The Biolinguistics of Autism: Emergent Perspectives

Nicolas Bourguignon, Aparna Nadig & Daniel Valois

This contribution attempts to import the study of autism into the biolinguistics program by reviewing the current state of knowledge on its neurobiology, physiology, and verbal phenotypes from a comparative vantage point. A closer look at alternative approaches to the primacy of social cognition impairments in autism spectrum disorders suggests fundamental differences in every aspect of language comprehension and production, suggesting productive directions of research in auditory and visual speech processing as well as executive control. Strong emphasis is put on the great heterogeneity of autism phenotypes, raising important caveats towards an all-or-nothing classification of autism. The study of autism brings interesting clues about the nature and evolution of language, in particular its ontological connections with musical and visual perception as well as executive functions and generativity. Success in this endeavor hinges upon expanding beyond the received wisdom of autism as a purely social disorder and favoring a 'cognitive style'-approach increasingly called for both inside and outside the autistic community.

Keywords: autism spectrum disorders; executive functions; language processing; music; vision

> Saying "person with autism" suggests that the autism can be separated from the person. But this is not the case. I can be separated from things that are not part of me, and I am still the same person. I am usually a "person with a blue shirt" one day, and a "person with a yellow shirt" the next day and I would still be the same person, because my clothing is not part of me. But autism **is** part of me. Autism is hard-wired into the ways my brain works. I am autistic because I **cannot** be separated from how my brain works.
>
> (from J. Sinclair, 1999, "*Why I dislike person first language*"[1])

We are grateful to Kleanthes Grohmann, Cedric Boeckx, Antonio Benítez-Burraco, and another anonymous reviewer for very fruitful exchanges in preparing this manuscript.

[1] The full version of this text is available under http://autismmythbusters.com/general-public/autistic-vs-people-with-autism/jim-sinclair-why-i-dislike-person-first-language. Mention of this reference to justify the use of the word 'autistic' rather than 'person with autism' was first made in Dawson *et al*. (2007). The term *autistic* will be used accordingly throughout the present article.

biolinguistics
© The Author (2012)

ISSN 1450–3417

Biolinguistics 6.2: 124–165, 2012
http://www.biolinguistics.eu

1. Introduction

The present article aims to make the study of Autism Spectrum Disorders (ASD) a chapter of the biolinguistic program, i.e. the study of language as an internal system of human biology (Jenkins 2000). It is argued that a cognitive neuroscience of ASD, in light of recent advances in neurolinguistics and cognitive psychology, can deepen our knowledge of the constitutive features of language and its evolution.

This paper has two explicit motivations. The first is to raise awareness of a view of ASD within the framework of 'cognitive styles' (Happé 1999; see also Mottron 2003) defined by strengths and weaknesses equally worthy of investigation. The specific strength–weakness fraction to be dwelled upon in this discussion is that of enhanced auditory and visual perception contrasted with decreased integration of perception into higher-order representations. The existence of different cognitive styles within the human species, notably as a result of variations in genetic and neurobiological underpinnings, holds promise for refining the comparative work integral to biolinguistics and cognitive science (Hauser *et al*. 2002, de Waal & Ferrari 2010). Accordingly, the second motivation is to provide an alternative to the common view of ASD as deficits mainly affecting the socio-cognitive aspects of language, specifically 'theory of mind', or the ability to infer from a person's behavior their mental states, including beliefs, desires and emotions (Baron-Cohen 1995). Theory of mind and its precursor skills are taken to be important prerequisites for the acquisition and proper use of language in context (e.g., Bloom 2002). As a result, most early research on language in autistics focused on their striking *pragmatic* impairments, sometimes driven by the theory of mind model (Baltaxe 1977, Tager-Flusberg 1992, Surian *et al*. 1996), without undertaking — or paying full attention to — investigations of every aspect of language structure. Yet, despite its widespread success in the cognitive science culture and its recognized importance for early stages of language acquisition, theory of mind falls short as an explanatory account of ASD phenotypes (Frith & Happé 1994). ASD also involve symptoms and characteristics outside the realm of social cognition, which are addressed by alternative, domain-general and bottom-up approaches to ASD such as enhanced perceptual functioning (Mottron *et al*. 2006), weak central coherence (Happé & Frith 2006) and disruptions of executive functions (Ozonoff *et al*. 1991, Russo *et al*. 2007).

We argue that these theories reveal novel and important facts about language in ASD, in particular a generally different mode of language development possibly encompassing all levels of linguistic representation (e.g., phonology, semantics, syntax, in addition to pragmatics), rooted in important differences in neurobiological architecture. We present a synthesis of findings evaluating these alternative models, with a focus on the various neural discrepancies affecting perceptual functioning, central coherence, and executive function in ASD. We provide a discussion of their implications for the study of language structure and development in autism and hope to demonstrate how the rich, neurophysiologically grounded science of ASD can contribute to intrinsic developmental–evolutionary questions of biolinguistics.

2. Autism and Biolinguistics: Advantages and Challenges

Importing the study of ASD into the province of biolinguistics may further the advancement of comparative models of language development and evolution, principally their genetic and neurophysiological aspects. The main challenge to be faced in this enterprise, however, resides in the large genetic and neurophysiological heterogeneity of the autistic spectrum itself.

2.1. Advantages: Intra-Species Variability

From a genetic and neurobiological vantage point, the study of ASD has allowed for significant forays into the 'emergence hypothesis' (Casanova & Tillquist 2008), whereby the advent of language is thought to have endowed human populations with the cognitive armamentarium to ignite their dramatic social and cultural development (Tattersall 2004, Chomsky 2006, 2007). In the wake of seminal approaches put forth to study language evolution despite the paucity of reliable biological artifacts, cognitive biologists ventured to compare human and animal cognition as a means of inferring which of the building blocks of language may be shared between humans and animals on the one hand (Hauser *et al*. 2002, deWaal & Ferrari 2010) and between language and social cognition on the other (Fitch *et al*. 2010). Nevertheless, while cross-species comparisons and animal models certainly are useful in tracing back the "foundational abstractions" of human language and intelligence (Gallistel 2009), comparative work would be incomplete without consideration of the differences emerging *from within* the human species. As the Human Genome Project reached its first significant milestones, it has become incontrovertible that genetic variations, and the interaction thereof with the organism's environment, lie at the source of many psychiatric conditions, including autism (Cowan *et al*. 2002). It follows that genetically-based conditions affecting the neural building blocks of language constitute a promising means to explore its nature and origins, along with the ontological connections between language and other constituents of the human mind (Fisher & Marcus 2006, Marcus & Rabagliati 2006). Given the co-occurrence of the linguistic and social atypicalities that characterize autistic phenotypes, the study of ASD has long been considered a candidate of choice. Although the question of autism as a proxy to investigate the relationship between language and social cognition is not excluded, a central goal of the present article is to show that social cognition is not the only aspect of language in autism that deserves consideration.

2.2. Challenges: Different Routes to the Same Outcome

Despite the aforementioned merits of studying autism as part of biolinguistics, the most likely challenge to be faced in that enterprise is the large genotypic and phenotypic heterogeneity observed in the autistic spectrum, which leads one to expect great variability at the neurophysiological level as well. Textbook descriptions of

autism (DSM-IV; APA 1997) as a triad of *reduced social interactions, delayed or atypical language, and repetitive and restricted interests and behavior* portray only in broad strokes a highly heterogeneous set of symptoms and degrees of severity that often goes beyond the large unevenness in verbal and nonverbal performance across autistic individuals, how it comes to reorganize itself differently from individual to individual in the course of development, and how this reorganization should be explained at the neurobiological level (Joseph *et al*. 2002). A description of the functioning and abilities of autistics needs to incorporate many dimensions such as age, verbal and nonverbal intelligence, and the settings in which behavior takes place (e.g., experimental vs. natural settings; Klin *et al.* 2003).

This patchwork-like picture of autism brings about several caveats and empirical hurdles: First, any investigation of cognitive abilities in ASD must ideally discriminate the broad categories of *high-functioning* autism (which characterizes a substantial 45–60% of individuals with ASD in recent reports (Newschaffer *et al*. 2007; see also Steiman *et al*. 2011), or individuals without intellectual delay, as measured by standardized intelligence tests, and with functional or fluent language abilities, from autism accompanied by mild or severe intellectual delay and minimal or generally non-functional language. Yet, surveying current evidence in both high- and low-functioning autism may provide important information about the potential endophenotypes of ASD as a whole.

Second, many of the neurophysiological studies to date test individuals with a very broad age range and there is little comparability across tasks employed. Focusing on tighter age spans but testing hypotheses over the course of development, and selecting tasks and methods that complement prior findings would provide a clearer picture of how and why language may or may not develop in subpopulations of the autism spectrum.

Third, a careful understanding of language design in autism requires that one consider the distinction between autistics *with* and those *without* formal language impairment. To that effect, while the former may have genetic overlap with specific language impairment (Kjelgaard & Tager-Flusberg 2001; but see Whitehouse *et al*. 2007 for a counterargument[2]), the forthcoming review of neurophysiological data suggests that autistics without behaviorally-defined language impairment may also display patterns of language acquisition and processing that depart from that of typical populations.

This third point highlights that a complete understanding of individual differences in language acquisition and processing demands comparisons across language

[2] With regard to the debate on the genetic relationship between autism and SLI, a series of genetic analyses have broadened the focus of attention from the well-known FOXP2 gene to the neurexin-encoding gene CNTNAP2 by suggesting that mutations affecting the former, while not being a major susceptibility gene for autism or language impairment (Newbury *et al*. 2002), may nevertheless have upstream consequences on the latter's regulation (Vernes *et al*. 2008). By bringing in autism together with other common types of language disorders, this type of evidence suggests that language development (and evolution) might result from a cascade-like interaction of different genetic factors. See also Benítez-Burraco (in press) for discussion.

disorders to determine which aspects (beyond decreased pre-verbal social communication in early development, Tager-Flusberg *et al*. 2005) are ASD-specific, rather than common to individuals with language impairment more generally.

In fact, the heterogeneity of ASD phenotypes yields a vexing tension for scientists keen on developing a generalized model of autism. After intensive efforts to formulate a unitary explanation of these complex phenotypic characteristics, the current state of knowledge has converged on a more fragmented etiology of autism (Happé *et al*. 2006), notably for reasons including its very intricate and still incompletely understood genetic and neural underpinnings. Indeed, existing evidence points to several dozen different genetic mutations associated with autistic behavior (Geshwind 2008, Walsh *et al*. 2008). This, along with the behavioral diversity of ASD (Volkmar & Klin 2005), calls for an approach to autism as a collection of multiple genotypic and phenotypic traits and subgroups rather than a unitary cognitive disorder or condition. Yet, we must still account for the aforementioned triad of features that define ASD. Neuroanatomically, a possible explanation for this is that initially distinct genetic mutations hold analogous consequences for general cortical design or the development of neural networks (Geshwind & Levitt 2007, Walsh *et al*. 2008). In the next section we review findings on brain structure in ASD populations at the levels of minicolumns, hemispheric lateralization and functional connectivity. This overview will serve as a basis upon which the various linguistic discrepancies of ASD can be introduced in light of nonsocial approaches to autism.

3. Brain Architecture in ASD

Discrepancies have been observed at various levels of neurobiological architecture in autistic populations, in particular minicolumnar organization, hemispheric lateralization and connectivity. Although these levels have been studied independently, unified models of autistic neurobiology are beginning to emerge.

3.1. Minicolumns

Casanova *et al*.'s (2002) postmortem morphometric studies on the columnar architecture of the superior and middle temporal gyri in nine autistic patients revealed that their minicolumns were more numerous, smaller and less compact (i.e. more dispersed) than in non autistic individuals. The dorsal and middle portions of these areas typically support the spectro-temporal analyses of speech sounds, while more posterior and ventral parts are involved in accessing lexical representations (Hickok & Poeppel 2007). Minicolumns are vertical bundles of approximately 100 neurons that constitute the basic units of information processing in the brain (Mountcastle 1997). Among other mechanisms[3], these assemblies bind their temporal activity via

[3] For reasons of space, we do not address the issue of columnar functioning at a molecular level, although evidence points to the impact of columnar disorganization on several neurotrans-

different levels of oscillatory coherence, allowing for top-down sensory integration across distant cortical areas (cf. Senkowski *et al*. 2008, Gray *et al*. 1989).

Studies on cortical oscillatory rhythms during sound and speech processing report an asymmetric and hierarchical temporal sensitivity of auditory cortices, with increased left temporal and premotor sensitivity to segmental (i.e. phonemic) information (~40 ms, the duration of the gamma-band), but greater tuning to suprasegmental (i.e. syllabic) information in the right temporal auditory and premotor cortices, correlated with the duration of the theta-band (~200 ms; Luo & Poeppel 2007, Giraud *et al*. 2007). Other studies show that neurons in the right hemisphere are preferentially sensitive to more basic features of auditory processing such as pitch (Belin *et al*. 1998) and slower modulations of sounds typical of musical and prosodic phrases (Belin *et al*. 2002). This hemispheric asymmetry is presumably attributable to differences in the structure and physiology of neuronal assemblies in the left and right hemispheres (Giraud *et al*. 2007).

Under normal circumstances, minicolumns in the left hemisphere contain a greater number of large pyramidal neurons than those in the right (Hutsler 2003). These large neurons typically fire at higher temporal frequencies than the smaller neurons on the right. However, in line with Casanova *et al.*'s findings, several studies report significantly reduced cell size in autistic adults' brains (Kemper & Bauman 1998), including in the hippocampus (Raymond *et al*. 1996), the main source of theta oscillations (Vertes 2005, in Giraud *et al*. 2007). These data suggest that decreased cell size might mostly be detrimental to the phonemic perceptual functions of the left hemisphere, while preserving the right hemisphere's tuning to the syllabic and prosodic characteristics of speech. The 'left-ear' dominance hypothesis of auditory perception in autism (formulated as early as Blackstock 1978) is explored in section 5.

3.2. Hemispheric Lateralization

Given the close links existing between columnar development and brain lateralization (Stephan *et al*. 2007), the features of columnar organization in autism outlined above are likely to impact hemispheric lateralization generally, affecting particularly the large cortical network of language processing (Chugani 2008). Using an MRI regional cortical volume analysis in 16 autistic boys, Herbert *et al*. (2002) reported reversed brain asymmetry in anterior cortical areas traditionally linked to language processing. A region included in Broca's area (pars opercularis), active during syntactic processing (Embick *et al*. 2000) and verbal working memory (Smith & Jonides 1999), appeared 27% larger in the right hemisphere in the ASD group relative to 17% larger in the left hemisphere in controls.

