Language impairments in autism: evidence against mind-blindness
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Introduction
Autism is a developmental disorder which causes severe cognitive and social deficits and affects four in every ten thousands individuals. A triad of impairments — impairments in social interaction, impairments in verbal and non verbal communication and an inappropriately restrictive behaviour — are found across the entire autistic population and are critical for a diagnostic. Characteristically, the severity of these impairments varies from individuals to others and even, within the same individual. Because autism is marked by great variation, it is mostly referred to as the Autistic Spectrum of Disorders (ASD). Although not universal in autism, deficits in language, particularly delays in language acquisition, are the principal early manifestation of the disorder. Again, the range of language abilities varies and goes from total muteness to the use of an apparent grammatically complex language. But it appears that over half of the autistic population is affected by some sort of language impairments. In many cases, these impairments involve all aspects of language — pragmatic, lexical, syntactic, morphological, phonological and phonetic.

The heterogeneity of the autistic population does not make the task of finding a cause for the spectrum of disorders easy. But one theory, Mind-blindness, has been very popular among psychologists working on ASD. The Mind-blindness hypothesis holds that one unique deficit, in the cognitive function of mentalizing, causes the range of deficits found in autism. Children with autism are prevented from a normal development because they lack a Theory of Mind, which is the ability to recognize that one and others have mental states, such as thoughts, wishes, intentions or beliefs and that these can be different from self’s. Language impairments found in autism come as consequences of more general deficiencies in communication caused by this deficit in the Theory of Mind.

In this paper, we argue that recent research into language impairments in autism and other developmental disorders invites us to re-evaluate this common consensus.

1 Tager-Flusberg, 2000a
2 Baron-Cohen et al., 1985
3 Frith, 2003; Tager-Flusberg, Ibid; Howlin, 2003
4 Hill & Frith, 2003
5 Happé, Ibid
6 Risi et al., Ibid
7 Aarons & Gittens, 1999
8 Aarons & Gittens, 1999; Young et al., 2005
9 Lord et al., Ibid
10 Tager-Flusberg, 2000b; Tager-Flusberg & Joseph, 2005
11 Tager-Flusberg, 2004
12 Baron-Cohen, 1995; Kjelgaard & Tager-Flusberg, 2001
13 Tager-Flusberg, 2004b
14 Baron-Cohen et al., 1985; Baron-Cohen, 1995; Gillot, Furniss & Walter, 2004
15 Frith & Happé, 1998; Frith, 1998
course, the aim of our re-evaluation is not to deny that a mentalizing deficit effectively underlies the range of social and communicative disorders. But Mind-blindness does not account for the whole picture of language deficits found in autism\textsuperscript{16}. And in the context of research findings in different fields (Linguistics, Genetics and Neurobiology) concerning the possible modular nature of the mind, a specific language disorder should, perhaps, be included into the set of cognitive deficits brought forward to explain the Autistic Spectrum of Disorders.

1. Autism and Language-Specific Research

A range of language impairments are characteristic of verbal autistic children. One category of impairments, Pragmatic Language Disorders (PLDs), affects all autistic children (Young et al., 2005). Those Pragmatic Language Disorders are characterised by deficits in comprehension, in particular (i) a low understanding of non-literal sequences such as metaphors, jokes or irony; (ii) a poor command of indirect speech acts such as questions (Aarons & Gittens, 1999) and (iii) difficulties with presuppositions and other conversational conventions such as politeness, turn-taking or “levels of formality” (Young et al., 2005). Linked to PLDs are further impairments in the production of speech. Among those are (i) personal pronouns reversal — for instance the use of “I” instead of “you” and vice-versa —, (ii) the misuse of such prepositions as “in”, “on”, “under”, “next to” (…), and (iii) the prevalence, in speech, of echolalia — formal repetition of other’s utterances (Fay, 1988; Aarons & Gittens, 1999).

\textsuperscript{16} Tager-Flusberg, 2004; Williams et al., forthcoming
Because children with autism have severe impairments in communication and are often reluctant to speak, especially around unknown people, a broad analysis of their verbal skills remains challenging. As a consequence only a few studies have focused on non-pragmatic aspects of the autistic speech. (Tager-Flusberg, 2000a; Tager-Flusberg, 2004b) But other levels of language are also affected. Thus, children with autism have been found to have impaired phonological skills. For instance, their speech systematically lacks intonation and can best be described as monotonous (Baron-Cohen, 1995). More specific problems with the analysis of phonological representation of original phonetic sequences have also been reported (Kjelgaard & Tager-Flusberg, 2001).

