

## Developmental Change in Neural Processing of Words by Children with Autism

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This study examined the development of neural processing of auditorally presented words in high functioning children with autism. The purpose was to test the hypothesis that electrophysiological abnormalities associated with impairments in early cortical processing and in semantic processing persist into early adolescence in autistic individuals. Eighteen children with autism and 18 normally developing children participated in the study. Ten of the children in each group were 8–9 years old, and 8 in each group were 11–12 years old ( $n = 36$ ). Lists of words were presented auditorally; half were words belonging to a specified semantic category and half were words outside the category. Results revealed that while early cortical processing abnormalities appeared to resolve with development, children with autism in both age groups failed to exhibit differential semantic processing of in-versus out-of-category words. Further, while 8 year-olds with autism generated a large N4 (a late cognitive ERP component, which is sensitive to semantic deviance from a context) to words in both stimulus classes the 11 year-olds showed attenuated N4 relative to normal controls in response to both stimulus types. An attempt is made to integrate findings with current cognitive theories toward a parsimonious explanation of semantic classification deficits in autism.

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**KEY WORDS:** Intelligence; Shortforms; Assessment.

Autism is a developmental, neurobiological disorder of heterogeneous etiology that becomes manifest by or before the age of three (DSM-IV). It involves dysfunction of one or more brain systems, and is characterized by a constellation of cognitive and behavioral deficits, such as failure to achieve typical developmental milestones in verbal and non-verbal communication, imaginative play, and peer relationships. Preoccupations and circumscribed interests are also observed in the child with autism. These impairments may be present to a greater or lesser degree, and can occur in the presence or absence of mental retardation. Children with autism are also frequently characterized as having auditory

processing and motor impairments. However, a “triad” of symptoms—impaired socialization, cognitive rigidity, and deficits in verbal communication—is consistently observed in all individuals with autism, regardless of IQ or severity of symptoms (Bartak & Rutter, 1976; Wing & Gould, 1979). An important goal of neurobiological, neuropathologic, and brain imaging studies has been to relate the pattern of cognitive and behavioral deficits observed in autism to underlying neural or structural aberrations (Courchesne, 1987; Kemper & Bauman, 1993; Piven & O’Leary, 1997).

Language deficits in children with autism vary widely in association with a number of developmental variables such as IQ, age of onset, language acquisition history, and remedial intervention (Baltaxe & Simmons, 1992; Baron-Cohen, 1988). However, high-functioning children with autism (individuals with an IQ greater than 70: Happé & Frith, 1996; Rapin, 1997) exhibit a distinctive pattern of strengths and

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weaknesses; while they may or may not show deficits in phonology and syntax, they are invariably impaired with respect to the semantic and pragmatic aspects of language (Tager-Flusberg, 1989; Twachtman-Cullen, 1998).

Semantic deficits are observable in individuals with autism in several distinct ways: (1) failure to employ semantic information to aid encoding of verbal material and later recall of word sequences (Bowler, Matthews, & Gardner, 1997; Hermelin & O'Connor, 1967, 1970; Tager-Flusberg, 1985b, 1991; Toichi & Kamio, 1998); (2) tendency to employ syntactic word order strategies rather than semantic comprehension strategies in interpreting connected speech (Paul, Fisher, & Cohen, 1988; Strohner & Nelson, 1974; Tager-Flusberg, 1981); (3) failure to interpret words according to semantic context (Minshew & Rattan, 1992; Tsai, 1992; Twachtman-Cullen, 1998); and (4) tendency to produce less prototypic exemplars of categories than normally developing children in word fluency tasks (Dunn, Gomes, & Sebastian, 1996). Thus, semantic deficits are evident at the lexical level as well as at the level of connected speech. The focus of the current investigation is on aspects of semantic processing that are deficient at the lexical/conceptual level.

Semantic deficits appear to persist into adolescence and adulthood even in high-functioning individuals, who generally exhibit a decline in the severity of other symptoms over time. This is particularly true for comprehension of verbal/auditory information (Paul & Cohen, 1986; Strandburg *et al.*, 1993; Tager-Flusberg, 1991; Toichi & Kamio, 1998).

However, as with other areas of cognitive functioning, children with autism have pockets of preserved ability for semantic processing. Evidence from past studies suggests that semantic processing impairments in high functioning autism exist at the more complex, higher-order level of language organization. Children with autism are able to correctly categorize objects and words according to basic and superordinate categories (Tager-Flusberg, 1985a; 1985b), and along perceptual and functional parameters (Ungerer & Sigman, 1987), with accuracy comparable to that of typically developing children. Children with autism demonstrate susceptibility to the Stroop effect and the abstract/concrete word effect (where concrete words are easier to read than frequency- and length-matched abstract words) to the same degree as age and reading level-matched typically developing controls and children with dyslexia (Bryson, 1983; Eskes, Bryson, & McCormick, 1990; Snowling & Frith, 1983). This has been

interpreted to mean that they can automatically access the meanings of single words for simple concepts (at least in the visual modality).

In two recent studies, comparable performance on semantic processing tasks was reported for adolescents and adults with autism and neurotypical controls. Kamio and Toichi (2000) presented either pictures or printed words as the primes, which were followed by a visual word fragment that had either a categorical, non-categorical, emotional, or physical/sensational semantic relationship to the prime or was completely unrelated. The task was to verbally complete the word fragment. Both groups performed significantly better when the fragments were related to the prime word and demonstrated comparable accuracy. Interestingly, the autism group benefited significantly more from the picture primes than from the word primes, whereas the control group benefited equally from each type of prime. The interpretation proposed by the authors is that pictorial semantic access in autism is superior to verbal access. In a similar paradigm Toichi and Kamio (2001) found, in the autism group only, a significant positive correlation between scores on the non-verbal intelligence measures and percent correct word identification. This was interpreted to indicate that the two groups may achieve the same accuracy rate via different neurocognitive strategies; specifically, that the autism group might engage visual analytic processes to a greater extent.

These findings suggest that basic semantic relationships among words facilitate word recognition in adolescents and adults with autism. However, it must be noted that only correct response rates, rather than reaction times were measured, so that these studies did not assess semantic distance among lexical items and their findings may not represent automatic facilitation of semantic processing.

Although there is significant evidence that individuals with autism comprehend basic concepts and word meanings, they do not appear to extract and apply commonalities among category members. In one of the most comprehensive and well-controlled neuropsychological studies to date, Minshew, Goldstein, and Siegel (1997) showed that adults with autism do not demonstrate deficits in basic information acquisition, or when processing information requiring procedural skills. Rather, their impairments are limited to more complex information processing. Failure to extract the common features of the meaning of words or concepts has implications not only for use of semantic information to aid learning and memory, but also for the development of a

conceptual base. That is, the organizational structure of the lexicon develops in an aberrant way in children with autism. In a word fluency study (Dunn *et al.*, 1996) high-functioning children with autism were asked to provide exemplars of a specific superordinate category. While the children with autism accessed category members as rapidly and accurately as language matched children with typical development and with developmental language disorders, the words they provided were significantly less prototypic (e.g., “ocelot,” and “hedgehog,” rather than more prototypical examples of animals, such as, “dog,” and “cat.”). This suggests that semantic organization *within* lexical categories (rather than at the categorical level) may be aberrant in children with autism. This hypothesis parallels that of Klinger and Dawson (1995, 2001), who propose that, while children with autism can categorize, they do so via a different mental process than children with normal development. Using a visual pattern paradigm, these investigators found that children with autism and typically developing children could all learn new categories when given rules about category membership. However, when the children had to extract commonalities between exemplars in order to determine category membership (i.e., had to form prototypes), only the typically developing children could learn the new category.

In sum, although children with autism can classify by basic and superordinate category and are susceptible to the Stroop and Abstract/Concrete word effects, it should not be assumed that these skills are accomplished via the same cognitive processes, or by the same neurophysiologic processes, as in children with typical development.

Neurophysiology provides a sensitive method for investigating neural mechanisms underlying the cognition. Event-related potentials (ERPs) offer a non-invasive, continuous, real-time measure of neurophysiological activity that is time-locked to the onset of the stimulus. It is therefore possible to monitor the immediate effects of a particular experimental manipulation as well as its consequences downstream. Furthermore, ERPs are particularly valuable for the study of clinical populations, since reliable effects can be obtained in passive listening paradigms, in the absence of a behavioral response.

The N4 is a late cognitive ERP component, which is sensitive to semantic deviance from a context. Single-word priming studies of cortical ERPs in normal adults and children have shown that N4 amplitude is largest in response to the second of two unrelated words (Bentin, McCarthy, & Wood, 1985),

and small or non-existent in response to the second in a pair of similar words (Coles & Rugg, 1995). Dunn, Vaughan, Kreuzer, and Kurtzberg (1999) found that N4 could be evoked in typically developing children in response to non-animal words when instruction set and stimulus list composition set up an expectancy for animal words. N4 is most strongly evoked by words whose meaning is unrelated to, or not predicted by, the context set up by a prior word or words (Kutas & Hillyard, 1980). N4 also appears to reflect the extent of the brain's search through the lexicon during the process of recognizing the meaning of a word, as observed in a larger N4 amplitude in response to words that are less predicted by the context in which they are presented (Nobre & McCarthy, 1995).

Very few studies have been undertaken to provide a developmental profile of the maturation of the N4 component. Byrne *et al.* (1999) found a significant N4 effect in typically developing children in response to word–picture pairs presented visually that were incongruous (the word did not match the picture), and no N4 for congruous word–picture pairs. This finding was evident in each of the four age groups (5–6, 7–8, 9–10 and 11–12 years). Gonzalez-Garrido *et al.* (1997) assessed differential N4 responses in typical children from three age groups: ages 7–8, 9–10 and 11–12 years, and found no difference in amplitude across all age groups. Onset latency did decrease with age.

Likewise, there are few studies examining the N4 component in children with autism. While typically developing children generate an increase in N4 amplitude in response to unexpected changes in stimulus location, children with autism do not (Verbaten, Roelofs, van Engeland, Kenemans, & Slangen, 1991). In our previous auditory N4 study (Dunn *et al.*, 1999), young children with autism (mean age 8–10) failed to demonstrate differential neural processing of auditorally presented words as indexed by N4 in a task that required them to distinguish category members from non-category members. The N4 effect was elicited in children with typical development in response to this task.

Interestingly, N4 abnormalities in these children were associated with abnormal delays in the latency of an earlier ERP component, the N1c (also known as the T-complex) (Dunn *et al.*, 1999). Other studies of N1c in individuals with autism have found that this component is consistently delayed in latency over the left hemisphere in response to verbal auditory stimuli, but not in response to tones (Dunn & Gravel, 1999; Klein *et al.*, 1995; Narita & Koga, 1987). It

should be noted, however, that Bruneau, Roux, Adrien, and Barthélémy (1999) found an abnormal latency shift in N1c to tones in very young children with autism (ages 4–8 years).

The N1b and N1c components of the ERP appear early after stimulus onset. N1b is seen at approximately 100–110 milliseconds, N1c appears at approximately 130–170 milliseconds. Topographically, N1b is observed maximally over fronto-central areas of the scalp, while N1c peaks over temporal regions. The N1b is primarily associated with activation of generators in the supratemporal plane of the auditory cortex (Vaughan & Ritter, 1970). N1c is generated from sources in the auditory association cortex on the lateral aspect of the temporal lobe (Wood & Wolpaw, 1982). These components are believed to be elicited by the physical aspects of the stimulus, since these responses vary with changes in the intensity or frequency of the stimulus, and do not appear in response to an omitted stimulus (Courchesne, 1987). Studies examining the N1b component in persons with autism identified no abnormal prolongation of latency in response to verbal stimuli (Bruneau, Garreau, Roux, & Lelord, 1987; Kemner, Verbaten, Cuperus, Camfferman, & van Engleland, 1995; Lincoln, Courchesne, Harms, & Allen 1995; Novick, Vaughan, Kurtzberg, & Simson, 1980). The functional significance of this latency shift over left temporal lobe (N1c) in response to language but not tones, and in the absence of abnormal N1b latency, is surmised from current knowledge of neural generators associated with these components, located on the lateral surface of the temporal lobes, implicating dysfunction of auditory association cortex in children with autism. Thus it has been hypothesized that this pattern of impairment could be indicative of delayed transmission of verbal auditory information along the neuronal pathways or synaptic connections in the auditory association cortex in children with autism (Bruneau *et al.*, 1999).

The current study examines whether N4 abnormalities persist and whether the association of slowed early cortical processing and N4 abnormalities persists with development.

## METHOD

### Participants

Twenty-two children with autism and 22 children with typical development participated in the

study. Four children from the autism group had to be excluded from the analyses due to impaired peripheral audition at the time of the evaluation (see Discussion section); the final number of children in each group was 18. These two groups were further divided by age: 8–9 year-olds ( $n=10$ ), and 11–12 year-olds ( $n=8$ ). The rationale for selecting these two age ranges was based on findings from our earlier investigation with younger children with autism (ages 7–10 years; Dunn *et al.*, 1999). A goal of the present study was to replicate those results, as well as extend them by including a group of older children with the same diagnosis in order to look for the presence of differential N4 processing with development.

No child had a history of seizures or other medical or neurologic disorder. All children in the final subject groups had normal peripheral hearing bilaterally and middle ear function. If a child displayed either a conductive or cochlear hearing loss, testing was suspended. In the case of a conductive loss (secondary to otitis media with effusion), the parent was informed and advised to seek medical/otologic consultation. Following resolution of the condition, the child returned to the protocol.

Children with autism were diagnosed through interview with the primary caregiver using the Autism Diagnostic Interview-Revised (ADI-R, Lord, Rutter, & LeCouteur, 1994), direct observation of the child using the Autism Diagnostic Observational Scale (ADOS, Lord *et al.*, 1989), and clinical observation based on DSM-IV criteria. Only children meeting full criteria for autism based on ADI, ADOS and clinical interview were included in the autism group for this study. Autistic participants within the normal range of nonverbal intelligence were tested, since this group affords the possibility of discovering differences in brain functioning in autism without the potential confound of mental retardation.

Control subjects were drawn from classes for normally developing children in the greater New York metropolitan area. None had a history of special education services. Age appropriate ability in cognition, language, and academic skills was confirmed in the control children by normal range scores on a battery of standardized tests. Complete developmental history obtained from the parents of control children confirmed normal pre- and post-natal history, and age-appropriate achievement of developmental milestones. The Wing Autistic Disorders Interview Checklist (WADIC; Wing, 1996) probes for (1) impairments in social relatedness, (2) impairments in social communication, and (3)

restricted or repetitive activities. Any potential control child with two or more positive responses on this checklist would be excluded from participation. (In our sample, none of the control children had any positive responses reported on the WAD-IC). Additionally, scores in at least the average range on both the Vineland Socialization and Communication Domains (Sparrow, Balla, & Cicchetti, 1984), a parent interview, were required for assignment to the control group.

Two groups of children with autism were matched by chronological age and non-verbal IQ with 2 groups of children with typical development, in a cross-sectional study design. Mean age of the younger group of children with autism was 9–1 ( $SD = 6$  mo., range = 8.33–9.83 years), and the mean age of the 8–9 year-old controls was 9–4 ( $SD = 4$  mo., range = 8.58–9.75 years). For the older children with autism, the mean age was 11–6 ( $SD = 7$  mo., range = 10.5–12.5 years); the controls had a mean age of 11–6 ( $SD = 6$  mo., range = 10.42–12.42 years). See below for a discussion and analysis of differences in cognitive skills between the groups.

In terms of gender and handedness, the 8 year-old groups were identical, with six males, 4 females, and 10 right-handed children in each group. The 11 year-old groups were slightly different from each other. In the autism group, there were six males, two females, six right-handed and two left-handed children. In the control group, there were five males, three females, seven right-handers and one left-handed child. All children in the study were monolingual (English) speakers.

## PROCEDURE

### Cognitive and Language Testing

In addition to diagnostic interviews, all children were administered a battery of standardized cognitive and language tests. This comprehensive testing was done in order to characterize our autism samples and to ascertain average range abilities in our typically developing samples. The Stanford-Binet (4th ed.) (Thorndike, Hagen & Sattler, 1986) was used to obtain overall, non-verbal, and verbal intelligence quotients. Children received a short battery of language tests including the Clinical Evaluation of Language Fundamentals—Third edition (CELF-3) (Semel, Wiig, & Secord, 1995), the Vineland Adaptive Behavior Scales—Communication and Socialization Domains (Sparrow *et al.*, 1984), and the Peabody Picture Vocabulary Test—III (Dunn & Dunn, 1981). Handedness was assessed with the Harris Test of Laterality (Harris, 1974).

Cognitive and language data showing the standard scores achieved by each group are displayed in Tables I and II. A multivariate analysis of variance between the 8 year-old groups revealed that the children in the autism group obtained significantly lower scores on full-scale and verbal IQ, Peabody Picture Vocabulary Test, and the Vineland Communication and Socialization Domains. There were no significant differences between the 8 year-old groups on non-verbal IQ and receptive language. The 11 year-old groups differed significantly on full-scale IQ, overall receptive language, and Vineland Communication and Socialization Domains. While originally matched only on non-verbal IQ, the 11 year-old

**Table I.** Cognitive and Language Data for 8 Year-Olds: Mean Standard Scores, Standard Deviations, and Between-group  $p$ -values

	Autism 8–9	Control 8–9	$F$ & $p$ -values
Full-scale IQ	96 (21), range: 77–148	113 (9), range: 103–127	$F[1,18]$ 4.734, $p = .043$
Verbal IQ	92 (23), range: 65–151	116 (8), range: 97–126	$F[1,18]$ 10.064, $p = .005$
Non-verbal IQ	98 (14), range: 76–126	112 (16), range: 97–142	NS
Receptive lang. <sup>a</sup>	93 (31)	110 (14)	NS
Word classes <sup>b</sup>	93 (21)	117 (10)	NS
Peabody <sup>c</sup>	9 (4)	11 (3)	$F[1,18]$ 10.52, $p = .005$
Vineland, Com <sup>d</sup>	85 (18)	106 (12)	$F[1,18]$ 6.833, $p = .018$
Vineland, Soc <sup>e</sup>	74 (12)	110 (11)	$F[1,18]$ 39.483, $p = .000$

<sup>a</sup> Note: The Stanford-Binet Test of Intelligence, 4th Edition, was used to obtain IQ scores.

<sup>b</sup> Receptive language subdomain.

Word Classes, CELF-3.

<sup>c</sup> Peabody Picture Vocabulary Test.

<sup>d</sup> Vineland Adaptive Behavior Scales—Communication Domain.

<sup>e</sup> Vineland Adaptive Behavior Scales—Socialization Domain.

**Table II.** Cognitive and Language Data for 11 Year-Olds: Mean Standard Scores, Standard Deviations, and Between-group *p*-values

	Autism 11–12	Cntrl 11–12	<i>F</i> & <i>p</i> -values
Full-scale IQ	93 (17), range: 65–116	109 (12), range: 93–125	$F[1,14] 4.724, p = .047$
Verbal IQ	92 (23), range: 55–120	111 (13), range: 92–134	NS
Non-Verbal IQ	95 (13), range: 74–110	105 (13), range: 85–125	NS
Receptive lang. <sup>a</sup>	85 (25)	109 (11)	$F[1,14] 6.659, p = .022$
Word classes <sup>b</sup>	9 (5)	12 (3)	NS
Peabody <sup>c</sup>	102 (26)	118 (21)	NS
Vineland, Com <sup>d</sup>	74 (14)	109 (7)	$F[1,14] 40.423, p = .000$
Vineland, Soc <sup>e</sup>	64 (13)	99 (13)	$F[1,14] 29.119, p = .000$

Note: The Stanford–Binet Test of Intelligence, 4th Edition, was used to obtain IQ scores.

<sup>a</sup> Receptive language subdomain.

<sup>b</sup> Word Classes, CELF-3.

<sup>c</sup> Peabody Picture Vocabulary Test.

<sup>d</sup> Vineland Adaptive Behavior Scales—Communication Domain.

<sup>e</sup> Vineland Adaptive Behavior Scales—Socialization Domain.

groups in our sample were also comparable on verbal IQ. The lack of difference in verbal IQ between the older children is consistent with findings from recent studies, which indicate that the discrepancy between verbal and non-verbal IQ in children with autism (i.e., non-verbal IQ > verbal IQ) lessens with age (Mayes & Calhoun, 2003).

Additionally, all the children with autism showed the typical pattern in high-functioning autism of lower Comprehension scores relative to their own scores on tests of non-verbal reasoning, such as Pattern Analysis (Goldstein, Beers, Siegel, & Minshew, 2001; Lockyer, Lazenby, Donnelly, Wilkenson, & Schoonheydt, 1999).

## Electrophysiologic Testing

### Stimuli

Stimuli were auditorally presented single words. They were delivered by a female voice, digitized at a rate of 44.1 kHz and edited to 400 milliseconds in (msec) duration. Stimuli were delivered at 83 (=/-2) dB sound pressure level (SPL) through insert earphones (ER-3A; 10 Ω). Stimulus levels were calibrated using a Bruel & Kjaer sound level meter and HA2 (2 cc) coupler.

### Procedure

Fifty percent of the words were animal labels and 50% were labels for non-animal items. All words were C–V–C syllables beginning with stop consonants; the final consonant provided the distinctive cue for whether a stimulus was an animal word or a

non-animal word. In-category and out-of-category words were matched pairwise for initial consonant and vowel, and onset of the cue for determination of category membership. Mean word frequency (Carroll, Davies, & Richman, 1971) for in-category words is 195.35 ( $SD = 267.24$ ), and for out-of-category words mean frequency is 367.35 ( $SD = 469.99$ ). Frequency between the two categories did not differ significantly ( $F[1,66] 3.441, p = .068$ ).

The stimuli were presented in 5 runs of 38 randomly ordered single words for a total of 190 stimuli, 95 targets and 95 non-targets. The interstimulus interval (ISI) was variable, ranging from 1400 to 2100 milliseconds. Table III shows the mean number of trials in each average for each group.

### Electrode Placement and Recording Techniques

An electrode cap (Electro-Cap International) was used with tin electrodes at sites determined by the International 10-20 system (American Electroencephalographic Society, 1990). Thirty-two electrodes were applied, referenced to the nose. Vertical eye movements were recorded with a bipolar configuration of FP2 and an electrode below the right eye. Horizontal eye movements were monitored at F7 and F8. All

**Table III.** Mean Number of Trials per Average (*SD*)

	In-category	Out-of-category
Autism, 8–9	38 (9)	31 (9)
Control, 8–9	65 (26)	52 (22)
Autism, 11–12	53 (29)	42 (23)
Control, 11–12	61 (25)	46 (18)

impedances were maintained below 5 kΩ. The EEG was amplified 50k with filter settings of 0.05–70 Hz. The continuous EEG for all channels was monitored during the recordings so that problems with electrodes could be identified and corrected and feedback about excessive motor movement could be given to the subject.

Digital stimulus markers were generated by a stimulus delivery computer program and sent to a Neuroscan signal-averaging computer for tagging EEG samples (single sweeps) associated with each stimulus presentation. Analysis time was 1500 milliseconds. Pre-stimulus baseline was 100 milliseconds. ERPs were averaged off line according to stimulus class within a run (i.e., words that fit with the semantic context vs. words that did not). Artifact rejection was done offline by software that detected and marked trials with peak deflection that exceeded 100 μV produced by eye or muscle movement. Only artifact-free trials were included in the averaged ERPs.

*Behavioral Procedure*

During ERP recording, the child was instructed to listen to the list of words and told that some of the words would be animal labels and some would not. The child was instructed to stare at a fixation point as s/he listened. Between each run, all participants were reminded to listen for animal words. The purpose of removing the requirement of a behavioral response during recording of ERPs was to minimize the effects of the P3 component on the morphology of the N4. An experimenter sat with the child throughout each run to ensure minimal physical movement, and continuous attention to task. After each run the children were given short breaks, during which they were encouraged to rest their eyes, blink, stretch, and yawn.

The child’s ability to classify the words as belonging to the specified category was assessed in a separate task. In the behavioral task, the children listened to words through ear inserts and were instructed to press a button whenever they heard a

target that belonged to the specified category. The categories used were animals and foods presented in separate lists. In each list 50% of the words belonged to the category and 50% were distracter words where the distinctive cue for determining whether the word was in category or out was the final consonant. Results of this task (mean reaction time, hits, misses, and false alarms) are shown in Table IV. The results of a MANOVA between the 8 year-old groups suggest very similar rates of responding among these children ( $F[1,18] .012, p = .915$ ). This was also true of the 11 year-olds ( $F[1,14] 2.149, p = .165$ ). The apparently elevated rate and variability of false alarms in the group of older children with autism appears to have resulted from impulsive responding in 4 of the 8 children (who responded with a button press to non-category members between 22 and 67 times over the course of the five runs), and a very low rate of false alarms in the rest of the children.

*ERP Data Analysis*

Offline average ERPs for each subject for each condition within each run were constructed. Grand mean waveforms were constructed and examined for each diagnostic group, for each condition.

*ERP Variables*

N4 was quantified with area measures (i.e. mean amplitude within a set latency window). Early latency ERP components, N1b, and N1c were quantified through baseline to peak amplitude and peak latency measurement. Based on the peak of N4 in the grand means from all groups, the latency window set for N4 was from 353 to 653 milliseconds post-stimulus onset. Measurements were taken at 32 standard electrode locations: FPZ, FZ, CZ, PZ, OZ, FP1, FP2, F3, F4, F7, F8, FC1, FC2, FC5, FC6, T3, T4, T5, T6, C3, C4, CP1, CP2, CP5, CP6, P3, P4, O1, O2, EOG, left and right mastoids, using nose as reference, according to the augmented International System (American Electroencephalographic Society, 1990). Repeated measures analyses of variance (ANOVA)

**Table IV.** Average Reaction Times, Percent Hits, Misses, and False Alarms for Behavioral Task

	Avg. RT, milliseconds (SD)	% Hits (SD)	% Misses (SD)	% False Alarms (SD)
Autism, 8–9	860 (9)	81.05 (13.21)	19.08 (13.14)	8.73 (15.83)
Control, 8–9	860 (8)	91.32 (3.41)	8.55 (3.31)	5.00 (5.25)
Autism, 11–12	800 (8)	91.12 (6.84)	9.05 (6.88)	10.36 (24.85)
Control, 11–12	790 (5)	93.59 (2.53)	6.25 (2.49)	2.85 (4.75)

were employed to evaluate differences between groups (age and diagnosis) and in-category vs. out-of-category conditions. For repeated measures with greater than one degree of freedom the Geisser–Greenhouse procedure was used to correct for violations of the sphericity assumption (Geisser & Greenhouse, 1959).

## RESULTS

### Behavioral Measure

While there was no behavioral response required during the semantic classification ERP paradigm, behavioral responses were measured for each participant in a separate task, as described above. Table IV shows the mean reaction times, as well as percentage of hits, misses, and false alarms for each group. There were no significant differences between the groups along these parameters.

### N4 Component

Figures 1–4 show the topographical distribution of the differences in N4 amplitude of in-versus out-of-category responses within each of the four groups. All statistical analyses were performed using

data from the 11 electrode sites where N4 was largest: CZ, FZ, PZ, C3, C4, CP1, CP2, F3, F4, FC1, and FC2. A repeated measures ANOVA was used in all analyses. Due to the small sample sizes, power values are provided for the within groups analyses.

### Within Groups

Figure 1 shows waveforms from the 8 year-old control group. While they exhibit a negativity at approximately 400 milliseconds post-stimulus for in-category words, a clear, more negative-going waveform is observable within the same time frame in response to the out-of-category words. Repeated measures ANOVA yielded a significant main effect of condition ( $F[1,9] = 7.654$ ,  $p = .022$ , Power,  $.05 = .693$ ).

In Fig. 2, the ERP responses from the 8 year-old children with autism are shown. Similarly to the 8 year-old controls, these children exhibit a negativity at approximately 400 milliseconds post-stimulus. Unlike their controls, there is no N4 effect; stimuli outside the constraints of context did not elicit an increase in N4 amplitude. In fact, the reverse pattern is observed in the grand means, with in-category stimuli eliciting the larger response. However, statistical

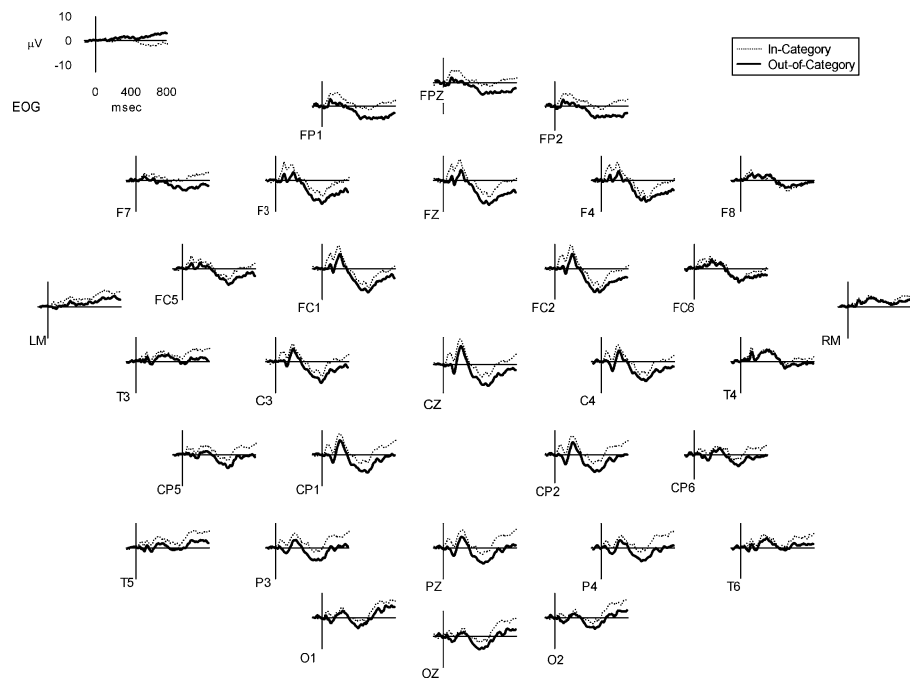


Fig. 1. Eight–nine year-old controls: topographical distribution of N4 responses: in-category vs. out-of-category.



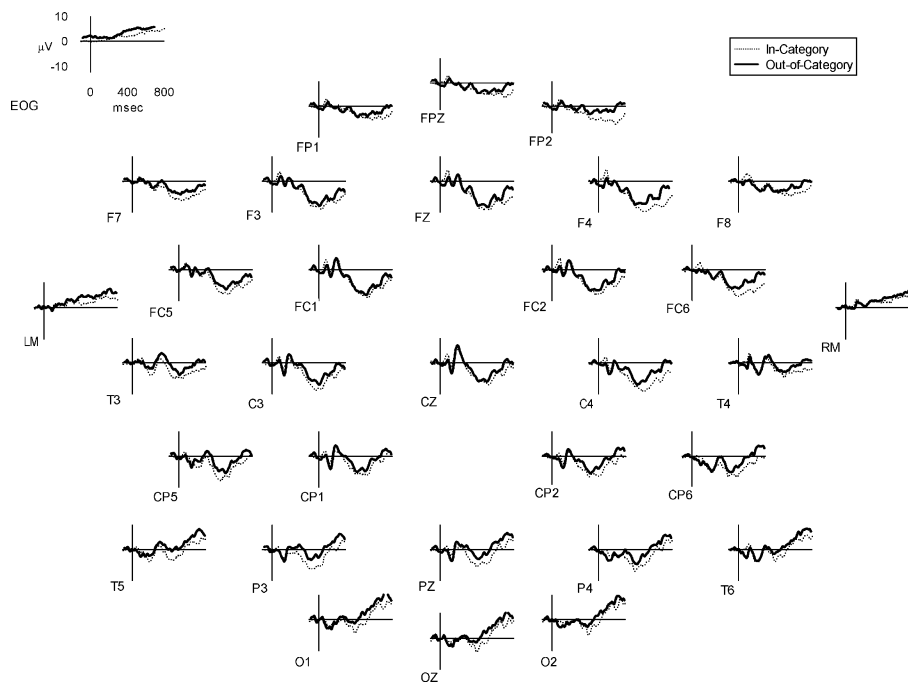


Fig. 2. Eight–nine year-old children with autism: topographical distribution of N4 responses: in-category vs. out-of-category.

analyses of the difference between conditions yielded non-significant values ( $F[1,9] = 0.670$ ,  $p = .434$ , Power, .05, = .114).

