Commentary on Drash and Tudor: An Analysis of Autism as a Contingency-Shaped Disorder of Verbal Behavior

**Raymond G. Romanczyk, Ph.D., BCBA & Jennifer M. Gillis, M.A.,
BCBA, Institute for Child Development, State University
of New York at Binghamton**

A critique is presented of the etiological model for autism as presented by Drash and Tudor. The model is rejected based upon conceptual and methodological grounds. The major points raised concern overgeneralization of research findings, limitations of single subject methodology to answer population based questions, current neurobiological research, and the danger of uni-dimensional models. Specific examples of cases that do not fit Drash and Tudor model are presented.

The field of autism has been a fertile ground for innumerable etiological theories and treatment approaches. Few have stood the test of empirical scrutiny (Romanczyk, Arnstein, Soorya, & J. Gillis, 2002). Thus new etiological and treatment proposals must be viewed with caution and examined with care using the conceptual and methodological tools of science.

As a preface, let us state that the behavioral approach to autism is an incredible conceptual and empirical achievement that is constantly developing. The benefit to countless children and families is without question. Our own work with children and families as well as our undergraduate and graduate training programs are emphatically behavioral. Thus we are firmly anchored in the behavioral tradition, and view the specific conceptual and methodological contributions of behavior analysis, applied behavior analysis, and verbal behavior analysis as essential to the understanding and treatment of autism. That said, any field must guard against too insular a perspective. It is in this context we provide the following comments on the Drash and Tudor paper.

Over-Generalization

The authors present research concerning the effectiveness of applied behavior analysis and

the clear impact of adult reinforcement on child behavior, both in promoting appropriate and inappropriate behavior. They then argue that parents could shape a child's behavior with the result being the symptomatology of autism. Part of the argument is the effectiveness of applied behavior analysis as it relates to their proposal for preventative intervention. This thesis is flawed and presents an example of the logical error known as affirming the consequent. As an example, if taking an aspirin causes remission of a headache, one cannot conclude that the cause of headaches is an absence of aspirin. The presentation by the authors makes tenuous connections between differing areas of research, and then extrapolates, in our opinion, well beyond the data.

Methodological Limitations

Single subject methodology is a very powerful tool, especially in the context of applied behavior analysis. Particularly with respect to the treatment of autism, single subject research has been acknowledged as an important source of information with respect to evaluating treatment efficacy (NYSDOH Clinical Practice Guideline, 1999). However, this methodology has limitations, particularly with respect to population issues. Information from group and longitudinal studies are critical in investigations of etiological hypotheses. There are generally accepted procedures for evaluating scientific evidence and weighting the contributions from various research designs that have been developed by the Agency for Health Care Policy and Research of the Public Health Service, U.S. Department of Health and Human Services

Address correspondence to Raymond G. Romanczyk, Ph.D., BCBA, Institute for Child Development, State University of New York at Binghamton, Binghamton, NY 13902-6000; e-mail: rromanc@binghamton.edu.

(Woolf, 1991, 1994). In our opinion, the reliance on a small number of single subject research studies in the current paper to argue for an exclusively behavioral analysis of etiology of autism is inappropriate and insufficient.

Neurobiological Perspective

While the authors give brief reference to the neurobiological research on the etiology of autism, the references and information provided are limited. While the authors are correct that specific pathophysiological mechanisms have not been fully identified for the symptomatology associated with autism, it is nevertheless the case that the authors significantly understate the current work in the field of neurobiology. More recent reports, such as Gilberg (2000) and Rodier (2000) illustrate the progress that is being made. In particular, Rodier's work, in the analysis of the timing of developmental anomalies during gestation and their association with known biological insults, has added greatly to our understanding of etiology. Further, research into disorders that have significant comorbidity with autism, such as Fragile-X and tuberous sclerosis, as well as related disorders with well-studied pathophysiology such as Rett Syndrome, aid in the search for specific pathophysiological mechanisms. Further, the continuing progress in identifying candidate genes for autism clearly points to a neurobiological etiology. While we do not have the space to provide a full review of the contributions of research in neurobiology, neuroimaging, and genetics to the etiology of autism, this is a large and productive area of research that cannot be ignored or minimized.

Uni-dimensional Models

Historically there has been significant debate over the role of nature versus nurture. While consuming much energy and resulting in heated and bitter debates, the time for such uni-dimensional models is long past. Skinner was clear concerning the importance and influence of biology on an organism's behavior. Contemporary multi-dimensional models seek to understand the interplay of biology and the impact of the environment on acquisition and expression of behavior. Both are critical components. This has very important implications for analysis of behavior and parsing the con-

tributing influences of neurobiology and environment.

Thought Experiments

Einstein used and recommended a process he termed "thought experiments" to examine the logic and premises of hypotheses that would be extremely difficult to conduct in an actual laboratory setting. We offer the following observations that serve to question the validity of the Drash & Tudor proposal:

Normally hearing children born to deaf parents develop spoken language even though the parents cannot reinforce spoken language.

Parents of children with autism reflect the full spectrum of ethnicity, culture, and education. There are parents of children with autism who are skilled professionals in the area of autism, and also applied behavior analysis. From this diversity to assert a consistent parent-child interaction causes autism is untenable.

One traditional control that researchers use to examine environmental influences is twin studies. Interestingly, unlike monozygotic twins, the majority of dizygotic twins are not concordant when one has autism. This does not point to environmental etiology as twins are raised in the same environment (e.g., household, family, parents, etc.)

Reports of children raised under extreme conditions of abuse and neglect do not support a completely environmental account for autism.

A significant proportion of children with autism do not achieve clinically significant improvement, particularly in language, using applied behavior analysis.

CONCLUSION

The paper by Drash & Tudor presents an extreme position concerning the etiology of autism. The research support for such a position is absent. In contrast the more contemporary neurobiological-behavioral model has extensive research support. Behavior analysis has much to offer in continuing to contribute to an understanding of the expression of behavior of both typically developing individuals as well as those who present with a wide range of developmental disorders, but it must take place within the context of the full breadth of our knowledge base.

REFERENCES

- Gillberg, C. (2000). *The Biology of the Autistic Syndromes* (3rd ed.) Cambridge, MA: Cambridge University Press.
- New York State Department of Health Clinical Practice Guideline: *The Guideline Technical Report. Autism/Pervasive Developmental Disorders, Assessment, and Intervention for Young Children (Age 0-3 Years)*. Publication No. 4217. New York State Department of Health, 1999.
- Rodier, P. M. (2000, February). The Early Origins of Autism, *Scientific American*, 282 (2), 56–63.
- Romanczyk, R. G., Arnstein, L., Soorya, L., & Gillis, J. (2002). The Myriad of Controversial Treatments for Autism: A Critical Evaluation of Efficacy. In Lilienfeld, Lohr, and Lynn (Eds.), *Science and Pseudo-science in Contemporary Clinical Psychology*. New York: Guilford Press.
- Woolf, S. H. (1991) *AHCPR Interim Manual for Clinical Practice Guideline Development*. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services. (AHCPR Publication No. 91-0018).
- Woolf, S. H. (1994). An organized analytic framework for practice guideline development: Using the analytic logic as a guide for reviewing evidence, developing recommendations, and explaining the rationale. In McCormick, K. A., Moore, S. R., & Siegel, R. A. (Eds.), *Clinical Practice Guideline Development: Methodology Perspectives*. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services (AHCPR Publication No. 95-0009).