9 Language in Autism

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INTRODUCTION

Autism, often referred to as autism spectrum disorder (ASD), is a developmental disorder of the brain that is strongly associated with deficits in language and communication as well as a variety of other impairments, including abnormal social interaction and motor function.[1] Roughly 20% of children with autism are essentially nonverbal, using fewer than five words per day.[2] Others acquire functional language to varying degrees, although the exact profile of language and communicative abilities appears to be somewhat heterogeneous.

In this chapter, we first summarize the evidence regarding language abilities in ASD. (Note that we use the terms autism and ASD in the broadest sense, including diagnoses of autism and Asperger's syndrome.) The data suggest that ASD is associated with a particular pattern of relatively spared and impaired language functions. We then examine in depth two explanatory theories of language in ASD. The two theories are complementary in that they focus on different sets of language functions. However, both theories take the same broad approach in that they address relations between language and nonlanguage domains with a view to exploring whether similar behavioral profiles across apparently distinct cognitive domains can be explained by common neurocognitive substrates.

According to one theory, deficits of "theory of mind" in ASD can explain *pragmatic* impairments of language and communication in terms of social deficits and their neurocognitive underpinnings.[3–5] In contrast, the procedural deficit hypothesis (PDH) posits that *grammatical* impairments in the disorder — including syntax, morphology, and phonology — can be largely explained by neurocognitive abnormalities of the procedural memory system, whereas lexical knowledge, which depends on the declarative memory system, remains relatively spared.[6,7] Thus, rather than investigating language deficits in isolation, we examine integrative explanatory theories that attempt to account for these deficits in the broader context of brain and behavior in ASD.

LANGUAGE AND COMMUNICATION IN ASD: THE EVIDENCE

Here we first present data related to pragmatic linguistic functions and then turn to evidence pertaining to grammatical and lexical aspects of language. Formulaic speech in ASD, which may be related to more than one of these domains, is addressed at the end of this section. Our discussion will concentrate on cross-sectional studies and, by necessity, on those children who have acquired at least some functional language.

Pragmatic Deficits

Pragmatics concerns the practical knowledge that is necessary to use and interpret language appropriately for the social and real-world contexts in which utterances are made. *Social* aspects of pragmatics crucially include knowledge of the social rules that govern speaker–hearer interactions (which often involves interpreting a speaker's intended meaning across different social contexts), whereas *real-world* aspects of pragmatics include knowledge of people and objects and how they are likely to interact (e.g., knowing that girls are more likely than boys to play with dolls). Pragmatics encompasses both verbal and nonverbal aspects of communication, including gestures, prosodic cues (i.e., of intonation and rhythm), and facial expressions, all of which combine to enhance effective communication in face-to-face social contexts. A range of impairments consistent with pragmatic deficits are inherent to ASD. These impairments are widespread and are found in both children and adults, with diagnoses ranging from classic autism to Asperger's syndrome.

Nonverbal Communicative Gesture

Young ASD children present with well-documented problems in nonverbal communication.[8–10] Data based on parental report indicate significant delays in the use of early gestures.[11] Studies of nonverbal intentional gestural communication in young ASD children consistently show that pointing gestures that are used to communicate requests are generally produced and understood, whereas pointing gestures that are used to share interest in an object or to direct attention to an event are virtually absent (even though these emerge at the same time as requesting gestures in typically developing children).[12]

Speech Acts

Speech acts are utterances that serve a communicative function, such as requests, comments, or commands. Importantly, they require knowledge of how language is used within a culture.[13] Evidence suggests that ASD children are missing speech acts that emphasize social engagement rather than speech acts that regulate others' behavior.[14] Wetherby and Prutting[15] examined the range of speech acts that were expressed by ASD children in both gestural and spoken language at early stages of development in comparison to language-matched typically developing children. They found that the ASD children were not significantly different from the controls in their use of language for requesting objects or actions, for protests, and for self-regulation (e.g., "Don't do that."). However, speech acts with social functions, such as comments, showing off, acknowledging the listener, and requesting information, were completely absent from ASD discourse. In another study, ASD children used declarative sentences that were direct responses to questions but did not make other types of declarative statements or comments, which are thought to involve more significant social awareness.[16] Compared to children with specific language impairment (SLI), ASD children have been found to use fewer affirming or agreeing utterances.[17] Finally, in a study comparing ASD children to Down syndrome children, the ASD children rarely communicated about objects that were the focus of their mothers' attention.[18]

Conversational Discourse

Deficits in conversational ability are found throughout childhood and adolescence in ASD.[19] Older, higher-functioning ASD adolescents are likely to speak too much and in a monologue style during interviews.[20,21] They have problems responding adequately to questions, especially when discussing an unusual event or personal narrative,[22] and tend to have difficulty making clear reference in their conversations to people or places.[23] These latter findings are consistent with evidence of impaired performance on referential communication tasks.[24] They also have difficulty judging the amount of information that needs to be included for effective communication.[25]

Other studies have shown that people with autism seem less able to shift their discourse when there are failures in communication — for example, if a listener has not heard or understood an utterance. Paul and Cohen[26] found that although adults

with autism were just as likely to respond to requests for clarification as mentally retarded adults matched on nonverbal IQ, their answers were less specific than those of the nonautistic participants. They were also less likely to add information that might be of help to the listener, suggesting problems in judging the relevance of a piece of information. This finding was recently replicated in high-functioning school-age children with autism.[27] Finally, we note that several studies have demonstrated abnormalities in autism in other kinds of discourse skills, especially in telling stories or recounting personal narratives.[28–30]

Pragmatic Functions of Prosody

Prosody describes the timing, rhythm, and intonation of speech. It has numerous pragmatic functions, including nongrammatical pauses and the use of stress to direct attention to words or other elements. In a recent survey of investigations of prosody in ASD, all studies (ten of ten) in which pragmatic functions of prosody could be isolated reported deficits in those functions.[31]

