories of two children from the same family.

Similarly, I don't think we should be too impressed when an untrained parent says, "I treated Susie and Johnny exactly alike, and Susie had absolutely no problems, but Johnny has autism." We know enough about self-reports to suspect that even if we professional behavior analysts were that parent, we should not be too confident in the validity of our own report. It may be more-or-less impossible to casually monitor those subtle behavioral interactions with any reliability; this is a case where a set of independent observers with their clip boards is crucial.

However, the difference between the classroom and the home is that behavior analysts, using their independent observers and clip boards, have filled volumes with experimental analyses of classroom contingencies but barely a notebook with experimental analyses or even behavior-analytic field studies of infant-rearing contingencies. And because of this lack of empirical research on the role of behavioral history in the etiology of autism, it takes a much greater leap of faith to believe that successful child rearing lies in the subtle details of the contingencies, just as successful classroom teaching lies in the subtle details of the contingencies. The angels as well as the devil are in the details.

That some behavior-analytic autism experts have children with autistic repertoires is interesting, but I don't see it as very convincing evidence for the biological basis of such repertoires. I've known many professional behavior analysts who are skilled at doing behavior analysis in the lab, in other people's classrooms, and in other peoples homes. But few have impressed me as being much more skilled than the average person in sticking to their behavior-analytic guns when they are in their own classroom or home. It is much easier to help others apply appropriate behavioral contingencies in their lives than to apply them in our own, where we must now wear not only the sole hat

of behavior analyst but the multiple hats of teacher, parent, spouse, friend, etc., enough hats to cause serious headaches.

BIOLOGICAL DETERMINISM

Along the same lines, I don't think we should be overly impressed with the implications of a biological etiology when giving a drug to a child classified as having ADHD disrupts his ADHD repertoire. We would not conclude that the etiology of a pigeon's fixed interval scallop is biological rather than contingent just because that scallop can be disrupted by drug administration. So I think we should be reluctant to jump from the effects of drugs on ADHD repertoires to the importance of biological determinism in the acquisition of autistic repertoires and values.¹

Bijou and Ghezzi (1999) suggest that (a) some children may be hypersensitive to the aversive qualities of auditory and tactile stimulation, and (b) the aversiveness of such stimuli might form the basis of the contingencies that shape autistic repertoires and values. But even if these interesting speculations are verified, they still don't point to a biological etiology of those autistic repertoires and values in the usual sense of *biological etiology*. Such verification does not mean there is a gene or a combination of genes that causes the child to acquire autistic repertoires and values. That would be like saying there is a genetic etiology of the acquisition of avoidance behavior in the Skinner box because electric shock is inherently aversive. It's the contingencies, not the gene, nor the neurotransmitters, nor any other biological entity, that results in the acquisition of an avoidance repertoire; and it may be the contingencies that result in the acquisition of an autistic repertoire.² However, Bijou and Ghezzi's

¹By *values*, I mean unlearned and learned reinforcers. Simply talking about *autistic repertoire* does not seem inclusive enough; therefore, I also talk about *autistic values*.

²Of course, all behavior is emitted by a living organism and involves genes, neurotransmitters, and a host of other biological entities; but it is the behavioral contingencies, not those biological entities, that explains why the rat exposed to the avoidance contingencies in the Skinner box acquires an avoidance repertoire and the rat not exposed to those contingencies does not acquire that repertoire.

speculation does suggest that we might do well to screen infants for hypersensitivity to auditory and tactile stimulation and carefully monitor the behavioral contingencies in the environment of those infants found to have such hypersensitivities.

There are other possible candidates for pathological reinforcers and aversive-conditions than hypersensitivity to auditory and tactile stimulation; for example, under sensitivity to social reinforcers (such as a smile or verbal praise) and under sensitivity to social aversive conditions (such as a frown or verbal disapproval). However, we know enough about the acquisition of learned reinforcers and learned aversive conditions that we might readily suspect the lack of those particular crucial reinforcing and aversive values is a result of a lack of effective pairing of neutral social stimuli (e.g., smiles) with effective reinforcers and a lack of effective pairing of other neutral social stimuli (e.g., frowns) with effective aversive conditions. So we needn't rush to the search for neurobiological causes of these deficits. In fact, when working with children labeled *autistic*, the first thing we behavior analysts do is try to provide effective pairing of neutral social stimuli with reinforcers so as to establish those learned reinforcers. (However, we seem to actively avoid effective pairing of other neutral social stimuli with aversive conditions, so as to establish those learned aversive conditions. And this might be a serious omission in our behavioral curricula, if we want the children to ultimately be sensitive to the socially inappropriateness of various subtle behaviors.)

OTHER ISSUES

It is true that we behavior analysts should guard against an insular perspective, one that results in our ignorance of the large amount of biological research dedicated to finding the neurobiological basis of autism. However, we should also guard against being uncritically swept away by the rising tide of biological research (and speculation). We should view proposed biological etiologies as the explanations of last resort until we have thoroughly exhausted our search for pathological behavioral contingencies. If it works, the contingency analysis is the most parsimonious explanation of behavior problems of all sorts, because it

makes use of no hypothetical processes or reification and relies only on known, well-documented concepts, principles, and processes.

If pathological behavioral contingencies cause autistic repertoires and values, then it is likely that replacing the pathological contingencies with appropriate contingencies would replace the autistic repertoires with appropriate repertoires. (But the success of this contingency replacement is likely only if the appropriate contingencies plus remedial contingencies are implemented with the skilled help of behavior-analytic autism experts.) However, it is a logical error called affirming the consequent, if we argue the reverse, that successful implementation of appropriate and remedial contingencies proves that pathological contingencies created the pathological repertoires in the first place. But, it is not a logical error to argue that the success of such behavioral-contingency interventions strongly suggests that pathological behavioral contingencies are good candidates for the cause of the problems.

Most behavior analysts have had a tremendous amount of high-quality training in research methodology and a relatively small amount of training in conceptual analysis; probably as a result of a misunderstanding of Skinner's objection to hypothetico-deductive theory construction. As a result we must take great care not to miss the conceptual forest for the methodological trees. For example, in writing reviews, we must take care that we not fall into the trap of comfortably writing critiques of research methodology, with little regard for conceptual integrity or conceptual significance of the literature we are reviewing. I suggest that the Drash and Tudor analysis has great conceptual integrity and significance.

CONCLUSIONS

I ask my colleagues to apply their highly developed research muscles to helping Drash and Tudor carry the heavy load of empirically determining the role of behavioral contingencies in the etiology of autistic repertoires and values. I ask my colleagues to actively participate in implementing Drash's (2004) brave, new research proposal, *Preventing Autism Now: A Possible Next Step for Behavior Analysis*.

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