Discussion

Co-morbidity of autism and SLI: kinds, kin and complexity

Bruce Tomblin
Communication Sciences and Disorders, University of Iowa, Iowa City, IA, USA

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Abstract

There has been a long-standing interest in the relationship between specific language impairment (SLI) and autism spectrum disorder (ASD). In the last decade Tager-Flusberg and colleagues have proposed that this relationship consists of a partial overlap between the two. Therefore, among children with ASD there exists a subgroup who have SLI and ASD which has been called 'ALI'. Tager-Flusberg’s laboratory has presented several papers showing similar language profiles and brain structure abnormalities in both SLI and ALI. Others (Bishop, Whitehouse, Botting, Williams) have been less convinced that these ALI children have both ASD and SLI. Although they generally agree that the two groups are grossly similar, careful inspection of the data shows that there are differences. I will argue that many of the problems in this debate stem from a view of SLI that represents a particular kind of language learner and therefore a particular and unique profile can be assumed. I argue for recognizing that SLI is not likely to be a unique kind of language learner. Many of the features reported to be characteristic of SLI are also found in other forms of neurodevelopmental disorders. Other features are the outgrowth of studying clinically identified children with SLI and thus the profile appears to reflect biases and practices in the clinical service system. As a result it may be more reasonable to conclude that there is a large group of children with ASD who have poor language skills. The question then remains why are there so many children with ASD who also have poor language? There are several factors that collectively are strong candidates for answers to this question.

Keywords: autism spectrum disorders (ASD), specific language impairment (SLI), co-morbidity.

Introduction

Recently, there have been several papers published regarding the relationship of the language and communication problems of children with autism and autism spectrum disorder (ASD) and children with specific language impairment (SLI). The basic question that has been raised concerns whether there is an excess of SLI within children with ASD? For the practicing clinician this question may appear to be a bit esoteric. Within the day-to-day practice of a speech-language clinician, each client is largely viewed as a unique entity. As such, clinical work is much more about the particular and the individual than it is about generalities and groups. The clinician is concerned most about the welfare of each person served and as such promoting effective communication in each client. Research is often quite the opposite. In this enterprise generalities concerning patterns and principles are the focus. Individuals are important insofar as they provide information about these patterns; however, the general-
mechanisms are, we should be able to develop better methods of intervention. This basic premise underlies nearly all fields of clinical endeavour and provides the basic scientific underpinnings of our discipline. In many respects, those of us who are engaged in applied or clinical research must admit that we are a long way from being able to deliver on this promise, but the fact that this model has been successful in other disciplines such as medicine and engineering suggests that the basic approach will yield benefits to our field and ultimately to each of our clients.

Those children who present with developmental language disorders come with a variety of language abilities and limitations. As noted above, we can assume that these variations are not just there by chance, but reflect systematic processes that shape the development of language and communication in these children. Thus, we can ask, how many different ways are there for children to have poor development of language and communication? In recent years a debate has surfaced around this question with respect to children with SLI and ASD. Children with SLI and ASD share the common feature of poor spoken communication skills. Thus, the question arises as to whether SLI and ASD are related in some fashion. At the extremes we could say that these are distinctly different forms of developmental communication disorder or we could say that these are closely related if not the same. As we will see, each of these positions has been posed as well as variations in between.

Defining and characterizing SLI and autism

SLI

Currently, most clinicians and researchers distinguish between SLI and ASD. SLI is a common neurodevelopmental disorder that involves persistent limitations in the acquisition and use of spoken language. The aetiology of SLI is unknown; however, in the past several years there has been a growing body of evidence supporting a genetic contribution (Tomblin 1996). The diagnostic standards for SLI involve language achievement levels below cut-off values of −1.0 to −1.5 standard deviations of age expectations. Additionally, minimum non-verbal IQ levels as high as 85 have often been employed, although this standard has been questioned by several researchers (Tager-Flusberg and Cooper 1999, Plante 1998). These criteria may be viewed as inclusionary criteria in that a child must meet these conditions for a diagnosis of SLI. Additionally, children with SLI are required to be free from developmental or sensory impairments. This last criterion has been referred to as an exclusionary criterion and ordinarily, ASD is viewed as an exclusionary condition for SLI. In this regard, SLI and ASD cannot overlap. But this exclusionary condition may be forcing SLI to be different from other communication disorders on arbitrary grounds.