Interpreting Nonliteral Language

Spoken and written discourse often includes different forms of nonliteral language, including idioms, metaphors, lies, jokes, and so forth. To understand nonliteral forms of language, one must infer the speaker's intended meaning (as in jokes or metaphors, for example) or understand cultural–linguistic expressions (as in idioms). Both of these entail pragmatic knowledge of how language is used in different social contexts. ASD individuals, even those who are older and high functioning, have great difficulty interpreting nonliteral or figurative speech.[32–35] These difficulties include problems with idioms,[36] with metaphor and irony,[32,37] and with the ability to explain nonliteral utterances embedded in stories (e.g., lies, jokes, pretence, irony, sarcasm, or double bluff).[33,38,39] Using a more structured task, it has also been found that children with autism have difficulty interpreting a speaker's intended meaning in a conversational context and, unlike matched controls, they interpret utterances in a literal way instead of in relation to the speaker's stated desire.[40]

Grammar and Lexicon

Language depends upon two mental abilities.[41,42] Idiosyncratic information must be memorized in some sort of mental dictionary, which is often referred to as the mental *lexicon*. The lexicon necessarily includes all words with arbitrary sound–meaning pairings, such as the noncompositional ("simple") word *cat*. But language also consists of regularities, which can be captured by rules of *grammar*. The rules constrain how lexical forms and other basic units in language combine to make complex representations, including phrases and sentences (e.g., *the cat took the train to work*; syntax), complex words (e.g., *walk* + *-ed* -> *walked*; morphology), and the structured sound patterns of words (e.g., *t* + *u* + *p* -> *tup*; phonology). Importantly, although complex representations (such as the phrase *the cat*) could be computed anew each time (*the* + *cat*), they could in principle also be stored in the mental lexicon (e.g., *the cat* could be stored as a single unit). As we will see, the evidence

suggests that grammatical composition is impaired in ASD, whereas lexical knowledge remains relatively spared.

Grammatical Abilities

Syntax underlies the rule-governed combination of words into the sequential and hierarchical structures of phrases and sentences. Impairments of sentence comprehension have been widely reported in ASD in both auditory and visual (i.e., reading) domains.[43–50] However, not all subjects show these deficits.[44,46] Intriguingly, one study of ASD adults reports normal accuracy but abnormally fast reaction times in a sentence comprehension task.[51]

In expressive language (i.e., in speaking), studies have found that the spontaneous speech of ASD children shows reduced syntactic complexity compared to children with Down syndrome or to developmentally delayed or typically developing control children.[52–54] Similarly, ASD children have been found to omit required closed-class items (e.g., definite articles such as *the*), which play important grammatical roles.[55,56] Children with ASD have also been shown to have lower rates of novel, nonimitative utterances compared to typically developing and Down syndrome children, instead relying more on "formulaic" utterances (see following text for further discussion on formulaic speech).[57] Finally, ASD children have shown impairments on the portion of the Clinical Evaluation of Language Fundamentals (CELF) that tests immediate sentence repetition and reflects (at least in part) expressive syntax.[46,58]

Morphology, which refers to the structure of words with respect to their meaningful parts, comes in two flavors. *Derivational morphology* (the creation of new words; e.g., *solemnity* and *toughness* are derived from *solemn* and *tough*) has, to our knowledge, not been investigated in ASD. Several studies of ASD have examined *inflectional morphology*, which concerns the modification of a word to fit its grammatical role (e.g., *sang* and *walked* are past-tense inflected). It is important to note that inflectional morphology involves both morphosyntax (the choice of inflection based on the syntax of a sentence — for example, choosing present tense or past tense, depending on the syntactic context) and morphophonology (phonological changes to a word that reflect morphological processes, such as the vowel change in the irregular past tense formation of *dug* from *dig*). Although morphosyntax strongly depends upon combinatorial rule-governed (i.e., grammatical) processes, this is not necessarily the case for morphophonology. Whereas regular morphophonological transformations, as in English regular past tenses, follow rule-governed compositional patterns (*walk* + *-ed*), irregular morphophonology is at least partly unpredictable (e.g., *sing–sang*, *bring–brought*), and therefore must rely on stored lexical knowledge.

In two studies that examined samples of spontaneous speech, ASD children omitted inflectional morphemes (e.g., they produced *play* instead of *playing*) more often than unimpaired or mentally retarded control subjects.[55,56] This pattern appeared to hold particularly for certain regularly inflected forms (especially *-ing*-suffixed forms, such as *playing*, but also for regular past tenses in the Bartolucci et al. study), whereas irregular past-tenses were relatively spared in both studies.

Similarly, a more recent study of spontaneous speech reported impairments on third-person singular present tense (e.g., in the production of *washes*), which is almost completely regular.[59]

A recent examination of elicited verbal morphology (subjects were asked to produce inflected forms in past tense picture sentence contexts) found impaired production of past tense forms in ASD (regulars and irregulars were not distinguished).[58] Similarly, high rates of omissions and incorrect inflections in present tense and (both regular and irregular) past tense production (e.g., *wash* or *washing* for *washes*; *catch* or *catching* for *caught*) were found in an elicitation study of language-impaired ASD children compared to ASD children without evident language deficits.[60] A more recent study of elicited past tense production in high-functioning ASD and age-matched typically developing children found normal accuracy scores for regular, irregular, and novel (e.g., *plag–plagged*) verbs. However, response times revealed important differences between the groups: the production of regularized (stem + *-ed*, e.g., in *walked*, *plagged*, *digged*) but not irregularized (e.g., *dug*, *splim–splam*) forms was abnormally fast in ASD compared to controls.[61]

In summary, all six studies of spontaneous or elicited speech reported abnormalities in the production of inflectional morphology. All but one reported actual impairments. The remaining study, in which ASD children were abnormally fast at producing regularized forms, differed from the others in having the highest-functioning ASD subjects (the highest IQs) and in having little or no requirement for social interaction during production, as the items were presented visually on a computer screen (for more discussion of this last point see Reference 62). Additionally, accuracy or response time differences between ASD and control subjects were found in all six studies for regulars, but were not observed for irregulars in three of the four studies that distinguished regular, and irregular morphological forms, including the one study in which regular and irregular forms were explicitly well-matched on frequency and other factors.[61] Thus, ASD seems to be associated with abnormalities of inflectional morphology, particularly for regular forms, although it is not yet clear to what extent this is due to problems of regular morphophonology, morphosyntax, or both.

Phonology refers to the sound patterns of language. In ASD, certain aspects of phonology may be relatively preserved, whereas others are somewhat impaired. Phonology is concerned both with individual speech sounds (i.e., phonemes, such as the */a/* sound in *father*) and how they are combined sequentially and hierarchically into syllables and words. Several studies have reported no particular ASD deficits with individual phonemes in either receptive or expressive language[46] (but see Reference 31; for a review of earlier studies see Reference 63). Impairments in ASD have more often been reported in the combination of sounds into complex structures. Thus, deficits have been found in the repetition of auditorily presented nonsense words (e.g., *barrazon*),[46,64] though the presence of such impairments is less clear in some studies.[53,58] Because this task is posited to involve both disassembling the input into smaller units (e.g., phonemes or syllables) and then reassembling these units in production, it is expected to involve compositional processes. The evidence therefore suggests that at least compositional aspects of phonology may be somewhat impaired.

Lexical Abilities

Behavioral studies suggest that lexical knowledge remains largely normal in ASD. First of all, word-learning abilities seem to be essentially intact.[53] Second, performance has been found to be unimpaired on a range of receptive lexical tasks, such as word–picture correspondence ("Is this a ... ?"), word–picture matching ("Pick the correct picture to match ..."), picture selection ("Show me all the ..."), and word definition ("What is a ...?").[44,46,65] Third, expressive lexical abilities seem to remain largely spared in single-word production tasks, such as picture naming, synonym and antonym generation, and reading single letters or words out loud.[45,46,48,66] When ASD subjects are asked to name pictures as rapidly as possible (so-called "rapid automatic naming"), a mixed profile can be seen, with some subjects showing normal performance, whereas others are impaired.[47] Similarly, performance on verbal fluency tasks, in which subjects are asked to name as many words as possible in a given period of time, seems to be generally, but not always, spared. Normal performance has been found in both letter fluency (e.g., "Name as many words as you can that begin with the letter F.") and category fluency (e.g., "Name as many animals as you can.").[44,47,48,67] Other studies, in contrast, have reported deficits in both types of verbal fluency tasks[49,66] as well as in unconstrained ("miscellaneous") verbal fluency (e.g., "Say as many words as you can think of, any words at all.").[67] Finally, as we have seen earlier, the production of irregular past-tense forms (which depend on memorized lexical knowledge) is generally, but not always, spared. Thus, the evidence suggests that although lexical knowledge itself may remain spared in ASD, there seem to be some deficits in retrieving or searching for this knowledge, perhaps particularly under speeded conditions.

Neuroimaging Studies

There have been few neuroimaging studies of lexicon or grammar in ASD. We are aware of only two functional neuroimaging studies of either domain, both of which examined syntactic processing. (A study of conceptual processing in ASD is discussed below.) In addition, one structural magnetic resonance imaging (MRI) experiment examined the relation between brain abnormalities and language impairments in ASD. In presenting these three studies, we focus specifically on structures previously implicated in language functions (i.e., we do not discuss visual areas, the pons, etc.). In the section on the PDH, we discuss the potential significance of these structures, specifically the relation between frontal, basal ganglia, and cerebellar structures and procedural memory and grammar on the one hand and between temporal/temporo-parietal structures and declarative memory and lexical memory on the other.

A functional MRI (fMRI) study of visual sentence comprehension (as compared to visual fixation) found that ASD adults showed greater activation than unimpaired age- and IQ-matched controls in the left posterior superior temporal sulcus (between the superior and middle temporal gyri, i.e., between Brodmann's areas — BA – 22 and 21), in right temporo-parietal/inferior-parietal cortex (BA 39), and in the parahippocampal gyrus bilaterally; but less activation than controls in the left inferior frontal gyrus ("Broca's region," i.e., BA 44, 45, and 47), right inferior frontal gyrus,

and left lateral and medial premotor cortex and nearby frontal cortex, including the SMA/pre-SMA (SMA refers to the supplementary motor area in medial BA 6; pre-SMA lies just anterior to the SMA).[51]

Similarly, a positron emission tomography (PET) study of auditory sentence comprehension (in comparison to silence) reported greater mean regional activation (we do not discuss deactivations, which are difficult to interpret) in adult ASD subjects (who were impaired at an off-line measure of auditory sentence comprehension) than age-matched healthy controls in the right superior temporal gyrus (BA 22, 41/42) and right inferior parietal cortex (BA 40), but less activation than controls in left premotor cortex (middle/inferior BA 6) as well as in the mid portion (i.e., not posterior) of the left middle temporal gyrus (BA 21).[47] Additionally, within left temporal cortex, the ASD subjects showed a peak activation in a posterior portion of this region, whereas the control subjects showed peak activations in mid and anterior portions. Similar to the fMRI study, there was also less activation in ASD than control subjects in left inferior frontal gyrus (within Broca's region, i.e., in BA 44, 45, and posterior 47), as well as in the left caudate nucleus and the left lentiform nucleus (both of which are part of the basal ganglia), although these differences did not reach significance. In a follow-up reanalysis of a subset of this data,[68] the authors reported reduced activation in the ASD subjects, compared to the controls, in left dorsolateral prefrontal cortex (BA 46) and in the right dentate nucleus of the cerebellum, suggesting abnormalities of frontal–cerebellar circuitry, in particular between left frontal cortex and the right cerebellum (note that the right cerebellum is connected primarily to the left cerebrum).

These functional imaging findings are paralleled by findings from a structural MRI study that related neuroanatomical volumes to performance on the CELF (which probes multiple domains of language) and a nonword repetition task.[69] ASD children were first divided into those who performed within the normal range on these two language tests and those who did not. These two ASD groups were compared against two age-matched comparison groups, one with specific language impairment (SLI) and one composed of typically developing control children. Both language-impaired groups (ASD with impaired language and SLI) had significant right hemisphere asymmetry (i.e., right larger than left), as compared to the two comparison groups, in classical Broca's area — that is, the pars triangularis (BA 45) and pars opercularis (BA 44) of the inferior frontal gyrus. Reported volumes from this region in each hemisphere suggest that the increased asymmetry stemmed from both decreased left hemisphere volume and increased right hemisphere volume in the two language-impaired groups. In contrast, both language-impaired groups showed left hemisphere asymmetry (left larger than right) in the planum temporale (the posterior portion of the upper bank of the temporal lobe), whereas the two comparison groups did not. Analogously to the Broca's area asymmetry, this increased leftward asymmetry in the planum temporale seems to be explained by a combination of decreased right hemisphere volume and increased left hemisphere volume in the two language-impaired groups.

In sum, in both of the functional neuroimaging studies of receptive syntax, ASD subjects showed greater activation in posterior superior-temporal/inferior-parietal cortex — including the posterior superior temporal gyrus and sulcus and temporo-parietal/inferior-parietal cortex — but less in certain left frontal regions, i.e., left

premotor cortex, dorsolateral prefrontal cortex, and Broca's region, as well as in left basal ganglia structures and the right cerebellum (note that only one of the studies addressed subcortical or cerebellar structures). The structural MRI study found that the language-impaired ASD (and SLI) subjects showed a volumetric decrease in left frontal regions, especially in Broca's area, but an increase in a left temporal lobe region — an intriguingly similar pattern to the patterns of increased and decreased activation found in the two functional neuroimaging studies. Interpretations of these findings are discussed below.

Formulaic Speech

ASD speech is marked by several striking features: repetitive and stereotyped utterances (e.g., overuse of routine utterances such as "thank you" or "you're welcome"); idiosyncratic sound–meaning associations (i.e., "metaphorical language" such as "I want to go blue" to express a desire to go outside); excessively literal language (e.g., responding "No, it's raining water" to the statement "It's raining cats and dogs"); difficulty with pronouns (e.g., a child with autism may say "Would you like a cookie?" to request one) and with other deictic terms (i.e., terms whose reference depends on contextual factors, such as *this*, *that*, *here*, *there*, etc.); and immediate or delayed echolalia (lexically, prosodically, and syntactically faithful repetitions).[57,63,70]

These various aspects of speech are highly characteristic of ASD and feature among the diagnostic criteria in DSM-IV for qualitative communication impairments in the disorder.[1] They are observed at much higher rates in ASD than in typically developing children (for a review, see Reference 63) or children with other developmental disorders, including SLI[71] and Down's syndrome.[57]

These aspects of autistic speech are often described together as *formulaic speech*.[57,72] *Formulas* are defined as prefabricated sequences of words that are stored and retrieved whole from memory (in any population, impaired or unimpaired).[73] It has been suggested that at least certain aspects of formulaic speech in ASD (e.g., echolalia and an overreliance on a restricted range of formulaic forms) may reflect pragmatic or social deficits in language use.[63,72] It has also been suggested that formulas offer a shortcut (via memorization) by which to bypass grammatical processing, particularly when grammatical processing is difficult.[73] Thus the overreliance in ASD on these forms may reflect more than one underlying deficit.

INTEGRATIVE THEORIES OF LANGUAGE IN ASD

Here we present two explanatory neurocognitive theories of language in ASD. Both theories integrate findings from language and nonlanguage domains. One focuses on pragmatics and the other on lexicon and grammar.

Pragmatics and Theory of Mind

As we have seen earlier, ASD involves pervasive impairments in the pragmatic aspects of language use. The disorder is also strongly associated with nonverbal social deficits.[74,75] The *theory of mind hypothesis of autism* has been proposed to

integrate deficits across these domains.[76,77] According to this hypothesis, people with ASD are fundamentally impaired at causally linking their own and other people's behavior to mental states. These deficits of theory of mind are posited to underlie impairments both of pragmatics and of nonverbal social abilities.[33]

The evidence suggests that individuals with ASD have theory of mind impairments, as indicated by deficits in tasks that tap their understanding of minds and mental states such as belief (e.g., false belief), knowledge, and emotion.[78,79] Crucially, these impairments appear to be linked not only to social deficits in ASD[75] but also to pragmatic deficits.[33] Several lines of evidence support an association between impairments of theory of mind and of pragmatics.

First of all, whereas ASD is associated with impairments of pragmatics that involve viewing people as mental beings, certain other aspects of language use that do not require knowledge of people's mental states tend to remain relatively spared — such as turn-taking skills, requesting behavior, or regulatory speech acts (see preceding text).

A second line of evidence comes from studies of joint attention in young children. Joint attention involves the triadic engagement between a child, another person, and an object or event of interest. It has been viewed as an early theory of mind ability in that it demonstrates the child's capacity to monitor and manipulate the attentional focus of another person. Deficits in joint attention are evident in looking behavior, communicative pointing, and sharing interest with another person.[8,10] Impairments in joint attention may explain why language is delayed in ASD, as joint attention skills are important developmental predictors of the rate of language development in the disorder.[80]

Third, deficits in conversational discourse in ASD seem to reflect problems in understanding that communication is both about exchanging information with others and about the expression and interpretation of the speaker's intended meaning.[32,81] For example, Capps et al.[19] found a positive correlation between performance on theory of mind tasks and the ability to maintain an ongoing topic of conversation among children with autism. Similarly, Hale and Tager-Flusberg[82] found an inverse relationship between performance at theory of mind tasks and the frequency of non-topic related utterances, a relationship that was independent of overall language ability. Difficulties with nonliteral language also seem to stem from impairments with theory of mind problems, in particular with understanding intentional aspects of communication. For example, Happé's studies have demonstrated a direct relationship between theory of mind performance and the ability to interpret nonliteral meaning in people with autism.[32,33]

Finally, it is interesting to note that functional neuroimaging studies in ASD suggest abnormalities in the neural structures that underlie the processing of theory of mind. Several brain structures have been implicated in typically developing individuals in theory of mind processing, in particular medial frontal cortex (BA 8/9, bordering on the cingulate gyrus), lateral inferior frontal cortex (primarily Broca's area, namely BA 44/45), and posterior superior temporal/temporo-parietal cortex.[83,84] Intriguingly, the small number of neuroimaging studies of theory of mind in ASD suggest a tendency for decreased activation (relative to controls) in both of these frontal regions and increased activation (relative to controls) in the temporal

region.[84–87] This pattern of decreased activation in Broca's area and increased activation in posterior superior temporal/temporo-parietal cortex is strikingly similar to that found in the functional neuroimaging studies of language discussed earlier. It remains to be seen whether similar patterns are found in the processing of pragmatic aspects of language.

Grammar, Lexicon, and the PDH

As we have seen in the preceding text, ASD is associated with impairments of combinatorial aspects of grammar, across syntax, morphology, and phonology. According to the procedural deficit hypothesis (PDH) of autism, these language impairments are explained by abnormalities of the brain structures that underlie the *procedural memory system*, resulting in impairments of the particular language and nonlanguage functions that depend on these structures. For a discussion of the PDH with respect to a number of different disorders, including autism, SLI, attention deficit hyperactivity disorder (ADHD), and dyslexia, see Reference 6. For an in-depth examination of the hypothesis as it applies to SLI, see Reference 7. For a related hypothesis of procedural learning deficits in ASD, see Reference 88.

The procedural memory system (for the sake of simplicity, we also refer to the system as the "procedural system") is implicated in the learning of new, and the control of long-established, motor and cognitive "skills," "habits," and other procedures, such as typing, riding a bicycle, or skilled game playing.[89–91] The system is particularly important for acquiring and performing skills involving sequences[91,92] and has been shown to underlie nonlinguistic rule learning.[93,94] Evidence also suggests that the system subserves aspects of the learning and use of grammatical rule-governed combination, across syntax, morphology, and phonology, in both receptive and expressive language.[6,95]

The procedural system is composed of a network of several interconnected brain structures. It depends especially on structures in the left hemisphere of the cerebrum.[96,97] It is rooted in neural circuits that encompass the frontal lobes and the basal ganglia (a set of subcortical structures that include the caudate nucleus and the putamen, which together constitute the neostriatum). Within frontal cortex, two areas play particularly important roles: premotor areas (especially the region of the supplementary motor area [SMA] and pre-SMA) and Broca's area. Other brain structures also form part of the procedural system network, including portions of both inferior parietal cortex and the cerebellum (including the dentate nucleus).[6,89,91,98,99] Finally, the procedural system appears to be closely related to the so-called "dorsal" stream pathway.[6,100]

The PDH of autism posits that ASD is associated with neural abnormalities of the procedural memory system, resulting in impairments of the various functions that depend on this system. Thus, one should expect motor and cognitive deficits, especially those involving sequencing. In the linguistic domain, one should observe deficits of grammar, across syntax, morphology and phonology. One should also find impairments of any other functions that depend on the brain structures that underlie the procedural system, such as aspects of temporal processing and working memory (whether or not these functions are related to procedural memory).[7]

Thus the PDH is a *brain*-based hypothesis. That is, unlike many other accounts of language deficits in autism or other developmental disorders, the PDH is based on the premise that that certain brain structures are abnormal, and that the functions that depend on these structures are therefore likely to be impaired.

The nature of the observed impairments is expected to vary with the precise nature of the neural dysfunction, potentially leading to substantial heterogeneity in the linguistic and nonlinguistic impairments in ASD. First of all, because each brain structure in the procedural system plays a somewhat different functional role, the dysfunction of different structures should lead to different types of impairment. For example, abnormalities of structures thought to underlie the acquisition of procedural knowledge, such as the basal ganglia, should yield different behavioral phenotypes than abnormalities of structures that may subserve the execution of procedural skills, which might be the case for certain frontal regions.[101] We expect that a portion of the heterogeneity of language (and other cognitive functions) in ASD may be explained by variation across individuals as to which structures are affected and to what degree.

Just as abnormalities of different structures within the procedural memory system should lead to behavioral heterogeneity, so should the dysfunction of different portions of structures — especially of those structures that constitute frontal/basal ganglia and frontal/cerebellar circuits. These circuits are composed of parallel and largely functionally segregated "channels" (also referred to as "loops").[102] For example, in the basal ganglia, each channel receives projections at the neostriatum — some channels primarily at the caudate and others at the putamen — from a particular set of cortical and subcortical structures. Each channel then follows the same set of internal connections within the basal ganglia and then projects outward via the thalamus to a particular cortical region (from which it also receives input), primarily in frontal cortex. A somewhat analogous architecture can be found in the circuitry projecting from the cerebellum via the thalamus to frontal cortex. Each of the frontal/basal ganglia and frontal/cerebellar channels underlies functions associated with the cortical region to which it projects. For example, the frontal/basal ganglia channel projecting to the primary motor cortex subserves motor functions.

Dysfunction in a given structure in this circuitry (e.g., in the neostriatum) is unlikely to be limited to a single channel. Rather, evidence from neurodegenerative and neurodevelopmental disorders suggests a tendency for dysfunction to co-occur across multiple channels.[103,104] However, it is unlikely that exactly the same channels will be affected in all individuals with a particular disorder. Therefore abnormalities in these structures in ASD should result in variability across individuals with respect to the combination of channels that are affected and the severity of their dysfunction. Nevertheless, evidence from other disorders suggests that within a given disorder, certain channels should be more likely than others to be dysfunctional.[103] So, although variability with regard to which channels are affected should lead to some functional heterogeneity across subjects, an important degree of similarity among individuals with the disorder is to be expected.

Additionally, different types of deficits should be associated with the dysfunction of different pathways within these circuits, in particular with differential dysfunction of the "direct" and "indirect" pathways within the basal ganglia. These two pathways

have opposing effects on the basal ganglia's influence on frontal cortex. Via a series of inhibitory and excitatory projections, the direct pathway ultimately disinhibits frontal cortical activity, whereas the indirect pathway ultimately inhibits it. Imbalances between the two pathways can lead to the excessive inhibition or disinhibition of functions that depend on the frontal cortical regions to which the basal ganglia project.[105,106] This is thought to explain the inhibited/suppressed ("hypo") and disinhibited/unsuppressed ("hyper") motor, grammar, and other behaviors found in various neurodevelopmental as well as neurodegenerative disorders affecting the basal ganglia, including Parkinson's disease, Huntington's disease, Tourette syndrome, obsessive-compulsive disorder (OCD) and ADHD.[104,105,107,108] For example, Huntington's disease patients show unsuppressible grammatical rule use (e.g., in affixation, saying *walkeded* and *dugged*) as well as unsuppressible movements, whereas Parkinson's disease patients show the suppression of both grammatical rule use and of movement.[108]

Given the highly plastic nature of the developing brain, compensation is likely to occur. It has been shown that the functions of abnormal neural tissue can be taken over by similar or proximate intact tissue.[109] Thus, abnormalities of specific portions of the striatum or frontal cortex may be compensated for by other portions of these structures in the same, or perhaps even the contralateral, hemisphere. In addition, if a function can be performed by more than one computational mechanism, it could be taken over by a brain structure whose type of computation is distinct from that of the abnormal tissue. Indeed, we posit that the *declarative memory system* can and will take over certain grammatical functions from the abnormal procedural memory system.

The declarative memory system normally subserves the long-term learning, representation, and use of knowledge about facts (conceptual–semantic knowledge, i.e., "semantic memory") and personally experienced events ("episodic memory").[110,111] The knowledge learned in this system is at least partly (but not completely) explicit — that is, available to conscious awareness.[111,112] Medial temporal structures (including the hippocampus and nearby regions such as the parahippocampal gyrus) consolidate new memories, which eventually depend largely on neocortical regions, particularly in the temporal lobes.[110,111,113,114] Other brain structures also play a role in declarative memory, including portions of Broca's region (BA 44, 45, and 47) and frontal polar cortex (BA 10), which seem to underlie aspects of the selection or retrieval of declarative memories,[115–117] and the right cerebellum, which may underlie searching for declarative knowledge.[118] Importantly, some of these other structures also constitute part of the procedural memory system, suggesting that procedural system abnormalities may be expected to result in impairments of lexical search or retrieval — though not necessarily of the acquisition and organization of lexical knowledge.[7] Finally, evidence suggests that the declarative memory system subserves the learning and use not only of fact and event knowledge but also of lexical knowledge. Middle and inferior aspects of the temporal lobe may be particularly important for storing word meanings, whereas superior temporal and temporo-parietal regions may be more important for storing phonological word forms, and possibly also for stored morphological and syntactic structures (e.g., in formulaic speech).[6,119]

The declarative and procedural memory systems do not function completely independently from each other. Rather, evidence suggests that they interact, yielding both cooperative and competitive learning and processing.[6,120–122] First, the two systems can complement each other in acquiring the same or analogous knowledge, including knowledge of sequences. Second, animal and human studies suggest that the two systems also interact competitively. This leads to a "seesaw effect,"[6] such that a dysfunction of one system results in an enhancement of the other, or that learning in one system depresses the function of the other.

We posit that in ASD the declarative memory system will tend to take over certain grammatical functions from the dysfunctional procedural memory system. In particular, complex structures that can be composed by the grammatical/procedural system (e.g., *walk* + *-ed*) in normally developing individuals may simply be stored as chunks (e.g., *walked*) in lexical/declarative memory in individuals with ASD. Structures that are easier to memorize should be more likely to be stored. Thus, forms that are of higher frequency, shorter, and perhaps less complex should be particularly likely to be memorized. Moreover, ASD individuals should be able to compensate for their grammatical deficits by learning explicit rules in declarative memory, such as "add -ed to the end of the verb when the event has already happened." Such increases in reliance on the lexical/declarative memory system in ASD should also be reflected in measures at the level of the brain, such as activation changes (compared to controls) in lexical/declarative memory brain structures in functional neuroimaging studies and possibly even in changes in the neuroanatomy of these structures (e.g., in their volumes or areas). Indeed, both behavioral and brain evidence suggests that such declarative-memory compensation takes place in other populations who appear to be afflicted with a grammatical/procedural dysfunction, including children with SLI [7] and agrammatic aphasics.[123]

Finally, such compensation should depend on the extent to which declarative memory abilities remain intact and should vary with respect to the functionality of this system. Thus, where declarative memory is dysfunctional, such compensation should be less available. In the extreme, a lack of such compensation in the context of a highly dysfunctional procedural memory system would be expected to result in a virtual absence of language. However, we suggest that declarative memory is often (though not necessarily) largely spared in ASD, resulting in a relative sparing of lexical knowledge, though the retrieval or search of this knowledge may tend to be problematic (see preceding text).

In the following two sections we provide brief overviews of the status in ASD of language and other functions that depend on the brain structures that underlie the procedural and declarative memory systems.

ASD Profile of Procedural System Functions

Grammar

As we have seen, rule-governed compositional aspects of grammar seem to be largely abnormal in ASD. This pattern holds across language domains, from syntax to morphology to phonology. In contrast, aspects of knowledge or processing in these domains that do not as clearly involve composition, such as irregularly

inflected forms or individual phonemes, seem to remain relatively spared. These contrasting patterns support the prediction of the PDH that compositional aspects of grammar, which are posited to depend on the procedural system, should be impaired in ASD.

Evidence also suggests that ASD individuals compensate for these impairments by relying on lexical/declarative memory. The widespread dependence on formulaic speech indicates a dependence on the use of memorized complex representations, even of structures that would normally be composed in syntax.[7,73] It is important to point out that even ASD language that appears to be normal may actually depend more heavily on lexical/declarative memory than it does in typically developing individuals. Although such a dependence may be difficult to detect in spontaneous language samples or in many standardized tests, it can be revealed by a variety of behavioral measures or analyses (e.g., "frequency effect" correlations between the forms' frequency of use and production or recognition times, suggesting storage of these forms) as well as by neuroimaging techniques.[7] Indeed, we have seen that neuroimaging studies of grammatical processing find not only reduced activation and volumes in procedural system brain structures but also increased activation and volumes in certain declarative memory structures, which may reflect an increased dependence on these structures due to compensation and the seesaw effect.

The two recent investigations that reported both normal accuracy and faster than normal grammatical processing in syntax and regular morphology are highly intriguing.[51,61] It is not yet clear what to make of these findings. One possibility is that these subjects are highly successful at compensating for compositional deficits with the lexical/declarative memory system. The increased temporal lobe activation in the Just et al. study is consistent with this view. Alternatively, these ASD subjects may possess a disinhibited/unsuppressed (hyper) profile, leading to particularly rapid responses, whereas subjects in other studies may instead display an inhibited/suppressed (hypo) profile, leading to impaired accuracy. Indeed, as we will see, both profiles seem to be found in ASD in certain nonlinguistic domains. A hyper profile is consistent with a different interpretation of the underactivation observed in procedural system brain structures during grammatical processing, namely that this reflects particularly efficient rather than deficient processing. Interestingly, the ASD subjects were high functioning in both of the investigations that reported fast processing, suggesting the possibility that such fast processing might be at least somewhat associated with high-functioning ASD.[124] Whether or not such an association turns out to hold, it is important to note that very few language studies of ASD report response times (we are not aware of any apart from the two discussed here), suggesting the possibility that such fast performance is actually not uncommon.

Nonlinguistic Procedural System Functions

Autism is strongly associated with a number of motor and nonmotor deficits that suggest abnormalities of the brain structures of the procedural system. First of all, the processing, as well as the acquisition of both verbal and nonverbal nonmotor sequences, has been reported to be abnormal in ASD, particularly for complex

sequences.[88,125,126] For example, abnormalities have been reported in the processing (production or recall) of hierarchically structured sequences, with relative sparing of linear, repetitive sequences.[125,126] This finding is particularly relevant to claims of grammatical impairments because grammar (and especially complex syntax) depends on the hierarchical rather than simply linear arrangement of smaller units into larger ones (e.g., the hierarchical combination of words to form phrases and sentences in syntax).[127,128] In addition to these findings concerning the processing of sequences, the one published study we are aware of that examined nonmotor sequence learning in a serial reaction time task found no evidence of learning in high-functioning ASD children and adolescents, whereas age- and IQ-matched typically developing control subjects indeed learned the sequence over the course of the task.[88]

A large number of studies have shown the existence of motor impairments in ASD (for reviews, see Reference 129 and Reference 130). Indeed, the prevalence of such findings has led to the suggestion that motor dysfunction in autism should be considered a core feature of the disorder.[131,132] Consistent with procedural memory abnormalities, deficits seem to be much worse for performing complex sequential motor skills than simple motor acts such as finger tapping.[44] A large number of studies have reported impairments in the pantomime and imitation of complex actions (for a review, see Reference 129). One particularly comprehensive study found that pantomiming actions (e.g., "Show how you would use a toothbrush.") was highly impaired, especially for sequential as compared to single actions; imitating actions was somewhat less impaired (but again, more so for sequential than single actions); and actual object use did not show deficits at all.[133] This is exactly the pattern seen in ideomotor apraxia, which is linked to left SMA, basal ganglia, and inferior parietal structures.[96] Finally, it has also been observed that individuals with ASD have difficulty learning complex sequential motor skills such as dancing or skipping.[129]

Other motor deficits in ASD are suggestive of abnormalities of specific brain structures of the procedural system. Impairments of balance and other motor-related functions linked to the cerebellum have often been reported in ASD.[124,134,135] Still other motor impairments suggest basal ganglia abnormalities and seem to indicate the existence of both hypo and hyper deficits in the disorder. On the one hand, a number of studies have reported hypokinetic (i.e., bradykinetic, or slow) movements similar to those of Parkinson's patients.[124,136] On the other hand, ASD is also strongly associated with unsuppressed motor activity, such as motor (and vocal) tics and stereotypies (repetitive movements or behaviors),[137] which have been linked to basal ganglia abnormalities not only in developmental disorders such as Tourette syndrome[138,139] but also in ASD itself[140,141] (but see Reference 142). Hyperkinetic (choreiform) movements such as isolated jerking of the extremities, which are characteristic of Huntington's disease, have also been observed in ASD.[135] Interestingly, in this last study, all ten subjects showed hyperkinetic movements, whereas none showed movement impairments typical of Parkinson's disease. Thus, there appears to be at least some separation between ASD groups that show hypo and hyper behaviors. Consistent with such a separation, Mari et al.[124] found that low-functioning ASD individuals showed bradykinetic movements similar to those found

in Parkinson's disease, whereas high-functioning individuals instead showed abnormally fast movements relative to normal controls. This lends credence to the hypothesis discussed earlier that hypo and hyper profiles may also be found in ASD in the domain of language.

Finally, other functions associated with the brain structures of the procedural system have also been found to be impaired. (For a discussion of the relationship of the following functions to procedural memory, see Reference 7.) Thus, investigations of ASD have reported deficits of rapid temporal processing[143] as well as impairments in the estimation of stimulus duration[144] (but see Reference 88). Additionally, working memory has been found to be impaired in ASD in some studies[145,146] (but see Reference 62). There have also been reports of deficits in ASD of motion processing, a dorsal stream function.[147,148] Indeed, one study[147] found deficits in motion processing, but not in form processing, which depends on the ventral stream (which is closely related to declarative memory[6]). Further examination in ASD of these and other functions that may depend on procedural system brain structures seems warranted.

ASD Profile of the Declarative Memory System

Lexical Memory

As we have seen, evidence suggests that word learning and lexical knowledge remain largely normal in ASD. For example, individuals with the disorder show intact performance at receptive lexical processing tasks. Similarly, expressive lexical abilities seem to be spared in single-word production tasks, though impairments have been found in both rapid naming and verbal fluency tasks, suggesting deficits in lexical retrieval or search, especially under speeded conditions. This profile of lexical abilities is consistent with a relatively normal lexical memory, accompanied by abnormalities to brain structures such as frontal and cerebellar regions that underlie lexical search and retrieval as well as aspects of rapid processing.[6,7]

Conceptual Knowledge

Conceptual knowledge appears to be largely spared in ASD. This seems to hold for both individual word meanings and their categorical organization.[65,149–152] For example, ASD children have been found to show a normal pattern of prototypicality ratings of members of numerous categories at both basic and superordinate levels, such as chairs, furniture, dogs, and animals.[149] Additionally, semantic priming (e.g., *nurse* will be responded to faster or more accurately following *doctor* than following *table* due to semantic associations between *doctor* and *nurse*) has been found to be normal,[153] suggesting intact conceptual–semantic representations in ASD. Another study reported normal interference in a Stroop task with a range of word categories, including not only color names but also concrete and abstract words.[150] Similarly, in a word–picture matching task, ASD subjects did not differ from controls on either concrete or abstract words.[151]

