

Clinical evidence that Asperger's disorder is a mild form of autism

Riva Ariella Ritvo^{a,*}, Edward R. Ritvo^b, Donald Guthrie^b, Max J. Ritvo^c

^a*Yale University School of Medicine, Yale Child Study Center, New Haven, CT 06520, USA*

^b*UCLA School of Medicine, The Neuropsychiatric Institute, Los Angeles, CA 90024, USA*

^c*Harvard Westlake School, Los Angeles, CA 90077, USA*

Abstract

Objective: The aim of this study is to obtain clinical evidence to test the hypothesis that Asperger's disorder (AD) is a mild form of autism (AU).

Method: A 78-item Likert scale (the RAADS) was administered to 25 adults with AD and 19 with AU (ages, 18-65 years) to assess presence, type, and duration of symptoms.

Results: The following results were found: (a) subjects with AD and AU have similar symptoms throughout adulthood (responses to 72 of 78 questions were not significantly different); (b) subjects with AD had a significantly fewer total number of symptoms; (c) subjects with AD reported nonsignificantly fewer symptoms in the *DSM-IV-TR* domains of social interaction and repetitive patterns of behavior; and (d) subjects with AD had significantly fewer symptoms in the communication domain.

Conclusions: The data support the hypothesis that AD is a mild form of AU, and that they share a common etiology and developmental neuropathology. It appears warranted in future diagnostic manuals to incorporate AU and AD into 1 diagnostic category such as, "Autism Spectrum Disorder, (with modifiers, severe, moderate, mild, atypical, and Asperger's type)."

© 2008 Elsevier Inc. All rights reserved.

1. Introduction

Shortly after Asperger's [1] description of patients with "autistic psychopathy" was translated into English by Lorna Wing in 1981 [2], debate arose regarding their relationship to patients Kanner [3] described as having "autistic disturbances of affective contact." Some theorized that they were separate disorders, whereas others stated that Asperger's disorder (AD) was a mild form of autistic disorder (AU) [4-8].

In the early 1980s, we began to study the relationship of the 2 disorders in an attempt to clarify the confusion in the field. Freeman et al [9] described children with mild developmental delays and mild symptoms, whom we labeled as having a "forme fruste" of autism. We also reported nondiagnosed parents of autistic children who had mild symptoms themselves [10,11]. These cases suggested the

need to broaden the phenotype of autism beyond those severely impaired cases initially described by Kanner. However, these studies were small and provided future research direction but not conclusive answers.

The initial debate was resolved for practical purposes in the early 1990s with the publication of *ICD-10* [12] and *DSM-IV* [13]. They established "official" clinical parameters for making the distinction between AU and AD based on the time of onset of language and certain cognitive functions. Designating differentiating criteria was done for heuristic purposes, with the expectation that ongoing research would soon provide pathognomonic markers that would determine whether they were distinct disorders or the same, differing only in degree of severity (personal communication, *DSM-IV* committee).

Recently, Baron-Cohen [14] introduced a new theory in an attempt to explain the relationship of the 2 disorders. It proposed that symptoms of AU and AD are not evidence of pathologic development but are normal traits that have become expressed to an extreme degree in certain individuals.

To date, no distinguishing markers have been identified, and the debate continues. One reason is the difficulty in applying the diagnostic criteria for AD as demonstrated in

Location of work: UCLA School of Medicine, Department of Child Psychiatry, Los Angeles, CA.

Previous presentation: A preliminary version of this paper (a poster session) was presented at the American Academy of Child and Adolescent Psychiatry meeting in San Diego, October 2006.

* Corresponding author.

E-mail address: ariella.ritvo@yale.edu (R. Ariella Ritvo).

2 recent studies. Tyron et al [15] carefully rediagnosed 26 children who had previously been diagnosed as having AD. Surprisingly, they were not able to reconfirm the diagnosis in even one of these 26 children. Klin et al [16], in a complex and detailed study, had 2 highly experienced clinicians independently diagnose 65 subjects, 8 to 32 years old. They used 3 definitions of AD (*DSM-IV*, Speech Delay, and a “New System”), as well as *DSM-IV* definitions of AU, Pervasive Developmental Disorder Not Otherwise Specified, and Non-Pervasive Developmental Disorder. They found that only 44% of the subjects received the diagnosis of AD by all 3 of the diagnostic criteria for AD.