Most striking, are recent analysis associating grammatical deficits to the disorder. Amongst others, are problems with morphological marking of finite verbs, such as the omission of the past tense morpheme -ed or -s marking the third person of the singular (Tager-Flusberg, 2004a). Other studies report problems with the parsing of subordinate clauses that follow verbs of cognition, such as thing, know or guess and may also have difficulties understanding complex wh-questions, such as (a), where the wh-phrase is extracted from a lower syntactic position in the embedded clause. (Tager-Flusberg, 2000b: 20; Joseph & Tager-Flusberg, 2005).

a) When did the girl say that she broke the radio when

Another cognitive module, associated with the language faculty and responsible for auditory processing, has been extensively investigated and found to be severely deficient in a few cases of autism (Siegal & Blades, 2003). In one particular study (Čeponienė et al., 2003), 9 children with high-functioning autism were examined on their abilities to discriminate various sequences of non-speech sounds, such as simple and complex tones, and sequences of speech-sounds, such as vowels. Their brain responses to each type of sounds were compared to those of 10 normal children without hearing impairments or learning disabilities. During the examination, one standard and one deviant stimulus for each class of sounds were conveyed to the children via two loud-speakers while they were watching a soundless video on television. Although, children were told not to pay attention to auditory stimuli projected by the speakers and to focus on the video instead, their attention to particular type of sounds was actually observed. Čeponienė et al. found that children in the autistic group, like those in the control group, were unimpaired in sensory sound processing. Both groups responded well to all types of stimuli and did not display major problems in discriminating simple and complex non-speech sounds. However, children with autism were found to have specific difficulties with the discrimination of vowels. This display of an impaired “attentional orientating for speech sounds” only, neither linked to a deficit in sensory processing or to the complexity of sounds, led to the suggestion that the auditory processor dealing with the analysis of speech sounds, such as vowels and maybe consonants, was not fully functional in autism. Hence, it was concluded that individuals with autism were impaired in auditory processing, but that this deficiency was entirely related to speech sounds. Impairments in language found in autism, or at least those related to phonology, could possibly be derived from such a deficient auditory processor.

17 Children with milder autistic symptoms and higher verbal and non verbal IQ.
The research on auditory processing is still new and more advanced investigations on this particular area are needed. Yet, primary results seem to be consistent with the possibility of a damaged language faculty being at the origin of some language deficits associated with autism. Also consistent with the proposal are recent findings from Tager-Flusberg and Joseph (2003) of a neurocognitive phenotype for autism characterised by language capacities. Looking at previous studies on language impairments in autism, they found that the autistic population could be divided into two subgroups: one with normal language skills and one with a severely impaired language. Those in the impaired language subgroup had discrepancies at all levels of language, including complex syntax and morphology. Furthermore, their profile was distinct from the profiles of other subgroups, defined by other criteria, such as verbal and non-verbal IQ discrepancies. Their classification of the autistic population highlights the fact that, unlike deficits in social interaction, communication and general behaviour, language deficiency is not universal in autism (Kjelgaard & Tager-Flusberg, 2001). It also puts the accent on the fact that some children affected by the core symptoms of autism develop an otherwise normal language. Therefore, in these cases, normal acquisition of language in autism is possible despite severe deficits in Theory of Mind. For Tager-Flusberg and Joseph (Ibid), language deficiency constitutes one of the various specific phenotypes of autism rather than a mere general consequence of other phenotypes.