However, not all conceptual categories seem to be spared in ASD. Specifically, ASD individuals have been found to show impairments at processing words that are

related to mental or emotional states[5,151] (but see Reference 154). Importantly, these impairments may be explained in terms of deficits of emotion or theory of mind.[5,155] Indeed, ASD deficits in the conceptual knowledge of verbs that are related to mental states ("cognition" verbs such as *know, think*) were found to be related to measures of theory of mind (but not to grammatical performance).[5]

We are aware of only one functional neuroimaging study of conceptual processing in ASD.[156] This fMRI study examined conceptual–semantic processing ("Judge if a word is positive or negative.") as compared to perceptual processing ("Judge if a word is upper or lower case."). In this comparison, control subjects robustly activated regions in the left BA 45, left BA 47 (and, borderline significantly, right BA 47), and left medial frontal cortex (two clusters of activation, one on the border of BA 6 and BA 8 — i.e., pre-SMA — and one more anterior in BA 8), as well as in the right cerebellum. In contrast, ASD subjects showed only minimal frontal activation, with a small cluster in left BA 47. They showed no activation in the cerebellum or in any other frontal region, including left BA 45, medial frontal cortex, or right BA 47. Moreover, and unlike the controls, they activated the posterior portion of the superior temporal sulcus/middle temporal gyrus. This is strikingly similar to the neuroimaging patterns discussed earlier and reinforces the view that the temporal lobe structures of lexical/declarative memory may not only be spared, but in fact might be relied on to a greater degree than in typically developing subjects, even for conceptual processing.

Learning in Declarative Memory

An increasing body of evidence suggests that the learning of verbal and nonverbal knowledge in declarative memory (i.e., the acquisition of knowledge in semantic memory) is essentially spared in ASD.[125,145,157,158] (Tasks probing immediate recall or recognition will not be discussed here as they may reflect processing in short-term or working memory rather than learning in declarative memory, which can perhaps be most clearly ascertained with recall or recognition after a delay; we only discuss such post-delay performance here.) First of all, numerous studies have suggested that learning in "rote memory" (i.e., memorizing individual items such as telephone numbers or addresses) is a strength in ASD.[145,159,160] Second, ASD subjects show normal performance on tasks of paired-associate learning (e.g., presenting a pair of words that are studied together, then later prompting with the first word of the pair and asking the subject to recall the second word).[44,49,157,161] Third, normal performance is also observed on cued recall measures after a delay (e.g., after the presentation of a list of words, subjects can be successfully cued with the initial sounds of the word, such as *fr* to cue the recall of *fruit*).[161–163]

However, evidence also suggests the existence of episodic memory impairments in ASD.[125,145,158,164] Thus, memory for recent, personally experienced events is consistently reported to be impaired in ASD relative to controls.[165–168] The basis of this episodic memory impairment in the context of spared semantic memory is not yet clear, although it has been suggested that these impairments may reflect the particular dependence of episodic memory on frontal structures.[158] Note that in any case such impairments would not be expected to impact the acquisition of lexical knowledge, which should depend on semantic rather than episodic memory.

The Neurobiology of Procedural and Declarative Memory Brain Structures in ASD

Although studies of the neurobiology of procedural and declarative memory brain structures have produced quite a few inconsistent results, some patterns are beginning to emerge (for overviews of these and other brain structures in ASD, see References 169, 170, and 171). Of particular interest here are abnormalities of left frontal cortex, and especially Broca's area, which have consistently been found in studies that have examined this region.[172–176] Interestingly, neuroanatomical abnormalities of frontal–cerebellar circuitry have also been implicated in several recent reports.[175–177] Moreover, postmortem studies reliably report reduced numbers of Purkinje cells in the cerebellum.[170] Nevertheless, results from experiments probing the cerebellum with other methods, such as structural MRI, have been more variable (for a recent review, see Reference 171). Studies of other brain structures of the procedural and declarative memory systems, including the hippocampus and the basal ganglia, have also produced somewhat inconsistent results, with some (but not other) studies suggesting abnormalities (see reviews mentioned earlier and discussion in Reference 142). (Note that the hippocampus was not examined in any of the language-related imaging studies discussed earlier, and the basal ganglia were only examined in one of these studies.) Further investigations are clearly needed to clarify the inconsistencies in these (and other) structures and to examine the functional consequences for language of any observed structural abnormalities.

SUMMARY AND CONCLUSION

In this chapter we have argued that language and communicative impairments in autism can be better understood in light of integrative explanatory frameworks that examine these deficits in the broader context of brain and behavior in ASD. First, impairments in pragmatic language abilities are argued to be related to theory of mind impairments. Second, we show that ASD is associated with impairments of compositional aspects of grammar, across linguistic domains, and argue that these impairments are related to abnormalities of brain structures of the procedural memory system, including at least Broca's area. In contrast, lexical and declarative (especially semantic) memory appear to be a relative strength, and evidence suggests that these capacities may be used to compensate for deficient grammatical processing in ASD.

The two classes of language and communication impairments (i.e., of pragmatics and grammar) have been presented as independent deficits. However, possible relations between the two have yet to be explored. Of particular interest is evidence suggesting that frontal cortex, including Broca's area, is implicated in both theory of mind and grammatical/procedural functions. Intriguingly, it has been suggested that theory of mind depends on the dorsal stream,[83] suggesting another potential neuroanatomical link between the two domains. Thus, it may be worthwhile to explore the possibility that pragmatic and grammatical deficits in ASD result from distinct functional deficits, which nevertheless both ultimately depend on the same or related underlying brain structures.

In conclusion, autism is associated with a particular profile of impaired (pragmatic, grammatical) and spared (lexical) language abilities. The status of these abilities in ASD can be at least partly explained in terms of their dependence on neurocognitive substrates that also subserve nonlanguage functions, specifically theory of mind (pragmatics), procedural memory (grammar), and declarative memory (lexicon). Whether or how the neurocognitive abnormalities underlying theory of mind and procedural memory are related to each other or to other abnormalities in ASD (e.g., underconnectivity,[51,178] weak central coherence,[179] or impaired executive function[180]) remains to be examined. Overall, the explanatory theories presented here not only provide wide-ranging accounts of linguistic and nonlinguistic data but also generate new questions and new directions for research.

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