The standard diagnostic conceptualization of language with the diagnosis of SLI has not distinguished between particular aspects of language; however, as research has been conducted with these children, a common profile has emerged. A large number of studies have shown that these children are particularly weak with respect to grammatical development. It has been within grammar that one finds particularly strong debate as to whether SLI represents a distinctly different type of learner or just a less adept learner. Several researchers who have examined the grammatical problems of children with SLI have concluded that these children have distinctive deficits with regard to grammar (Clahsen 1999, Gopnik 1990, Rice and Wexler 1996, van der Lely and Barratt 2003). One grammatical feature involving the failure to mark tense and aspect in obligatory contexts has been proposed as a marker for SLI (Rice and Wexler 1996). Additional markers for SLI have also been proposed with respect to poor sentence repetition and repetition of non-words (Borting and Conti-Ramsden 2003).

The notion that there is a marker for SLI suggests that there are features of SLI that are distinctive and largely unique to SLI. This raises a question as to how we conceive of SLI. The idea that there is a marker for SLI connotes the notion that SLI represents a distinct category of language learners that are different from other types of learners. Such a view therefore proposes that SLI is a natural kind. Quine (1969) defined a natural kind as a class or group that is bound together by natural law and thus has a common causal system. There is some reason to question whether these markers are unique to SLI. The grammatical problems associated with SLI are also found in other populations of children with developmental disorders such as Down syndrome and even Williams syndrome (for instance, Eadie et al. 2002, Grant et al. 1997). Furthermore, there is little evidence that the language skills of children with SLI are qualitatively distinct from children who are typically developing other than being simply less well developed (Dollaghan 2004, Leonard 1987, 1998, Tomblin and Zhang 1999). Findings such as these support the notion that children with SLI represent a region of poor grammatical skills that are at one end of a continuum of ability with children who are very good grammar learners are at the other end of the spectrum. Underlying this continuum, we might assume, are the same developmental processes that operate in a graded fashion over the whole continuum and thus generating faster or slower learning rates.

As we consider this notion of a continuum, we need to consider that the skills that go into communication
may be comprised of multiple continua as shown in figure 1. This concept is certainly not an original idea. Bishop (2000) has put forth very similar multidimensional accounts for communication disorder. As noted above, children with SLI are very likely to have poor grammatical skills and this is usually accompanied by poor vocabulary. Thus, these two aspects of language may arguably be a single dimension around which children can vary. In the literature on ASD and SLI this dimension has been called structural language. Bates and Goodman (2001) have clearly argued that these arise from a single system and thus represent a single dimension. As noted earlier, Pinker (1997) as well as van der Lely and Stollwerck (1997) have argued that these are distinct systems. Tomblin and Zhang (2006) have shown that these systems appear to be highly related, although in older children vocabulary becomes less associated with grammar, no doubt because grammatical development becomes limited whereas vocabulary does not.

There has been less certainty about whether speech sound disorder or expressive phonology is also a part of the language profile of SLI and thus as well perhaps a component of the same dimension as grammar and vocabulary. In an early study, Bishop and Edmundson (1987) proposed that speech-sound production (phonology) occupied the same dimension as grammar and semantics. In their model, these systems were differentially vulnerable and therefore one would find children with speech sound problems but spared grammar and semantics, but not the other way around. Therefore, all these aspects of language were related to each other and the strength or weakness of children’s skills could be placed along a single dimension. In recent years it has become more likely that the expressive phonological skills (speech-sound production) of children seem to be free to vary somewhat independently of vocabulary and grammar (Pennington and Bishop 2009, Shriberg et al. 1999, Tomblin et al. 2004). Thus, we may provisionally consider speech-sound production abilities to be a separate dimension that can vary among children largely independently from their grammatical and vocabulary abilities.

A third dimension of communication is concerned with the social use of language subsumed within the notion of pragmatics. Pragmatic development and skills are difficult to quantify and to portray in developmental terms in contrast with phonological, lexical or grammatical abilities. In contrast with their grammatical skills, the pragmatic skills of children with SLI have often been reported to be a relative strength (Craig and Evans 1993, Leonard et al. 1982, Schaeffer 2003), although this does not mean that social communication is unscathed in these children. Studies do show that they are less skilled in conversations and in their ability to adapt information content to the listener (Farrant et al. 2006, Adams and Bishop 1989, Marton et al. 2005). Some of these differences may be the outgrowth of their limited lexical and grammatical skills, but some social difficulties remain even when language skills are accounted for (Botting and Conti-Ramsden 2008).