Another study by De Fossé *et al*. (2004) comparing ASD children with or with-

---

mitters putatively involved in regulating important aspects of language development and brain plasticity, in particular the influence of GABA-ergic transmission during the critical period (Hensch 2005). Specific hypotheses on the correlates of minicolumnar disruption on GABA transmission in autism are formulated in Casanova *et al.* (2003).

out language impairments, children with specific language impairments (SLI), and typically developing children, suggests that reversed lateralization of frontal language areas is related to language impairments rather than autistic disorders per se. Herbert *et al*.'s (2004) comparison between ASD, language impaired children and typical controls reports that language impaired and autistic children had proportionally greater right hemisphere volume relative to typically developing and language-impaired participants, but that this right hemisphere bias was more pronounced in the autistic than the language impaired group. Detailed investigation of a shared rightward lateralization between ASD and SLI individuals is beyond the scope of this paper; based on neuroimaging and phenotypic data, Whitehouse and colleagues proposed that the brain asymmetry in SLI and ASD constitutes the same expression of different neurobiological etiologies (Whitehouse *et al*. 2007, 2008).

The lateralization of temporal regions implicated in the auditory and lexical processing of speech is less clear and probably depends in great part on variabilities in the exact anatomy and function of these areas as well as on methodological considerations. In Herbert *et al*.'s (2002) *a priori* analysis, a region corresponding to the Planum Temporale appeared 25% larger on the left in the autistic group relative to 5% larger on the left in the control group, but this difference was much less extreme than that observed in Broca's area. Post-hoc analyses revealed that the leftward lateralization in the autistic group was actually strongest and reached statistical significance in the posterior temporal fusiform gyrus, a region implicated in picture naming and lexical processing (cf. Indefrey & Levelt 2004 for review), which was 20% larger in the left in autistic subjects relative to 6% larger in the right in controls. Adjacent regions, however, showed a trend towards rightward lateralization in the ASD group, including the inferior fusiform gyrus implicated in face processing (Kanwisher *et al*. 1997). However, Jou *et al*. (2010) report significantly enhanced rightward cortical volume in the posterior superior temporal gyrus of ASD adolescents, and normal cortical volumes have been observed in the right Planum Temporale in ASD adults (Rojas *et al*. 2002) and children and adolescents (Rojas *et al*. 2005). Contrary to Herbert *et al*. (2002), Rojas *et al*.'s studies revealed decreased cortical volumes in the left Planum Temporale. Further research is needed to better establish the degrees of lateralization in Wernicke's area and the Planum Temporale in ASD, but existing evidence points to aberrant patterns of hemispheric lateralization in the cortical network of language in ASD populations.

3.3. Functional Connectivity

Besides its impact on hemispheric lateralization, atypical columnar development also has significant consequences on cortical connectivity (Casanova & Trippe 2009), in particular those that characterize large associative areas engaged in complex cognitive and linguistic functions. The large pyramidal cells of the left hemisphere mentioned earlier are thought to form the long-range connections between anterior and posterior language areas (Hutsler 2003). Accordingly, decreased amounts of magno-

pyramidal cells and correspondingly smaller minicolumns are likely to disrupt long-range connectivity. This was observed in fronto-parietal and parieto-temporal networks using structural and functional MRI (McAlonan *et al*. 2005, Just *et al*. 2007), as well as in central subcortical fiber structures such as the arcuate fasciculus using diffusion tensor imaging (Fletcher *et al*. 2010). By contrast, locally normal or enhanced short-range connectivity has been reported in posterior primary sensory cortices (occipital visual areas, cf. Belmonte & Yurgelun-Todd 2003; see also Buxhoeveden *et al*. 2004) and regions contained in Wernicke's area (Just *et al*. 2004).

Thus, studies on connectivity in autism distinguish between underconnectivity over large association areas and normal or enhanced connectivity of primary visual and posterior temporal areas. This distinction led several researchers to suggest that local overconnectivity might compensate for large-scale underconnectivity in the successful completion of specific cognitive tasks (Mottron *et al*. 2006, Just *et al*. 2004, Bertone *et al*. 2005, Williams & Casanova 2010). Interestingly, microstructural studies in typical brains indicate that the amount of large pyramidal cells in temporal language areas decreases as one moves posteriorly (Hutsler 2003), possibly making posterior areas less vulnerable to dysconnectivity and impaired developmental trajectories compared to more anterior brain regions (Carper *et al*. 2002). Also, the spacing of columnar assemblies in posterior language areas is greater in the left hemisphere than in the right in normal brains — an anatomical pattern similar to that observed in the visual cortex and suggesting stronger modular organization in the posterior parts of the left hemisphere (Galluske *et al*. 2000). Given the increased number and greater-than-normal dispersion of minicolumns observed in autistic brains by Casanova *et al*. (*op*. *cit*.), the hypothesis has emerged that autistic brains might be characterized by more numerous and hyperactive cortical modules, which may account for specific features of autistic behavior (Williams & Casanova 2010).

3.4. Hopes and Hurdles for Unification

Although the various discrepancies documented in the investigation of brain anatomy in autism have to a large extent been studied separately, one cannot afford to ignore the strong interdependencies between them. Attempts to integrate these observations in a single framework will prove useful, and necessary, in formulating empirically testable hypotheses on the distinctive cognitive processes that define autism (Coleman 2005). Geschwind (2008) expresses this expectation while also allowing for possible divergences in neural architecture within the autistic spectrum itself. Beyond the many developmental routes potentially related to multiple and divergent cases of autism, current integrated neurobiological hypotheses to date (e.g., Markram *et al*. 2007, Williams & Casanova 2010) managed to emphasize the following dichotomy to describe autistic cognition generally: On the one hand, skills requiring multimodal integration of information, for example language and social cognition, will likely be more vulnerable to dysfunction. For example, Damasio & Maurer (1978: 779) noticed that "the verbal defects of autism […] are seen only in a set of […]

transcortical aphasias that result from a more or less complete anatomical isolation of speech areas". On the other hand, principles of economy in wiring (Cherniak 1994; mentioned in Williams & Casanova 2010) may compensate for this large-scale under-connectivity with a local overconnectivity and hyper-functioning of modular cortical systems reacting to psychophysically 'simple' environmental features.

It is important at this point to clarify the particular meaning of the terms 'simple' or 'complex' as they are understood in our discussion. As in Samson *et al*. (2005), and in line with hierarchical cortical models of perception and learning (e.g., Friston 2005), we consider a neurocognitive system as 'complex' if it is organized into elemental but hierarchically nested units that encode correspondingly complex information. Accordingly, a decrease in the hierarchical organization of processing systems in autism may lead to the processing of narrower, possibly non-hierarchical units. In this sense, 'complexity' at the neurocognitive level should not be confounded with complexity at the level of a particular *task*, in that complex tasks may involve the manipulation of simple stimuli.

This propensity for complex manipulation of simple material is now often assumed to be a characteristic trait of autistic cognition. In its extreme form, it gives rise to special splinter skills (e.g., letter decoding, calculation, list memory, 2D- and 3D-drawing, and music) before functional language is attained at the cost of long, deliberate efforts in some individuals. Special talents are far from the rule in ASD, but are nonetheless particularly informative to the extent that they magnify cognitive trends that might be generally distributed across the autistic spectrum (Mottron *et al*. 2006), and provide important clues on the neuronal systems that may define autism as a whole. If such hypothesis holds, a crucial question arises for language — a prime example of hierarchical complexity at all levels of structure and use. In particular, individuals with ASD might extend their initial cognitive strengths in processing simple/unimodal stimuli to the learning and processing of higher-order and hierarchically complex cues over the course of their development, including those characterizing speech and natural syntax (Mottron *et al.* 2006). Yet, the dearth of longitudinal studies of neural development in autism makes it unclear if neuroanatomical differences reflect the end-state of years of living with a different phenotype and consequent differences in interaction with the environment, or a relative continuity of differences present in the 'initial state' of ASD. A crucial focus of current work in the neuroscience of autism should thus be to determine if these anatomical and functional differences are similarly observed in young children with ASD. In this scenario much work lies ahead in specifying how neuroanatomical differences modify the mechanisms of language acquisition, and, in turn, unraveling how atypical brain development determines language processing in autism.

4. Alternatives to Socio-Cognitive Models of Autism

The unifying hypotheses presented above echo several cognitive psychological mo-

dels of autism that do not consider social communication as its prime domain of deficit. To varying degrees, these models have accounted for autistic language processing in terms of the simple-complex dichotomy developed earlier: The models of enhanced perceptual functioning (EPF; Mottron *et al*. 2006) and weak central coherence (Happé & Frith 2006) have prominently shifted the focus of autism research to the *positive* impacts of autistics' processing bias towards simple, *non-hierarchical* cues. By contrast, models dwelling on autistics' weaknesses in executive functions (see Hill 2004, Russo *et al*. 2007) emphasize the possible difficulties autistics experience as a result of their limitations in processing and producing hierarchically complex stimuli, including sentences (Just *et al*. 2004).

In the remainder of this paper, we take each of these approaches as an illustration of how language in autism could be studied outside of its socio-cognitive aspects: Perceptual functioning in phonology, central coherence in word and sentence processing via visual imagery, and executive functions in the relation between language, thought, and action. We also endeavor to map these observations to those made in neurobiology. But before we proceed, we wish to emphasize that we do not treat these approaches as mutually exclusive in the sense that one (say, perceptual functioning) fares better than the other (say, central coherence) in accounting for a particular aspect of language (say, phonology). Given the theoretical proximity between some of these approaches, there is good reason to believe that they might end up complementing each other in explaining the same aspect of autistics' speech processing abilities. Nor do we claim that a particular discrepancy found at one level of language processing in autism necessarily entails a similar discrepancy at another level. Finally the great phenotypic variability so characteristic of ASD forces us to interpret any observed discrepancies as applying to the tested subgroup of individuals with ASD, without assuming that they should be found uniformly in all autistics. Resolving these issues will depend on the success of our predictions, on a better delineation of the various autistic phenotypes observed, and on how the aforementioned models of autism develop in the future.

5. Phonological Processing: Enhanced Perception of Local Auditory Features

Neurobiological and cognitive psychological evidence suggests a 'left-ear' preference of speech processing in autism as a result of smaller minicolumns, rightward hemispheric lateralization and decreased connectivity in left-hemispheric language areas. This might account for autistics' enhanced perception of phonological primitives processed preferentially in the right hemisphere and shorter neuronal assemblies, namely syllables and prosody, and suggests decreased hierarchical processing of phonemic within syllabic information. Developmental evidence shows that this pattern occurs early. Putative links with preserved or enhanced musical abilities in autism are discussed.

5.1. Neurophysiological Evidence for Rightward Dominance of Speech Processing in Autism

Beginning with adult data, decreased left-lateralization during auditory language processing was reported in a positron emission tomography (PET) study by Müller *et al*. (1999) with five high-functioning participants, and in an fMRI study with 26 young adults by Anderson *et al*. (2010).[4] In another PET study on the processing of 200 ms steady-state synthetic CVC speech-like sounds in five autistic adults, Boddaert *et al*. (2003) observed both significantly lower activity in the left superior temporal cortex and increased activation of the right superior temporal and frontal areas.

Directly addressing the question of when such pattern occurs in development, a follow-up study with intellectually delayed autistic children (Boddaert *et al*. 2004) reported decreased left-hemispheric activity but failed to replicate any right hemispheric effect, suggesting that rightward lateralization of speech processing might occur as a function of age, IQ, and/or verbal ability. ERP and MEG research on sound-related cortical components (in particular the N/M100 cortical response reflecting early auditory processing) and fMRI studies on speech processing in ASD children have begun to refine the relationship between rightward lateralization and development in autistics: Delays in the right hemispheric N/M100 responses to subtle tone contrasts in ASD children are taken as evidence for atypical maturational development of the auditory system in autism generally (Gage *et al*. 2003a, 2003b, Roberts *et al*. 2010).

Beyond these potential delays, other evidence goes along Boddaert *et al*.'s (2004) assumption that the development of autistics' speech recognition system might also follow distinctive maturational trajectories. Compared to the well-established route towards increased left-lateralization in typical children's cortical activation to speech, Flagg *et al*. (2005) found a significant, age-related rightward lateralization in ASD children. Bruneau *et al*.'s (1999) study with intellectually delayed children with autism, normal and intellectually delayed controls reported tone intensity effects on the N/M100 amplitude in the right hemisphere in the ASD group only. Bruneau *et al*. (2003) replicated these results and showed that the ampli-

[4] Interestingly, the reversed lateralization observed by Müller *et al*. (1999) in ASD participants was related only to speech perception, suggesting a dissociation between production and perception systems and lateralization in ASD. Subsequent imaging research on language production in ASD individuals remains scarce and offers mixed and oftentimes surprising results. In a response-naming fMRI study with ASD adolescents, Knaus *et al*. (2008) reported less left-lateralization but greater activation of Broca's area in the ASD relative to the control group. In a functional transcranial Doppler ultrasonography study on language production in adults with autism, adults with a history of SLI, language-impaired adults, and typical adults, Whitehouse *et al*. (2008) reported that the ASD group, like the typical and SLI-history group, had significant activation in the left hemisphere, while right-hemispheric or bilateral activation was mostly significant in the non-ASD language impaired groups. These results led the authors to suggest (in line with Whitehouse 2007) that the aberrant lateralization patterns shared between ASD and SLI individuals might be the similar expression of different neurobio-logical causes.

tude of the right temporal N/M100 was larger as participants' verbal and non-verbal communication abilities increased.

Along the same line, Redcay & Courchesne (2008) report that 2- to 3-year-old toddlers with provisional diagnosis of ASD showed greater rightward activity when presented with auditory bedtime stories during natural sleep (see also Eyler *et al*. 2010). Again, correlations showed that right-hemispheric activation was positively linked to verbal abilities and negatively correlated with autism severity. Interestingly, Wilson *et al.*'s (2007) MEG study reports reduced left-hemispheric steady state gamma-responses to non-speech sounds in autistic adolescents, while frequency power in the right hemisphere did not differ from controls. By contrast, Murias *et al*. (2007) observed significantly increased resting state theta rhythms in autistic relative to controls subjects. This increase in theta oscillations, most detectable in left temporal and frontal regions, is argued by the authors to reflect a decrease in long-range connectivity. The implications of these factors to autistics' language processing will be considered in turn.