The present study was designed to obtain clinical data to help further our understanding of the relationship of AD with AU. We postulated that (a) AD is a mild form (phenotype) of AU; (b) both are caused by the same abnormal genetic programming of brain development; and (c) both express the same developmental brain neuropathology that is manifested clinically by separation (lack of normal coordination) of the 3 main developmental pathways (social interaction, communication, and repetitive behaviors) and by delays, spurts, and plateaus of brain development [17].

The literature supports a common etiology due to abnormal genetic programming: (a) the original suggestions of both Kanner [3] and Asperger [1] that “familial factors are involved”; (b) the high concordance rate in monozygotic twins [18,19] and the high sibling recurrence risk estimate [20]; (c) evidence of delayed maturation of brain volume [21]; and (d) common abnormalities in neurotransmitters (ie, serotonin and melatonin) [22,23]. In addition, an international multicenter study recently identified candidate genes that may be associated with both AD and AU [24].

For this study, 3 specific hypotheses were established for empirical testing. They stated that if AD is a mild form of AU, then (a) similar symptoms will be present in both disorders throughout adulthood, (b) subjects with AD will have fewer overall symptoms than subjects with AU, and (c) subjects with AD will have fewer symptoms than subjects with AU in each of the 3 *DSM-IV-TR* symptom domains [25].

To obtain data on specific symptoms to test these hypotheses, we administered an empirically based 78-item, self-rating scale (the RAADS) [26] to 25 adults with AD and 19 with AU. It uses a developmentally based Likert scale to quantify the presence, distribution, and longevity of specific symptoms.

2. Method

2.1. Subjects

Forty-four subjects were recruited from (a) previously diagnosed patients of the investigators and clinicians known to the authors to be experts in developmental disabilities; (b) national autism and AU and AD support groups; (c) referrals

Table 1
Participant demographics

Participant group	Number	Male/ Female ratio	Mean age	Education high school (%)	Education college (%)	Married (%)
AD	25	2.13	38.2	28	64	17
AU	19	1.71	34.9	42	26	16

from AU and AD diagnostic clinics that were familiar with the project; and (d) advertisements on websites for adults with AU and AD. Subjects were enrolled in the order in which they volunteered (see Table 1 for demographics).

2.2. Diagnostic evaluations and subject assignments

Two diagnosticians on the research team who were blind to each other’s diagnoses evaluated all subjects independently. When the blind was broken, it was found that all their diagnoses were concordant. *ICD-10* [12] and *DSM-IV-TR* [25] criteria for AD or AU were used with the time of onset of language and cognitive functioning as distinguishing criteria. Each evaluation consisted of reviewing prior medical records when available, obtaining a developmental history, and conducting an interview and a mental status examination. Medical records were available on 41 subjects. In addition, parental interviews were conducted to confirm clinical course and history. (Parents of 35 subjects were available for interviews.)

Two months after the initial interviews, all subjects were evaluated using “a gold standard” diagnostic system—the ADI/ADOS. This was done to confirm validity of clinical diagnosis, as well as concurrent validity of *DSM-IV-TR* criteria and the RAADS. All subjects scored above the cutoff level for autism/Pervasive Developmental Disorder Not Otherwise Specified.

The following were the inclusion criteria: (a) a prior diagnosis of AD or AU; (b) a current rediagnosis of AU or AD reached by agreement between the 2 diagnosticians who were blind to each other’s conclusions; (c) confirmation of diagnosis on ADI/ADOS; (d) willingness to participate and to sign the informed consent document; (e) age 18 or older; (f) completed high school; (g) clinical evidence of a nonretarded verbal IQ; (h) demonstrated ability to read, understand, and appropriately answer questions on the scale; (i) a driver’s license; and (j) good general health now and in the past.

The following were the exclusion criteria: (a) a history or clinical evidence of a medical condition other than AU or AD that could affect normal brain development; (b) the presence of any other *DSM-IV-TR* Axis I diagnosis; and (c) taking any psychoactive or other medications that could interfere with cognition or answering the scale in any manner.

2.3. Informed consent

The California Institutional Review Board, Inc, located in Pasadena, CA, approved the consent form and scale (IRB

متن کامل مقاله

دریافت فوری ←

ISIArticles

مرجع مقالات تخصصی ایران

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان دانلود رایگان ۲ صفحه اول هر مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات