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Language in autism and specific language impairment: Where are the links?

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Abstract
It has been suggested that language impairment in autism is behaviourally, neurobiologically, and etiologically related to specific language impairment (SLI). In this paper, we review evidence at each level and argue that the vast majority of data does not support the view that language impairment in autism can be explained in terms of co-morbid SLI. We make recommendations for how this debate might be resolved and we suggest a shift in research focus. We recommend that researchers concentrate on those aspects of language impairment that predominate in each disorder rather than on those comparatively small areas of potential overlap.

Keywords: Autism Spectrum Disorder; Specific Language Impairment

Autism is defined in terms of a triad of core impairments in social interaction, communication, and behavioural flexibility (American Psychiatric Association, 2000). The severity of each symptom varies on a continuum, and partial or atypical forms also occur. Moreover, structural language impairment and intellectual disability commonly co-occur in autism. Some individuals have mild language impairment and mild intellectual disability, whereas others are non-verbal with severe or profound cognitive impairments. Less frequently, language impairment co-occurs with autism independently of intellectual disability as measured by Performance IQ or Full Scale IQ. Other co-morbid physical, medical, mental-health, and developmental anomalies are common, especially in less able individuals. This heterogeneity has led to the widely accepted view of autism as a spectrum of related disorders and we use the terms 'autism' and 'autistic spectrum disorder' (ASD) interchangeably here. We use the term ASD-LI to refer to individuals with autism who have impairments in structural language, regardless of overall cognitive ability.

Specific language impairment (SLI), sometimes referred to as developmental language disorder, is diagnosed in children who display markedly impaired spoken language functioning with test scores at least 1.25 standard deviations below the mean, despite normal nonverbal intelligence, and with no apparent sensory or neurological dysfunction. SLI is not diagnosed if an autism-related disorder is present (American Psychiatric Association, 2000). Specific language impairment, like autism, consists of several subtypes. The most common subtypes are expressive language disorder (leaving comprehension relatively unimpaired), mixed receptive-expressive language disorder, and articulatory or phonological disorder (American Psychiatric Association, 2000; World Health Organisation, 1992). Studies utilising cluster analyses yield a larger set of more subtly differentiated subtypes, including one characterised by selective impairment of language use (pragmatics). In the current paper, pragmatic language impairment will be considered separately from more common forms of SLI that are characterized by structural language impairments (see discussion below). In summary, SLI, like autism, is a heterogeneous disorder with different aspects of language being more or less affected across individuals. The picture of SLI is further complicated by the fact that a notable minority of individuals with SLI move across subtypes throughout development (Conti-Ramsden & Botting, 1999).

Researchers have long thought that autism and SLI are related. One early hypothesis was that autism (then only diagnosed in people with structural language impairments) resulted from a severe developmental language disorder leading to social withdrawal, disorientation, and reactive rigidity of behaviour (Churchill, 1972; Rutter, 1967). Boucher (1976) argued against this hypothesis, however, on the grounds that severe developmental language disorder can occur independently of autism and that social impairments are usually more marked and persistent than structural language impairments among individuals with autism. In addition, detailed studies by Bartak, Rutter, and Cox (1975) and Cantwell, Baker and Rutter (1978) investigating language, cognition, and behaviour in children with autism and children with SLI suggested clear differences between the two diagnostic groups, although a subgroup of individuals with 'mixed' autism and SLI was identified.

When the concept of autism broadened to include Asperger syndrome, the suggestion that autism resulted from language impairment became untenable. This is because Asperger syndrome is, by definition, a triad of autism-related behaviours in the absence of clinically significant structural language impairment. Research into structural language impairments in

autism became sparse in subsequent years. Bartak et al.'s (1975) conclusions that language impairments in autism and in SLI are qualitatively different, but that mixed forms can occur, were widely accepted. Moreover, differences between language impairments in the two conditions appeared to be supported by studies showing that language impairment in autism is characterised by delay rather than deviancy (Tager-Flusberg, 1981; Tager-Flusberg et al., 1990).

In the 1990s, evidence from family studies suggested a genetic relation between vulnerability to autism and vulnerability to language-related developmental difficulties of various kinds, including SLI (e.g., Bolton et al., 1994; Folstein et al., 1999; Fombonne et al., 1997; Piven et al., 1997; Szatmari et al., 2000). These findings revived interest in the behavioural relation between language impairments in autism and in SLI and new studies suggested that the linguistic profiles in ASD-LI were more similar to the language profiles commonly seen in SLI than had been previously recognised (Kjelgaard & Tager-Flusberg, 2001; Rapin & Dunn, 2003; Roberts, Rice, & Tager-Flusberg, 2004). In addition to evidence for shared etiological factors and similar linguistic profiles, reports of shared neurobiology also began to appear (e.g., De Fossé et al., 2004; Herbert et al., 2002, 2005; Rojas et al., 2002).

In sum, evidence has accumulated to suggest that language impairments in autism and in SLI are more closely related than was once thought to be the case. Indeed, Tager-Flusberg and her colleagues (e.g., Roberts et al., 2004) have tentatively proposed that autism with accompanying language impairment can be explained in terms of co-morbid SLI. The aim of this paper is to evaluate evidence concerning the relation between the two disorders at the behavioural, neurobiological, and etiological levels and to evaluate the hypothesis that co-morbid SLI is the main cause of structural language impairment in autism.

Behavioural Aspects of Language Impairment in Specific Language Impairment and in Autism

Recent evidence suggests that the linguistic profiles of individuals with ASD-LI are more similar to the language profiles commonly seen in SLI than was assumed over recent decades to be the case (Kjelgaard & Tager-Flusberg, 2001; Rapin & Dunn, 2003; Roberts et al., 2004). Moreover, recent studies have shown that individuals with ASD-LI perform poorly on cognitive measures that are sensitive clinical markers for, and perhaps cognitive endophenotypes of, SLI. These studies have been cited in favour of the argument that ASD-LI and SLI are partially overlapping disorders (Botting & Conti-Ramsden, 2003; Kjelgaard & Tager-Flusberg, 2001; Roberts et al., 2004). In this section, evidence for this claim is critically reviewed. Detailed descriptions of language impairments in SLI and in ASD-LI will be presented followed by a discussion of the cognitive markers that may underlie these impairments.

Clinical Presentations of Language Impairment in SLI and ASD-LI

Impairment in the acquisition of *spoken* language is predominant in SLI; that is, deficits are seen in aspects of language that depend on the input-output systems of hearing and speech[1]. Spontaneous use of compensatory gestures and signs by children with SLI who have expressive language impairment is sometimes reported (Evans, Alibali & McNeil, 2001) and children with the rare condition known as verbal auditory agnosia, for whom spoken language acquisition is virtually impossible, can acquire language in other modalities, including written or signed language (Rapin, 1996b). Although there is now evidence suggesting that school-aged (i.e., 6;0

[1] SLI and developmental dyslexia are commonly associated, but through a common dependence on phonology (e.g., Bishop & Snowling, 2004).

years and above) children with SLI may have wider difficulties with, for example, non-verbal memory (Bavin et al., 2005), it is both clinically and theoretically relevant that these children are identified as having spoken language as their primary difficulty. Also, although literacy ability is often affected in SLI, the difficulties appear to be qualitatively different to those in autism and a considerable minority of children with SLI have competent literacy skills (e.g., Botting, 2007).

As mentioned previously, various subtypes of spoken language impairment are found in SLI. These are defined in terms of profiles of ability across comprehension and expression and according to the degree to which phonology, grammar (morphology and syntax), semantics, and pragmatics are affected. The most common profile in SLI involves problems in language production and comprehension. Moreover, deficits in phonology and syntax are more severe than are deficits in higher-order, lexical or pragmatic language skills (Leonard, 2000). This generalisation, however, conceals the fact that several subtypes emerge from studies using cluster analyses, in addition to rare cases of verbal auditory agnosia (Conti-Ramsden, Crutchley & Botting, 1997; Rapin & Dunn, 2003)[2]: These include

- Expressive phonological (articulatory) impairment, with comprehension relatively unimpaired.
- Mixed receptive-expressive phonologic and syntactic impairment (as previously described).
- Lexical-syntactic problems with word finding difficulties and immature syntax.
- Semantic and pragmatic impairments with unimpaired phonology and syntax.

The most common classification for preschool (Rapin & Dunn, 2003) and school-aged (Conti-Ramsden & Botting, 1999) children with SLI is phonologic-syntactic disorder. Although changes in classification across development are not uncommon, deficits in phonology and syntax appear to be persistent in SLI. Conti-Ramsden and Botting found that 45.7% (n = 84) of 7-year-olds with a diagnosis of SLI, and who were attending a specialist language unit, could be classified in the phonologic-syntactic category. When reassessed at age 8 years, 53.5% (n = 77) were classified with phonologic-syntactic disorder, confirming the mixed receptive-expressive difficulties experienced by a significant proportion of individuals with SLI. Tables 1 and 2 show the percentage of children from Conti-Ramsden and Botting's sample falling into each subtype of SLI (again excluding groups with verbal dyspraxia and normal levels of language), according to Rapin and Allen's (1987) and Rapin's (1996b) classification system, respectively.

Tables 1 and 2 about here

Semantic-pragmatic disorder constitutes a form of higher-order processing impairment according to Rapin and Allen's (1987) and Rapin's (1996b) classification system. It is important to note, however, that pragmatic language impairment (impaired language use) can occur independently of semantic impairments and independently of the social and behavioural flexibility impairments associated with autism (Bishop, 1998; Bishop & Norbury, 2002; Botting & Conti-Ramsden, 1999). Thus, pragmatic language impairment appears to be, in part, dissociable from the structural language impairments of SLI (affecting phonology, grammar, and

[2] Following Rapin and Allen's (1987) classifications, this list excludes a cluster labelled 'verbal dyspraxia' which constitutes a speech output problem rather than an impairment of structural language acquisition. Children in this cluster were also excluded from the analyses conducted by Conti-Ramsden and Botting, (1999), as discussed below.

semantics) and also dissociable from full forms of autism, although sharing one diagnostic feature with autism. The status of pragmatic language impairment as either a subtype of SLI or a form of ASD has been discussed for many years (Bishop, 1989; Boucher, 1998; Brook & Bowler, 1992). A link between pragmatic language impairment and ASD, however, has not been extensively investigated in recent research. Rather, the theoretical zeitgeist has been to focus on links among prototypical forms of SLI and ASD-LI. This hypothesis and related evidence is, therefore, the central focus of the current paper.

As mentioned above, the structural language profiles in individuals with ASD-LI vary considerably as they do in SLI, partly because co-morbid conditions such as hearing impairment, Down's syndrome, and Fragile X syndrome are sometimes present and have their own distinctive effects on language acquisition. Moreover, a small minority of individuals with ASD-LI have normal intellectual abilities. Thus, language impairment in these individuals may differ qualitatively from language impairment in less able individuals (Boucher, Mayes & Bigham, 2008).

The most striking characteristic of the structural language impairment in autism as it occurs in lower functioning individuals is that it is rarely specific to the speech modality. Acquisition of language through writing or signing is generally as affected as the acquisition of spoken language. There are exceptions to this, generally having to do with co-morbid problems affecting specific input-output systems. For example, signed language may be more easily acquired than spoken language in hearing impaired individuals. However, signing or gesturing will not be spontaneously used or as easily acquired as it is by hearing impaired children who are not autistic (Wing, 1996). In one study of preschool children with ASD-LI reviewed by Rapin and Dunn (2003), a small number of children with autism were identified as having verbal auditory agnosia (as described in the section on language in SLI). For these children, as for children with ASD plus hearing impairment, signed language may be easier to acquire than spoken language, but will not be acquired spontaneously or easily.

Descriptions of spoken language acquisition in ASD-LI also vary depending on the age and ability of the groups. Tager-Flusberg, Paul & Lord's (2005) review of language impairments associated with autism presents what may be described as the classical picture based on older individuals with some language. Tager-Flusberg et al. stressed the marked problems of comprehension in these individuals, especially comprehension of speech in everyday situations as opposed to single word comprehension on clinical assessment tests. Regarding expressive language, Tager-Flusberg et al. concluded the following: (1) articulation (expressive use of phonological knowledge in speech) and syntax are generally unimpaired, or else delayed, but mental-age appropriate; (2) morphological errors occur including omission of articles and some tense markers; (3) word use can be idiosyncratic, pedantic, or over-concrete, although categorical knowledge appears relatively normal; and (4) pronoun reversal is common in young or less able individuals.

The first and most comprehensive assessment of language profiles in older children with ASD-LI, which included a comparison group of children with SLI, was conducted by Bartak et al., (1975, 1977). Forty-seven boys with severe developmental language disorder were initially considered for inclusion. Out of the original 47, 19 individuals with ASD-LI and 23 individuals with SLI were selected for detailed assessment. The remaining 5 boys were described as having mixed SLI and autism and were excluded from the main investigation. The mean age of the selected autism group was 7;0 years compared with 8;2 years in the SLI group and the groups were matched for nonverbal intelligence (which was in the low normal range), for mean length

of utterance, and for expressive language on a formal test. Formal assessments of numerous linguistic and other behaviours were conducted, alongside a structured parental interview (Bartak et al., 1975). A follow-up study was conducted two years after the original. Corpora of spontaneous speech were collected from 12 boys in each group, matched for age and nonverbal IQ, and the speech samples were subjected to syntactic and functional analyses (Cantwell et al., 1978).

One potential concern about these studies is the degree to which children in the SLI group, who attended specialist schools and units attached to hospitals, are comparable to children with SLI, who attend language schools and clinics. Despite this concern, the study is interesting because the findings on language anticipate many of the findings from more recent studies with respect to both similarities and differences amongst the language abilities of individuals with ASD-LI and SLI. Bartak et al. (1975) found that more than 50% of the children in both groups had syntax that was 'primitive or lacking'. Detailed linguistic analyses in the follow-up study showed similar patterns of spared and impaired morphological and syntactic abilities. One difference in grammatical abilities between the two groups, however, was that children with ASD-LI were less likely to make errors of 3rd person singular tense marking than children with SLI (Cantwell et al., 1978). Tense marking errors are a reliable clinical index of SLI. Thus, this dissimilarity is notable (see below for a full discussion). In addition to mainly shared grammatical problems, both groups had similar histories of delayed developmental milestones including deviant or absent language babble and delayed language onset. Both groups also had a family history of speech or language disorder according to self-report (Bartak et al., 1975).

There were also clear linguistic differences between the two groups, however. Children with ASD-LI were less able than the children with SLI to understand or use gestural communication, either spontaneously or in a test situation (Bartak et al., 1975). Their spoken language comprehension was impaired relative to the SLI group, as was their reading comprehension (despite having better mechanical reading ability than the SLI group). They were less likely than children with SLI to have, or to have had, defective articulation and they produced significantly more utterances that were echolalic, bizarre, or inappropriate than the SLI group. Discriminant function analyses showed that the ASD-LI and SLI groups could be clearly differentiated in terms of linguistic differences, as well as in terms of cognitive and behavioural differences (Bartak et al., 1977).

Howlin, Mawhood, and Rutter (2000) and Mawhood, Howlin, and Rutter (2000) followed 38 (19 with ASD-LI, 19 with SLI) of the original participants from Bartak et al.'s (1975) study to adulthood, administering detailed language, cognitive and behavioural assessments when the participants were in their mid-20s. Discriminant function analyses continued to clearly differentiate the two groups both in terms of structural language skills and communicative language usage. However, Howlin et al. and Mawhood et al. found greater overlap between the two groups than did Bartak et al. in the original study. For example, the structural language abilities of the ASD-LI group were no longer significantly worse than those of the SLI participants. Also, whilst the participants with ASD-LI continued to display significantly lower levels of social and communicative functioning than the participants with SLI, several individuals in the latter group displayed some impairment in these areas, including difficulties in establishing/maintaining peer relationships and sustaining conversation. By adulthood, the participants with SLI also displayed a somewhat narrow range of interests and some showed a tendency toward stereotyped behaviours, including engaging in rituals and

displaying motor mannerisms. Again, however, the participants with ASD-LI continued to be more widely and severely affected than the SLI group, in this regard.

Mawhood et al. (2000, p.556) argued that their findings suggest a close connection between ASD-LI and SLI, although they also suggested that "in autism the focus needs to be on abnormalities of language *usage*, not simply *delay* as measured by formal language tests". Some overlap in symptomatology between the two disorders at the behavioural level leaves open the question of whether or not the two disorders are related at neurobiological and/or etiological levels. In fact, etiological overlap is contraindicated by different developmental trajectories in each disorder (see below for further discussion).

In a recent study of language profiles in school-aged children with ASD, Kjelgaard and Tager-Flusberg (2001) explored the performance of 89 children using the Clinical Evaluation of Language Fundamentals (Wiig, Secord & Semel, 1992), plus additional tests of articulation, single word vocabulary comprehension and expression. The group as a whole showed no significant difference between comprehension and expression and articulation was unimpaired. However, there was wide variability in scores and considerable heterogeneity among individual profiles. For this reason, Kjelgaard and Tager-Flusberg selected a sample of 44 children with ASD who were able to complete the preschool version of the Clinical Evaluation of Language Fundamentals and divided this sample into three subgroups according to scores on the test. Children whose standard scores were 85 or higher were designated as having normal language (n = 10); those with standard scores of 70 to 84 (or 1 *SD* below the mean) and below 70 (more than 2 *SD* below the mean) were designated as borderline language impaired (n = 13) and language impaired (n = 21) respectively. The subgroup with normal language on the Clinical Evaluation of Language Fundamentals had no impairment on any of the other tests and a flat language profile overall. The borderline group had normal articulation with vocabulary comprehension and expression scores 1 *SD* below the mean. The language impaired group had articulation within the normal range and vocabulary comprehension and expression 2 *SD* below the mean. Differences in vocabulary scores between the borderline and language impaired groups were highly significant.

Kjelgaard and Tager-Flusberg (2001) concluded that the language profiles in language impaired individuals with autism resemble the language profile 'that defines' SLI. This interpretation is only partly justified, however, because phonological (articulatory) impairments are a relatively common feature of language profiles in SLI (even when cases of 'verbal apraxia' are excluded), but articulatory impairments were not found in the children with ASD-LI, consistent with findings from numerous other studies (as reviewed by Tager-Flusberg et al., 2005).

Another study exploring performance on the Clinical Evaluation of Language Fundamentals by school-aged children with ASD-LI was conducted by Lloyd, Paintin, and Botting (2006). Although smaller in scale than Kjelgaard and Tager-Flusberg's (2001) study, it had the advantage of including a group of (n = 18) children with SLI, enabling direct comparisons between language profiles in each disorder. Participants with ASD-LI were similar to those with SLI in finding the Recalling Sentences subtest of the Clinical Evaluation of Language Fundamentals most difficult (cf. Botting & Conti-Ramsden, 2003) and the Listening to Paragraphs subtest the least difficult. However, the overall patterns of difficulty were different in the two groups, albeit not significantly (possibly due to a lack of power): The SLI group showed poorer expressive than receptive skills, whereas the ASD-LI group showed the opposite

pattern, with higher scores observed on expressive language subtests than on receptive language subtests.

All of the studies reviewed so far show that the characteristic language profiles of school-aged (i.e., 6;0 years of age, upwards) children with ASD-LI share some features with the typical profiles of school-aged children with SLI. Nonetheless, several significant differences are evident, one notable example being widespread impairments in expressive language (particularly expressive phonology) in SLI, but not in ASD-LI.

Rapin and Dunn (2003) reviewed the combined results of two large-scale studies of language impaired preschool children with ASD (excluding the most severely learning disabled) and a follow-up study of a subgroup of children. They reported that approximately 61% of *pre*schoolers with ASD-LI could be clinically described as having mixed receptive-expressive spoken language disorder, a recognised subtype of SLI. The remaining 39% of the children had higher-order processing disorders, characterised by difficulties in comprehension and the acquisition of word meaning (semantics), with secondary delay in the acquisition of phonology, morphology, and syntax. None of the preschool children with ASD-LI (N = 496), however, had a purely expressive language disorder.

Findings from the follow-up study of 92 7;0 – 9;0 year old children suggested that the proportion of ASD-LI children with mixed receptive-expressive problems declined with age, leaving the majority of children with no clinically significant phonological impairments, but with higher-order processing difficulties and residual grammatical impairments. According to Rapin and Dunn (2003), this description resembles the classic pattern of language impairments in older children and adolescents with ASD-LI (as summarised above). Thus, Rapin and Dunn's observations are similar to those of most other studies. Rapin & Dunn stress the importance of taking a developmental perspective when considering the nature and causes of language impairment – whether in children with ASD or those with SLI.

The studies reported by Rapin and Dunn (2003) are also important because they included large numbers of language impaired preschool children without autism, enabling comparisons between the clinical descriptions of language impairments in the two broadly defined groups. Approximately 52% of the preschoolers with SLI could be classified as mixed receptive-expressive language disorder, with 14% classified as higher-order processing disorder, and 34% classified as expressive disorder only. The comparable percentages in the children with ASD-LI, as highlighted above, were 61%, 39% and 0%, respectively. The lower rate of higher-order processing problems and the higher rate of purely expressive disorder in preschoolers with SLI relative to those with ASD-LI are notable and represent a significant difference between language profiles in the two disorders. However, the fact that early language profiles in ASD-LI overlap with language profiles commonly seen in SLI to a greater extent than later language profiles may be important for understanding relations between the two disorders. One possibility is that, over time, the structural language impairments in ASD-LI are modulated by the effects of autistic symptomatology, masking their similarity to the impairments in SLI. Whilst it is almost certain, however, that ASD-specific impairments in social interaction and imagination influence the language profiles in ASD-LI (e.g., Baron-Cohen, Baldwin & Crowson, 1997; Preissler, 2008), it seems unlikely that these impairments would or could lead to the *resolution* of clinical impairments in phonology and grammar. Nor does it seem likely that the additional symptomatology present in ASD-LI, but not SLI, would increase impairments in some domains of language while simultaneously replacing phonologic-syntactic problems with higher-order processing impairments.

Rapin and Dunn's clinical observations of preschool children with ASD-LI were confirmed in another study of structural language functioning in young children with ASD-LI, aged 3;0 to 6;0 years (Eigsti, Bennetto & Dadlani, 2007). Participants with ASD-LI were matched for receptive vocabulary with typically developing children and were matched for age, receptive vocabulary, and non-verbal intelligence with children who had a learning disability. Transcriptions of 100 utterances made by each child during a joint play session with an experimenter were rated for morpho-syntactic complexity according to the 'Index of Productive Syntax' (Scarborough, 1990). This index was developed to reflect the typical order of acquisition of grammatical items. The children with ASD-LI produced utterances that were less grammatically complex than those made by either learning disabled or typically developing participants, despite using a greater variety of words in their interactions. The developmental trajectory of grammar acquisition in each group was evaluated by calculating an overall score and then comparing this to the difficulty of the items in the participants' utterances. Eigsti et al. found that the fit between the overall index score and the complexity of items was poorer in children with ASD-LI than in the other groups, where learning disabled and typically developing participants produced grammatical items that were concordant with their overall level of grammar. Although this study was cross-sectional, and thus cannot speak directly to developmental issues, the latter result suggests that grammatical knowledge may be acquired in an atypical fashion, in addition to being developmentally delayed, in ASD-LI.

Whilst the morpho-syntactic deficits observed in young children with ASD-LI in Eigsti et al.'s (2007) study resemble those typically seen in SLI, one important difference is notable: children with ASD-LI, unlike those with learning disability, did not omit grammatical morphemes when marking tense. As discussed below, deficits in tense marking are a sensitive clinical marker of SLI. Thus, the finding that young language impaired children with ASD do not show these omission errors, despite other limitations in their grammatical knowledge, is theoretically as well as clinically informative.

Summary and Discussion

The extent to which the phenotypic language profiles in ASD-LI and SLI should be viewed as similar or different depends, to some degree, on the age at which affected individuals are assessed. In school-aged individuals with SLI, language difficulties are predominantly of a mixed receptive-expressive form, affecting all levels of comprehension and production (e.g., Conti-Ramsden & Botting, 1999). In contrast, the language difficulties experienced by school-aged individuals with ASD-LI are best characterised as higher-order processing deficits involving major impairments in comprehension and production of discourse, but relatively unimpaired phonology and mild to moderate impairments of grammar. Echolalic and idiosyncratic utterances as well as pronoun reversals are common in children with ASD-LI, but rare in individuals with SLI. At school-age, therefore, the typical profile in each disorder is predominantly different, despite some areas of overlap. In preschool children, however, the typical profiles of linguistic impairments substantially overlap in each disorder. The majority of pre-school children with ASD-LI, like preschool and school-aged children with SLI, show difficulties at all levels of structural language and are best classified as having mixed receptive-expressive difficulties.

The question then arises as to why the two disorders share different developmental trajectories. The phonological and, to some extent, the grammatical deficits seen in young children with ASD-LI resolve over time, whereas deficits in these areas continue to be

substantial in a large number of individuals with SLI. This is not to say that these areas of language remain statically impaired in all children with SLI children. Unpublished data from the Conti-Ramsden Manchester Language Study suggest that teachers and/or speech and language therapists report difficulties in expressive phonology (articulation) in approximately 48% of 7- and 8-year-olds with SLI, with a 72% stability rate. Although phonological problems do resolve to some extent in SLI, this improvement is nothing like that seen in ASD-LI in which normal, or mental-age appropriate, articulation is seen in virtually all children, even when other areas of language are severely affected (e.g., Kjelgaard & Tager-Flusberg, 2001).

The issue of differing developmental trajectories in ASD-LI and SLI also arose in follow-up studies of Bartack et al.'s (1975) sample, conducted by Howlin et al. (2000) and Mawhood et al. (2000). Individuals with ASD-LI were easily differentiated from those with SLI in terms of their linguistic, cognitive, and behavioural profiles during childhood, but were not as well differentiated in adulthood. This presents an intriguing antithesis, in that SLI can develop into something like autism in some individuals, whereas individuals with ASD-LI tend to grow out of their more SLI-like language impairments. It is, of course, unlikely that the causal chain from etiology, neurobiology, and cognition to behaviour will be a straightforward one, nor common to all cases in either ASD-LI or SLI. Thus, the finding that each disorder follows a behaviourally different developmental trajectory does not rule out the possibility that the two disorders may be related at one or more lower levels of description. One possibility, favoured by Mawhood et al., is that SLI represents a 'lesser variant' form of ASD such that social and pragmatic problems go undetected early on, but 'snowball' with time when cognitive and linguistic demands increase. This possibility leaves unexplained, however, the fact that the language skills of participants with ASD-LI in the Mawhood et al. study improved over time, whereas those of participants with SLI showed a relative decline. If linguistic deficits in each disorder have the same underlying basis then it is not obvious why the group with more severe and widespread initial deficits (i.e., the autism group in Bartak et al.'s study) should show greater compensatory flexibility than the SLI group, whose early social, communicative, and linguistic abilities were all superior to those of the ASD group. It may be that individuals in the SLI group had autistic features of behaviour from the outset, but that these went unnoticed during the selection process. This seems unlikely for at least two reasons, however. First, several potential participants were excluded during the selection stage precisely because they were perceived to have 'mixed autism and SLI'. Second, discriminant function analyses, utilising both language and behavioural measures, differentiated between the participant groups in both the childhood study and the adult follow-up study. It is more parsimonious to suggest that whilst social and pragmatic difficulties are primary impairments in ASD, such difficulties are secondary to primary language difficulties in individuals with SLI, affecting only a subset of those with an early diagnosis (Botting & Conti-Ramsden, 2008; Durkin & Conti-Ramsden, 2007).

Nevertheless, the observation of behavioural overlap with SLI in over half of all young children with ASD-LI has obvious implications for treatment and intervention efforts. Thus, it represents an important observation. Whether the observed similarities between the two disorders are surface manifestations of the same underlying neuropsychological dysfunction, or whether they result from distinct pathologies, is however an open question. 'Marker' behaviours and the study of their underlying causes, considered next, shed some light on this question.

Clinical Markers for Language Impairment in SLI and ASD-LI

A great deal of research has been carried out to investigate the characteristics and

psychological causes of SLI in its most common forms, i.e., those involving phonological and/or grammatical impairments. An informative approach, increasingly used in research on developmental disorders, is to search for clinical/cognitive markers that are reliably associated with aspects of behavioural impairment (e.g., Bishop & Snowling, 2004; Frith, 1997). Regarding SLI, specific errors of tense marking, namely omission of the past tense marker /-ed/ and the 3rd-person present tense marker /-s/ have been shown in several studies and are considered a reliable marker of SLI (e.g., Leonard et al., 1992; Oetting & Herchov, 1997; Rice, Wexler & Cleave, 1995). Sentence repetition, a task on which children with SLI perform poorly, also discriminates children with SLI from typically developing children (e.g., Conti-Ramsden et al., 2001). Much of the research investigating causes of SLI has focussed on auditory perception and phonological short-term memory (for reviews of early studies see Leonard, 2000; for later discussion see e.g., Gathercole, 2006; Tallal, 2004). The most robust finding in this literature is impaired non-word repetition. Impaired non-word repetition, like tense-errors and poor sentence repetition, is an excellent clinical marker for SLI (see Coady & Evans, 2008 for a recent review), distinguishing individuals with SLI from typically developing individuals. These tasks also distinguish individuals who have a history of SLI, but who now show normal levels of performance on standard language assessments, from individuals with no history of SLI (Bishop, North & Donlan, 1996). There is still some debate about the causal role of phonological processes in more severe language impairments and recent research has suggested other possible causes. These include more general memory impairments (e.g., Marton, 2008; Archibald & Gathercole, 2006a) and higher-order processing capacity (e.g., Montgomery, 2006). What is not disputed, however, is the sensitivity of these measures as clinical markers of SLI. If ASD-LI and SLI represent (partially) overlapping disorders at a deeper level than surface presentation, similarities in performance on clinical markers should be evident.

Roberts, Rice, & Tager-Flusberg (2004) assessed tense marking in 62 children with ASD who were divided into normal, borderline, and language impaired subgroups using the same criteria as Kjelgaard and Tager-Flusberg (2001). Regular past tense marking (e.g., 'He talk/ed/') and 3rd-person singular present tense marking (e.g., 'She talk/s/') were assessed. The language impaired subgroup made fewer correct responses on the past tense and 3rd person singular tests than either the normal or borderline groups. Roberts et al. interpreted this finding as indicating a significant similarity between language impairment in ASD-LI and in SLI, consistent with the hypothesis that co-morbid SLI is the main cause of language impairment in autism. However, the types of error made by the children with ASD-LI differed from the errors made typically by children with SLI. This was masked by the error analysis provided by Roberts et al., as detailed below.

Regarding errors on the 3rd-person present task, Roberts et al. report that *across groups* the main errors were bare stem (i.e., unmarked/omitted) forms, typical of the errors seen in SLI. However, the prediction being tested was that children in the language impaired group (and to a lesser extent the borderline group) would show a preponderance of bare stem errors resembling those seen in SLI, whereas children in the normal language group would show few errors of any kind. The data on error types reproduced in Table 3 suggest that these predictions were not supported.

Table 3 about here

No significant differences in the proportion of bare stem errors between language sub-groups were found on the third-person singular task. In contrast, the impaired sub-group was

more likely than the normal-language sub-group not to respond at all to the probe or to produce an echolalic response (such errors were collectively classified as *'no response'*). In fact, as seen in Table 3, the majority of the incorrect responses made by children in the impaired subgroup were classified as *'other verb'* responses. As the authors note, 'other verb' responses, involving answers such as "he's a hero" when questioned about what a cowboy does, are a likely consequence of pragmatic difficulties, unique to autism, rather than structural language difficulties. The same is true of the echolalic responses produced by these participants.

In sum, the two main types of error (*'no response'* and *'other verb'*) made by language impaired children with autism in marking third person singular tense are unique patterns in autism and are not typically seen in SLI. Rather, errors in SLI are largely restricted to bare stem errors. Thus, it is difficult to conclude that similar patterns of marking verbs with third person singular tense are seen in SLI and ASD-LI. Although such marking was less frequent in the language impaired ASD group than the other subgroups, their errors were of a qualitatively different kind than those made by children with SLI.

Regarding performance on the past tense task, the same error patterns were predicted as for the 3rd person present task. The data on error types are reproduced in Table 4. The data suggest that these predictions were also not supported. The error patterns in the three groups resembled those that occurred in the 3rd-person present task.

Table 4 about here

As with third-person singular marking, *'other verb'* responses constituted the most prevalent error type in the language impaired sub-group. *'No response'* errors were also more prevalent in the impaired group than the other two sub-groups. However, bare stem errors were made significantly more often by the language impaired sub-group than the other sub-groups. Importantly, though, these errors were only made for irregular verbs. This pattern of past tense marking in language impaired children with autism is uncharacteristic of that in SLI in which bare stem errors are characteristically observed only with regular forms (Leonard et al., 1992; Leonard, Eyer, Bedore & Grela, 1997; Oetting & Horohov, 1997). In sum, the kinds of errors in tense marking of regular verbs made by language impaired children with ASD do not closely resemble the kinds of errors made by children with SLI (cf. Bartak et al, 1975; Eigsti et al., 2007). Rather, language impaired children with ASD are most likely to produce errors of the kind 'other verb' or 'no response'.

Botting and Conti-Ramsden (2003) also assessed tense marking, as well as non-word repetition and sentence repetition, in (n = 13) children with ASD-LI. Their study included a group of (n = 29) children with SLI, allowing direct comparisons to be made between the performance of participants with each disorder. Participants with ASD-LI performed poorly on the non-word repetition task (the group achieved median scores between 1 and 2 *SD* below the normative mean for age). However, participants with SLI performed even worse (achieving median scores more than 2 *SD* below the mean for age). In contrast, no significant group differences in absolute performance were seen on either sentence recall or tense marking.

Botting and Conti-Ramsden's (2003) finding that individuals with ASD-LI performed as poorly on sentence repetition as individuals with SLI is important and suggestive of a common cognitive substrate to language impairment. Similarly, poor non-word repetition performance by individuals with ASD-LI has been observed in a number of independent studies (Bishop et al., 2004; Kjelgaard & Tager-Flusberg, 2001; Whitehouse, Barry & Bishop, 2008). Kjelgaard and

Tager-Flusberg (p. 304) interpreted these findings as indicating a 'theoretically significant' overlap between language impairments in autism and in SLI. Coincidence of poor scores on such tests in-and-of-itself, however, amounts to a quite limited descriptive similarity between the two disorders. Kjelgaard and Tager-Flusberg's conclusion may not be justified since tasks such as non-word repetition tap a number of skills including phonological memory, speech perception, the construction of phonological representations, and articulation (Snowling, Chiat & Hulme, 1991). Deficits in any of these areas could cause impaired non-word repetition. Indeed, non-word repetition is also impaired in quite different populations such as in individuals with Down syndrome (Laws & Gunn, 2004). A similar point can be made concerning Botting and Conti-Ramsden's observation of impaired sentence recall in groups with ASD-LI and classic SLI. Our analysis of Roberts' et al.'s findings on tense marking suggests that the overlap is superficial, here also, and may involve different underlying causes.

Only one study, by Whitehouse Barry and Bishop (2008), has directly examined whether poor non-word repetition in individuals with ASD-LI and SLI results from the same underlying neuropsychological dysfunction. Participants with ASD were subdivided into those with structural language impairment (n = 18), who scored below the 10th percentile on two or more tests from a comprehensive language assessment battery, and those with normal language (n = 16), who scored in the normal range. In line with previous findings, participants with ASD-LI, as well as participants with SLI, performed significantly less well on the non-word repetition task than participants with ASD who were not language impaired. However, unlike previous studies in which only overall non-word repetition performance has been examined, Whitehouse et al. explored *patterns* of errors across stimuli with different syllable-lengths. In line with previous research (e.g., Coady & Evans, 2008), they found an effect of syllable-length in the SLI group, with errors increasing as syllable-length increased from two to five syllables. In contrast, syllable-length had little affect on performance in the ASD-LI group. Thus, participants with SLI and ASD-LI performed similarly when stimuli were two and three syllables in length, but the SLI group made significantly more errors than the ASD-LI group when stimuli were five syllables in length. The SLI group also made more errors than the ASD-LI group when stimuli were four syllables, although this difference failed to reach significance. The different patterns of error in the two groups provide some evidence, as Whitehouse et al. argue, that the underlying basis of the non-word repetition deficit is different in each disorder. This particular aspect of Whitehouse et al.'s results should be treated with some caution, however, because only a small number of individuals with ASD (n = 8) performed poorly enough on the non-word repetition measure to be included in the analyses.

Finally, in contrast to Botting and Conti-Ramsden's (2003) findings, Whitehouse et al. (2008) found that participants with SLI performed worse than both language impaired and language-normal participants with ASD on a sentence repetition task. The reliability of sentence repetition as a marker for language-impairment in ASD is therefore open to question, although future research involving larger samples of participants with ASD-LI may well establish its reliability.

Neurobiology Associated With Specific Language Impairment and With Language Impairment in Autism

Research on SLI has documented abnormalities in language-related brain centres including the planum temporale, a region in the superior temporal gyrus, and Broca's area, part of the inferior frontal gyrus (e.g., Clark & Plante, 1998; Gauger, Lombardino & Leonard, 1997;

Plante, Swisher, Vance & Rapcsak, 1991). These regions are larger in the left hemisphere than in the right hemisphere in approximately 70-75% of neurotypical individuals (see Steinmetz et al., 1990 for a review). The studies cited above, however, have found reduced left-right asymmetry or reversed (i.e., R > L) asymmetry of these regions in individuals with SLI. Plante et al. (1991), for instance, found six out of eight children with SLI to have atypical perisylvian (a structure containing the planum temporale) asymmetries, with the right hemisphere showing either larger or equal volume to the left hemisphere. In contrast, L > R asymmetry was seen in six out of eight typically developing participants. Proportional volume data also showed significant differences between groups; the right perisylvian areas were larger in the SLI group than in the comparison group. Abnormal asymmetries have also been found in functional imaging studies (Hugdahl et al., 2004; Shafer et al., 2000). Moreover, Plante (1991) found abnormal structural asymmetry in the parents of children with SLI, suggesting a familial basis to the disorder. These abnormalities were over-represented in parents who reported positive histories of language impairment.

Neurobiology in Autism as Compared to SLI

If ASD-LI and SLI are partially overlapping disorders then similarities in neuroanatomy might be predicted, including abnormalities of size and structure of the planum temporale and Broca's area, associated with reduced or reversed hemispheric asymmetry. The most specific test of these predictions is to compare ASD groups with and without language impairments to an SLI group. Similarities in neuroanatomy should only be seen between those with SLI and those with ASD who have additional language deficits.

One relatively early study of head circumference in ASD included a comparison group of individuals with language disorder, allowing similarities in overall brain size to be explored (Woodhouse et al., 1996). In keeping with other findings (e.g., Bailey et al., 1995), almost half (48.7%) of the children with ASD had a head circumference above the 90th percentile. This compared to only 13.6% of children with language disorder. One interesting result was that children in the SLI group who had been diagnosed with semantic-pragmatic disorder were similar to children with ASD in having unusually large heads, 63.6% having head circumferences above the 97th percentile. This provides some evidence of a neuroanatomical link between semantic-pragmatic disorder and ASD. It is unclear, however, whether the children with semantic-pragmatic disorder, in fact, had ASD given that they had not been assessed formally and had been given a diagnosis of semantic-pragmatic disorder only on the basis of 'unusual' language usage. It is also important to note that abnormal head/brain size is age-related in ASD, with the greatest atypicality apparent in preschool children, declining to a non-significant trend in late adolescence/early adulthood (Redcay & Courchesne, 2004). Woodhouse et al. do not provide details about the average age of participants in their study, stating merely that all were 'under 16 years of age' (p.666). Thus, it is difficult to evaluate the significance of their results.

Only one study directly comparing language-related brain areas in ASD groups with and without language impairment has been reported. De Fossé et al. (2004) included groups of language impaired and non-language impaired boys with autism, a group of boys with SLI, and a typically developing comparison group. The ASD-LI group resembled the SLI group in showing R > L inferior frontal gyrus asymmetry. The ASD-language-normal group, in contrast, showed the same L > R asymmetry as typically developing participants. De Fossé et al. (2004) concluded that R > L asymmetry in boys with ASD-LI relate specifically to language impairment

rather than to autism *per se*. Further, they argued that their findings support a common neurobiological basis of language impairment in autism and SLI.

One difficulty with De Fosse et al.'s (2004) conclusion, however, is that the planum temporale asymmetry in the two language impaired groups was not as predicted. Contrary to other findings on children with SLI, De Fosse et al. found *exaggerated* L > R asymmetry of the planum temporale rather than *reduced* or *reversed* asymmetry in this group. Moreover, the ASD-LI group had exaggerated L > R planum temporale asymmetry relative to typically developing and language-normal children with ASD. De Fossé et al. suggest that variations across different studies could result from differences in ages of the participants, bearing in mind that language lateralisation increases with age and minor differences in the ages of comparison groups can alter findings (Schultz et al., 1994). This argument cuts both ways, however. If the ages of the children in De Fossé et al.'s study were related to the unexpected findings with respect to the planum temporale, then they might also have contributed to the predicted findings relating to the inferior frontal gyrus.

The inconsistency of findings with respect to language-related neuroanatomy in ASD-LI, and of their interpretation as indicating a shared neurobiological basis for language impairments in ASD-LI and SLI, is underscored by findings from other studies focussing on ASD. None of these additional studies included an exclusively ASD-LI group, but all found abnormalities in language-related brain regions. For example, Rojas et al. (2002) found reduced left hemisphere planum temporale volume in a mixed ability group of adults with autism compared with a neurotypical group matched for age and handedness. Herbert et al. (2002) found that the inferior lateral frontal language cortices (which included the inferior frontal gyrus /Broca's area) showed significantly reversed asymmetry in relatively high functioning children with autism (minimum nonverbal IQ 80) compared to an age- and handedness-matched comparison group. This region was 27% larger on the right side in the ASD group compared to 17% larger on the left side in the comparison group. Posterior regions (including the planum temporale) also differed significantly between the groups; the left planum temporale was 25% larger than the right in the ASD group but only 5% larger in controls, consistent with the unexpected finding from De Fossé et al.'s (2004) study. In an extensive study of brain asymmetries in children with autism, children with SLI, and a normal comparison group, all of whom had normal nonverbal intelligence, Herbert et al. (2005) found both similarities and differences in asymmetry in high-functioning children with autism and children with SLI. Both groups showed more asymmetry than typically developing children, but the abnormalities were not confined to language areas.

Researchers have also examined the neurophysiological underpinnings of language impairment in ASD-LI and SLI using event-related potentials. Much of this work, however, has focused on assessments of temporal auditory perception, a topic that is beyond the scope of this paper. Other studies have produced somewhat inconsistent results, varying widely with respect to type of task and measurement used. Furthermore, to the authors' knowledge, no studies have directly compared ASD-LI and SLI using electrophysiological techniques. Thus, we do not consider this evidence further.

Finally, studies using Positon Emission Tomography or fMRI to assess brain activity during sentence processing have shown reduced activation in Broca's area in high functioning individuals with ASD, accompanied by increased activation in Wernicke's area, which includes the planum temporale (Harris et al., 2006; Just et al., 2004; Muller et al., 1998). This finding makes it somewhat difficult to argue that overlap in neurobiology between SLI and ASD-LI (even were it to be robustly demonstrated) is causally related to the behavioural overlap

described in the previous section. It is true that language comprehension and semantic processing are not entirely normal in high-functioning individuals with autism or Asperger's syndrome (Kamio et al., 2007). However, clinically significant language delay or impairment is, by definition, absent in Asperger's syndrome and it is rare for individuals with high-functioning autism to have a clinically significant language disorder. Functional abnormalities in language-related brain regions in high-functioning autism groups or in Asperger's syndrome must, therefore, relate either to sub-clinical anomalies of comprehension and semantic processing or to some other aspect of autism.

There is also a more general difficulty with drawing firm conclusions about links among disorders from these studies. On the one hand, similar neuroanatomical anomalies in ASD-LI and SLI do not provide clear evidence of a common neurological basis to the language impairments associated with each disorder because it is never clear whether structural abnormalities are the basis of behavioural difficulties or a consequence of them. On the other hand, the failure to find similarities between disorders does not necessarily mean that they are not related. As Karmiloff and Karmiloff-Smith (2001) caution, "language processing can be very different in atypical brains… there are numerous alternative pathways to the ultimate goal of achieving adult-like language" (p.209). These difficulties of interpretation are further compounded by the fact that brain abnormalities do not always result in atypical or impaired behaviour. Hence, not all individuals with right hemisphere language lateralisation manifest language impairments: approximately one quarter of children with early onset left hemisphere temporal lobe epilepsy show subsequent right hemisphere language dominance with no noticeable language difficulties (Brázdil et al., 2003). Some typically developing individuals also show atypical cortical asymmetry with no obvious effects on their language development (e.g., Jernigan et al., 1991).

Genetic Factors in the Etiology of Specific Language Impairment and Autism

Family studies exploring potential patterns of familial transmission of language impairments in ASD and SLI and genetic linkage studies searching for underlying susceptibility to such impairments at the molecular genetic level are the main sources of evidence regarding the possibility of etiological overlap. Evidence from family studies will be considered first.

Family Studies

If the language impairments that sometimes co-occur with autism share etiological origins with SLI, family studies should produce a number of specific results. First, they should confirm that language impairments are heritable in each disorder because it is theoretically possible that language impairment in one or the other condition is not heritable. Having established this, family studies should also find (a) increased prevalence of SLI in families of autism probands and (b) increased prevalence of ASD in families of SLI probands. These findings are required because it is possible for language impairments in each disorder to be heritable but genetically unrelated. Evidence relating to each of these issues is considered below.

Heritability of Language Impairment in SLI

Several studies have demonstrated high rates of language problems in the relatives of SLI probands according to self-report (e.g., Neils & Aram, 1986; Tallal, Ross & Curtiss, 1989; Tomblin, 1989). Tomblin (1989) confirmed elevated rates of language difficulties in relatives of SLI probands in a clinical assessment. More importantly, the relatives of SLI probands also

show measurable impairments on direct language assessments. Plante, Shenkman and Clark (1996), for example, found 63% of SLI-proband parents, compared to only 17% of typically developing children's parents, performed at levels comparable to adults with known histories of SLI on a battery of language tests, including measures of expressive and receptive vocabulary and sentence comprehension. Tomblin and Buckwalter (1994) found that 21% of relatives (siblings and parents) of an SLI proband displayed language levels consistent with a formal diagnosis of SLI. Conti-Ramsden, Simkin, and Pickles (2006) found that 35% of relatives of children with SLI had language or literacy difficulties. This figure was similar regardless of whether self-report or objective measurement was used.

Specific aspects of the SLI phenotype also appear to be highly heritable. Bishop, North, and Donlan (1996), for example, studied phonological processing skills as measured by non-word repetition in a sample of twin pairs, some of whom were monozygotic and others dizygotic. Probands in these twin-pairs were considered language impaired if they showed poor performance (scaled scores of 80 or below) on at least one language assessment in a battery including measures of receptive and expressive vocabulary and grammar. Importantly, probands who had primary difficulties with expressive phonology were not included in the sample. A second group of probands consisted of children with a resolved language impairment who now scored within the normal range on all language measures. Finally, a group of control probands was included, who had no history of impairment and no measurable difficulty in any area of language. The control probands were matched closely with the SLI probands in terms of age and nonverbal IQ.

Bishop et al.'s (1996) first finding of note was that the resolved SLI group had normal lexical and grammatical knowledge, but their non-word repetition scores were in the language impaired range and as poor as the scores of probands with concurrent SLI. This highlights the sensitivity of phonological processing tasks in identifying resolved cases of language impairment. To examine the heritability of non-word repetition impairments, Bishop et al. subjected their data to a procedure developed by DeFries and Fulker (1988). This involved entering the non-word repetition scores of the co-twins of probands as the dependent variable in a regression analysis, with proband non-word repetition scores and the degree of their relationship to the co-twin (i.e., 50% for dizygotic twins or 100% for monozygotic twins) as predictor variables. If genetic influences underlie similarities in the non-word repetition performance of probands and their co-twins, then the scores of dizygotic co-twins should be significantly closer to the mean score of the control group than the scores of the monozygotic co-twins. This was exactly what Bishop et al. found. The non-word repetition scores of dizygotic co-twins regressed significantly more toward the mean than those of monozygotic co-twins, confirming the heritable basis of non-word repetition performance in SLI. Further confirming this conclusion was their finding that the monozygotic co-twins of language impaired probands had significantly lower non-word repetition scores than the dizygotic co-twins of language impaired probands.

Bishop et al.'s (1996) findings have since been replicated by Bishop et al. (1999) and extended in a study by Barry, Yasin and Bishop (2006). Barry et al. explored non-word repetition ability, among other language skills, in the parents of individuals with SLI. These parents were significantly impaired relative to parents of typically developing children on a measure of non-word repetition, tests of digit span (also tapping verbal short term memory), and oro-motor coordination ability (e.g., repeating tongue-twisters). When parents who met the criteria for language impairment (either by self-report or by direct testing) were excluded from

analyses, group differences on these measures were still highly significant. In other words, parents of SLI probands were still impaired on these measures even when they did not have any manifest language difficulties. In each analysis, though, non-word repetition performance was the only factor that was needed to discriminate one group of parents from the other, yielding a specificity of approximately 78% and a sensitivity of approximately 70%. These findings, along with those of Bishop et al. (1996, 1999), provide strong evidence for the familial aggregation of phonological processing difficulties in language impairment of kinds generally recognised as characteristic of SLI.

Heritability of Language Impairment in ASD

Self-reported problems in early language development by relatives of ASD probands have been noted in several studies (Bolton et al., 1994; Folstein et al., 1999; Fombonne et al., 1997; Piven et al., 1997; Szatmari et al., 2000). Evidence of language deficits with direct testing, however, is less reliable.

Compared to relatives of probands without autism, relatives of ASD probands show impairment in some studies using verbal intelligence quotients (VIQs) as a measure of higher-order, lexical language skills (Folstein et al., 1999; Fombonne, et al., 1997; Lindgren et al., 2006). The statistical differences that have been reported, however, appear to be due to unusually high scores in comparison groups, rather than by unusually low scores of autism relatives. The mean VIQ scores of autism relatives, although significantly lower than those of comparison groups, were above 100 in each study and do not reflect absolute impairment. VIQ, however, may not be sufficiently sensitive to subtle language difficulties in the families of ASD probands. VIQ (as measured by the commonly used Wechsler Scales; Wechsler, 1992, 1997) assesses higher-order lexical knowledge directly only on the Similarities and Vocabulary subtests. The Arithmetic, Digit Span, and Information subtests do not assess higher-order language processing directly, although they have a verbal loading. VIQ also appears insensitive to phonological and grammatical impairments as evident from the fact that individuals with a history of SLI show deficits in these aspects of language, particularly phonology, even when VIQ is relatively unimpaired (Bishop et al., 1996; Bishop et al., 1999; Lewis & Freebairn, 1992; Tomblin, Freese & Records, 1992).

Studies using specific linguistic measures, like similar studies using VIQ, have failed to find clear evidence for language deficits in the relatives of ASD probands. For example, Pilowski et al. (2003) found no impairments in siblings of autism probands on tests that are used to diagnose SLI. Plumet, Goldblum and Leboyer (1995) found no differences between autism relatives and comparison relatives in word repetition, phonological skills, and digit span. Happé, Briskman, and Frith (2001) found no impairments in autism relatives on a spoonerism task (measuring phonological awareness) or on a digit span task. Piven and Palmer (1997) found no impairment on a test of nonsense word reading, another measure of phonological processing, although Folstein et al. (1999) did report impairment on this measure.

Adopting a different approach, Dworzynski et al. (2008) explored genetic links between early language difficulties and later autistic-like traits in a population-based twin sample. The language abilities of all twin-pairs in the Twins Early Development Study (Oliver & Plomin, 2007) were assessed by parent-completed questionnaires concerning expressive vocabulary and grammar, at ages 2, 3, and 4 years. Parents also completed an autism screening questionnaire – the Childhood Asperger Syndrome Test (Scott, Baron-Cohen, Bolton & Brayne, 2002) – detailing their children's social, communicative, and imaginative development at age 8 years.

Dworzynski et al. selected a sub-sample of probands whose scores on the Childhood Asperger Syndrome Test indicated that they were at risk of ASD and then explored the language abilities of their co-twins, using a composite language score derived from the measures taken in early childhood. Using a DeFries and Fulker (1988) extremes analysis (described above), Dworzynski et al. found that the language composite scores of dizygotic co-twins regressed more toward the population mean than did the composite score of monozygotic co-twins, indicating that extreme autistic-like traits are genetically linked to language abilities. This finding confirms Dworzynski et al.'s (2007) results who, adopting a quantitative trait approach, explored the link between autistic-like traits and language abilities in the whole sample of the Twins Early Development Study.

In addition to assessing whether the phenotypic associations between two measures (or traits) are due to genetic factors, twin analysis techniques can also assess whether the genetic factors that influence performance on one measure are the same as those that affect performance on the other measure. Hence, 'genetic correlations' (derived from path tracing in structural equation modeling) quantify how *much* genetic factors influence trait overlap. In Dworzynski et al.'s (2008) study, the genetic correlation between the composite language score and the total score on the Childhood Asperger Syndrome Test was notably modest at 0.33, leading the authors to conclude that although genetic factors underlie most of the phenotypic association between early language skills and later autism-like traits, "the majority of genetic influences [between the two traits] are not shared, with the highest level of genetic overlap being only just over one-third". In other words, Dworzynski et al. observed relatively little overlap in genetic factors *common* to both language skills and autistic symptomatology.

Dworzynski et al.'s (2007, 2008) results should be interpreted with caution, however. The inevitable drawback to population-based studies such as Dworzynski et al's, that includes an impressively large sample of participants and implements sophisticated statistical techniques designed to assess genetic influences on behavioural traits, is that the results are based on parent-report measures. The Childhood Asperger Syndrome Test (e.g., Williams et al., 2005) and the early language measures (e.g., Fenson et al., 2000) are more or less valid measures of autistic symptomatology and language competence, respectively. However most researchers would agree that experimenter-/clinician-based administration of 'gold standard' measures, such as the Autism Diagnostic Observation Schedule-Generic (Lord et al., 2000), or standardised language tests, would be preferable. For example, the Childhood Asperger Screening Test, as Dworzynski et al. point out, is a measure designed to screen for possible autistic symptomatology in non-clinical samples: achieving a high score on this measure is not synonymous with a diagnosis of ASD, 36.4% of those scoring above the high-risk cut-off fail to meet criteria for ASD on direct assessment (Scott et al., 2002). Dworzynski et al.'s results indicate, therefore, that language competence is genetically related to autistic-*like* or autism-*related* traits, not to ASD per se. Likewise, the parental language indices used in this study were designed to document language development generally and to screen for marked difficulties. These measures may underestimate clinical language difficulties because parents may overestimate the size of their children's vocabulary.

Firm conclusions regarding the heritability of language impairment in ASD should not be drawn on the basis of the studies cited above, for two reasons. The first is that the studies may not have included the most appropriate participant group. The second reason is that language impairment occurs in only a proportion of individuals with ASD. Any study aiming to assess the heritability of such impairments should restrict their sample of families to those including a

proband with ASD-LI. We would not expect to see, on any obvious model of familiality, language impairments in the relatives of language-normal probands. None of the studies cited above distinguished between language impaired and language-normal probands. Thus, it may be that the generally negative findings result from an over-inclusive participant group.

Three family studies have focussed on more narrowly selected groups, with mixed results. Bishop et al. (2004) divided their ASD probands into 'language impaired' and 'language-normal' subgroups in two ways. The first method identified subgroups on the basis of VIQ; probands with VIQs of 77 or less on the Wechsler Intelligence Scales were classified as language impaired, whereas those with VIQs of 78 or higher were classified as language-normal. The second method was based on non-word repetition performance. Bishop et al. found that the relatives of low-VIQ probands had significantly lower VIQ scores than the relatives of normal-VIQ probands when mothers, fathers, and siblings were grouped together. Bishop et al. pointed out, however, that the verbal skills of the relatives of low-VIQ probands were not impaired by any absolute standard, their mean VIQs being close to 100, in each case. Moreover, Bishop et al. found no differences in non-word repetition ability between the relatives of probands who showed impairments in non-word repetition and the relatives of probands who showed normal non-word repetition skills.

Bishop et al.'s (2004) findings were generally confirmed by Lindgren et al. (2006). These authors divided the language status of ASD probands into impaired and normal according to performance on the Clinical Evaluation of Language Fundamentals–III (Semel, Wiig & Secord, 1995) and on a test of non-word repetition. Probands with standard scores of greater than 1 *SD* below the mean on either measure were classified as language impaired. Relatives of language impaired and language-normal probands were compared with respect to their language skills. VIQ was lower in relatives of language-impaired probands than relatives of language normal probands, although they were not impaired by any absolute standard. Scores were over 100 in all cases, replicating Bishop et al.'s (2004) findings. Lindgren et al. also found that the siblings of language impaired probands had significantly lower scores than the siblings of language-normal probands on a 'total language composite' derived from performance on the Clinical Evaluation of Language Fundamentals–III. Here again, however, the standard scores of these siblings were all over 100 and thus not even below average, let alone clinically impaired. A third finding from Lindgren et al.'s study replicated Bishop et al.'s (2004) finding that mean non-word repetition scores of relatives of language impaired probands were not significantly different from those of relatives of language-normal probands.

A fourth finding reported by Lindgren et al. (2006) was that the relatives of language impaired probands were more likely than the relatives of language normal probands to score in the language impaired range (defined as greater than 1 *SD* below the mean) on the Clinical Evaluation of Language Fundamentals–III or the non-word repetition test. These differences appeared to be due to the poor performance of mothers of language impaired probands. Neither the siblings nor the fathers of these probands
were significantly more likely to be classified as language impaired than were the siblings and fathers of language-normal probands. A difficulty with this final, potentially interesting, finding is that it is not possible to determine whether differences between the two sets of relatives are due to difficulties in general language ability (as measured by the Clinical Evaluation of Language Fundamentals–III) or in phonological processing (as measured by the non-word repetition task). Poor performance on either measure was considered sufficient to be classified

as language impaired. Lindgren et al.'s findings are not yet in the public domain. Thus, their results should be treated with some caution, despite their potential importance.

Finally, Whitehouse, Barry, and Bishop (2007) reported negative findings from a study assessing parents of ASD and ASD-LI probands using a battery of tests sensitive to language deficits in SLI. ASD probands were subdivided into language impaired and language-normal groups according to their performance on a test of non-word repetition. The parents of these two groups of ASD participants were then compared for language competency. No differences were seen between the two parent groups on any language measure. Overall, Whitehouse et al. classified 8 out of 30 parents of SLI probands as language impaired (defined as scoring greater than 1 *SD* below the mean on at least two language tests). This compared to only 2 out of 30 parents of ASD probands and 2 out of 30 parents of typically developing probands. This difference was statistically significant, reflecting the stronger heritability of structural language impairments in SLI than in ASD.

The second methodological problem with the studies cited earlier in this section concerns the measures used to assess language heritability. Measures have been derived from a hypothetical analogy with SLI and from the multifaceted VIQ measure. However, these may not be sensitive to the language-related impairments that parents report. Whitehouse et al. (2007), described above, found no phonological processing impairments in parents of ASD probands (or ASD-LI probands) compared to parents of children with SLI and parents of typically developing children using direct measures. This same study, however, found communication impairments according to parents' self reports. The authors suggest that their findings highlight a double dissociation between heritable structural language difficulties in SLI but not in ASD (or ASD-LI) and heritable pragmatic/communication difficulties in ASD (and ASD-LI) but not in SLI.

Several studies have directly assessed comparative difficulties with structural language versus communication experienced by relatives of individuals with ASD and SLI. Landa, Folstein, and Isaacs (1991), for example, explored the narrative-discourse ability of parents with children who have ASD by asking them to generate a short story after the presentation of a brief prompt designed to elicit an adventure-based narrative. The stories were rated for several features including length, completeness, overall quality and, importantly, the presence of semantic-syntactic errors, such as verb-tense errors and incorrect use of words. Landa et al. found the stories of all groups of parents similar in overall length. However, the stories that were generated by ASD parents contained significantly fewer complete episodes and were lower in quality, overall, than the stories generated by (comparison) parents of children with Down-syndrome or typical development. ASD parents frequently made asides (e.g., tangential statements about themselves) that departed from 'the text and rhetorical mode of story telling', suggesting an insensitivity to narrative context and listener expectations. Furthermore, 14 (34%) of the ASD parents, compared to only three (13%) of the comparison parents, produced stories that received an overall quality rating of zero (out of three). Indeed, two ASD parents, but no comparison parent, refused to produce spontaneous narratives at all, claiming that they could not 'tell stories'. In spite of these obvious difficulties in narrative production, ASD parents did not produce *structural*, semantic-syntactic errors. However, Landa et al. do not provide information about the language characteristics of the ASD probands. This latter result is therefore difficult to interpret because structural language anomalies should be expected only in the relatives of language impaired individuals. The suggestion, however, that communication impairments are heritable across the whole spectrum of autism-related conditions, not just in a subgroup with structural language impairment, is supported by these findings.

Bishop et al. (2006) explored language and communicative functioning in the siblings of children with ASD using the Children's Communication Checklist-2 (Bishop, 2003). This study included details of proband language abilities. Only one of 46 (2.2%) siblings of typically developing probands scored in the impaired range on the 'general communication composite' of the checklist (i.e., more than 2 *SD* below the mean), but almost one quarter (10/43) of the siblings of probands with ASD did so. This result was statistically significant even when three (of 43) ASD siblings who manifested autistic symptomatology consistent with a diagnosis of ASD were excluded from the analyses. Furthermore, eight of the 10 ASD siblings who were classified as impaired on the general communication composite had negative scores on the 'social interaction deviance composite' of the Checklist, indicating greater difficulties with social communication than with structural language. This adds weight to Whitehouse et al.'s (2007) suggestion that communicative difficulties rather than structural language impairments most clearly aggregate in the families of ASD probands. However, an analysis of sibling performance on the individual subscales of the checklist revealed a pattern of between-group differences not entirely consistent with this hypothesis. A number of ASD siblings scored poorly on a proportion of the pragmatic language subscales ('use of context', 'non-verbal communication') *and* on two of the subscales assessing structural language ability ('speech', 'coherence'). This suggests that certain aspects of structural language difficulty may form part of the heritable ASD phenotype. It should be remembered, however, that the Children's Communication Checklist-2 is a parent report measure. Therefore, this latter result might be expected on the basis of previous studies using report measures rather than direct testing.