5.2. 'Left-Ear' Bias in Speech Processing: Syllables and Prosody

Samson *et al.*'s (2005) review of the literature on auditory processing in ASD points out autistic populations' enhanced performance in tasks involving spectrally and temporally simple material, accounting for their superiority in identifying pitch changes (i.e. absolute pitch, Heaton *et al*. 1999), pure tone discrimination (Bonnel *et al*. 2003, Heaton *et al*. 1998), detection of local changes in contour-preserved melodies (Mottron *et al*. 2000), or — more occasionally — exquisite musical talent (Miller 1999). Other research has applied this hypothesis directly to language processing.

In a study comparing the perception and comprehension, by fluent autistic adolescents and non-autistic controls, of simple sentences with specific prosodic modulations and analogous musical sequences, Järvinen-Pasley *et al*. (2008, Study 1) observed that autistic adolescents performed significantly better than the control group in perceiving prosodic variations in both the linguistic and non-linguistic perceptual samples.[5] Enhanced perceptual processing in autistics has also been found at the word and syllable levels. Mottron *et al.*'s (2001) study of word recall comparing high-functioning autistic and typical individuals reported that whereas typical individuals benefited more from semantic cueing in word recall, the autistic group was equally biased by semantic and syllabic cueing, suggesting that autistics "benefit equally from superficial (syllabic) and deep (semantic) recall cues" (p. 258).

[5] Enhanced perception of prosody may appear as a striking contrast to reports of aberrant expressive prosody produced by autistic speakers (Nadig & Shaw 2012, Peppé *et al*. 2007, Shriberg *et al*. 2001). Global pitch production as well as different functional types of prosody (affective, grammatical, pragmatic) appear to be more disregulated than comprehension of prosody in ASD. Recent work documents atypical production of pitch and duration in non-social situations as well (e.g., Bonneh *et al*. 2011, naming; Diehl *et al*. 2011, imitation), suggesting that basic motor planning or production-perception feedback mechanisms (Russo *et al*. 2008) contribute to differences in prosodic production in ASD.

Using slightly larger groups and narrower age-ranges, Järvinen-Pasley *et al*. (2008, Study 2) compared typical and high-functioning ASD children's perception and comprehension of short sentences displaying specific syllabic rhythms. The autistic group performed significantly better than controls in perceiving syllabic rhythmicity, while the control group showed higher levels of sentence comprehension. Although these data point to enhanced perception of syllabic and prosodic patterns in autistics, it is difficult for now to know whether this pattern might ultimately be detrimental to language comprehension (see McCleery *et al*. 2010 for potential neurophysiological effects of auditory processing on the N400 ERP component in autistic children).

5.3. Neurophysiological Evidence for Decreased Hemispheric Synchronization

As neurophysiological research on phonological processing suggests that large neurons in the left hemisphere show increased sensitivity to phonemic variations (Giraud *et al*., 2007), reports of long-range connectivity disruption (Fletcher *et al*. 2010) and smaller columnar units in auditory cortices (Casanova *et al*. 2002) in autism lead one to predict that autistics may show reduced sensitivity to subtle phonemic variations within syllabic tiers, as in the detection of consonant (e.g., /dɪp/ vs. /tɪp/) or vowel changes (e.g., /ɑ/ vs. /æ/). A recent fMRI study by Dinstein *et al*. (2011) comparing brain activation in autistic, language-delayed, and typically developing toddlers during verbal and non-verbal auditory stimuli presentation in natural sleep found significant evidence of hemispheric desynchronization in the ASD group.[6]

At a more fine-grained level, Event Related Brain Potentials (ERPs) studies provide evidence of decreased sensitivity to phonemic modulations, including those embedded in syllabic units. Ceponienè *et al*.'s (2003) ERP study on autistic participants' sensory and attentional integration of deviances involving simple tones, complex tones, and natural speech vowels in an 'oddball' paradigm (i.e. the detection of unpredictable events in otherwise consistent auditory sequences; cf. Näätänen *et al*. 1978, 1990) reports intact *sensory* processing of all sound categories but no *attentional* processing of vowel modulation, confirming ASD participants' atypical processing of phonemic variations but intact processing of non-speech sounds. Subsequent neurophysiological research corroborates atypicalities in attentional processing of phonemic changes contrasted with greater sensitivity to pitch (Lepistö *et al*. 2005, 2008) but decreased tuning to phonemic changes within syllables (discriminating /taa/ from /kaa/, for example; cf. Jansson-Verkasalo *et al*. 2003).

5.4. Summary and Prospective Research Questions

Atypical right-hemispheric dominance in auditory speech processing in autism has come to be increasingly consensual (see Haesen *et al*. 2011 for another review). Coupling such observations to those made on hemispheric specialization for speech pro-

[6] It is important to note here that Dinstein's study did not allow the authors to determine the directionality of lateralization between the groups.

cessing leads us to formulate the following predictions: Autistics might show a 'left-ear' bias towards syllabic and prosodic patterns, a feature possibly shared in their preserved or enhanced processing of rhythmic and melodic patterns. By contrast, evidence suggests decreased sensitivity to primitives typically subserved by the left hemisphere, namely subtle phonemic variations, whether or not nested in syllabic constituents. This pattern appears to occur early in development, but the extent to which it is compensatory or detrimental to speech perception remains an open question. Beyond possible maturational delays in cortical activity of the right-hemisphere in autistic children without intellectual impairments (Roberts 2010), positive correlations between rightward lateralization of speech/non-speech sound perception and age (Flagg 2005) or verbal abilities in autistic children with intellectual delay (Bruneau *et al*. 2003) suggest that right hemisphere processing of speech is a compensatory mechanism in at least some subgroups of autistic participants.

Answers to the question as to how auditory language processing functions in autism might contribute a good deal to our understanding of how the evolution of complex auditory abilities could have furthered communication, hence social interactions. As Siegal & Blades (2003) point out, discrepancies in complex sound processing in autism, and their impact on autistics' social abilities, may well be more adequately accounted for through investigations of brain structures supporting human voice processing than by appeal to social-cognitive models of autism (see also Gervais *et al.* 2004). On the other hand, autistics' peculiar strengths in auditory perception and their link to language ability might appear quite valuable in studying the relationship between spoken language and cognitive capacities relying on the right hemisphere such as music (Levitin & Tirovolas 2009).

Detailed investigations of the link between musical capacities or enhanced perception of rhythmic/melodic patterns in autistics and their potential ability to exploit these skills in the perception of speech (syllabic vocalization, rhythm and prosody) could shed significant light on the evolutionary connection between these domains of human cognition. In any event, approaches to phonological perception in autism based on discrepancies at the structural and functional levels of neuronal assemblies seem to be gaining promising speed (Giraud & Poeppel 2012).

6. Word and Sentence-Level Processing: Greater Reliance on Visual Imagery in Lexical and Sentential Processing

Evidence shows that some autistics' visual processing is atypically active during performance in tasks of higher cognition, including language comprehension. Increased visual imagery might be particularly important, if not compensatory, in their integration of verbal material, in particular at the levels of words and sentences. Parallels with savant visual abilities and implications for language comprehension are addressed.

6.1. Behavioral and Neurophysiological Evidence for Enhanced Visual Imagery

Early reports of some autistics' strengths in visual processing were based on their enhanced performance on measures of visual intelligence such as the Embedded Figure Task (EFT; Shah & Frith 1983, Joliffe & Baron-Cohen 1997), whereby participants must detect geometric figures contained in more complex visual patterns. In particular, their success on the EFT indexes a tendency to ignore the global properties of images to the benefit of their local features. This local bias in visual integration contrasts radically from typical visual perception, which rather proceeds from global features to hierarchically organized subparts (Navon 1977). Interestingly, autistics' performance in the EFT is correlated with greater cortical activity in occipital areas relative to comparison participants (Ring *et al*. 1999), providing the neurophysiological basis for a 'visual imagery' approach to problem solving.

On a more general basis, several studies demonstrated that ASD individuals' level of intellectual functioning reached significantly higher results when measured through minimally verbal visual tasks such as the Wechsler Block Design subtest or the Raven's Progressive Matrices than through verbal subtests (Happé 1994, Dawson *et al*. 2007). Soulières *et al*. (2009) also demonstrated that autistics' performance in the Raven's matrices was linked to higher activation of occipital regions, while performance in the control group was linked to increased activity of prefrontal areas supporting working memory (Postle *et al*. 1999, Smith & Jonides 1999). A patent example of autism as a visual cognitive style nevertheless comes from autistic draftsmen able to reproduce scenes and objects with exquisite fidelity (Mottron & Belleville 1993) but evidence also shows that autistics' visual integration abilities decrease whenever second-order visual information is involved (Bertone *et al*. 2003), indicating that visual strengths in autism are restricted to simple, non-hierarchical visual material. This latter observation may explain autistic individuals' impaired perception of hierarchically-organized stimuli such as biological motion (Blake *et al.* 2003) or facial masks (Deruelle *et al*. 2010).

It must be reiterated yet again, however, that cognitive peaks in visual abilities are not always found in ASD. Higher verbal than visual abilities are found as well and these profiles may in fact specify different subgroups of autistic individuals (Black *et al*. 2009). Several studies using EFT did not replicate visual facilitation in autistic children, and researchers have recently come to criticize this task and its application to autism on a number of counts (see White & Saldaña 2011). Although neural imaging confirms enhanced activity of the visual cortex in autistics, careful replication of visual processing tasks in ASD individuals is needed to strengthen this argument.

In the late 1980s, autistics' islets of visual abilities figured as evidence for the development of the central coherence approach to autism (Frith 1989, Frith & Happé 2006). On a par with EPF, this approach also stresses the prevalence of simple over complex perception and derives from this perceptual hallmark autistic populations' typical attraction for small, isolated features of the environment and obsessive drive

for sameness. Extended to general cognitive processes (including auditory processing; see Frith & Happé 2006 for a synthesis), this perspective thus emphasizes that autistic perceptual processes are primarily not hierarchical, favoring fragmentary over holistic processing.

Here we focus on the primary findings that spawned the development of weak central coherence, namely peculiarities in visuo-spatial tasks, but findings of decreased hierarchical configuration and enhanced visual imagery have had ramifications in the description of language phenotypes in ASD (see Happé 1999 for review). Specifically, they predict that ASD individuals should show intact processing of isolated lexical items and would be inferior in processing hierarchically structured sentential constituents (see Frith & Snowling 1983 for early evidence).

An ancillary prediction linking facilitated lexical access and enhanced first-order visual processing is that people with autism should show near intact, even enhanced lexical access *via* visual imagery. Neuroanatomically, this phenomenon may find its roots in the greater activation of vision-related areas of the brain during the EFT, Block Design, or Raven's tasks mentioned above, but also in reports of aberrant lateralization of posterior temporal regions (Herbert *et al.* 2002), which are engaged in picture-naming tasks (Indefrey & Levelt 2003), mental image generation (D'Esposito *et al*. 1997), and reading (Dehaene & Cohen 2007) on the left, and in face processing on the right (Kanwisher *et al*. 1997), including during audio-visual speech processing in degraded auditory environments (Kawase *et al*. 1997). Interestingly, face-processing areas in autism show remarkably weak activation during face scanning (Pierce *et al*. 2001), suggesting the possibility that audio-visual perception of speech might be problematic in ASD (see section 6.4 below).

6.2. Visual Imagery Enhances Lexical Access

Existing behavioral and neurophysiological evidence with autistic participants supports the prediction that lexical access and visual imagery can be intact or superior in autism. Autistics appear to show relative strengths in lexical acquisition relative to other aspects of language (Tager-Flusberg *et al*. 2005) and are advantaged in word access in the pictorial (Kamio & Toichi 2000) and orthographic modalities (Toichi & Kamio 2002). Interestingly, Walenski *et al*.'s (2008) picture-naming study comparing high-functioning autistic and typical children report faster naming performance in the ASD compared to the typically developing group, providing evidence for more efficient lexical access in autism.

Current imaging research also suggests that facilitation in lexical access in autistics is related to increased activation of posterior temporal and occipital areas, even in the absence of pictorial prompts. In an fMRI study on word classification in ASD adults, Harris *et al*. (2006) observed increased activation of left posterior temporal areas (Wernicke's area) in the ASD group compared to the control group. Gaffrey *et al*.'s (2007) fMRI study on word classification in ASD participants and typical controls reported significantly increased bilateral activation in the visual cortex in

the ASD compared to the control group. Finally, in their fMRI study comparing performance in a pictorial reasoning task in 12 children with high-functioning autism and 12 age- and IQ-matched controls, Sahyoun *et al*. (2009) showed that although the two groups displayed similar activation in the typical language areas when verbal mediation was necessary, the autism group had substantially greater activation of occipital and ventro-temporal areas in the tasks requiring verbal mediation, while greater activation was found in temporo-frontal language regions in the typical group. The authors suggest that enhanced engagement of posterior regions across tasks in the autistic group indicates greater "reliance on visual mediation […] in tasks of higher cognition".

6.3. Visual Imagery at the Sentence Level

While current evidence supports the view that visual imagery might be linked to greater performance at the word level in ASD, evidence for decreased integration of words in hierarchically structured expressions is mixed, and questions remain unresolved as to whether autistic populations may achieve similar performance as typical, yet through different strategies. Early claims of weak central coherence effects in sentence processing come from studies reporting autistics' decreased ability to choose the appropriate pronunciation of homographs according to their sentential context (e.g., *In her eyes/dress there was a big* ***tear***; Frith & Snowling 1983, Happé 1997, Jolliffe & Baron-Cohen 1999, Lopez & Leekam 2003).

However, these claims have been challenged and/or refined on a number of counts. In a disambiguation study comparing children with autism and concomitant language impairment, children with autism but without language impairment, language-impaired children, and typically developing children using a picture selection paradigm, Norbury (2005) reported that both the autism group with language impairment and the language-impaired group performed equally worse than the ASD group without language impairments and the typically developing group, indicating that decreased ability to use context for disambiguation may stem from language impairment rather than autism per se. This effect was replicated in a lexical ambiguity resolution study by Nadig (2011), where children with high-functioning autism did not differ from typically developing peers matched on language level in being able to use a sentential context to disambiguate a homophone (e.g., *fan*, *bank*, *cell*) when pictures of each versions of the homophone were presented, as reflected by their anticipatory eye-movements.

Brock *et al*.'s (2008) findings from an eye-tracking study of sentence processing in 24 ASD adolescents and 24 controls brings fine-grained evidence that impairments in the use of sentential context to identify a particular word might be attributable to language impairment irrespective of whether or not participants are autistic. In one condition, a visual display accompanying an auditory sentence (e.g., *He stroked the hamster)* presented only the picture of a phonological competitor for the object noun (e.g., *hammer*) and unrelated pictures. Importantly, these sentences were semantically

constraining, such that the phonological competitor (*hammer)* was not a viable object for the verb *stroke*. ASD participants without language impairment and the language unimpaired control group inhibited looks to the hammer following constraining versus neutral verbs such as *chose*, demonstrating online use of sentential context. However, for constraining sentences both autistics with poor language skills and language-impaired controls continued to look at the hammer as candidate based on its phonological onset, despite the lack of fit with the semantics of the verb.

Taken together, these findings are at odds with the prediction of local, piecemeal processing of words in autism, and the consequent prediction of insensitivity to global sentential context. However the question remains as to whether underlying processing strategies are similar between autistics and typicals. Notably, given autistics' putatively intact or enhanced visual processing abilities, it is possible that the use of visual stimuli in lexical disambiguation or phonological competition tasks would have advantaged or facilitated processing in the autism groups.[7] Earlier homograph disambiguation studies (e.g., Happé 1997) that found poorer performance in ASD groups did not present pictorial stimuli. Importantly, other research suggests that superior visual processing might not be sufficient for the comprehension of complex hierarchical structures and operations such as c-command or A-movement. For example, Perovic *et al*. (2007) tested autistic children's comprehension of actional vs. non-actional passives (e.g., *Mary was pushed by Thom*; *Mary was loved by Thom*) and anaphora vs. pronoun structures (e.g., identifying the antecedent in $Bart_i$*'s* dad_j *is washing himself*$_j$/*him*$_i$) using a sentence-picture matching task. Autistics' poor performance at these tasks despite the use of pictorial material indicates that visual imagery may not be sufficient to compensate for core aspects of (Reuland 2001), at least in the early stages of language development.

Nevertheless, neural imaging has brought significant evidence that the use of visual imagery and enhanced lexical access still seems to constitute a key factor in autistics' sentence interpretation. For example, Kana *et al*.'s (2006) fMRI study compared brain activation between high-functioning autistic individuals and normal adults in processing sentences with high-imagery (e.g., *The number eight when rotated 90 degrees looks like a pair of eyeglasses*) vs. low-imagery (e.g., *Addition, subtraction, and multiplications are all math skills*) semantic content. In typical individuals, the processing of high-imagery sentences had already been shown to simultaneously engage areas typically activated during language comprehension and posterior areas subserving visuo-spatial processing, while processing low-imagery sentences activates language-related areas only (Just *et al*. 2004a), suggesting that large-scale integration of visual and verbal information is required when sentences have high imageability

[7] By design, the majority of the target-competitor word pairs in Brock *et al*.'s study began with the same syllable (e.g., *bucket – butter; medal – medicine*), while Happé's (1997) stimuli contained phonemic variations within syllables (e.g., *There was a big* ***tear*** *in her* ***eye/dress***). According to the hypotheses formulated in section 4, the fact that the ASD group performed as well as the control group in Brock *et al*.'s study but not in Happé's may be explained by their presumably intact perception of syllabic patterns but reduced perception of phonemic variations within hierarchically larger units.

content. In Kana *et al.*'s study, by contrast, whereas the simultaneous activation of language- and vision-related areas was triggered only by high-imagery sentences in the control group, ASD participants had increased activation of occipital and parietal areas for *both* high- and low-imagery sentences, while the language network was significantly less activated.

Based on these findings, the authors suggested that "there is a tendency in people with autism to use more visuo-spatial processing by recruiting posterior brain regions in accomplishing even language tasks" (p. 2485). Importantly, they propose to consider this effect as "an adaptation to the underconnectivity in autism, making greater use of parietal and occipital areas and relying less on frontal regions for linguistic processing" (p. 2492). A lexically- (and perhaps visual imagery-) rather than syntactically-based account of sentence processing in autism was also provided in an earlier fMRI study by the same group (Just *et al.* 2004b), in which enhanced activity in the posterior parts of the left superior and middle temporal gyri (i.e. Wernicke's area) in the ASD group contrasted with significantly increased activity of frontal areas in the control group. These results suggest that, "autistic participants may rely more on an enhanced word-processing ability and less on integrating processes that bring the words of a sentence together into an integrated syntactic and semantic structure".

Similar hypotheses on language processing in autism have already been formulated within the framework of other research agendas (e.g., Ullman 2004), but open questions persist as to the proper characterization of autistics' visually/lexically-based sentence processing strategies. First, we must still determine *what* particular visual representations are indeed activated in autistics' processing of verbal material, namely images of *words* or other, more abstract representations (if not both). Many of the studies described above involved reading written sentences or watching pictorial representations. As such, it is difficult to tell if the activation of visual and multimodal language areas reflected activation of graphemes or images with transparent semantic content. Also, warnings about heterogeneity in visual processing across the autistic spectrum must damper the claim that *all* autistics profit from enhanced visual imagery to process language. In effect, these two issues might at some point end up confronting each other: If the hypothesis that activation of visual cortices in sentence processing actually reflects enhanced grapheme decoding turns out to be correct, then it must readily take into account the great heterogeneity of reading skills in autistics, ranging from floor to ceiling (Nation *et al*. 2006).

6.4. Summary and Prospective Research

Many questions remain open with regard to the place vision occupies in language design. These questions have often been the centre of much attention in language sciences, from lexical semantics (Jackendoff 1983) to language acquisition (Gleitman 1990) or speech processing (van Wassenhove *et al*. 2005) and language evolution generally (Corballis 2009). Studying the nature and use of visual imagery during

speech integration in ASD individuals may thus prove valuable on several counts. Notably, could autistic individuals' greater reliance on neural areas subserving visual processing to extract the meaning of words and sentences tell us anything about the mechanisms by which lexical concepts are acquired, processed and combined over time? Does there exist a correspondence between levels of visual complexity and particular levels of linguistic representation, and is it necessary, or even correct, to explain this correspondence by appealing to autistics' social deficits instead of the core mechanisms underlying their visual abilities?

From a computational point of view, the study of autism may help enlighten many grey areas regarding the computational origins of speech and language, in particular when these are assumed to have emerged from the 'social experience' of visually presented information (Gallese 2008). For example, autistic individuals seem to show resistance to McGurk effects (McGurk & McDonald 1976), involving cross-sensory integration of speech and facial articulatory movements (e.g., Mongillo *et al*. 2008). Should this phenomenon be explained in terms of autistic individuals' impaired social comprehension of facial masks, by their putatively deficient 'mirror neuron' detector (Williams *et al*. 2004) or rather by their decreased ability to use facial movements as hierarchical predictors of the speech input? While theory of mind may limit the explanation of this phenomenon to a failure to sense the social significance of face perception, an account centered on the levels of visual complexity in autism would allow for an exploration of the possible connections between visual intelligence and the underlying computational principles of natural languages. Naturally, exploring this territory will necessarily involve a deeper understanding of the computations of audio-visual speech. Luckily, evidence in this domain grows at a rather fast rate (Arnal *et al*. 2011).

On another line of thinking about the significance of graphical evidence in the evolution of language and mind, autistic draftsmen's accurate reproductions of visual scenes have led several authors to note that sophistication in human graphic feats may not necessarily be the sign of verbal intelligence as it is characterized in typical individuals today (Humphrey 1998 contra Tattersall 1998),[8] sparking both new ideas and new doubts about early artistic artifacts as tokens of full-fledged human intelligence. In this respect, autism presents an undeniable comparative advantage. Importantly, one can view the study of autism as an opportunity to identify the distinctive roles that vision and language might have (had) with regard to internal thought processes, and what their respective benefits or disadvantages could be for human consciousness (Dennett 1992: Chap. 7).

That language and vision constitute initially independent but complementary

[8] Among the most suggestive parallels drawn by Humphrey (1998) between cave art and savant drawings is the striking lack of symbolism, which puts into question interpretations of cave art as evidence for the emergence of a symbolic, hence possibly computational mind. It is also worth pointing out, as Humphrey does, that these parallels serve as arguments on what "we should not assume about the mental capacities of the cave artists" (p. 171) and constitute in no case the basis for speculations about common clinical phenotypes between modern autistic populations and cave artists.

tools for thought is reflected in anecdotes from autistic savant artists. For example, Lorna Selfe (1995) tells us the story of Nadia, a gifted autistic child born in 1967, whose drawing abilities ultimately waned follow-ing her first steps in actual linguistic communication at the age of eight. Temple Grandin's (1996) book *Thinking in Pictures,* by emphasizing the primacy of visual over verbal information in her daily stream of consciousness, has a similar sort of flavor. If these personal stories turn out to be correct, we believe that certain types of autism as being at one extreme of the 'verbalizer–visualizer' cognitive continuum, where the cognitive functions of 'inner speech' (Carruthers 2002) could be compared to those of 'private diagram-drawing' (Dennett 1992), set the stage for a direct investigation of their respective advantages and weaknesses.

Empirical research in this area is obviously challenging, and therefore scant (see Hulburt *et al*. 1994 for an early attempt with ASD individuals), but the issues at stake have begun to emerge along with an adequate research framework. Two questions deserve consideration: First, if private speech allows for cognitive functions that private diagram-drawing does not, autistics' performance should be decreased in tasks tapping the former, but not the latter. Second, if private diagram-drawing allows for roundabout strategies to solve problems typically hinging upon inner speech, as seems to be the case for sentence processing, neural imaging should provide ways to discover how this happens in autism. As for the particular research framework within which these questions can be addressed, Hinzen's (2008: 355) mention of the "systems of *executive control* that both human and non-human animals exercise when planning a sequence of actions so as to achieve a particular goal" (italics ours) provides an ideal entry into the problem. In the last section of this paper we sketch out how an Executive Function (EF) approach to autism might serve the purposes of biolinguistics. This section is admittedly the most speculative part of our discussion, so we will limit ourselves to a brief description the areas of EF in autism that we think merit close attention.

7. Executive Functions in Autism: Connectivity and the Prefrontal Cortex

Aberrant neural organization in the prefrontal cortex in autism is linked to weaknesses in higher-order executive control of thought and action, with possible ramifications for several aspects of language comprehension and production, specifically the role of inner speech in complex planning, monitoring of verbal information along its various dimensions, and generativity.

7.1. Neurophysiological and Behavioral Evidence for Executive Function Discrepancies in Autistic Speech

The most striking patterns of aberrant developmental trajectories and cortical architecture in autism appear in the prefrontal cortex (Carper *et al*. 2002). Among other

discrepancies, Courchesne & Pierce (2005) point out excessive and disorganized connectivity *within* the frontal lobes and poor connectivity *between* the frontal lobes and other cortical areas. The importance of the prefrontal cortex and the long-range connections it shares with virtually all other regions of the brain has long been acknowledged in subserving complex EF such as problem solving, language, decision, attention, planning, and goal-directed behavior (Fuster 2008). It is therefore unsurprising that autistic populations show several deficits in mental flexibility and planning, or perseveration (Hill 2004). Regions of the prefrontal cortex for which aberrant lateralization has been reported, such as Broca's area, are not only tonically active in processing language-like hierarchical structures (Musso *et al*. 2003) but also seem to play a critical role in the hierarchical organization of human behavior generally, leading to the conjecture that language may share the same hierarchical properties as those underlying complex human activities (Koechlin & Jubault 2006, Fuster 2008).

Hypotheses of EF as the 'private speech' underlying human thought and behavior (Vygotsky 1962, Luria 1979) not only echo the linguists' suggestions that language may constitute the very "skeleton of thought" (Hinzen 2009), but also conflate the ideas of EF and language as workspace of human planning and decision-making (Hinzen 2008). The rapprochement appears equally well as language and EF have both been assumed to constitute the basis of human creativity, in particular the generative properties so typical of natural languages (Goldberg 2009, see also Fuster 2008: Chap. 5). The proposed limited use of inner speech in autistic populations (Whitehouse *et al*. 2006) resulting from their EF impairments therefore raises at least three questions: Do autistics' "deficits in planning and discourse processing" (Hinzen 2008) tell us anything about the role of language in regulating human thought? (2) Do autistics' superior skills in visual processing lead them to manipulate verbal information in peculiar ways? And (3) do autistics EF impairments have connections to language generativity? We will briefly touch on these points in turn.[9]

7.2. Inner Speech and Planning

Regarding question (1), if inner speech has a role to play in an individual's decision-making ability, autistics should show specific impairments in planning as a result of limited use of inner speech. Poor performance on the Wisconsin Card Sorting Task (WCST), tapping into participants' rule and set-shifting ability, was part of the first evidence to have motivated the development of executive theories of autism (Ozonoff *et al*. 1991). Impaired performance on WCST is believed to reflect an inability to establish goal hierarchies and flexibly shift attention from one set of rules to another. Interestingly, neuropsychological studies suggest that WCST performance is verbally

[9] It is important to note that there are multiple components of executive function and that atypical EF profiles are present in neurodevelopmental disorders more generally (cf. Happé *et al*. 2006, Ozonoff & Jensen 1999). Future work should pinpoint more clearly the profile specific to ASD, and how this set of EF strengths may be related to enhanced performance on visual imagery tasks (cf. Eigsti 2011).

mediated and depends on the integrity of crucial language brain regions (Baldo *et al*. 2005, but see Konishi 1998). It is intriguing to note from Baldo *et al*.'s (2005) study that inner speech impairments in aphasic patients provoked perseverations, or repetitive responses not related to the changing problem presented, not only in WCST, but also in the Raven's, even though both tasks initially tap into visual processing.

However, a proportion of high-functioning autistic individuals are impaired in the former, but unimpaired or superior in the latter, suggesting that enhanced visual processing could compensate or successfully replace weaker inner speech in solving certain visual problems but not others (Kunda & Goel 2011). A possible answer lies in the fact that whereas WCST requires fluctuant application of different rules to the same input, the Raven's Matrices do not. If this turns out to be the critical factor, one could infer that inner speech (or lack thereof) specifically supports (or impair) the ability to flexibly switch from one task to the other (see Emerson & Miyake 2000 for experimental evidence). Further research is needed to explore this question.