2. Autism and other Developmental Disorders
Similarities between the language deficits found in autism and those found in other developmental disorders, such as Down syndrome and Specific Language Impairment also support this hypothesis. Children diagnosed with Down syndrome, a genetic disorder caused by an abnormal trisomy of chromosome 21, display some language impairments similar to those found in ASD. The disorder, as autism, is commonly associated with specific cognitive impairments. Attention span, auditory processing, short term memory and symbolization are assessed as very poor. Furthermore, mental retardation seems to be systematic within the population affected, with the majority of individuals not going beyond a mental age of five years. Language acquisition is also delayed compared to normally developing children but phonological, lexical and pragmatic skills are on average acquired. In the case of morphosyntax, children with Down syndrome are slightly deficient. As children with autism, they have difficulties with function words, such as prepositions, auxiliaries and pronouns. They either do not employ them or use them inappropriately. Besides, their misuses of pronouns often affect the cohesive logic of their discourse. Furthermore, they tend to make mistakes with morphological tense markers and assign verbs with the right inflection in only half of their utterances. Children with Down syndrome also only rarely use morphemes denoting gender. Like autistic children, who tend to use a very basic syntax (Tager-Flusberg, 2000a), children with Down syndrome mostly produce telegraphic sentences. (Rondal, 1988:165-176; Abbeduto & Murphy, 2004: 77-86). But, unlike children with autism, children with

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Down syndrome have normal capacities in mind-reading and *Theory of Mind* (Baron-Cohen et al., 1985). Moreover, social interaction and both verbal and non-verbal communication are not impaired. On the contrary, they are amongst the strongest qualities of individuals with the disorder. (Laws & Bishop, 2004) Such facts, indeed, support the claim that impairments in the non-social aspects of the autistic language may not result from deficits in the *Theory of Mind* and may not come as consequences of more general problems in communication.

The idea that language impairments found in autism might be caused by a specific deficit affecting the language faculty, finds additional support in the recently recognized common linguistic characteristics shared by children with autism and children with SLI. Specific language impairment is a disorder present in nearly 7% of children and which prevents normal acquisition of language from taking place. It is not associated with any other cognitive or social discrepancies and occurs in children whose intellectual skills appear to be perfectly normal. Commonly, SLI is thought to be caused by a deficient language module. The range of language impairments associated with SLI is varied (van der Lely & Ullman, 2001), yet two types of deficits are considered to be at the heart of the disorder’s phenotype. Thus, all children with SLI have difficulties with the processing of phonological sequences and with the analysis of inflectional morphemes. Phonological deficiencies, particularly in building phonological representations for acoustic signals, are especially visible in tasks involving the repetition of nonsense words. In fact, these types of exercises are very poorly executed by all children with SLI. Problems with inflectional morphology, for their part, are demonstrated by a strong pattern of errors in the grammatical marking of tense - past tense among others - and the assignment of third person markers. (Tager-Flusberg, 2004a; van der Lely & Ullman, Ibid)

Tager-Flusberg & her colleagues investigated the connection of autism with SLI. In particular, they looked at whether core impairments found in the language disorder were also present in autism (Tager-Flusberg & Joseph, 2003). In a first study (Tager-Flusberg & Kjelgaard, 2001), they analysed the performances of 89 autistic children, aged between four and fourteen years old, on a battery of language tests normally used to diagnose SLI. Special attention was paid to their results on the non-word repetition task, involving oral reproductions of sequences of nonsense words. More than half of the population observed did not show satisfying competence. Contrasting these poor performances in non-word repetition with the high occurrence of direct and mitigated echolalia in autistic speech, Tager-Flusberg and Kjelgaard concluded that children with autism did not have specific problems of articulation. Although, autistic individuals did slightly better than children with SLI, who are reported to all systematically fail non-word repetition tasks, overall they were, like them, unable to analyse the phonetic characteristics of original sequences in order to get their phonological representations. In a second study19, 62 children from the original 89 who had taken part in the first study were tested on their morphosyntactic skills. The paradigms presented to the participants were particularly designed to (i) obtain regular and irregular past tense forms for series of lexical verbs such as *wash* or *fall* and (ii) bring out the morpheme for the third person singular in the present tense. Among the 62 children, 20 were classified as language-impaired. Overall, their performances in both tasks were found to be inferior to those in the normal language group. Most of their errors, like those noted in children with SLI, were to omit the past tense and third person

morphemes in contexts when they were obligatorily required. Convinced by their consistent findings on both studies, Tager-Flusberg and her peers proposed that the language deficits found in a subtype of autism were caused by the same language disorder affecting subjects with SLI.

