We wish to correct certain errors of fact and misrepresentations of our research that appeared in a recent article in this journal entitled "Autism and Pervasive Developmental Disorders: Concepts and Diagnostic Issues (Rutter & Schopler, 1987).

1. The authors state: "Although informed by scientific research, the National Society for Children and Adults with Autism (NSAC) definition was developed to shape favorable social policies rather than scientific synthesis." This misstatement echoes a similar inaccurate comment published in a discussion to the original publication of the NSAC definition in 1978 (Ritvo & Freeman, 1978). We did not feel it warranted correcting at that time but do now.

The facts are these: The NSAC definition was reached by consensus of the NSAC Professional Advisory Board (PAB) from 1975 through 1977. Members of the PAB represented the disciplines of medicine, education, and psychology and parents of autistic persons. It was formulated upon the most current scientific data available, discussed and modified over 1½ years' time, and finalized only after careful and critical discussions. The purpose of the members of the PAB, as professionals, was to provide a definition of autism for scientists throughout the world to use to assure as much homogeneity among research populations and comparability of research data and results as could be achieved given our limited state of scientific knowledge. It included the statement "This is a working definition. It will be altered if indicated by the results of ongoing research." The Board of Directors of NSAC voted to adopt the definition of the PAB as "official" in July 1977, to answer requests NSAC received from governmental, medical, educational, and other organizations as to "what is autism?" In this way the NSAC definition became written into and referred to in various federal, state, and local laws and regulations. We are certain of this chain of events, from formulation for scientific purposes to advocacy use, because we personally instigated and facilitated the entire process.

2. The authors, in a pejorative footnote (page 178), misrepresent and misinterpret the results of our initial exploratory studies on genetic factors
in autism. They state, "Their data are based on biased samples . . . and inappropriate analyses . . . accordingly, little weight can be attached to their findings; moreover, their own data include some that are inconsistent with the hypotheses . . . it is quite premature to postulate specific models of inheritance before the necessary data for such models are available."

The facts are as follows: In the articles referred to we (a) stressed the preliminary nature of the research, (b) explicitly delineated the ascertainment biases that limit generalizing from our study populations to other autistic populations and autism in general, and (c) explicitly stated that our speculations concerning possible genetic models are highly tentative and meant primarily to serve heuristic purposes. For example, we state in our twin study (Ritvo, Freeman, Mason-Brothers, Mo, & Ritvo, 1985), "it remains to be determined what, if any, proportion of cases are caused by pathogenic genes. However, the assumption that pathogenic genes are present poses the fascinating task of determining where on the gene map they reside, precisely what pathologic ciphers they transmit, and whether we can deduce their presence from clinical clues." In our paper "Evidence for Autosomal Recessive Inheritance in 46 Families with Multiple Incidences of Autism" (Ritvo, Spence, et al., 1985) we state, "The results of this study are limited strictly to the multiple-incidence population ascertained for this study. They cannot be generalized to other multiple-incidence families or to families with only one autistic proband . . . Finally, autism is a syndrome with many possible etiologies. Thus, genetic factors, if operable, may account for only a small subset of patients, whether from single- or multiple-incidence families." And again, "The results of this study are applicable only to the 46 families ascertained for having at least two siblings affected with the syndrome of autism."

3. The authors state, "Whatever benefits there may be from fenfluramine administration, they do not result from normalization of pathologically high serotonin levels."

The facts are these: We do not yet know the specific neurotransmitter systems affected or by what mechanisms fenfluramine produces symptomatic remissions. Research is under way on several continents that we hope will shed light on postulated biochemical abnormalities in autism and other developmental disabilities. It is certainly too soon to claim that abnormal levels of serotonin were normalized or not, and to conclude that this did or did not have anything to do with the observed symptomatic improvements.

4. The authors state, "There have been very few neuropathological studies, but these, too, have shown either quite subtle histological changes or no detectable abnormalities."

The facts are that in 1986 we published the first study based on the NSAC autopsy research project. We reported decreased cerebella Purkinje cell counts in each of the four autistic decedents obtained since the project began in 1976 (Ritvo et al., 1986). A review of the neuropathological litera-