Finally, a study by Ruser et al. (2007) explored language and communication abilities in the siblings of individuals with ASD, SLI, and Down-syndrome, using a modified version of the Pragmatic Ratings Scale. The Pragmatic Ratings Scale is an interview-based assessment in which a conversation between participant and experimenter is recorded and rated for aspects of social-pragmatic language use. Studies using the original Scale found marked communicative impairments in the parents of ASD probands (Landa et al., 1992; Piven et al., 1997). Ruser et al. modified the original Scale, which assessed few aspects of structural language and non-verbal communication, by adding items to assess grammatical errors and verbal-emotional expressions. A principle component analysis yielded four clusters (subscales) relating to 'emotional expressiveness and awareness of the other', 'communicative performance', 'over-talkativeness' and 'language'. Parents of both ASD and SLI probands performed significantly worse, overall, than parents of Down-syndrome probands. Indeed, Down-syndrome parents were superior to the other parents on each of the subscales apart from 'communicative performance'. No significant differences between ASD and SLI parents were found, leading Ruser et al. (p.1331) to argue that communicative impairments form part of the broader SLI phenotype and that these findings 'add to the evidence that autism and SLI share aspects of their etiology'.

Ruser et al.'s (2007) results support the notion that communicative difficulties in SLI have a familial basis, as they do in ASD. However, they do not necessarily show that the inherited communicative impairments are of the same form. Indeed, Ruser et al. found that different items predicted group membership for ASD and SLI parents, respectively. The items 'failure to reference' (part of the 'emotional expressiveness and awareness of other' subscale) and 'reformulations' (part of the 'language' subscale) best distinguished ASD parents from Down-syndrome parents. In contrast, 'grammatical errors' (part of the 'language' subscale) and 'dominating conversation' (part of the 'over-talkativeness' subscale) best distinguished SLI parents from Down-syndrome parents. It is notable that grammatical errors were made only by

SLI parents. This is consistent with the hypothesis that *structural* language deficits form part of the heritable phenotype in SLI but not ASD. Indeed, the fact that different characteristics predicted group membership for SLI and ASD parents led Ruser et al. (p.1331) to concede that there are 'qualitative differences between the hypothesised broader SLI phenotype and broader autism phenotype'.

In sum, relatives of ASD probands often report a history of language difficulties. However, there is negligible evidence from direct testing that structural language impairments aggregate in the families of individuals with ASD-LI or ASD, more generally. There is, therefore, no clear evidence of a raised prevalence of SLI in families of individuals with an ASD. Specifically, difficulties with phonological processing, characteristic of the most prevalent forms of SLI and clearly heritable, do not form part of the heritable phenotype in ASD. This does not necessarily mean, however, that no aspect of linguistic difficulty in ASD-LI has a heritable basis. There is some evidence for familial aggregation of general verbal-cognitive ability in ASD, as assessed by verbal intelligence tests. Verbal intelligence tests assess a variety of language-related cognitive skills (as discussed above), however, and only partially assess aspects of higher-order linguistic processing ability that, according to the evidence reviewed above, may have a heritable basis in ASD. No study, to date, has assessed the familiality of higher-order lexical knowledge in ASD using a sensitive measure. We predict that impairments should be seen in relatives of ASD-LI probands when using such a measure. In contrast, difficulties in language *use* probably characterise the relatives of all ASD probands, regardless of the proband's structural language ability. If this suggestion is confirmed by further studies employing direct measures of pragmatic language, it might explain the current discrepancy between findings of heritable structural language impairments from self-reports and the failure to find heritable impairments using direct testing with ASD relatives. Thus, the language-related difficulties often reported by ASD relatives may reflect communication or *pragmatic* language impairment rather than *structural* language difficulties.

Prevalence of SLI in Families of ASD Probands

Evidence to date suggests that the prevalence of SLI, or of subclinical features of SLI, in families of ASD probands is not significantly raised. Difficulties with phonological processing, characteristic of the most prevalent forms of SLI and which are clearly heritable in that disorder, do not form part of the heritable phenotype in ASD. Evidence regarding the heritability of other features of language impairment in ASD will become clearer as more research is conducted, assessing only the relatives of language impaired autism probands rather than unselected groups of relatives.

Prevalence of ASD in Families of SLI Probands

Rapin (1996a) examined the rate of autism diagnoses among family members of probands with SLI, high-functioning autism, low-functioning autism, or intellectual disability without autism. She found that 5% of the families with a low-functioning proband with autism reported at least one other family member with an autism diagnosis. This compared to 3.9% of families with a high-functioning proband, 2.1% of families with an SLI proband, and 0% of families with an intellectually disabled proband. Rapin concluded that autism and SLI should be viewed as etiologically distinct. Tomblin, Hafeman, and O'Brien (2003) pointed out, however, that Rapin's results may indicate rates of autism diagnoses in the families of SLI probands that

are intermediate between families of autism probands and families of intellectually disabled probands.

Tomblin et al. (2003) found that of 292 siblings of SLI probands, three (1%) met the diagnostic criteria for autism. They argued that this prevalence rate was significantly higher than the population estimate of autism diagnoses of approximately 0.1% and represents an association between autism and SLI. However, the reliability of Tomblin et al.'s findings can be disputed. Chance findings seem likely in a prevalence study that discovers only a small number of 'affected' cases. Moreover, a comparison group consisting of 230 siblings of typically developing probands also contained one child who met the criteria for a diagnosis of autism. This prevalence rate of 0.4% was not significantly different from the population estimate, suggesting no significantly increased risk in typically developing siblings. However, it was also not significantly different from the prevalence rate seen in SLI siblings (1%). Thus, the high prevalence of autism found in SLI siblings might be a sampling artefact given no significant differences in the rates of autism in SLI siblings and comparison siblings. The fact that (a) mean scores on the Autism Behaviour Checklist (a measure of autistic features; Krug, Arick, & Almond, 1980) did not differ between SLI siblings and typically developing siblings and that (b) there was no difference in the number of high checklist scorers (which indicates an increased risk of autism) provides some support for this suggestion.

Summary of findings from family studies

In the introduction to this section, we suggested that if the language impairments that sometimes co-occur with autism share genetic origins with SLI, then family studies should confirm that language impairments are heritable in each disorder. Family studies should also find raised prevalence of ASD in families of SLI probands and raised prevalence of ASD in families of SLI probands if language impairments in the two disorders share genetic origins. We noted that language impairments in both SLI and ASD-LI might be heritable but unrelated, in which case the raised prevalence of SLI in autism families and of autism in SLI families would not occur.

The evidence that we reviewed seems most consistent with this latter conclusion. The heritability of phonological processing abilities in SLI, but not in ASD is strongly supported. There is some evidence, however, for the heritability of verbal-cognitive abilities and for pragmatic (use of language) impairment in relatives of ASD probands. These findings need to be confirmed and expanded to include tests of higher-order verbal processing and pragmatic abilities in families of SLI probands. Failure to find evidence for the heritability of pragmatic impairments in SLI would confirm that *communication* impairments in common forms of SLI and the broad spectrum of autism-related conditions are etiologically unrelated. The most critical tests of the genetic relation between *structural* language impairment in SLI and in ASD-LI, however, concern the heritability of higher-order lexical processing abilities, in which there is greater potential for overlap than in either phonological-grammatical impairments (most common in SLI) or pragmatic impairments (universal in of ASD).

Molecular Genetic Studies

Molecular genetic studies usually involve genome-wide scans in search of chromosomal regions that are shared more often by individuals affected by a particular disorder than would be expected by chance. The strength of any 'linkage' to a particular region is expressed as a logarithmic odds ratio, or 'LOD score'. This is the odds of the association arising because of a

reliable link to the disorder (or trait) in question compared to the odds of the association arising if the region is not linked to the disorder. Significant linkage to a chromosomal region does not mean that a gene (or set of genes) that is causally responsible for a trait has been isolated. Rather, it suggests that the responsible gene(s) will be uncovered at that locus, upon further sequencing.

If shared genetic factors underlie the language impairments associated with autism and those characteristic of SLI, then molecular genetic studies will show that language impairment in each case is associated with specific genetic variations, one or more of which is common to the two conditions. Evidence relating to the genetic bases of language impairment in SLI and in autism will be considered separately, followed by a section on evidence relating to possible overlapping factors.

Genetic Factors Linked to SLI

The most reliable linkage signals in SLI have come from sites on chromosomes 16q (SLI consortium, 2002, 2004) and 19q (Bartlett et al., 2003; SLI consortium, 2002, 2004). Neither of these has been reliably indicated in studies of autism. The most significant aspect of these studies is that performance on a test of non-word repetition accounted solely for the linkage to chromosome 16q. When data from the independent samples studied by the SLI consortium (2002, 2004) were combined, chromosome 16q yielded a multipoint mean LOD score of 7.46. This exceeds Lander and Kruglyak's (1995) threshold for highly significant linkage (defined as a LOD score of over 5.4) and is among the highest ever recorded for a behavioural trait in any molecular genetic study. It seems likely, therefore, that a gene or a small set of genes on chromosome 16q play a significant role in the non-word repetition deficits in SLI.

Genetic Factors Linked to Language Impairment in ASD

Molecular studies of autism have shown linkage to a large number of chromosomal sites (including 1p, 2q, 4p, 4q, 6q, 7q, 10p, 13q, 16p, 19q, 22q), although few results have been reliably replicated (Barnby & Monaco, 2003). The most consistent linkage signals in autism have come from sites on chromosomes 7q (Ashley-Koch et al., 1999; Bradford et al., 2001; International Molecular Genetic Study of Autism Consortium (IMGSAC), 1998, 2001, 2005; Shao et al., 2002; Trikalinos et al., 2006) and 2q (Buxbaum et al., 2001; IMGSAC, 2001; Shao et al., 2002). Importantly, the signals to these sites increased when only the relatives of language impaired probands with autism were included in the analyses (Buxbaum et al., 2001; Bradford et al., 2001; Shao et al., 2002). Linkage to site 13q21 was also strengthened under these conditions (Bradford et al., 2001). The findings suggest that these sites represent good candidates for susceptibility loci related to language development in autism. They also provide some evidence for the heritability of language impairment in autism, strengthening the point made above that heritability of language impairment in autism should not yet be rejected based on weak evidence from family studies.

Evidence Relating to Shared Genetic Risk Factors

One potential chromosomal overlap between autism and SLI is at 13q21. Bradford et al. (2001) found linkage to this site in autism, particularly when analyses were restricted to language impaired phenotypes. Bartlett et al. (2002, 2004) found evidence for the involvement of this same locus in language and literacy impairment. It should be noted, however, that linkage to 13q21 in Bartlett et al.'s studies was found only in individuals classified as having a reading

impairment (defined as single non-word reading scores more than 1 *SD* below performance IQ scores). Linkage to 13q21 was not found in individuals with a language impairment (defined as standard language quotients below 85). There are undoubtedly phenotypic similarities between SLI and reading disability. However, few researchers suggest a potential overlap between reading disability and the kinds of language impairment associated with autism. Bartlett et al. (2002) argue that the reading impairments shown by participants in their sample reflect underlying language impairments and, hence, that the chromosomal overlap with autism is valid. This argument leaves unexplained, however, why linkage to 13q21 was not found under any model of language impairment in their (2002, 2004) studies or why other studies, using populations of language impaired rather than reading impaired individuals, have not found linkage to this site.