Another EF task possibly requiring covert vocalization and for which individuals with autism show particular impairments is the Tower of London task or its variants (Ozonoff & McEvoy 1991).[10] It is possible that the Tower of London and WCST both necessitate inner speech to a greater extent than the Raven's matrices as a result of requiring more complex planning abilities. If so, this would support the hypothesis that language is an important tool for setting long-term goals. Along similar lines, Carruthers (2002) proposes that EF and inner speech have an important part to play in perceiving and planning the behavior of other people, making them important components of theory of mind (Carruthers 2002, Newton & deVilliers 2007, but see Forgeot d'Arc & Ramus 2011).

These hypotheses parallel those of studies attempting to link autistics' ability to pass false-belief tasks and their acquisition of complement syntax (Tager-Flusberg & Joseph 2005; for an original argument on the relationship between complementation and theory of mind, see de Villiers & Pyers 2002) or other striking reports of autistics' success at false-belief tasks after achieving a certain verbal mental age (Happé 1995). Regarding social cognition generally, authors have observed that autistics' level of social functioning was significantly linked to their verbal abilities (Joseph *et al*. 2002), possibly making linguistic competence a crucial compensatory mechanism of their deficit in social cognition, perhaps more so than in typical children, strengthening further the link between language and social cognition.

7.3. Monitoring Verbal Information across its Various Dimensions

With regard to question (2), EF and the prefrontal cortex are important for the flexible selection of stimuli according to their nature, context and cross-temporal contingencies (Koechlin *et al*. 2003), for example when subjects are asked to judge the same verbal item along its different levels of representation, e.g., orthography, phon-

[10] For an application of the Tower of London to prefrontal functions, see Shallice (1982). A variant of this task is the Tower of Hanoi.

ology, and meaning. Research on working memory and EF also shows hemispheric selectivity between left and right prefrontal regions, with the left frontal cortex subserving verbal information, and the right visuo-spatial stimuli (Smith & Jonides 1999). Accordingly, autistics' enhanced perceptual bias towards the visual features of words along with their rightward bias in Broca's area might lead them to perseverate on their orthographic rather than phonological or semantic aspects. This was shown in Toichi & Kamio (2002), who compared autistic and learning-disabled adults and adolescents' discrimination of words based on their orthographic properties, pronunciation, or meaning.

Results indicated not only that the autistic group had no level-of-processing effect compared to the control group, but also that the autistic group performed better than the control group in the orthographic task, suggesting a processing perseverance at the orthographic relative to phonological and semantic level. Interestingly, Harris *et al.*'s (2006) fMRI study on levels-of-processing effects in autistic and control participants reports that while activation of Broca's area was significant for the semantic relative to the orthographic task in the control group, its activation was undifferentiated between the two conditions in the ASD group. Koshino *et al.*'s (2005) fMRI study on verbal working memory comparing high-functioning and control participants provides even more compelling evidence. The authors observed that the control group had substantially more activation in the left and right prefrontal regions, while the autistic group had significant activation in right prefrontal and parietal regions, suggesting that autistic participants would have used a "visual-graphical approach […] in which they coded the shape of the alphabet letters without naming them" (p. 818).

Such conclusions are interesting but raise a few parallel issues to be worked through. First, the link between right prefrontal regions and 'letter decoding' must be checked against neurophysiological theories that locate letter decoding in left inferior temporal regions (Dehaene & Cohen 2007), which — interestingly enough — also showed signs of significantly greater activation in the ASD relative to the control group (see also hypotheses on visual imagery sketched in section 6). Second, that visuo-spatial strategies could somewhat supplant manipulation of verbal information does not entail that inner speech is totally absent in ASD populations (Williams *et al*. 2008), nor that visuo-spatial working memory capacity is exempt from impairments as a function of stimulus complexity (Williams *et al*. 2005). Further research will be needed to refine this question, taking into account age, functioning, task demands, and neurophysiological factors.

7.4. Generativity

We wish to end this section with a brief mention of the studies that have investigated *generativity* in ASD populations. The notion of EF as an important contributing factor of creativity (Shallice 1988, Goldberg 2009, Fuster 2008) has been used to account for autistics' impaired ideational fluency in play (Lewis & Boucher 1995) and, more

recently, language production (Turner 1999). These characteristics might be visible at varying degrees in the development of intellectually unimpaired and impaired individuals. Tager-Flusberg *et al.*'s (1990) longitudinal study on language development between autistic children and children with Down syndrome remarks that "autistic children […] tend to rely on a narrower range of grammatical structure in their spontaneous speech" (p. 17), despite similar levels of syntactic development as, and higher IQ levels than, children with Down syndrome.

Other research points to autistics' lack of flexibility in structural levels of linguistic representation, as reflected in extreme forms of echolalia[11] (Roberts 1989), 'stereotyped language', and "gestalt language learning patterns exhibited by autistic individuals who, unlike unimpaired children, may not develop a truly flexible syntactic rule system" (Landa 2000: 127). Interestingly, cases of limited syntactic flexibility must also be contrasted with instances of exaggerated lexical creativity such as the production of neologisms and idiosyncratic language (Volden & Lord 1991). Facts such as these are difficult to accommodate within a socio-cognitive account of autism but certainly deserve closer inspection from a 'generative' perspective.

8. Spreading the Net: Conceptual Payoffs for the Biolinguistic Program

Granted some consensus emerges on the topics we have discussed, we believe that the perspective advocated in the present article might help advance some of the core theoretical work in biolinguistics in a more concrete and observable way. In particular, more light could eventually be shed on the definition and the relative contribution of the conceptual divide between the Faculty of Language in the broad and narrow sense (FLB vs. FLN; Hauser *et al*. 2002) as well as on the relationship between language and other facets of cognition. Importantly, the constructs brought forth by alternative models of autism — *central coherence* in auditory and visual perception; *visual imagery* in concept acquisition and audiovisual language; *generativity* and *monitoring* in executive functions — may all in our view be part of the infrastructure of FL. We see two significant advantages to their introduction into boilinguistics: one related to the constituents of cognition that could have served and interacted as precursors to this faculty altogether; the other related to the importance of embedding ASD and its features into solid computational theories of neural functioning. We will briefly exemplify them in turn.

First, the cognitive phenomena highlighted throughout this text turn out to be necessary for other cognitive abilities likely to form part of FL broadly and narrowly defined. For example, prefrontal executive functions are necessary components of

[11] One must note that echolalia takes different forms in autism, with different levels of severity and functional roles as a result of different levels of development or functioning. Early studies on echolalia in autism have proposed interesting ways of using autistic echolalia as an indicator of propositional speech development (Baltaxe & Simmons 1977). Accordingly, we speculate that various forms of echolalia could be related to different levels of sophistication in grammatical generative power.

meta-representation (Stuss *et al*. 2001, Ozonoff *et al*. 1991), which is in turn deemed to be an important requisite for sophisticated intra-species communication. Furthermore, the view that executive functions are the "generative capability of the frontal lobes that made complex propositional structures possible" (Goldberg 2009) points to yet new bases for complex recursive thinking. At a lower level, central coherence could be analogous to the temporal *binding* of sensorimotor information necessary to construct higher-order representational hierarchies across neural networks, be it for auditory language or for other cognitive abilities (Engel *et al*. 2001). As a case in point, our discussion of the possible impacts of underconnectivity on cortical oscillations and phonological processing is only part of the broader discussion on the role of endogenous cortical cycles in perception and cognition (Fries *et al*. 2007), providing strong empirical and theoretical extensions of central coherence in autism. Similarly, we mentioned that impairments in the hierarchical integration of audio-visual information could contribute to autistics' resistance to McGurk illusions. Rather than appealing to socio-cognitive explanations of this phenomenon, our understanding of this impairment would gain significant depth through hierarchical cortical models of perception (Friston 2005, Rao & Ballard 1999), especially if it is confirmed that cortical hierarchies are precisely what may be jeopardized in ASD. One advantage for taking these factors into account in characterizing FL is to understand not only what the precursors to language are (e.g., vision, central coherence, generativity, etc.), but how they interface hierarchically with one another within the constraints of neural architecture to eventually give rise to a full-fledged capability for language structure and use.

The second advantage follows directly from the first: A very exciting move in the study of language in ASD would be to look at central coherence, enhanced perceptual functioning, and executive function in light of existing computational theories. For example, the study of central coherence could be embedded within fine-grained and biologically realistic models of binding, asymmetric sampling and predictive coding (Engel *et al*. 2001, Bever & Poeppel 2011, Giraud & Poeppel 2012).[12] The same is arguably true for the computational principles underlying EF, which have received much support both from a theoretical (Dehaene & Changeux 1997) and empirical point of view (Koechlin *et al*. 2003) but remain largely absent from the literature on autism. In effect, the reason why autism research has been so hard to reconcile with contemporary language science beyond its socio-cognitive considerations is possibly the failure to appreciate that autism, much like social cognition or language, is a collection of different perceptual and cognitive factors, each of which is altered in its own computational and neurobiological machinery. If, by contrast, the multiple perceptual and cognitive facets of autism — and, for that matter, of *every* developmental disorder implicating language — are understood and specified through grounded explanatory theories of neural computation, biolinguistics could go a long way into the reverse-engineering agenda it has set out to pursue.

[12] We are grateful to one of the reviewers for bringing this point to our attention.

9. Conclusion

The present article was an attempt to integrate the study of autism within the framework of the biolinguistic program along two interconnected perspectives, namely that of autism as a cognitive style, on the one hand, and of autism as a heterogeneous set of verbal and nonverbal behaviors outside the realm of social cognition, on the other. These perspectives have led us to consider three alternative approaches of autistic cognition that focus on differences in perception and cognition (driven by differences in neural architecture), and their application to linguistic traits observed in autism. We propose that these traits hold promise for understanding individual linguistic differences if they are explored in the neurosciences of language: brain lateralization in auditory language processing, the role of visual intelligence in defining the nature and trajectories of language design and evolution, and the parallel between language and executive functions.

Importantly, we emphasize that our paper should be construed less as a discussion *on autism* than as a review of the *ways in which autism can feed the research program pursued in biolinguistics*. It is therefore neither comprehensive, nor integrative. Its primary goal is to show that the use of comparisons with autism to elucidate only pragmatic aspects of language is an insufficient and unnecessarily limited approach, and that this should be complemented with bottom-up, alternative, and empirically testable hypotheses that do not necessarily appeal to social cognition. In short, we hope to have shown that there is more to study about language in autistic populations than their assumed "blindness to Gricean Maxims" (Surian *et al.* 1996) and that thorough understanding of linguistic phenotypes in autism requires domain-general, neuroscientifically explainable, and ultimately *computational* hypotheses encompassing *every* level of linguistic representation.

This is not to say, however, that studying the interface between language and social cognition through autism is no longer worthwhile. To the contrary, we argue that the perspective defended here might bring pending research questions back to the forefront: Where are the links, both biological and psychological, between social cognition and language to be found? *Are* there any such links? Are these links a "spandrel" or otherwise characterized "cultural recycling" of the brain (Dehaene & Cohen 2007)? More particularly, did the computational complexity of social cognition, if any, feed into language or vice versa (Fitch 2005)? Addressing these issues also requires recognizing that to fully understand the social phenotype in autism, one must strive to tease apart aspects of autistics' social cognition that do present deficits from those that don't. As Sinclair's epigraph expresses quite clearly, a growing number of people within the autistic community struggle for their recognition within society as 'another intelligence', where their preoccupations and interests deserve as much heed as our common habits of verbal interchanges (Wollman 2008). As in any other fields of science, this paradox certainly summarizes how complex the problem becomes when looked at carefully, but comes yet again with novel and exciting questions about the place of language within human nature and society.

References

Anderson, Jeffrey S., Nicholas Lange, Alyson Froehlich, Molly DuBray B., Jason Druzgal, Michael P. Froimowitz, Andrew L. Alexander, Erin D. Bigler & Janet E. Lainhart. 2010. Decreased left posterior insular activity during auditory language in autism. *American Journal of Neuroradiology* 31, 131–139.

Arnal, Luc H., Valentin Wyart & Anne-Lise Giraud. 2011. Transitions in neural oscillations reflect prediction errors generated in audiovisual speech. *Nature Neuroscience* 14, 797–803.

Baldo, Juliana V., Nina F. Dronkers, David Wilkins, Carl Ludy, Patricia Raskin & Jiye Kim. 2005. Is problem solving dependent on language? *Brain and Language* 92, 240–250.

Baltaxe, Christiane A. 1977. Pragmatic deficits in the language of autistic adolescents. *Journal of Pediatric Psychology* 2, 176–180.

Baltaxe, Christiane A. & James Q. Simmons. 1977. Bedtime soliloquies and linguistic competence in autism. *Journal of Speech and Hearing Disorders* 42, 476–393.

Baron-Cohen, Simon. 1995. *Mindblindness: An Essay on Autism and Theory of Mind.* Cambridge, MA: MIT Press.

Belin, Pascal, Stephen McAdams, Bennet Smith, Sophie Savel, Lionel Thivard, Séverine Samson & Yves Samson. 1998. The functional anatomy of sound intensity discrimination. *The Journal of Neuroscience* 18, 6388–6394.

Belin, Pascal, Stephen McAdams, Lionel Thivard, Bennet Smith, Sophie Savel, Mônica Zilbovicius, Séverine Samson & Yves Samson. 2002. The neuroanatomical substrate of sound duration discrimination. *Neuropyschologia* 40, 1956–1964.

Belmonte, Matthew K. & Deborah Yurgelun-Todd. 2003. Functional anatomy of impaired selective attention and compensatory processing in autism. *Cognitive Brain Research* 17, 651–664.

Bertone, Armando, Laurent Mottron, Patricia Jelenic & Jocelyn Faubert. 2003. Motion perception in autism: A "complex" issue. *Journal of Cognitive Neuroscience* 15, 219–225.

Bever, Thomas G. & David Poeppel. 2010. Analysis by synthesis: A (re-)emerging program of research for language and vision. *Biolinguistics* 4, 174–200.

Bishop, Somer L., Whitney Guthrie, Mia Coffing & Catherine Lord. 2011. Convergent validity of the Mullen scales of early learning and the differential ability scales in children with autism spectrum disorders. *American Journal of Intellectual and Developmental Disorders* 116, 331–343.

Black, David O., Gregory L. Wallace, Jennifer L. Sokoloff & Lauren Kenworthy. 2009. Brief report: IQ split predicts social symptoms and communication abilities in high-functioning children with autism spectrum disorders. *Journal of Autism and Developmental Disorders* 39, 1613–1619.