Within the perspective just outlined, we have evidence that individual differences in spoken communication development seem to occur within a three-dimensional space. One dimension is defined by grammar and vocabulary and we can call this structural language. The next is speech-sound production; and the third is social communication or pragmatics. These dimensions are not completely independent of each other, but children are moderately free to vary in their accomplishment on each. Within this model SLI then occupies a region of individual differences primarily on the language dimension. In this regard, I am saying that SLI does not represent a natural kind of language learner. In fact, I would say that SLI only represents a convenient, arbitrarily constructed group of language learners who by this grouping are more similar to each other than learners who fall in some other region of the three-dimensional space just described.

I have just proposed that there appear to be three dimensions that contain individual differences in communication development. Each of these dimensions seems to be somewhat independent of the other and if this is correct then we might conclude that each dimension has its own set of developmental mechanisms. This perspective has to be incorrect on two grounds. First, there is no reason that some mechanisms can be shared across dimensions; but even more importantly, it is hard to believe that the
developmental status of one system such as speech-sound production does not influence another such as structural language. It is quite reasonable in my mind to believe that poor intelligibility will have an effect on structural language development. Thus, although these dimensions have the potential to be independent across a wide range of ability, there may be local effects in this relationship where poor ability in one domain has a direct effect on another domain. This interactive effect is likely non-linear and could therefore warp the local individual differences space much the way a gravitational field can warp the linear trajectory of light. This warping effect will have the effect of creating elevated rates of co-morbidity within what otherwise might be a set of relatively independent dimensions of communication development. This same notion of possible local interactions that could generate elevated co-morbidity will come up again when we begin to consider the relationship of SLI and ASD.

**Autism Spectrum Disorder (ASD)**

The diagnosis of ASD has come to be defined in terms of a triad of developmental deficits involving qualitative impairments of social interaction, communication, and restricted, repetitive and stereotyped behaviour (for instance, Happé and Ronald 2008). The language and communication profiles of children with ASD are highly variable across and within aspects of language and communication. As noted above, considerable research over the past several decades has taught us that pragmatic abilities appear to vary at least somewhat independently from structural aspects of language (phonology and grammar). This perspective has been reflected in Bishop’s (2002) distinction between pragmatic disorder and SLI. Children with ASD are characteristically challenged with respect to pragmatic ability. In contrast, we find considerable variation among these children with respect to their structural language abilities where some children are non-speaking, others present with poor language while still others have solid skills with respect to phonology, grammar and vocabulary. Interestingly, the aspects of communication that are characteristically limited in SLI—structural aspects—are most variable in ASD, and vice versa. This kind of profile contrast has often been used in neuropsychology as a double dissociation, which is used to argue that the two disorders are independent and therefore different natural kinds.

We can see this view of ASD as a natural kind rather clearly in the accounts of the aetiologies of ASD. It has been common to view ASD as a distinct form of child psychopathology that has a single unifying core deficit such as an impaired Theory of Mind (ToM), Central Coherence (CC) or Executive Function (EF). In this way of thinking there is a particular causal account for why children present with ASD that is different from one used with children who are typically developing, mentally disabled or who are SLI. Such a view of ASD has been challenged over the years simply by the recognition of the extent of variability in behaviours of children. We might ask whether ASD is a composite of several semi-independent dimensions such as we observed with speech and language disorders and that there may a continuum along which people can vary on these dimensions.