Chromosome 7q has also been suggested as a possible site of overlapping risk loci in autism and SLI (e.g., Tager-Flusberg, 2003). Evidence for its role in SLI came from the study of a three-generation family ('KE') of which 50% of its members manifest language disorder with orofacial dyspraxia (Gopnik & Crago, 1991; Ullman & Gopnik, 1999). These language impairments resulted from a mutation of a single gene ('FOXP2') located on chromosome 7q31 (Lai et al., 2001), leading to optimism that a 'single gene' cause of SLI had been discovered. Given the strong linkage signals to 7q31 in autism (e.g., IMGSAC, 1998), Folstein and Mankoski (2000), followed by Alarcón et al. (2002), argued that language impairments in autism might also be explained by mutations to FOXP2. Subsequent studies by Wassink et al. (2002) and Newbury et al. (2002) have shown conclusively, however, that defects in FOXP2 are not related to autism. Further, the study by Newbury et al. also provided good evidence that it is unlikely that mutations to FOXP2 result in common forms of SLI, outside the quite idiosyncratic disorder exhibited by the KE family.

Summary of findings from molecular genetic studies

Molecular genetic studies confirm evidence from family studies, showing SLI to be a heritable disorder. They also provide evidence for the heritability of language impairments in ASD that family studies have not shown. Candidate genes for language impairment in autism have been identified on chromosomes 2q, 7q, and 13q21, and for SLI on various genes, most notably 16q which shows strong linkage to non-word repetition. Sites on chromosome 7q, and 13q have been hypothesised as shared genetic risk factors for language impairment in autism and in SLI. However, there is little evidence to support this contention. Chromosome 16q has not been implicated in ASD, despite showing strong linkage to non-word repetition in individuals with SLI. This suggests that the genetic bases for language impairments in SLI are at least partly separable from those responsible for language impairments in ASD. It is important to stress, however, that failure to find linkage to the same chromosomal regions in different disorders does not mean that the two disorders are etiologically unrelated. Molecular genetic studies sometimes fail to find linkage to the same regions within the same disorder. This prevents firm conclusions regarding cross-disorder patterns of linkage. It is nonetheless striking that, despite an intense research focus, studies have not found overlapping susceptibility loci for language impairments in ASD and SLI.

Overview

The aim of this review was to evaluate the extent to which structural language impairment in ASD-LI could, or should, be explained in terms of co-morbid structural language

impairment, commonly associated with SLI. At the behavioural level of description, we asked whether the two disorders share dimensions of structural language impairment, as assessed by performance on standardised language tests. It is clear from the evidence that some language difficulties in school-aged children with ASD-LI are similar to those experienced in 'classic' forms of SLI. However, the dimensions on which children with each disorder are most clearly impaired are not the same. In contrast, the linguistic deficits seen in ASD-LI during the *preschool* years share many characteristics of those seen in SLI, including marked difficulties with receptive and expressive phonology, grammar, and semantics.

The evidence suggests that ASD-LI and SLI share dimensions of behavioural impairment at a certain point in development. Similarities between two disorders, however, do not necessarily mean that the underlying causes are the same. At neurobiological and genetic levels, there appears to be little support for the suggestion that ASD-LI and SLI are overlapping disorders. The evidence suggests, rather, that abnormalities in cortical asymmetry characteristic of SLI are not clearly present in ASD-LI. Furthermore, the familial transmission of structural language impairments in SLI emerges clearly from the evidence, whereas the evidence for heritability in individuals with ASD-LI is much less robust. Similarly, molecular genetic research suggests that structural language impairments in each disorder have a genetic basis, but that these bases are different.

In sum, we suggest that there is remarkably little evidence that structural language impairments in ASD-LI can be explained in terms of co-morbid SLI despite a strong theoretical and empirical drive to explore this hypothesis at multiple levels and from multiple perspectives. However, drawing *absolute* conclusions from the available evidence is not currently possible given the difficulties identified with the research designs at each level of investigation. Notably, future research must employ and compare groups of participants with clearly defined dimensions of language impairment. Identifying subgroups through the successive clarification of the phenotype has already proven successful in elucidating the genetic basis of structural language impairment in ASD (Buxbaum et al., 2001; Shao et al., 2002). It seems likely that such a strategy could prove successful in revealing potential links between ASD-LI and SLI at the behavioural and neurobiological levels, should they exist. Thus, it may be that abnormal cortical asymmetries will occur only in those individuals with ASD-LI who have concurrent, or a history of, mixed receptive-expressive impairments, rather than higher-order processing difficulties. It may also be that semantic impairments that characterise the language profiles of individuals with higher-order processing deficits may be heritable in ASD-LI as well as in SLI, a fact masked by the use of over-inclusive participant groups in the studies cited above.

An alternative strategy to sub-grouping participants according to evermore narrowly defined behavioural categories is to focus on the endophenotypic processes underlying behavioural impairment. Poor non-word repetition, sentence repetition, and errors of grammatical tense marking are well established cognitive markers of SLI, even in resolved cases who show no clinically significant behavioural impairment. Thus, the implication for future research using these measures in studies of individuals with ASD-LI and their families is clear. Recent research investigating marker behaviours in groups of children with ASD-LI and SLI has produced mixed results. There is evidence that children with ASD-LI perform poorly overall on these tasks. However, there is no evidence that their difficulties have a heritable basis, as they do in SLI, nor that the pattern of difficulties shown by affected individuals is the same as the pattern shown by children with SLI. Only a few studies have explored performance on these measures in both individuals with ASD-LI and SLI. Thus, we suggest that the field would be advanced

significantly by conducting further experiments aimed at establishing specific forms of impairment with respect to each of these markers. For example, Whitehouse et al.'s (2008) finding that children with SLI, but not ASD-LI, are sensitive to the syllable length of stimuli in non-word repetition tasks should be replicated and extended to explore whether manipulations to other features of stimuli affect the groups similarly or dissimilarly. For instance, non-word stimuli with syllable-structures that are reminiscent of English may facilitate performance in children with SLI (Archibald & Gathercole, 2006b), but not in children with ASD-LI. This would be evidence for a more general impairment in the latter group. Different patterns of performance on these tasks would indicate differences in the neuropsychological dysfunction that underlies poor performance in the two groups.

The above strategies could help resolve the debate about whether structural language impairments in ASD-LI, or in some forms of ASD-LI, represent co-morbid SLI. In addition, we suggest that future research exploring possible links between ASD-LI and SLI focus on higher-order language processing impairments that may be common to ASD and some subtypes of SLI. Here again, the behavioural overlap may be superficial. This issue remains to be fully investigated with measures that are sensitive to higher-order impairment.

Although we have made several recommendations for resolving the debate about co-morbidity in ASD-LI and SLI, we suggest that the debate itself may be hindering progress in elucidating the basis and cause of impairments that predominate in each disorder. There is very little indication that structural language impairments characteristic of, and heritable in, the most prevalent forms of SLI aggregate in the families of individuals with ASD. However, we strongly suspect that pragmatic and, to a lesser extent, semantic difficulties are heritable in ASD (cf. Whitehouse et al., 2007). It is on this basis that conceptualising ASD-LI and SLI as (partially) overlapping disorders and, hence, concentrating efforts on finding similarities between the two, may result in us missing more about each disorder than we explain.

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Table 1

Percentage of participants from Conti-Ramsden and Botting's (1999) sample falling into each sub-type of SLI, according to Rapin and Allen's (1987) classification system

	SLI Subtype			
Age at Testing	Phonological Programming Disorder	Phonologic-Syntactic Disorder	Lexical-Syntactic Disorder	Semantic-Pragmatic Disorder
7 years (N = 184)	12.5	45.7	28.3	13.5
8 years (N = 184)	13.2	53.5	20.8	13.5

Note: The data in this table are from **"**Classification of children with specific language impairment: longitudinal considerations" by G. Conti-Ramsden & N. Botting, 1999, *Journal of Speech Language and Hearing Research, 42*(5), p.1199.

Table 2

Percentage of participants from Conti-Ramsden and Botting's (1999) sample falling into each sub-type of SLI, according to Rapin's (1996b) classification system

	SLI Subtype		
Age at Testing	Expressive Disorder	Mixed Receptive-Expressive Disorder	Higher-order Processing Disorder
7 years (N = 164)	24.4	45.7	29.9
8 years (N = 164)	28.7	43.9	27.4

Note: The data in this table are from **"**Classification of children with specific language impairment: longitudinal considerations" by G. Conti-Ramsden & N. Botting, 1999, *Journal of Speech Language and Hearing Research, 42*(5), p.1199.

Table 3

Mean (SD) percentage of responses on 3rd-person singular probes by children with ASD or ASD-LI in the study by Roberts, Rice and Tager-Flusberg, (2004)

Language Group	Response type					
	Correct	Bare Stem	Other Verb	No Response	No Verb	Incorrect Irregular
Normal (*n* = 27)	76.3 (28.8)	14.1 (22.4)	8.9 (16.9)	<1 (2.6)	1.1 (4.2)	<1 (1.9)
Borderline (*n* = 16)	61.3 (32.2)	21.9 (17.2)	14.4 (20.3)	2.5 (5.7)	4.4 (8.1)	<1 (2.5)
Impaired (*n* = 19)	36.8 (23.3)	21.9 (17.2)	23.2 (25.0)	11.6 (18.0)	6.3 (11.1)	4.7 (8.4)

Note: The table is adapted from **"**Tense marking in children with autism" by J. A. Roberts, M. L. Rice, & H. Tager-Flusberg, 2004, *Applied Psycholinguistics*, *25*, p.434. Copyright 2004 by Cambridge University Press. Adapted with permission.

Table 4

Mean (SD) percentage of responses on past tense probes by children with ASD or ASD-LI in the study by Roberts, Rice and Tager-Flusberg, (2004)

Language Group	Response type				
	Correct	Bare Stem	Other Verb	No Response	No Verb
Normal (*n* = 27)	63.8 (29.2)	12.3 (16.3)	10.0 (14.5)	4.7 (14.3)	<1 (3.0)
Borderline (*n* = 16)	58.2 (28.9)	12.2 (14.9)	12.5 (21.2)	1.6 (4.2)	7.2 (2.5)
Impaired (*n* = 19)	30.6 (26.5)	23.4 (16.6)	25.7 (32.6)	12.3 (18.3)	2.3 (5.1)

Note: The table is adapted from **"**Tense marking in children with autism" by J. A. Roberts, M. L. Rice, & H. Tager-Flusberg, 2004, *Applied Psycholinguistics, 25,* p.436.