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<th></th>
<th>IQ</th>
<th>Theory of Mind</th>
<th>Language</th>
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<tbody>
<tr>
<td>ASD</td>
<td>Low (under 70) to high</td>
<td>Severely impaired</td>
<td>From severely impaired to normal acquisition</td>
</tr>
<tr>
<td>SLI</td>
<td>Normal</td>
<td>Normal</td>
<td>Severely impaired</td>
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<tr>
<td>Down syndrome</td>
<td>Low</td>
<td>High</td>
<td>Impaired to normal acquisition</td>
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*Figure 2. Overlap between ASD, SLI and Down syndrome: IQ discrepancies, Theory of Mind, and language deficits*

Research on the overlap between autism, Down syndrome and SLI is still new and further analyses are needed before one brings out a definite conclusion (Tager-Flusberg, 2004). But the striking similarities between language impairments found in the three disorders, despite very different intellectual and social capabilities (as illustrated in Figure 2.1 below), here again, argue for the recognition of an additional cognitive deficit, involving the language module, to account for the wide range of autistic disorders. Of course, the fact that no consistent genotypic description for autism exists, adds to the difficulty in finding the origin of language impairments. Some topical issues in genetics, however, offer a motivating perspective.

3. Evidence from Genetic Studies

In recent years, research for the genetic origin of the cognitive and behavioural anomalies found in autism has centred its attention on the area surrounding chromosome 7q (Baron-Cohen, 2004). In 2002, Alarcón et al. investigated the presence of a possible Quantitative Trait Locus (QTLs), associated with the language phenotype of autism, on chromosome 7q. Because siblings of autistic individuals with language impairments have more difficulties linked to language — delayed acquisition of language, problems with spelling and slightly poorer articulation skills — than siblings of autistic subjects with normal linguistic skills, it was hypothesised that such QTLs could “underlie significant regions of autism linkage”. DNA samples were collected from members of 152 families from the Autism Genetic Resource Exchange and compared to measurements of three features of
the ADI-R\textsuperscript{20}: the age at production of first word, the age at production of first phrase and restrictive behaviours. Alarcón and her colleagues discovered that the three features of the ADI-R they had measured were consistently present amongst direct relatives of children with autism. Although, the age at first phrase did not seem to be related to a QTL on chromosome 7q, a strong connection between the age at production of first word and the chromosomal region was established. Relegating the absence of a correlation between chromosome 7q and first phrase onset to fallibilities in their methodological organisation and finding support in previous studies which had found a Quantitative Trait Locus for language on chromosome 7q 35-36, Alarcón et al. put forward two claims. First, a specific Locus for language is indeed located in the region of chromosome 7q. Second, anomalies in specific genes situated on this chromosome may be at the origin of language disorders found within the Autistic Spectrum of Disorders.

In another study, O'Brien et al. (2003) also analysed DNA samples, but this time from children with SLI and their siblings. Like Alarcón and her peers, they found a vivid correlation between the language phenotype of SLI and the region of chromosome 7q. Particularly, they established that the locus was situated nearby FOXP2 on chromosome 7q31 and that language impairments in SLI could be due to a genetic vulnerability in that region. Of course, categorical theories linking chromosome 7q with the language module are still premature. But, these initial breakthroughs should not be ignored by theorists looking for the cognitive causes of autism. And, as suggested by Frith (1998), we trust that a model of research into the origin of autism should recognise not one, but several distinct cognitive deficits. Such a model should integrate deficits in Theory of Mind, Executive functions\textsuperscript{21} and Central Coherence\textsuperscript{22}, as well as vulnerabilities of the language faculty as underlying deficits causing the Autistic Spectrum of Disorders.

4. The neurological perspective
Autism is associated with severe anomalies in the configuration of the cerebral organ (Hill & Frith, 2003). Such regions of the brain as the amygdala, the cerebellum and the frontal cortex — said to implicate the mentalising function — have been proved by neuroimaging and post-mortem analyses to function abnormally (Happé & Frith, 1996; Baron-Cohen, 2004). So far, language deficits have not consistently been linked to any specific brain abnormalities. Nevertheless, it is possible that another cerebral region, critical for normal language growth, is damaged in autism. Herbert et al. (2002, reviewed in Tager-Flusberg & Joseph, 2003) looked at whether the language impaired phenotype of autism could be associated with specific irregular growth patterns of the brain. They compared the brain structures of 16 male children with autism, who were part of a language impaired subgroup, with the brain structures of 16 boys without any language disorder or learning disabilities. In particular, they observed the size of the language area