An early form of this perspective arose out of research on the family members of individuals with autism that resulted in the notion of a Broader Autism Phenotype (BAP) (Cox et al. 1975, Bolton et al. 1994, Le Couteur et al. 1996, Landa et al. 1996, Folstein et al. 1999, Fombonne et al. 1997). The BAP has been described by Bolton et al. (1994) as dysfunction in communication, social or stereotyped behaviours, and thus represents a lesser variant of autism that will be expressed in one or more of these domains. Thus, these researchers hypothesized that the triad of symptoms of autism were both dimensional in terms of severity and could be disassociated from one another. Furthermore, it was thought that different causal factors could influence each of these dimensions. More recently, others have presented a similar view. Studies are now showing that the traits that comprise ASD can be extended into the normal population and that there is little evidence that there is a discontinuity at some clinical boundary (Constantino and Todd 2003, Skuse et al. 2005). Furthermore, Ronald et al. (2006a, 2006b) have shown that these traits have considerable freedom to vary independently. This leads us to question whether ASD is properly viewed as a natural kind as well.

**SLI and autism: a long debate over kinship**

If SLI and ASD are natural kinds of developmental disorders, then it makes sense to consider whether they are in some way related. By definition, children with SLI cannot present with ASD; however, ASD does not exclude SLI and therefore this form of overlap has become a central issue of debate. Diagnostic overlap is often referred to as co-morbidity. Co-morbidity is the concept that two or more diseases occur together at rates that exceed chance levels. Classically, co-morbidity has referred to the co-occurrence of two or more independent diseases; however, this term is also used to refer to diseases that may be partially related (for instance, Neale and Kendler 1995) and it is in this sense that I will be using the term. If two clinical conditions with unknown aetiologies are co-morbid, we can hypothesize that they are likely to share common risk factors or aetiologies. Therefore, the question of co-morbidity of
SLI and ASD will ultimately rest on whether there are shared underlying mechanisms for the language deficits in these two conditions.

The notion that ASD was related to SLI extends back to at least 1967 when Rutter (1967) hypothesized that language impairment served as the primary abnormality in autism and that this abnormality might be similar to that of a severe receptive aphasia. In this respect ASD was a severe variant of receptive language impairment and thus the two were one and the same. In fact, Rutter considered the social difficulties in ASD to arise from the very poor language skills and therefore were secondary to language deficits. Rutter and colleagues tested this idea by contrasting the language and cognitive characteristics of children with receptive aphasia and children with autism (Bartak et al. 1975, 1977). They found that these two clinical groups were similar with regard to deficits in vocabulary and syntax; however, differences in the communication profiles of these children were found with respect to the use of gestures and spoken language for social communication purposes. That is, they identified the profile features described earlier wherein children with ASD were most prominently impaired in pragmatics and less affected in the structural aspects of language. Numerous other studies have demonstrated the marked impairment of social aspects of language in children with autism (Baltaxe 1977, Tager-Flusberg 1994). Because the profiles of ASD and receptive aphasia (SLI) were not the same particularly with respect to pragmatic skills, most researchers and clinicians recognized them as representing different developmental disorders.

**SLI and ASD as co-morbid**

Recognizing that two disorders are different; however, this does not necessarily mean they cannot be comorbid. As noted above, it remains possible for children with ASD to also present with SLI and this viewpoint was put forth about a decade ago by Tager-Flusberg and colleagues. In so doing, Tager-Flusberg’s claim has stimulated considerable debate over the last decade. This view was first expressed by Kjelgaard and Tager-Flusberg (2001) who emphasized the variability in structural language skills even among children with ASD who were verbal. This variability presented itself as a continuum from normal to very low structural language skills. They further noted that this variability in language contrasted with generally poor pragmatic skills and generally preserved speech-sound production abilities. In this respect children with ASD as a group presented with a different profile than children with SLI; however, a subgroup of children with ASD shared poor structural language problems with children with SLI, as depicted in figure 2.