Blackstock, Edward G. 1978. Cerebral asymmetry and the development of early infantile autism. *Journal of Autism and Childhood Schizophrenia* 8, 339–353.

Blake, Randolph, Lauren M. Turner, Moria J. Smoski, Stacie L. Pozdol & Wendy L.

Stone. 2003. Visual recognition of biological motion is impaired in children with autism. *Psychological Science* 14, 151–157.

Bloom, Paul. 2002. *How Children Acquire the Meaning of Words.* Cambridge, MA: MIT Press.

Boddaert, Nathalie, Pascal Belin, Nadia Chabane, Jean-Baptiste Poline, Catherine Barthélémy, Francis Brunelle, Marie-Christine Mouren-Simeoni, Yves Samson & Mônica Zilbovicius. 2003. Perception of complex sounds: Abnormal pattern of cortical activation in autism. *American Journal of Psychiatry* 160, 2057–2060.

Boddaert, Nathalie, Nadia Chabane, Pascal Belin, Marie Bougeois, Vincent Royer, Catherine Barthélémy, Marie-Christine Mouren-Simeoni, Anne Philippe, Francis Brunelle, Yves Samson & Mônica Zilbovicius. 2004. Perception of complex sounds in autism: Abnormal auditory cortical processing in children. *American Journal of Psychiatry* 161, 2117–2120.

Bonneh, Yoram S., Yoram Levanon, Omrit Dean-Pardo, Lan Lossos & Yael Adini. 2011. Abnormal speech spectrum and increased pitch variability in young autistic children. *Frontiers in Human Neuroscience* 4, 1–7.

Bonnel, Anna, Laurent Mottron, Isabelle Peretz, Manon Trudel, Erick Gallun & Anne-Marie Bonnel. 2003. Enhanced pitch sensitivity in individuals with autism: A signal detection analysis. *Journal of Cognitive Neuroscience* 15, 226–235.

Brock, Jon, Courtenay F. Norbury, Einav Shiri & Kate Nation. 2008. Do individuals with autism process words in context? Evidence from language-mediated eye-movements. *Cognition* 108, 896–904.

Bruneau, Nicole, Frédérique Bonnet-Bribault, Marie Gomot, Jean-Louis Adrien & Catherine Barthélémy. 2003. Cortical auditory processing and communication in children with autism: Electrophysiological/behavioral relations. *International Journal of Psychophysiology* 51, 17–25.

Bruneau, Nicole, Sylvie Roux, Jean-Louis Adrien & Catherine Barthélémy. 1999. Auditory associative cortex dysfunction in children with autism: Evidence from late auditory evoked potentials. *Clinical Neurophysiology* 110, 1927–1934.

Buxhoeveden, Daniel, Katerina Semendeferi, Natalie Schenker & Eric Courchesne. 2004. Decreased cell column spacing in autism. *Society for Neuroscience* 582, 6.

Carper, Ruth A., Pamela Moses, Zachary D. Tigue & Eric Courchesne. 2002. Cerebral lobes in autism: Early hyperplasia and abnormal age effects. *NeuroImage* 16, 1038–1051.

Carruthers, Peter. 2002. The cognitive functions of language. *Behavioral and Brain Sciences* 25, 657–726.

Casanova, Manuel F., Daniel P. Buxhoeveden & Juan Carlos Gomez. 2003. Disruption in the inhibitory architecture of the cell minicolumn: Implications for autism. *The Neuroscientist* 9, 496–507.

Casanova, Manuel F., Daniel P. Buxhoeveden, Andrew Switala & Emil Roy. 2002. Minicolumnar pathology in autism. *Neurology* 58, 428–432.

Casanova, Manuel F. & Christopher R. Tillquist. 2007. Encephalization, emergent properties, and psychiatry: A minicolumnar perspective. *The Neuroscientist* 14,

101–118.
Casanova, Manuel F. & Juan Trippe. 2009. Radial cytoarchitecture and patterns of cortical connectivity in autism. *Philosophical Transactions of the Royal Society* 364, 1433–1436.
Ceponienè, Rita, Tuulia Lepistö, Shestakova, A., Vanhala, R., Alku, P. & Risto Näätänen. 2003. Speech-sound selective auditory impairment in children with autism: They can perceive but do not attend. *Proceedings of the National Academy of Sciences of the United States of America* 100, 5567–5572.
Cherniak, Christopher. 1994. Component placement optimization in the brain. *The Journal of Neuroscience* 14, 2418–2427.
Chomsky, Noam. 2005. Three factors in language design. *Linguistic Inquiry* 36, 1–22.
Chomsky, Noam. 2006. *Language and Mind*. Cambridge, MA: Cambridge University Press.
Chomsky, Noam. 2007. Approaching UG from below. In Uli Sauerland & Hans-Martin Gärtner (eds.), *Interfaces + Recursion = Language*: *Chomsky's Minimalism and the View from Syntax-Semantics*, 1-29. Berlin: DeGruyter.
Chugani, Diane C. 2004. Serotonin in autism and pediatric epilepsies. *Mental Retardation and Developmental Disabilities Research Reviews* 10, 112–116.
Coleman, Mary. 2005. A neurological framework. In Mary Coleman (ed.), *The Neurology of Autism*, 40–74. Oxford: Oxford University Press.
Corballis, Michael C. 2009. Language as gesture. *Human Movement Science* 28, 556–565.
Courchesne, Eric & Karen Pierce. 2005. Brain overgrowth in autism during a critical time in development: Implication for frontal pyramidal neuron and interneuron development and connectivity. *International Journal of Developmental Neuroscience* 23, 153–170.
Cowan, Maxwell, Kathy L. Kopinsky & Steven E. Hyman. 2002. The human genome project and its Impact on psychiatry. *Annual Reviews in Neurosciences* 25, 1–50.
Craik, Fergus I. & Robert S. Lockhart. 1972. Levels of processing: A framework for memory research. *Journal of Verbal Learning and Verbal Behavior* 11, 671–684.
D'Esposito, Mark, John A. Detre, Geoffrey K. Aguirre, Matthew Stallcup, David C. Alsop, Lynette J. Tippet & Martha J. Farah. 1997. A functional MRI study of mental image generation. *Neuropsychologia* 35, 725–730.
Damasio, Antonio R. & Ralph G. Maurer. 1978. A neurological model for childhood autism. *Archives of Neurology* 35, 777–786.
Dawson, Geraldine, Karen Toth, Robert Abbott, Julie Osterling, Jeff Munson, Annette Estes & Jane Liaw. 2004. Early social attention impairments in autism: Social orienting, joint attention and attention to distress. *Developmental Psychology* 40, 271–283.
Dawson, Michele, Isabelle Soulières, Morton A. Gernsbacher & Laurent Mottron. 2007. The level and nature of autistic intelligence. *Psychological Science* 18, 657–662.
Dehaene, Stanislas & Jean-Pierre Changeux. 1997. A hierarchical framework for

planning behavior. *Proceedings of the National Academy of Sciences of the United States of America* 94, 13293–13298.

Dehaene, Stanislas & Laurent Cohen. 2007. Cultural recycling of cortical maps. *Neuron* 56, 384–398.

Dennett, Daniel C. 1992. *Consciousness Explained*. London: Penguin.

Deruelle, Christine, Cecilie Rondan, Bruno Gepner & Carole Tardif. 2004. Spatial frequency and face processing in children with autism and Asperger syndrome. *Journal of Autism and Developmental Disorders* 34, 199–210.

deWaal, Frans B.M. & Pier Francesco Ferrari. 2010. Towards a bottom-up perspective on animal and human cognition. *Trends in Cognitive Sciences* 14, 201–207.

Diehl, Joshua John & Rhea Paul. 2011. Acoustic differences in the imitation of prosodic patterns in children with autism spectrum disorders. *Research in Autism Spectrum Disorders* 6, 123–134.

Dinstein, Ilan, Karen Pierce, Lisa Eyler, Stephanie Solso, Raphael Malach, Marine Behrmann & Eric Courchesne. 2011. Disrupted neural synchronization in toddlers with autism. *Neuron* 70, 1218–1225.

Eigsti, Inge-Marie. Executive functions in ASD. In Deborah A. Fein (ed.), *The Neuropsychology of Autism*, 185–203. Oxford: Oxford University Press.

Embick, David, Alec Marantz, Yasushi Miyashita, Wayne O'Neil & Kuniyoshi L. Sakai. 2000. A syntactic specialization for Broca's area. *Proceedings of the National Academy of Sciences of the United States of America* 97, 6150–6154.

Engel, Andreas, Pascal Fries & Wolf Singer. 2001. Dynamic predictions: Oscillations and synchrony in top-down processing. *Nature Reviews Neuroscience* 2, 704–716.

Eyler, Lisa T., Karen Pierce & Eric Courchesne. 2010. Abnormal brain response to language stimuli in sleeping infants and toddlers with ASD. Paper presented at the *9th Annual International Meeting for Autism Research*, Philadelphia, PA (20–22 May 2010).

Fisher, Simon E. & Gary F. Marcus. 2006. The eloquent ape: Genes, brains and the evolution of language. *Nature Reviews Genetics* 7, 9–20.

Fitch, W. Tecumseh. 2005. Computation and cognition: Four distinctions and their implications. In Anne Cutler (ed.), *Twenty-First Century Psycholinguistics*, 381–400. New York: Lawrence Erlbaum.

Fitch, W. Tecumseh, Ludwig Huber & Thomas Bugnyar. 2010. Social cognition and the evolution of language: Constructing cognitive phylogenies. *Neuron* 366, 376–388.

Flagg, Elissa J., Janis E. Oram Cardy, Wendy Roberts, Timothy P.L. Roberts. 2005. Language lateralization development in children with autism: Insights from the late field magnetoencephalogram. *Neuroscience Letters* 386, 82–87.

Fletcher, Thomas, Ross T. Whitaker, Ran Tao, Molly B. DuBray, Alyson Froehlich, Caitlin Ravichandran, Andrew L. Alexander, Erin D. Bigler, Nicholas Lange, Janet E. Linhart. 2010. Microstructural connectivity of the arcuate fasciculus in adolescents with high-functioning autism. *NeuroImage* 51, 1117–1125.

Forgeot d'Arc, Baudouin & Frank Ramus. 2011. Belief attribution despite verbal

interference. *Journal of Experimental Psychology* 64, 975–990.
De Fossé, Lies, Steven M. Hodge, Nikos Makris, David N. Kennedy, Vernes S. Caviness, Lauren McGrath, Shelley Steele, David A. Ziegler, Marthia R. Herbert, Jean A. Frazier, Helen Tager-Flusberg & Gordon J. Harris. 2004. Language-association cortex asymmetry in autism and specific language impairment. *Annals of Neurology* 56, 757–766.
Fries, Pascal, Danko Nikolic & Wolf Singer. 2007. The gamma cycle. *Trends in Neurosciences* 30, 309–316.
Friston, Karl. 2005. A theory of cortical responses. *Philosophical Transactions of the Royal Society* 360, 815–836.
Frith, Uta. 1989. *Autism: Explaining the Enigma*. Hoboken: Wiley-Blackwell.
Frith, Uta & Francesca Happé. 1994. Autism: Beyond 'Theory of Mind'. *Cognition* 50, 115–132.
Frith, Uta & Margaret Snowling. 1983. Reading for meaning and reading for sound in autistic and dyslexic children. *British Journal of Developmental Psychology* 1, 329–342.
Fuster, Joaquin. 2008. *The Prefrontal Cortex*, 4th edn. London: Academic Press.
Gaffrey, Michael S., Natalia M. Kleinhans, Frank Hais, Natacha Akshoomoff, Ashley Campbell, Eric Courchesne & Ralph-Axel Müller. 2007. Atypical participation of visual cortex during word processing in autism: An fMRI study of semantic decision. *Neuropsychologia* 45, 1672–1684.
Gage, Nicole M., Bryna Siegel, Melanie Callen & Timothy P.L. Roberts. 2003a. Cortical sound processing in children with autism disorder: An MEG investigation. *NeuroReport* 14, 2047–2051.
Gage, Nicole M., Bryna Siegel & Timothy P.L. Roberts. 2003b. Cortical auditory system maturational abnormalities in children with autism disorder: An MEG investigation. *Developmental Brain Research* 144, 201–209.
Gallese, Vitorio. 2008. Mirror neurons and the social nature of language: The neural exploitation hypothesis. *Social Neuroscience* 3, 317–333.
Gallistel, Randy C.H. 2009. The foundational abstractions. In Massimo Piattelli-Palmarini, Juan Uriagereka & Pello Salaburu (eds.), *Of Minds and Language: A Dialogue with Noam Chomsky in the Basque Country*, 58–73. Oxford: Oxford University Press.
Gervais, Hélène, Pascal Belin, Natalie Boddaert, Marion Leboyer, Arnaud Coez, Ignacio Sfaello, Catherine Barthélémy, Francis Brunelle, Yves Samson & Mônica Zilbovicius. 2004. Abnormal cortical voice processing in autism. *Nature Neuroscience* 7, 801–802.
Geschwind, Daniel H. 2008. Autism: Many genes, common pathways? *Cell* 135, 391–395.
Geschwind, Daniel H. & Pat Levitt. 2007. Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinions in Neurobiology* 17, 103–111.
Giraud, Anne-Lise, Andreas Kleinschmidt, David Poeppel, Torben E. Lund, Richard

Frackowiack & Helmut Laufs. 2007. Endogenous cortical rhythms determine cerebral specialization for speech perception and production. *Neuron* 56, 1127–1134.

Giraud, Anne-Lise & David Poeppel. 2012. Cortical oscillations and speech processing: Emerging computational principles and operations. *Nature Neuroscience* 15, 511–517.

Gleitman, Lila. 1990. The structural sources of verb meanings. *Language Acquisition* 1, 3–55.

Goldberg, Ekholon. 2009. *The New Executive Brain: Frontal Lobes in a Complex World*. New York: Oxford University Press.

Grandin, Temple. 1996. *Thinking in Pictures: And Other Reports from My Life with Autism*. New York: Vintage Books.

Gray, Charles M., Peter König, Andreas K. Engel & Wolf Singer. 1989. Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties. *Nature* 338, 334–337.

Haesen, Brigitt, Bart Boets & Johan Wagemans. 2011. A review of behavioral and electrophysiological studies on auditory processing and speech perception in autism spectrum disorders. *Research in Autism Spectrum Disorders* 5, 701–714.