\textsuperscript{20} Autism Diagnostic Interview revised
\textsuperscript{21} Like impairments in language and Theory of Mind, deficiencies in Executive Functions also constitute a subpart of the autistic phenotype. Problems with mental operations such as planning, working memory, mental flexibility and prepotent inhibition are said to cause ASD repetitive behaviours. (cf. Hill (2004) and Joseph et al. (2005) for a review of Executive Dysfunctions in autism)
\textsuperscript{22} This universal part of information processing is also deficient in autism. (cf. Frith & Happé (1994) for a brief overview of Weak Central Coherence in autism)
situated in the left and right hemispheres of the cortex. They found that the lateral frontal language cortex in the right hemisphere was significantly larger in autistic children with language impairments than in normal children. Besides, important language regions in the left hemisphere — perisylvian area, planum temporale and Heschel’s gyrus — of children with autism were found to be smaller than those of the children in the control group. They concluded that these cerebral asymmetries, also found in children with SLI and other language disorder, could be directly linked to the language impairments found in this subgroup of autistic children.

Other deficits found in autism have not yet been linked to enlargement of specific parts of the brain. It is therefore hard to draw a conclusion on the correlation between irregular size of specific brain regions and particular cognitive deficits. Yet, Herbert’s findings suggest that irregularities in specific areas of the brain could be responsible for the malfunction of cognitive modules located in those regions (Tager-Flusberg & Joseph, Ibid). And the idea of autism being caused by a language deficit, amongst other cognitive discrepancies, is not inconsistent with the anatomy of the autistic brain. Significant anomalies are, indeed, prominent across the entire cerebral configuration, including around the regions involved in language production and comprehension. The reasons for the defect of some cerebral regions are not categorically established. Yet, because of the plasticity of the brain, a neuronal dysfunction preventing the necessary connection between different cerebral sections seems more likely (Happé & Frith, 1996).

Williams et al. (forthcoming), in particular, suggest that a group of neurons located in the frontal cortex - the mirror neurons (MNs) - are defective in autism and cause the spectrum of disorders. The primary function of MNs is to link the information processed by a higher-level visual module to the motor cortex. Other type of processed information, such as recognition of facial expressions (linked to Theory of Mind) or vocal utterances
may also run in direction of the motor cortex through these particular neurons. According to their proposal, a breakdown in the structure of mirror neurons, which causes “poor modulation” in the brain and malfunctioning of critical cognitive functions, underlies the range of deficits found in autism, including those linked to language.

From a neurobiological perspective, it seems possible that language impairments found in autism are caused by an underlying language disorder. The classification of the autistic population into subgroups according to their phenotypes (Tager-Flusberg and her group, 2002; 2003) demonstrates a correlation between language impairments and vulnerabilities in the language regions of the autistic brain - particularly, brain size discrepancies. Williams and his colleagues (Ibid), who provide a plausible explanation onto the ontogenetic cause of autism, even integrate a fault in the language faculty into their model. But most striking, are the consistent findings of vulnerabilities in the frontal lobes area of the brain across the autistic population (Happé & Frith, 1996; Tager-Flusberg & Joseph, Ibid; Baron-Cohen, 2004; Williams et al., Ibid). Indeed, individuals with damage to the frontal lobes, caused by various developmental disorders or head injuries, display some of the same impairments as those commonly found in autism (Hill, 2004). Given those facts, future studies into the cause of language impairments in autism, investigating this particular area of the brain, may bring important breakthroughs. (Joseph et al. 2005).

**Conclusion**

The nature of linguistic impairments found in autism and recent breakthroughs in Genetics powerfully suggest that the language module of individuals with autism is deficient. Given the anatomy of the autistic brain and its scattered cerebral vulnerabilities, a model recognising a deficit of the language faculty, together with deficits in the Theory of Mind, as underlying sources of the Autistic Spectrum of Disorders seems coherent. Future studies looking at specific neuronal breakdowns and at the correlation between frontal lobes damage and specific language deficits in autism may bring crucial findings and, perhaps, shed light into the origins of this terrible disorder.

**References**