Figure 3 shows the ERPs of the 11 year-old controls. In this figure, the differential N4 effect can clearly be seen centrally and bilaterally at fronto-

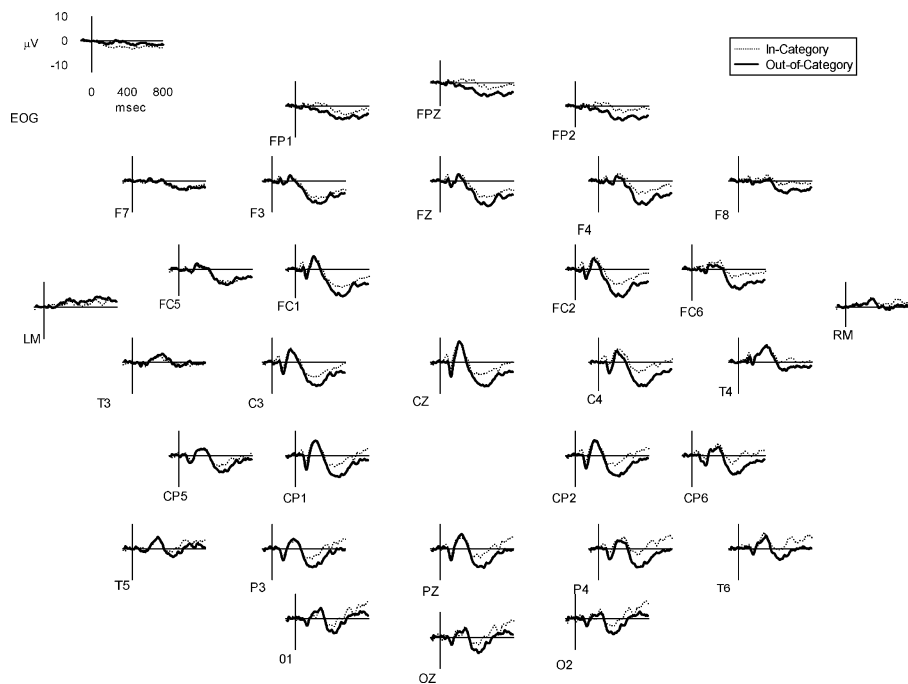


Fig. 3. Eleven–twelve year-old controls: topographical distribution of N4 responses: in-category vs. out-of-category.

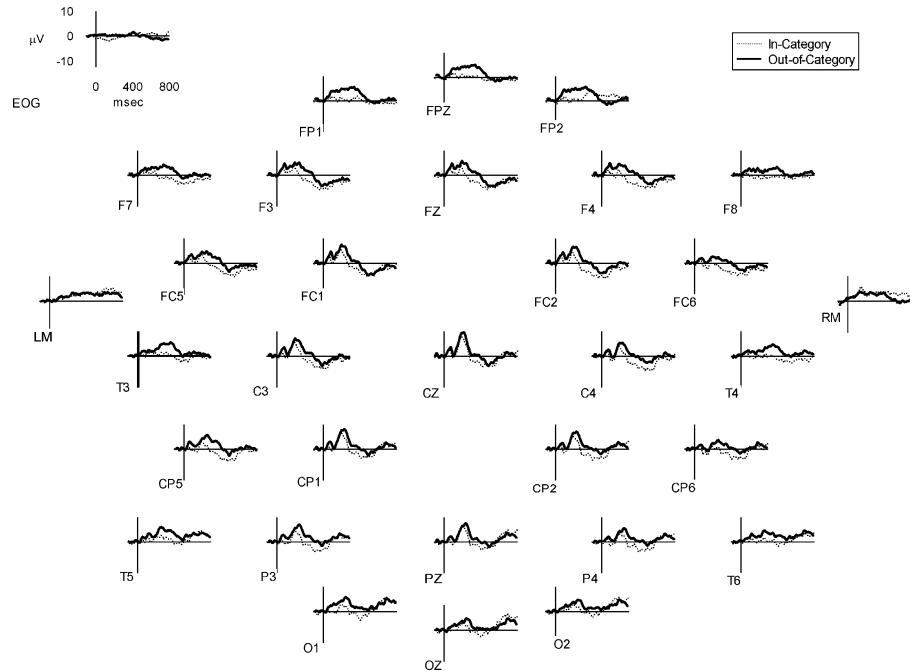


Fig. 4. Eleven-year-old children with autism: topographical distribution of N4 responses: in-category vs. out-of-category.

central and centro-parietal areas. Amplitude is larger over right hemisphere. The repeated measures ANOVA revealed a significant main effect of condition ( $F[1,7] = 11.794, p = .011, \text{Power}, .05, = .836$ ).

Figure 4 shows N4 responses from the 11-year-old children with autism. Attenuation of the waveform amplitude is readily observable. The reversed pattern of in-category responses appearing larger in amplitude than out-of-category responses is again evident in the grand means. Statistical analyses of the difference between conditions were non-significant ( $F[1,7] = 0.188, p = .677$ ).

#### Between Groups: Analyses by Condition and Age Group

*Eight year-olds:* A repeated measures ANOVA examining amplitude of N4 in both conditions with responses at all 11 electrode sites revealed a significant condition  $\times$  group interaction ( $F[1,18] = 6.052, p = .024$ ). This interaction was due entirely to the difference between the two groups in their responses to in-category stimuli ( $F[1,18] = 7.902, p = .012$ ) with the responses of the children with autism to in-category stimuli being larger. There was no statistical difference between the groups in their responses to out-of-category stimuli ( $F[1,18] = .123, p = .730$ ).

*Eleven year-olds:* A repeated measures ANOVA combining both conditions with responses at all 11 electrode sites revealed a significant condition  $\times$  group interaction ( $F[1,14] = 6.600, p = .022$ ). Further analysis (repeated measures ANOVA) indicated a trend toward a difference between the two groups in their responses to out-of-category stimuli with typical controls generating the larger N4 responses to the out-of-category stimuli ( $F[1,14] = 3.464, p = .08$ ).

#### N1b and N1c Components

*N1b:* Table V shows the mean latencies and amplitudes of the N1b component for all four groups, for both in-category and out-of-category conditions, as measured where maximal, at CZ. Repeated

Table V. N1b<sup>a</sup>: Mean Peak Latency, Amplitude

	In-category latency, milliseconds (amplitude, $\mu\text{V}$ )	Out-of-category latency, milliseconds (amplitude, $\mu\text{V}$ )
Autism, 8–9	118 (–1.9)	116 (–3.0)
Control, 8–9	110 (1.6)	120 (–5.5)
Autism, 11–12	111 (–2.6)	111 (–1.1)
Control, 11–12	108 (–2.9)	111 (–5.8)

measures analysis of variance yielded no significant differences within or between the four groups, for latency or amplitude, of the N1b component.

*N1c*: Tables VI and VII show the mean latencies and amplitudes of the N1c component measured at T3, T4, T5, and T6 for all four groups, for both in-category and out-of-category conditions. Repeated measures analysis of variance between the groups showed a significant group  $\times$  condition interaction ( $F[3,31] 5.281, p = .005$ ) for latency of N1c. *Post hoc* analysis using the Scheffé criterion revealed that the latency of N1c in the 8 year-old group with autism was significantly delayed, compared with all other groups. This occurred in their responses to in-category stimuli (animal words), at T3 (on average 48 milliseconds delay) and T5 (on average 63 milliseconds delay) (left hemisphere only). Significance levels at T3 and T5 were well within the .05 criterion for all comparisons between the 8 year-old group with autism and each of the other three groups. There was no statistically significant difference in N1c latency ( $F[1,13] 0.456, p = .511$ ) or amplitude ( $F[1,13] 1.308, p = .273$ ) between the 11 year-old children with autism and their typically developing controls.

A correlation analysis was done to assess whether the speed of early cortical processing is related to semantic expectancy, as indexed by the latency of N1c and amplitude of N4. These parameters were transformed to z-scores, and bivariate Pearson product moment correlation coefficients

were obtained. In all 4 groups, a relationship between latency of N1c and amplitude of N4 was not supported. These results must be interpreted with caution due to the small number of subjects in the analysis.

**DISCUSSION**

**N4 Component**

Findings from the current study lend neuro-physiologic support to the behavioral observations, as described in the introduction to this paper, that children with autism have persistent deficits in the semantic processing of auditory words as they enter pre-adolescence. In both the 8 and 11 year-old control groups there was a clear enhancement of the N4 ERP waveform in response to out-of-category words relative to in-category words. On the other hand in both the 8 and 11 year-old children with autism, the difference in N4 response between the two conditions was negligible.

Although aberrant processing was evident in both age groups with autism, developmental changes were noted between the 8 and 11 year-olds in both groups. While all controls produced a statistically significant N4 effect, a large negativity in response to in-category words was evident in the 8 year-old controls' grand mean waveforms as well. In the 11 year-old control group, N4 was more attenuated in response to in-category words than in response to out-of-category words. The 8 year-olds with autism produced a larger N4 to the in-category words than did their normal controls; in fact their responses to animal words were as large as their responses to non-animal words. For the 8 year-old children with autism, all words appear to be processed in a way that is detached from context, even when they were given the explicit categorical context. Conversely, the 11 year-olds with autism generated a significantly attenuated N4 in response to all stimuli; in this case it is as if all words were expected. Although there is a persistent lack of differential processing of words that fit and did not fit with lexical context in children with autism, overall changes in the amplitude of N4 perhaps reflect changes in expectancy based on more experience with language with increasing age.

**N1 Component**

The early cortical component N1c was delayed in latency in the 8-year-old children with autism over

**Table VI.** N1c: 8 Year-Olds' Mean Peak Latency (Amplitude)

	T3	T4	T5	T6
<i>In-category</i>				
Autism, 8-9	202 (-4.9)	219 (-4.7)	188 (-4.9)	187 (-5.0)
Control, 8-9	154 (0.6)	156 (1.0)	161 (1.4)	165 (-0.3)
<i>Out-of-category</i>				
Autism, 8-9	160 (-3.3)	168 (-3.9)	160 (-4.3)	169 (-4.9)
Control, 8-9	154 (-3.5)	157 (-1.7)	160 (-3.9)	164 (-3.6)

**Table VII.** N1c: 11 Year-Olds' Mean Peak Latency (Amplitude)

	T3	T4	T5	T6
<i>In-category</i>				
Autism, 11-12	153 (-1.5)	156 (-1.8)	154 (-1.4)	156 (2.3)
Control, 11-12	149 (-1.3)	158 (.36)	151 (-1.7)	160 (-1.2)
<i>Out-of-category</i>				
Autism, 11-12	159 (.10)	163 (0.8)	163 (.58)	165 (1.2)
Control, 11-12	154 (-1.5)	154 (-.96)	150 (-2.1)	158 (-2.9)

left temporal regions, in response to in-category words. The 11-year-old children with autism showed no such delay. This finding suggests that (1) speed of early cortical processing of auditory verbal stimuli improves with age in autism, and (2) aberrant contextual processing of words is not an immediate consequence of slowed early cortical auditory processing, (although, the role of slowed early cortical processing at a young age in the development of aberrant contextual processing remains to be investigated).

### Cognitive Explanations

The findings from this study provide an opportunity to assimilate empirical findings and theoretical work across several sources, in an attempt to parsimoniously explain semantic deficits in autism. On a cognitive level, if high-functioning children with autism learn basic and superordinate categories, then it is possible that semantic deficits in autism exist at the level of organization *within* lexical/conceptual categories. Support for this view is provided by a word fluency study comparing high functioning children with autism, language-impaired children, and normal controls. Autistic participants supplied an age-appropriate number of correct exemplars of specific superordinate categories, but the exemplars they provided were significantly less prototypic than those provided by the controls or language impaired children (Dunn *et al.*, 1996).

Prototype formation involves a process of mental abstraction. In typically developing children, prototype formation occurs through the child's increasing exposure to different category members. Over time, commonalities between category exemplars are extracted, and a single best example, or an "average" (Posner & Keele, 1970), of all category members is derived and stored in memory. An important functional aspect of developing prototypes is that it allows us to summarize information for storage in memory, so that we do not need to memorize information about every instance of a category or concept. This process occurs naturally; it is seldom explicitly taught. Results from studies with infants suggest that babies as young as 10 months old are able to form prototypic mental representations of category exemplars (Younger, 1990; Younger & Gottleib, 1988). Additional studies of prototype formation in the visual modality have shown that strong examples of a category are encoded faster, are remembered longer, and are preferred over weaker

exemplars of a category (Garner, 1974; Mervis & Rosch, 1981; Rosch, 1975, 1977). However, Klinger and Dawson (1995, 2001) found that children with autism *do not* categorize new (visual) information by forming abstract prototypes; instead they rely on an inflexible, rule-based strategy for categorizing new information. For example, when given explicit rules for categorizing novel animals, children with autism perform similarly to typical controls in their ability to use those rules to learn the new category (i.e., given concrete information). But when required to identify category membership of novel animals without being given explicit rules to define category membership (i.e., using abstraction to generalize information and form prototypes) children with autism were not able to learn the new category.

A related theory of cognitive deficits in autism is that of weak central coherence (Frith, 1989; Frith & Happé, 1994). Central coherence is the cognitive process whereby information across various instances of a category becomes integrated to create a new, "higher level of meaning in context." (Frith & Happé, 1994, p. 121). In typical development, this allows the child to create a general understanding of a concept or category as the sum of many parts. As pointed out by Klinger and Dawson (1995, 2001), this process is comparable to prototype formation, and likewise, failure to form prototypes is similar to the idea of weak central coherence in autism. That is, children with autism tend to process information in a "piecemeal" fashion, seeing only the details and missing the central, cohesive picture or idea that holds the parts together. In terms of semantic processing, failure to form prototypes and weak central coherence could explain why children with autism can learn and execute a classification task, but fail to show the neurophysiological signs (i.e., N4 differentiation) of the relationship of a word to its categorical context.

Lack of organization within lexical category could inhibit automatic access to more prototypical exemplars both expressively and receptively. Spreading activation theories of semantic processing (i.e., Collins & Loftus, 1975) describe concepts stored in memory as a network of "nodes." From each concept node there are links to other nodes that represent the properties of those concepts as well as to related words and concepts. The links between concepts have varying degrees of strength, or accessibility; the stronger the link between two nodes, the more likely it is that activation will facilitate that connection over others within the network. When context activates a

node, this activation spreads first along the strongest links, then on to less associated nodes. That is, “activation is like a signal from a source that is attenuated as it travels outward.” (Collins & Loftus, p. 411). Semantic relatedness between two words, then, would be determined by the combined weights on all the paths between the nodes that are activated by context.

If the N4 effect is a neurophysiologic index of the degree of semantic relatedness between a word and its context, its absence in the autism groups suggests an aberrant pattern in the spread of activation elicited by both the in- and out-of-category words. If the pathways to unrelated, or peripherally related, or running in parallel rather than as overlapping and distributed, concepts are as strong or stronger than the pathways to what would normally be highly related nodes, in individuals with autism, the result would be a lack of differential N4 effect. Typical organization would be reflected by strong neural differential reactions to words in context, as was observed in the control groups.

Three sub-processes of semantic processing are commonly described in the word recognition and ERP literature (Friederici, Steinhauer, & Frisch, 1999; Hinojosa, Martín-Loeches, & Rubia, 2001; Marslen-Wilson, 1989; Neely, 1991; Neely & Keefe, 1989; Sereno, Rayner, & Posner, 1998; Skrandies, 1998; Zwitserlood, 1989). The first sub-process is lexical access, which could be related to pre-semantic analysis, and is thought to occur 80–200 milliseconds post-stimulus. The earliest perceived features of the signal activate a set of compatible items in the mental lexicon (Sereno *et al.*, 1998; Skrandies, 1998). The second sub-process is lexical selection, occurring between 200 and 300 milliseconds post-stimulus, wherein the best candidate is selected from the items activated through lexical access. The last step is lexical integration, at 300–500 milliseconds post-stimulus. Here, the selected lexical item is incorporated into a higher order representation specific to the semantic constraints imposed by the context. Again it can be speculated that if organization within the mental lexicon is aberrant, processing at the earliest stages would be affected, in that the initial set of activated items in response to each presented word may be too large, and only peripherally related, so that choosing the best exemplar during lexical selection is impaired, and lexical integration thus prevented.

This could be tied in with the failure of children with autism to develop prototypes, and weak central

coherence. The extraction of commonalities between category exemplars and the conceptualization of a best exemplar is similar to the hierarchical structure of the lexical network in spreading activation theory, and lexical selection and integration in word recognition theories. Similarly, if central coherence—in typical development—allows the child to create a general understanding of a concept or category as the sum of many parts, this must be facilitated by a hierarchical organization of the mental lexicon.

### Physiological Explanations

Macrocephaly is a condition frequently observed in children with autism (Bauman, 1996; Bolton *et al.*, 1994; Filipek *et al.*, 1992; Kemper & Bauman, 1993; Piven *et al.*, 1992). There is recent evidence that macrocephaly may not develop until early childhood, and that it may peak much later in childhood, rather than being present at birth and peaking in early childhood than has been previously thought (Fombonne, Rogé, Claverie, Courty, & Frémolle, 1999). It is unknown whether macrocephaly is caused by increased neural growth or decreased neural pruning. Either way, if neural connections are increasing while language is developing, and frequently paired connections are not strengthened over others due to lack of competition, then an overlapping, distributed network is less likely to develop, and organization of the lexicon could be expected to develop aberrantly. This is consistent with the word fluency data and cognitive theories mentioned above, in that overgrowth or under-pruning could result in many connections or associations to each category label, without the benefit of some associations (e.g. prototypes) having stronger weights than others (less prototypical exemplars). If this were the case, then a deliberate, rule-based strategy would have to be employed by individuals with autism in order to determine membership of a stimulus in one category over another, resulting in loss of automaticity of the response, and no differential N4.

### CONCLUSIONS

In sum, children with autism show abnormalities in the N4 component of event-related potentials. Specifically, unlike typically developing controls, they consistently fail to show a differentiation in response to context-dependent words in a single-word semantic classification task. Additionally, this deficit

appears to persist into early adolescence. Theories of prototype formation during development as well as the central coherence identify specific abnormalities in cognition in autism that may in turn implicate categorical organization within the lexicon.

### Future Directions

Although this study replicates previous findings, a major caveat in the interpretation of this data is the small number of participants in the study providing insufficient power to reveal what might have been significant differences given a larger sample size. Replication with a larger number of children would be advisable for future study.

Another important caveat in the interpretation of data from the current study is that this was a cross-sectional study. Longitudinal, rather than cross-sectional data should be obtained to determine whether these neurophysiologic response deficits persist over time within the same individuals, and whether they are accompanied by continuing semantic processing deficits.

More information is needed about the nature of lexical/conceptual associations in children with autism. While our findings show that the foundation for the structure of the lexicon may be laid down in an aberrant way in children with autism, we do not know *how* the lexicon is organized in these children. Future studies could further understanding of lexical/semantic organization in autism by using behavioral paradigms such as word fluency, word associations, and speeded classification as well as semantic priming tasks. Much narrower age groups should also be examined; collapsing data across several age groups can obscure subtle developmental changes, such as those differences observed in this study between the older and younger children in both groups. A more complete understanding of the complex aspects of semantic processing will be necessary for the development of effective interventions for children with autism.

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### REFERENCES

- American Electroencephalographic Society (1990). *Standard electrode position nomenclature*, Bloomfield, CT.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Baltaxe, C., & Simmons, J. Q. (1992). A comparison of language issues in high-functioning autistics and related disorders with onset in childhood and adolescence. In E. Schopler, & G. B. Mesibov (Eds.), *High-functioning individuals with autism* (pp. 201–225). New York: Plenum Press.
- Baron-Cohen, S. (1988). Social and pragmatic deficits in autism: Cognitive or affective?. *Journal of Autism and Developmental Disorders*, 18(3), 379–402.
- Bartak, L., & Rutter, M. (1976). Differences between mentally retarded and normally intelligent autistic children. *Journal of Autism & Childhood Schizophrenia*, 6, 109–120.
- Bauman, M. L. (1996). Neuroanatomic observations of the brain in pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 26, 199–203.
- Bentin, S., McCarthy, G., & Wood, C. C. (1985). Event related potentials associated with semantic priming. *Electroencephalography and Clinical Neurophysiology*, 60, 343–355.
- Bolton, P., MacDonald, H., Pickles, A., Rios, P., Goode, S., & Crowson, M., (1994) A case-control family history study of autism. *Journal of Child Psychology and Psychiatry*, 35, 877–900.
- Bowler, D. M., Matthews, N. J., & Gardiner, J. M. (1997). Asperger's syndrome and memory: Similarity to autism but not amnesia. *Neuropsychologia*, 35(1), 65–70.
- Bruneau, N., Garreau, B., Roux, S., & Lelord, G. (1987). Modulation of auditory evoked potentials with increasing stimulus intensity in autistic children. *Electroencephalography and Clinical Neurophysiology Suppl.*, 40, 584–589.
- Bruneau, N., Roux, S., Adrien, J. L., & Barthélémy, C. (1999). Auditory associative cortex dysfunction in children with autism: Evidence from late auditory evoked potentials (N1 wave-T complex). *Clinical Neurophysiology*, 110, 1927–1934.
- Byrne, J. M., Connolly, J. F., MacLean, S. E., Dooley, J. M., Gordon, K. E., & Beattie, T. L. (1999). Brain activity and language assessment using event-related potentials: Development of a clinical protocol. *Developmental Medicine & Child Neurology*, 41(11), 740–747.
- Carroll, J. B., Davies, P., & Richman, B. (1971). *Word frequency book*. New York: American Heritage Publishing Co.
- Coles, M. G. H., & Rugg, M. D. (1995). Event-related brain potentials: An introduction. In M. G. H. Coles, & M. D. Rugg (Eds.), *Electrophysiology of mind: Event-related brain potential and cognition* (pp. 1–26). New York: Oxford University Press.
- Collins, A. M., & Loftus, E. F. (1975). A spreading activation theory of semantic processing. *Psychological Review*, 82(6), 407–428.
- Courchesne, E. (1987). A neurophysiological view of autism. In E. Schopler, & G. B. Mesibov (Eds.), *Neurobiological issues in autism* (pp. 285–324). New York: Plenum Press.
- Dunn, L. M., & Dunn, L. M. (1981). *Peabody Picture Vocabulary Test—Revised*. Circle Pines, MN: American Guidance Service.
- Dunn, M., Gomes, H., & Sebastian, M. (1996). Prototypicality of responses of autistic, language disordered, and normal children in a word fluency task. *Child Neuropsychology*, 2, 99–108.

- Dunn, M., Vaughan, H., Kreuzer, J., & Kurtzberg, D. (1999). Electrophysiological correlates of semantic classification in autistic and normal children. *Developmental Neuropsychology, 16*, 79–99.
- Filipek, P. A., Richelme, C., Kennedy, D. N., Rademacher, J., Pitcher, D. A., & Zidel, S. (1992). Morphometric analysis of the brain: Developmental language disorders and autism [abstract]. *Annals of Neurology, 32*, 475.
- Fombonne, E., Rogé, B., Claverie, J., Courty, S., & Frémolle, J. (1999). Microcephaly and macrocephaly in autism. *Journal of Autism and Developmental Disorders, 29*, 113–119.
- Friederici, A. D., Steinhauer, K., & Frisch, S. (1999). Lexical integration: Sequential effects of syntactic and semantic information. *Memory & Cognition, 27*(3), 438–453.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Basil Blackwell.
- Frith, U., & Happé, F. (1994). Autism: Beyond “theory of mind”. *Cognition, 50*, 115–132.
- Garner, W. R. (1974). *The processing of information and structure*. Potomac MD: Erlbaum.
- Geisser, S., & Greenhouse, S. (1959). On methods in the analysis of profile data. *Psychometric, 24*, 95–112.
- Gonzalez-Garrido, A. A., Oropezade Alba, J. L., Riestra Castaneda, R., Riestra Castaneda, J. M., Perez Avalos, M. C., & Valdes Sosa, M. (1997). Event-related brain potentials to semantically incongruent words in children of different ages. *Archives of Medical Research, 28*(1), 109–113.
- Goldstein, G., Beers, S. R., Siegel, D. J., & Minshew, N. J. (2001). A comparison of WAIS-R profiles in adults with high-functioning autism or differing subtypes of learning disability. *Applied Neuropsychology, 8*(3), 148–154.
- Happé, F., & Frith, U. (1996). The neuropsychology of autism. *Brain, 119*(4), 1377–1400.
- Harris, A. J. (1974). *Harris test of lateral dominance: Manual for administration and interpretation*. New York: Psychological Corporation.
- Hermelin, B., & O'Connor, N. (1967). Remembering of words by psychotic and subnormal children. *British Journal of Psychology, 58*, 213–218.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon.
- Hinojosa, J. A., Martín-Loeches, M., & Rubia, F. J. (2001). Event-related potentials and semantics: An overview and an integrative proposal. *Brain & Language, 78*, 128–139.
- Kamio, Y., & Toichi, M. (2000). Dual access to semantics in autism: Is pictorial access superior to verbal access? *Journal of Child Psychology & Psychiatry & Allied Disciplines, 41*(7), 859–867.
- Kemner, C., Verbaten, M. N., Cuperus, J. M., Camfferman, G., & van Engeland, H. (1995). Auditory event-related potentials in autistic children and three different control groups. *Biological Psychiatry, 38*(3), 150–165.
- Kemper, T. L., & Bauman, M. L. (1993). The contribution of neuropathologic studies to the understanding of autism. *Neurologic Clinics, 11*(1), 175–187.
- Klein, S. K., Kurtzberg, D., Brattson, A., Kreuzer, J. A., Stapells, D. R., Dunn, M. A., Rapin, I., & Vaughan, H. G. (1995). Electrophysiologic manifestations of impaired temporal lobe auditory processing in verbal auditory agnosia. *Brain and Language, 51*(3), 383–405.
- Klinger, L. G., & Dawson, G. (1995). A fresh look at categorization abilities in persons with autism. In E. Schopler, & G. B. Mesibov (Eds.), *Learning and cognition in autism*. New York: Plenum Press.
- Klinger, L. G., & Dawson, G. (2001). Prototype formation in autism. *Development and Psychopathology, 13*, 111–124.
- Kutas, M., & Hillyard, S. (1980). Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science, 207*, 203–205.
- Lincoln, A. J., Courchesne, E., Harms, L., & Allen, M. (1995). Sensory modulation of auditory stimuli in children with autism and receptive developmental language disorder: Event-related potential evidence. *Journal of Autism and Developmental Disorders, 25*(5), 521–539.
- Lockyer, D. M., Lazenby, A. L., Donnelly, R. E., Wilkenson, M., & Schoonheydt, W. (1999). Intelligence patterns among children with high-functioning autism, phenylketonuria, and childhood head injury. *Journal of Autism and Developmental Disorders, 29*(1), 5–17.
- Lord, C., Rutter, M. L., & LeCouteur, A. (1994). Autism diagnostic interview—Revised. A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*, 659–685.
- Lord, C., Rutter, M. L., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., & Schopler, E. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders, 19*, 185–212.
- Marslen-Wilson, W. D. (1989). Access and integration: Projecting sound onto meaning. In W. D. Marslen-Wilson (Ed.), *Lexical representation and process* (pp. 3–24). Cambridge, MA: MIT Press.
- Mayes, S. D., & Calhoun, S. L. (2003). Ability profiles in children with autism: Influence of age and IQ. *Autism, 7*(1), 65–80.
- Mervis, C. B., & Rosch, E. (1981). Categorization of natural objects. *Annual Review of Psychology, 32*, 89–115.
- Minshew, N. J., Goldstein, G., & Siegel, D. J. (1997). Neuropsychological functioning in autism: Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society, 3*, 303–316.
- Minshew, N. J., & Rattan A. I. (1992). The clinical syndrome of autism. In: S. J. Segalowitz & I. Rapin (Eds.), *Child neuropsychology. Handbook of neuropsychology* (Vol. 7, pp. 401–441). New York: Elsevier Science.
- Narita, T., & Koga, Y. (1987). Neuropsychological assessment of childhood autism. *Advances in Biological Psychiatry, 16*, 156–170.
- Neely, J. H. (1991). Semantic priming effects in visual word recognition: A selective review of current findings and theories. In D. Besner, & G. Humphreys (Eds.), *Basic processes in reading: Visual word recognition* (pp. 264–336). Hillsdale, NJ: Erlbaum.
- Neely, J. H., & Keefe, D. E. (1989). Semantic context effects on visual word processing: A hybrid prospective/retrospective processing theory. In G. H. Bower (Ed.), *The psychology of learning and motivation: Advances in research and theory* (pp. 207–248). New York: Academic Press.
- Nobre, A. C., & McCarthy, G. (1995). Language-related field potentials in the anterior-medial temporal lobe: II. Effects of word type and semantic priming. *The Journal of Neuroscience, 15*(2), 1090–1098.
- Novick, B., Vaughan, H. G., Kurtzberg, D., & Simson, R. (1980). An electrophysiological indication of auditory processing defects in autism. *Psychiatry Research, 30*, 107–114.
- Paul, R., & Cohen, D. J. (1986). Comprehension of indirect requests in adults with autistic disorders and mental retardation. *Journal of Speech and Hearing Research, 28*(4), 669–679.
- Paul, R., Fischer, M. L., & Cohen, D. J. (1988). Brief report: Sentence comprehension strategies in children with autism and specific language disorders. *Journal of Autism and Developmental Disorders, 18*, 669–679.
- Piven, J., Nehme, E., Simon, J., Barta, P., Pearlson, G., & Folstein, S. E. (1992). Magnetic resonance imaging in autism: Measurement of the cerebellum, pons, and fourth ventricle. *Biological Psychiatry, 31*, 491–504.
- Piven, J., & O'Leary, D. (1997). Neuroimaging in autism. *Child and Adolescent Psychiatric Clinics of North America, 6*, 305–323.
- Posner, M. I., & Keele, S. W. (1970). Retention of abstract ideas. *Journal of Experimental Psychology, 83*, 304–308.

- Rapin, I. (1997). Autism. *New England Journal of Medicine*, 337(2), 97–104.
- Rosch, E. (1975). Cognitive reference points. *Cognitive Psychology*, 7, 532–547.
- Rosch, E. (1977). Human categorization. In N. Warren (Ed.), *Studies in cross-cultural psychology* (pp. 1–49). London: Academic Press.
- Semel, E., Wiig, E. H., & Secord, W. A. (1995). *Clinical evaluation of language fundamentals* (3rd ed.). San Antonio: The Psychological Corporation.
- Sereno, C. C., Rayner, K., & Posner, M. I. (1998). Establishing a time-line of word recognition: Evidence from eye movement and event-related potentials. *Neuroreport*, 9, 2195–2200.
- Skrandies, W. (1998). Evoked potential correlates of semantic meaning: A brain mapping study. *Cognitive Brain Research*, 6, 173–183.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984). *Vineland adaptive behavior scale*. Circle Pines, MN: American Guidance Service.
- Strandburg, R. J., Marsh, J. T., Brown, W. S., Asarnow, R. F., Guthrie, D., & Higa, J. (1993). Event-related potentials in high-functioning adult autistics: Linguistic and nonlinguistic visual information processing tasks. *Neuropsychologia*, 31(5), 413–434.
- Strohner, H., & Nelson, K. E. (1974). The young child's development of sentence comprehension: Influence of event probability, nonverbal context, syntactic form, and strategies. *Child Development*, 45, 567–576.
- Tager-Flusberg, H. (1981). Sentence comprehension in autistic children. *Applied Psycholinguistics*, 2, 5–24.
- Tager-Flusberg, H. (1985a). Basic level and superordinate level categorization by autistic, mentally retarded and normal children. *Journal of Experimental Child Psychology*, 40, 450–469.
- Tager-Flusberg, H. (1985b). The conceptual basis for referential word meaning in children with autism. *Child Development*, 56, 1167–1178.
- Tager-Flusberg, H. (1989). A psycholinguistic perspective on language development in the autistic child. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 92–109). New York: Guilford Press.
- Tager-Flusberg, H. (1991). Semantic processing in the free recall of autistic children: Further evidence for a cognitive deficit. *British Journal of Developmental Psychology*, 9(3), 417–430.
- Thorndike, R. L., Hagen, E. P., & Sattler, J. M. (1986). *The Stanford-Binet intelligence scale* (4th ed.). Chicago: Riverside Publishing Company.
- Toichi, M., & Kamio, Y. (1998). Verbal memory in autistic adolescents. *Japanese Journal of Child and Adolescent Psychiatry*, 39(4), 364–373.
- Toichi, M., & Kamio, Y. (2001). Verbal associations for simple common words in high-functioning autism. *Journal of Autism and Developmental Disorders*, 31(5), 483–490.
- Tsai, L. Y. (1992). Diagnostic issues in high-functioning autism. In E. Schopler, & G. B. Mesibov (Eds.), *High-functioning individuals with autism* (pp. 11–40). New York: Plenum Press.
- Twachtman-Cullen, D. (1998). Language and communication in high-functioning autism and Asperger syndrome. In E. Schopler, G. B. Mesibov, & L. J. Kuncie (Eds.), *Asperger syndrome or high-functioning autism?* (pp. 199–225). New York: Plenum Press.
- Ungerer, J., & Sigman, M. (1987). Categorization skills and receptive language development in autistic children. *Journal of Autism and Developmental Disorders*, 17, 3–16.
- Vaughan, H. G., & Ritter, W. (1970). The sources of auditory evoked responses recorded from the human scalp. *Electroencephalography & Clinical Neurophysiology*, 28, 360–367.
- Verbaten, M. N., Roelofs, J. W., van Engeland, H., Kenemans, J. K., & Slangen, J. L. (1991). Abnormal visual event-related potentials of autistic children.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Wing, L. (1996). The Wing autistic disorders interview checklist (WADIC). In I. Rapin (Ed.), *Preschool children with inadequate communication: Developmental language disorder, autism, low IQ. Clinics in Developmental Medicine Series*. (Vol. 139) London: MacKeith Press, distributed by Cambridge University Press.
- Wood, C. C., & Wolpaw, J. R. (1982). Scalp distribution of human auditory evoked potentials. II. Evidence for overlapping sources and involvement of auditory cortex. *Electroencephalography & Clinical Neurophysiology*, 54, 25–38.
- Younger, B. (1990). Infant categorization: Memory for category-level and specific item information. *Journal of Experimental Child Psychology*, 50, 131–155.
- Younger, B., & Gotleib, S. (1988). Development of categorization skills: Changes in the nature or structure of infant form categories?. *Developmental Psychology*, 24, 611–619.
- Zwitserslood, P. (1989). The locus of the effects of sentential-semantic context in spoken-word processing. *Cognition*, 32, 25–64.