Happé, Francesca G.E. 1994. Wechsler IQ profile and Theory of Mind in autism: A research note. *Journal of Child Psychology and Psychiatry* 35, 1461–1471.

Happé, Francesca G.E. 1995. The role of age and verbal ability in the Theory of Mind task performance of subjects with autism. *Child Development* 68, 843–855.

Happé, Francesca G.E. 1999. Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences* 3, 216–222.

Happé, Francesca G.E., Rhonda Booth, Rebecca Charlton & Claire Hughes. 2006. Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. *Brain and Cognition* 61, 25–39.

Happé, Francesca G.E. & Uta Frith. 2006. The weak coherence account: Detail-focused cognitive style in Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders* 36, 6–25.

Happé, Francesca G.E., Angelica Ronald & Robert Plomin. 2006. Time to give up on a single explanation for autism. *Nature Neuroscience* 9, 1218–1220.

Harris, Gordon J., Christopher F. Chabris, Jill Clark, Trinity Urban, Aharon Itzhak, Shelley Steele, Lauren McGrath, Karen Condouris & Helen Tager-Flusberg. 2006. Brain activation during semantic processing in autism spectrum disorders via functional magnetic resonance imaging. *Brain and Cognition* 61, 54–68.

Hauser, Marc D., Noam Chomsky & W. Tecumseh Fitch. 2002. The faculty of language: What is it, who has it, and how did it evolve? *Science* 22, 1569–1579.

Heaton, Pamela, Beate Hermelin & Linda Pring. 1998. Autism and pitch processing: A precursor for savant musical ability? *Music Perception* 154, 291–305.

Heaton, Pamela, Linda Pring & Beate Hermelin. 1999. A pseudo-savant: A case of exceptional musical splinter skills. *Neurocase* 5, 503–509.

Hensch, Takao K. 2005. Critical period plasticity in local cortical circuits. *Nature Reviews Neuroscience* 6, 877–888.

Herbert, Martha R., Gordon J. Harris, Adrien Kristen, David A. Ziegler, Nikos Makris, David N. Kennedy, Nicholas T. Lange, Christopher F. Chabris, Anna Bakardjiev, James Hodgson, Takeoka Masanori, Helen Tager-Flusberg & Verne S. Caviness. 2002. Abnormal asymmetry in language association cortex in autism. *Annals of Neurology* 52, 588–596.

Herbert, Martha R., David A. Ziegler, Curtis Deutsch, Liam M. O'Brien, David N. Kennedy, Fillipek, P.A., Anna Bakardjiev, James Hodgson, Takeoka Masanori, Nikos Makris, Caviness & Verne S. 2004. Brain asymmetries in autism and developmental language disorders: A nested whole-brain analysis. *Brain* 128, 213–226.

Hickok, Gregory & David Poeppel. 2007. The cortical organization of speech processing. *Nature Reviews Neuroscience* 8, 393–402.

Hill, Elisabeth L. 2004. Executive dysfunction in autism. *Trends in Cognitive Sciences* 8, 26–32.

Hinzen, Wolfram. 2008. Prospects for an explanatory theory of semantics. *Biolinguistics* 2, 348–363.

Hinzen, Wolfram. 2009. Hierarchy, merge and truth. In Massimo Piattelli-Palmarini, Juan Uriagereka & Pello Salaburu (eds.), *Of Minds and Language: A Dialogue with Noam Chomsky in the Basque Country*, 123–140. Oxford: Oxford University Press.

Hoffman, Donald. 1998. *Visual Intelligence*. New York: Norton.

Hulburt, Russ, T., Happé, Francesca G.E. & Frith, Uta. 1994. Sampling the form of inner experience in three adults with Asperger syndrome. *Psychological Medicine* 24, 385–395.

Humphrey, Nicholas. 1998. Cave art, autism and the evolution of the human mind. *Cambridge Archaeological Journal* 8, 165–191.

Hutsler, Jeffrey. 2003. The specialized structure of human language cortex: Pyramidal cell size asymmetries within auditory and language-associated regions of the temporal lobes. *Brain and Language* 86, 226–242.

Hutsler, Jeffrey & Ralf A.W. Galluske. 2003. Hemispheric asymmetries in cerebral cortical networks. *Trends in Neuroscience* 36, 429–435.

Indefrey, Peter & Willem J. M. Levelt. 2004. The spatial and temporal signatures of word production components. *Cognition* 92, 101–144.

Jackendoff, Ray. 1983. *Semantics and Cognition*. Cambridge, MA: MIT Press.

Jansson-Verkasalo, Eira, Rita Ceponienè, Marko Kielinen, Kalervo Suominen, Ville Jäntti, Sirkka-Liisa Linna, Irma Moilanen & Risto Näätänen. 2003. Deficient auditory processing in children with Asperger syndrome, as indexed by event-related potentials. *Neuroscience Letters* 338, 197–200.

Järvninen-Palsey, Anna, Gregory L. Wallace, Franck Ramus, Francesca Happé & Pamela Heaton. 2008. Enhanced perceptual processing of speech in autism. *Developmental Science* 11, 109–121.

Jenkins, Lyle. 2000. *Biolinguistics: Exploring the Biology of Language*. Cambridge: Cambridge University Press.

Jolliffe, Therese & Simon Baron-Cohen. 1997. Are people with autism and Asperger syndrome faster than normal on the Embedded Figure Test. *Journal of Child Psychology and Psychiatry* 38, 527–534.

Joseph, Robert M., Helen Tager-Flusberg & Catherine Lord. 2002. Cognitive profiles and social-communicative functioning in children with autism spectrum disorder. *Journal of Child Psychology and Psychiatry* 43, 807–821.

Jou, Roger J., Nancy J. Minshew, Matcheri, S. Keshavan, Matthew P. Vitale & Antonio Y. Hardan. 2010. Enlarged right superior temporal gyrus in children and adolescent with autism. *Brain Research* 1360, 205–210.

Just, Marcel A., Vladimir L. Cherkassky, Timothy A. Keller, Rajesh K. Kana & Nancy J. Minshew. 2005. Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI Study of an executive function task and corpus callosum morphometry. *Cerebral Cortex* 17, 951–961.

Just Marcel A. Vladimir L. Cherkassky, Timothy A. Keller & Nancy J. Minshew. 2004b. Cortical activation and synchronization during sentence comprehension in high-functioning autism: evidence of underconnectivity. *Brain* 127, 1811–1821.

Just, Marcel A., Sharlene D. Newman, Timothy A. Keller, Alice McEleney & Patricia A. Carpenter. 2004a. Imagery in sentence comprehension: An fMRI study. *NeuroImage* 21, 112–124.

Kamio Yoko & Motomi Toichi. 2000. Dual access to semantics in autism: Is pictorial access superior to verbal access? *Journal of Child Psychology and Psychiatry* 41, 859–867.

Kana, Rajesh K., Timothy Keller, Vladimir L. Cherkassky, Nancy J. Minshew & Just, Marcel A. 2006. Sentence comprehension in autism: Thinking in pictures with decreased functional connectivity. *Brain* 129, 2484–2493.

Kemper, Thomas L. & Margaret L. Bauman. 1998. Neuropathology in infantile autism. *Journal of Neuropathology and Experimental Neurology* 57, 645–652.

Kjelgaard, Margaret M. & Helen Tager-Flusberg. 2001. An investigation of language impairment in autism: Implications for genetic subgroups. *Language and Cognitive Processes* 16, 287–308.

Koechlin, Etienne, Chrystèle Ody & Frédérique Kouneiher. 2003. The architecture of cognitive control in the human prefrontal cortex. *Science* 302, 1181–1185.

Koechlin, Etienne & Thomas Jubault. 2006. Broca's area and the hierarchical organization of human behavior. *Neuron* 50, 963–974.

Konishi, Seiki, Kyochi Nakajima, Idai Uchida, Masashi Kamevama, Kiyoshi Nakahara, Kensuke Sekihara & Yasushi Miyashita. 1998. Transient activation of inferior prefrontal cortex during cognitive set shifting. *Nature Neuroscience* 1, 89–94.

Koshino, Hideya, Patricia A. Carpenter, Nancy J. Minshew, Vladimir L. Cherkassky, Timothy A. Keller & Marcel A. Just. 2005. Functional connectivity in an fMRI

working memory task in high-functioning autism. *NeuroImage* 24, 810–821.
Kunda, Maithilee & Ashok K. Goel. 2011. Thinking in pictures as a cognitive account of autism. *Journal of Autism and Developmental Developmental Disorders* 41, 1157–1177.
Landa, Rebecca. 2000. Social Language use in Asperger syndrome and high-functioning autism. In Ami Klin, Fred R. Volkmar & Sara S. Sparrow (eds.), *Asperger Syndrome*, 159–171. New York: The Guilford Press.
Lepistö, Tuulia, Marika Kajander, Raija Vanhala, Paavo Alku, Minna Huotilainen, Risto Näätänen & Teija Kujala. 2008. The perception of invariant speech features in children with autism. *Biological Psychology* 77, 25–31.
Lepistö, Tuulia, Teija Kujala, Raija Vahala, Paavo Alku, Minna Huotilainen & Risto Näätänen. 2005. The discrimination of and orienting to speech and non-speech sounds in children with autism. *Brain Research* 1066, 147–157.
Levitin, Daniel J. & Anna K. Tirovolas. 2009. Current advances in the cognitive neuroscience of music. *Annals of the New York Academy of Sciences* 1156, 211–231.
Lewis, Vicky & Jill Boucher. 1995. Generativity in the play of young people with autism. *Journal of Autism and Developmental Disorders* 25, 105–121.
Lopez, Beatriz & Susan R. Leekam. 2003. Do children with autism fail to process information in context? *Journal of Child Psychology and Psychiatry* 44, 285–300.
Luo, Huan, Zuxiang Liu & David Poeppel. 2010. Auditory cortex tracks both auditory and visual stimulus dynamics using low-frequency neuronal phase modulation. *PLoS Biology* 8, e1000445.
Luo, Huan & David Poeppel. 2007. Phase patterns of neuronal responses reliably discriminate speech in human auditory cortex. *Neuron* 54, 1001–1010.
Marcus, Gary F. & Hugh Rabagliati. 2006. What developmental disorders can tell us about the nature and origins of language. *Nature Neuroscience* 9, 1226–1229.
Markram, Henry, Tania Rinaldi & Kamila Markram. 2007. The Intense World Syndrome — an alternative hypothesis for autism. *Frontiers in Neuroscience* 1, 77–96.
McAlonan, Grainne M., Vinci Cheung, Charlotte Cheung, John Suckling, Lam, Grace Y., K.S. Tai, L. Yip, Declan G.M. Murphy & Siew E. Chua. 2005. Mapping the brain in autism. A voxel-based MRI study of volumetric differences and intercorrelations in autism. *Brain* 128, 268–276.
McCleery, Joseph, Rita Ceponienè, Karen M. Burner, Jeanne Townsend, Mikaela Kinnear & Laura Schreibman. 2010. Neural correlates of verbal and nonverbal semantic integration in children with autism spectrum disorders. *Journal of Child Psychology and Psychiatry* 51, 277–286.
McGurk, Harry & John McDonald. 1976. Hearing lips and seeing voices. *Nature* 264, 746–748.
Miller, Leon K. 1989. *Musical Savants: Exceptional Skills in the Mentally Retarded*. Hillsdale, NJ: Erlbaum.
Minshew, Nancy J., Gerald Goldstein & Don J. Siegel. 1997. Neuropsychologic functioning in autism: Profile of a complex information processing disorder. *Journal*

of the International Neuropsychological Society 3, 303–316.

Mongillo, Elizabeth A., Julia R. Irwin, Doug H. Whalen, Cheryl Klaiman, Alice S. Carter & Robert T. Schultz. 2008. Audiovisual processing in children with and without Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders* 38, 1349–1358.

Mottron, Laurent. 2003. *L'autisme: Une autre intelligence.* Bruxelles: Mardagas.

Mottron, Laurent & Sylvie Belleville. 1993. A study of perceptual analysis in a high-level autistic subject with exceptional graphic abilities. *Brain and Cognition* 23, 279–309.

Mottron Laurent, Michèle Dawson, Isabelle Soulières, Bénédicte Hubert & Jake Burack. 2006. Enhanced perceptual functioning in autism: An update and eight principles of autistic perception. *Journal of Autism and Developmental Disorders* 36, 23–47.

Mottron, Laurent, Karine Morasse & Sylvie Belleville. 2001. A study of memory functioning in individuals with autism. *Journal of Child Psychology and Psychiatry* 42, 253–260.

Mottron, Laurent, Isabelle Peretz & Élise Ménard. 2000. Local and global processing of music in high-functioning persons with autism: Beyond central coherence? *Journal of Child Psychology and Psychiatry* 41, 1057–1065.

Mountcastle, Vernon B. 1997. The columnar organization of the neocortex. *Brain* 120, 701–722.

Müller, Ralph-Axel, M.E. Behen, R.D. Rothermel, D.C. Chugani, O. Musik, T.J. Mangner, H.T. Chugani. 1999. Brain mapping of language and auditory perception in high-functioning autistic adults: A PET study. *Journal of Autism and Developmental Disorders* 29, 19–31.

Murias, Michael, Sara J. Webb, Jessica Greenson & Geraldine Dawson. 2007. Resting state cortical connectivity reflected in EEG coherence in individuals with autism. *Biological Psychiatry* 62, 270–273.

Musso, Mariacristina, Andrea Moro, Volkmar Glauche, Michel Rijntjes, Jürgen Reichenbach, Christian Büchel & Cornelius Weiller. 2003. Broca's area and the language instinct. *Nature Neuroscience* 6, 774–780.

Näätänen, Risto & Anthony W.K. Gaillard & Sirkka Mäntysalo. 1978. Early selective attention effect on evoked potential reinterpreted. *Acta Psychologica* 42, 313–329.

Näätänen, Risto. 1990. The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behavioral and Brain Sciences* 13, 201–288.

Nadig, Aparna. 2011. Use of sentence context for lexical ambiguity resolution in high functioning autism. Paper presented at the *12th International Congress for the Study of Child Language,* Montreal (19–23 July 2011).

Nadig, Aparna & Holly Shaw. 2012. Acoustic and perceptual measurement of expressive prosody in high-functioning autism: Increased pitch range and what it means to listeners. *Journal of Autism and Developmental Disorders* 42, 499–511.

Nation, Kate, Paula Clarke, Barry Wright & Christine Williams. 2006. Patterns of reading ability in children with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders* 36, 911–919.

Navon, David. 1977. Forest before trees: The precedence of global features in visual perception. *Cognitive Psychology* 9, 353–383.

Newbury, Dianne F., E. Bonora, J. Lamb, S.E Fisher, C.S.L. Lai, G. Baird, L. Jannoun, V. Slonims, C.M. Stott, M.J. Merricks, P.F. Bolton, A.J. Bailey, A.P. Monaco & the International Molecular Genetic Study of Autism Consortium. 2002. *FOXP2* is not a major susceptibility gene for autism or Specific Language Impairment. *American Journal of Human Genetics* 70, 1318–1327.

Newschaffer Craig J., Lisa A. Croen, Julie Daniels, Ellen Giarelli, Judith K. Grether, Susan E. Levy, David S. Mandell, Lisa A. Miller, Jennifer Pinto-Martin, Judy Reaven, Ann M. Reynolds, Catherine E. Rice, Diana Schendel & Gayle Windham. 2007. The epidemiology of Autism Spectrum Disorders. *Annual Reviews of Public Health* 28, 21–24.

Newton, Ashley & Jill G. deViliers. 2007. Thinking while talking: Adults fail false belief attribution. *Psychological Science* 18, 574–579.

Norbury, Courtenay F. 2005. Barking up the wrong tree? Lexical ambiguity resolution in children with language impairments and autistic spectrum disorders. *Journal of Experimental Child Psychology* 90, 142–171.

Ozonoff, Sally & Jenise Jensen. 1999. Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders* 29, 171–177.

Ozonoff, Sally & Ryan E. McEvoy. 1994. A longitudinal study of executive function and theory of mind development in autism. *Development and Psychopathology* 6, 415–431.

Ozonoff, Sally, Bruce F. Pennington & Sally Rogers. 1991. Executive function deficits in high-functioning autistic individuals: Relationship to Theory of Mind. *Journal of Child Psychology and Psychiatry* 32, 1081–1105.

Paul, Rhea, Amy Augustyn, Ami Klin & Fred R. Volkmar. 2005. Perception and production of prosody by speakers with Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders* 35, 205–220.

Peppé, Susan, Joanne McCann, Fiona Gibbon, Anne O'Hare & Mel Rutherford. 2007. Receptive and expressive prosodic ability in school-age children with high-functioning autism. *Journal of Speech, Language and Hearing Research* 50, 1015–1028.

Perovic, Alexandra, Nadia Modyanova & Kenneth Wexler. 2007. Knowledge of c-command and A-movement in children and adolescents with autism and with Asperger syndrome. Paper presented at *GALA 2007: Generative Approaches to Language Acquisition*, Barcelona (6–8 September 2007).

Pierce, Karen, Ralph-Axel Müller, J. Ambrose, Greg Allen, Eric Courchesne. 2001. Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain* 124, 2053–2073.

Postle, Bradley R., Jeffrey S. Berger, Mark D'Esposito. 1999. Functional neuroanatomical double dissociation of mnemonic and executive control processes contributing to working memory performance. *Proceedings of the National Academy of Sciences of the United States of America* 96, 12959–12964.

Rao, Rajesh P. N. & Dana H. Ballard. 1999. Predictive coding in the visual cortex: A functional interpretation of some extra-classical fields effects. *Nature Neuroscience* 2, 77–87.

Raymond, Gerald V., Margaret L. Bauman & Thomas L. Kemper. 1996. Hippocampus in autism: A Golgi analysis. *Acta Neuropathologica* 91, 117–119.

Redcay, Elizabeth, Eric Courchesne. 2008. Deviant functional magnetic resonance imaging patterns of brain activity to speech in 2-3-year-old-children with Autism Spectrum Disorders. *Biological Psychiatry* 64, 589–598.

Reuland, Eric. 2001. Primitives of binding. *Linguistic Inquiry* 32, 439–492.

Ring, Howard A., Simon Baron-Cohen, Sally Wheelwright, Steve C.R. Williams, Mick Brammer, Chris Andrew & Edward T. Bullmore. 1999. Cerebral correlates of preserved cognitive skills in autism. *Brain* 122, 1305–1315.

Roberts, Timothy P.L., Sarah Y. Khan, Michael M. Rey, Justin F. Monroe, Katelyn Cannon, Lisa Blaskey, Sarah Woldoff, Saba Quasmieh, Mike Gandal, Gwen L. Schmidt, Deborah Zarnow, Susan E. Levy & Christopher J. Edgar. 2010. MEG detection of delayed auditory evoked responses in autism spectrum disorders: Towards an imaging biomarker for autism. *Autism Research* 3, 8–18.

Rojas, D. C., S. D. Bawn, T. L. Benkers, M. L. Reite & S. J. Rogers. 2002. Smaller left hemisphere planum temporale in adults with autistic disorder. *Neuroscience Letters* 328, 237–240.

Rojas, Donald C., Suzanne L. Camou, Martine L. Reite & Sally J. Rogers. 2005. Planum temporale volume in children and adolescents with autism. *Journal of Autism and Developmental Disorders* 35, 479–486.

Russo, Natalie, Tara Flanagan, Grace Iarocci, Darlene Berringer, Philip D. Zelazo & Jacob A. Burack. 2007. Deconstructing executive deficits among persons with autism: Implications for cognitive neuroscience. *Brain and Cognition* 65, 77–86.

Russo, Nicole, Charles Larson & Nina Krauss. 2008. Audio-vocal system regulation in children with autism spectrum disorders. *Experimental Brain Research* 188, 111–124.

Sahyoun, Chérif P., John W. Belliveau, Isabelle Soulières, Shira Schwartz & Maria Mody. 2010. Neuroimaging of the functional and structural networks underlying visuospatial vs. linguistic reasoning in high-functional autism. *Neuropsychologia* 48, 86–95.

Samson, Fabienne, Laurent Mottron, Jemel Boutheina, Pascal Belin, Valter Ciocca. 2006. Can spectro-temporal complexity explain the autistic patterns of performance on auditory tasks? *Journal of Autism and Developmental Disorders* 36, 65–75.

Selfe, Lorna. 1995. *Nadia Revisited: A Longitudinal Study of an Autistic Savant*. New York: Taylor & Francis.

Senkowski, Daniel, Till R. Schneider, John J. Foxe & Andreas K. Engel. 2008. Crossmodal binding through neural coherence: Implication for multisensory processing. *Trends in Neuroscience* 31, 401–409.

Shah, Amitta & Uta Frith. 1983. An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry* 24, 613–620.

Shallice, Tim. 1982. Specific impairments of planning. *Philosophical Transaction of the Royal Society of London* 298, 199–209.

Shallice, Tim. 1988. *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.

Shriberg, Lawrence, Rhea Paul, Jane L. McSweeny, Ami Klin, Donald J. Cohen & Fred R. Volkmar. 2001. Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language and Hearing Research* 44, 1097–1115.

Siegal, Michael & Mark Blades. 2003. Language and auditory processing in autism. *Trends in Cognitive Sciences* 7, 378–380.

Smith, Edward E & John Jonides. 1999. Storage and executive processes in frontal lobes. *Science* 283, 1657–1661.

Soulières, Isabelle, Michelle Dawson, Fabienne Samson, Elise B. Barbeau, Cherif P. Sahyoun, Gary E. Strangman, Thomas A. Zeffiro & Laurent Mottron. 2009. Enhanced visual processing contributes to matrix reasoning in autism. *Human Brain Mapping* 30, 4082–4107.

Steiman, Mandy, Rebecca Simon, Lisa Reisinger & Eric Fombonne. 2011. Trends in autism rates — Is there an epidemic? In M. Elena Garralda & Jean-Philippe Raynaud (eds.), *Increasing Awareness of Child and Adolescent Mental Health*, 162–192. Lanham: Jason Aronson.

Stephan, Klaas E., Gereon R. Fink & John C. Marshall. 2007. Mechanisms of hemispheric specialization: Insights from analyses of connectivity. *Neuropsychologia* 45, 209–228.

Stuss, Donald T., Gordon G. Gallup Jr. & Michael P. Alexander. 2001. The frontal lobes are necessary for 'Theory of Mind'. *Brain* 124, 279–286.

Surian, Luca, Simon Baron-Cohen & Heather van der Lely. 1996. Are children with autism deaf to Gricean Maxims? *Cognitive Neuropsychiatry* 1, 55–71.

Tager-Flusberg, Helen. 1992. Autistic children's talk about psychological states: Deficits in the early acquisition of a Theory of Mind. *Child Development* 63, 161–172.

Tager-Flusberg, Helen, Susan Calkins, Tina Nolin, Therese Baumberger, Marcia Anderson & Ann Chadwick-Dias. 1990. A longitudinal study of language acquisition in autistic and Down syndrome children. *Journal of Autism and Developmental Disorders* 20, 1–21.

Tager-Flusberg, Helen & Robert M. Joseph. 2003. Identifying neurocognitive phenotypes in autism. *Philosophical Transaction of the Royal Society* 358, 303–314.

Tager-Flusberg, Helen & Robert M. Joseph. 2005. How language facilitates the acquisition of false-belief understanding in children with autism. In Janet

Wilde & Jodie A. Baird (eds.), *Why Language Matters for Theory of Mind*, 298–318. New York: Oxford University Press.

Tager-Flusberg, Helen, Rhea Paul & Catherine Lord. 2005. Language and communication in autism. In Fred R. Volkmar, Rhea Paul, Ami Klin & Donald L. Cohen (eds.), *Handbook of Autism and Pervasive Developmental Disorders,* 3rd edn., 335–364. Hoboken, NJ: Wiley & Sons.

Tattersall, Ian. 1998. *Becoming Human: Evolution of Human Uniqueness*. New York: Harcourt Brace.

Tattersall, Ian. 2004. What happened in the origin of human consciousness? *The Anatomical Records* 276B, 19–26.

Toichi, Motomi & Yoko Kamio. 2002. Long-term memory and levels of processing in autism. *Neuropsychologia* 40, 964–969.

Turner, Michelle A. 1999. Generating novel ideas: Fluency performance in high-functioning and learning disabled individuals with autism. *Journal of Child Psychology and Psychiatry* 40, 189–201.

Ullman, Michael T. 2004. Contributions of memory circuits to language: The declarative/procedural model. *Cognition* 92, 231–270.

Vernes Sonja C., Dianne F. Newbury, Brett S. Abrahams, Laura Winchester, Jérôme Nicod, Matthias Groszer, Marciela Alarcon, Peter L. Olivier, Kay E. Davies, Daniel H. Geschwind, Anthony P. Monaco & Simon E. Fisher. 2008. A functional genetic link between distinct developmental language disorders. *The New England Journal of Medicine* 359, 2337–2345.

Vertes, Robert P. 2005. Hippocampal theta rhythm: A tag for short-term memory. *Hippocampus* 15, 923–935.

de Villiers, Jill G. & Jennie E. Pyers. 2002. Complements to cognition: A longitudinal study of the relationship between complex syntax and false-belief-understanding. *Cognitive Development* 17, 1037–1060.

Volden, Joanne & Catherine Lord. 1991. Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders* 21, 109–130.

Volkmar, Fred R. & Ami Klin. 2005. Issues in the classification of autism and related conditions. In Fred R. Volkmar, Rhea Paul, Ami Klin & Donald L. Cohen (eds.), *Handbook of Autism and Pervasive Developmental Disorders,* 3rd edn., 5–41. Hoboken, NJ: Wiley & Sons.

Vygotsky, Lev. 1962. *Thought and Language*. Cambridge, MA: MIT Press.

Walenski, Matthew, Stewart H. Mostofsky, Jennifer C. Gidley-Larson & Michael T. Ullman. 2008. Brief report: Enhanced picture naming in autism. *Journal of Autism and Developmental Disorders* 38, 1395–1399.

Walsh, Christopher A., Eric M. Morrow & John L. R. Rubenstein. 2008. Autism and brain development. *Cell* 135, 396–400.

van Wassenhove, Virginie, Ken Grant & David Poeppel. 2005. Visual speech speeds up the neural processing of auditory speech. *Proceedings of the National Academy of Sciences of the United States of America* 102, 1181–1186.

White, Sarah J. & David Saldaña. 2011. Performance of children with autism on the

embedded figure test: A closer look at a popular task. *Journal of Autism and Developmental Disorders* 41, 1565–1572.

Whitehouse, Andrew J.O. & Dorothy V.M. Bishop. 2008. Cerebral dominance for language function in adults with specific language impairment or autism. *Brain* 131, 3293–3200.

Whitehouse, Andrew J.O., Johanna G. Barry & Dorothy V.M. Bishop. 2007. The broader language phenotype of autism: A comparison with specific language impairment. *Journal of Child Psychology and Psychiatry* 48, 822–830.

Williams, David, Francesca G.E. Happé & Christopher Jarrold. 2008. Intact inner speech use in autism spectrum disorder: Evidence from a short-term memory task. *Journal of Child Psychology and Psychiatry* 49, 51–58.

Williams, Diane L., Gerald Goldstein, Patricia A. Carpenter & Nancy J. Minshew. 2005. Verbal and spatial working memory in autism. *Journal of Autism and Developmental Disorders* 35, 747–756.

Williams, Emily L. & Manuel F. Casanova. 2010. Autism and dyslexia: A spectrum of cognitive styles as defined by minicolumnar morphometry. *Medical Hypotheses* 74, 59–62.

Williams, Justin H. G., Dominic W. Massaro, Natalie J. Peel, Alexis Bosseler & Thomas Suddendorf. 2004. Visual-auditory integration during speech imitation in autism. *Research in Developmental Disabilities* 25, 559–575.

Wilson, Tony W., Donald C. Rojas, Martin L. Reite, Peter D. Teale & Sally J. Rogers. 2007. Children and adolescents with autism exhibit reduced MEG steady-state gamma responses. *Biological Psychiatry* 62, 192–197.

Wollman, David. 2008. Yeah, I'm autistic. You got a problem with that? *Wired Magazine* (16 March 2008).

Nicolas Bourguignon & Daniel Valois
University of Montreal
Department of Linguistics
C.P. 6128, succursale Centre-ville
Montreal, QC H3C 3J7
Canada

Aparna S. Nadig
McGill University
School of Communication Sciences & Disorders
1266, Pine Av. West
Montreal, QC H3G 1A8
Canada

nicolas.bourguignon@umontreal.ca, daniel.valois@umontreal.ca, aparna.nadig@mcgill.ca