These authors then focused on the dimension of variability that comprised structural language that provided this overlap. The continuum of language ability was therefore cut-up into three groups based on the Peabody Picture Vocabulary Test with cut-points placed at $-1$ and $-2$ SD, where normal levels were above $-1$ SD (ALN) and impaired levels (ALI) were below $-2$ SD, and those between $-1$ and $-2$ were borderline. They reported that 50 of the 82 children were in the ALI category, whereas only 22 were ALN. In a sample of 82 children we would expect that less than 3% of the population would score below $-2$ SD, and yet in this sample 61% scored below this threshold. This shows a substantial excess of children with poor language. Not only was there an excess of poor language learners, but also the authors presented data that argued that the language profiles of these children in the borderline group and impaired group match profiles reported for children with SLI. Additionally, they also noted that both SLI and ASD had been shown to be heritable and that the research on the BAP had suggested that near relatives of children with autism were prone to language-learning disorders. So they hypothesized that a subgroup of children with ASD may also have SLI. As such, SLI and ASD were therefore likely to have related if not shared aetiologies at least within this subgroup. Subsequent to this paper, Tager-Flusberg and Roberts (2003) expanded on this idea by bringing together literature on cognition neuroimaging and genetics in ASD to show greater evidence that a subgroup of children with autism may also have SLI. Two pieces of evidence in that paper focused on studies that examined for the presence of two markers of SLI: poor tense use and poor non-word repetition. In these studies they compared children with ASD and normal
language (ALN), children with ASD and apparent SLI (ALI), and a group of children with SLI. Tager-Flusberg (2006) as well as Tager-Flusberg and Roberts (2003) described the non-word repetition skills of children with ALI and ALN. Although both groups showed very good speech-sound production abilities on the Goldman Fristoe Test of Articulation, the ALI children were less capable of repeating syllable strings in non-words. Roberts et al. (2004) used an elicitation task that probed for tense usage in these children and found the ALI children and the SLI children were poor on tense usage. These findings that the children with ALI presented with linguistic characteristics of SLI added additional support to the view that SLI and ASD were co-morbid. I want to remind readers that I previously noted that grammatical deficits, even those just concerned with tense marking, are found in poor language users of other populations. A similar observation can be made with respect to non-word repetition. Poor non-word repetition is often observed in children with dyslexia and, indeed, Catts et al. (2005) have presented evidence that poor non-word repetition is more likely a marker for dyslexia than SLI. Thus, these SLI markers may not be good markers for identifying a particular natural kind of poor language learner but rather are associated with poor language skills in general. If so, then by selecting a group of children with ASD who have poor language, it should not be surprising to see that they show the signs of poor language learners. Having said this, we have not resolved the question as to why there is an excess of poor language learners regardless of what we call them.

Brain imaging

Unquestionably, language arises from and through brain systems. The structural properties of the brains of SLI and ASD could therefore reveal underlying biological markers of SLI in children with ASD. Research into the brain characteristics of children with ASD has been extensive; however, far fewer brain studies can be found for children with SLI, but some general patterns seem to emerge. Several studies have provided evidence for an abnormal left–right hemispheric relationship in children with SLI (Jernigan et al. 1991, Plante et al. 1989, 1991, Gauger et al. 1997) resulting in smaller than expected left perisylvian (posterior language areas) volumes relative to the right hemisphere. However, Preis et al. (1998) did not find abnormal symmetry in the perisylvian region. Gauger et al. (1997) also extended the regions of interest for SLI to the inferior frontal cortex where they also found the volume of the left hemisphere in this region to be smaller in children with SLI than controls.

Tager-Flusberg et al. (2004) recently reported a rightward asymmetry in the inferior frontal cortex of children with SLI than controls thus replicating the Gauger et al. study. Also, surprisingly given the prior results, the planum temporale was found to have more leftward asymmetry in the children with SLI than among typically developing children in this study. That is, children with SLI had larger left hemispheric development than controls. Herbert et al. (2005) performed a nested hierarchical analysis ranging from whole brain through major anatomical lobes to smaller regions defined as parcellation units with images from children with SLI. Similar to De Fosse et al. (2004), they found more rightward asymmetry in the frontal language areas and more leftward asymmetry in the parietal language areas.

Among the studies of brain structure there is a small set of studies that have directly contrasted children with SLI and children with ASD. In addition to reporting on findings concerned with SLI, De Fosse et al. (2004) compared children with SLI with children with ASD who either presented with poor language (ALI) or had normal language abilities (ALN). Patterns of abnormal asymmetry in the inferior frontal cortex were found for the children with SLI and the children with ALI. Likewise both the SLI and children who were ALI showed the unexpected exaggerated L > R asymmetry described earlier in the perisylvian area. The children with normal language and those with ALN did not show these abnormal asymmetric relationships. Herbert et al. (2005) used children with ASD who were described as being high functioning and therefore would be likely to have had normal structural language abilities and may have been similar to De Fosse et al.’s normal language ASD group. Despite this, they also found similar patterns of asymmetry to those of De Fosse et al.’s study for both anterior and posterior parcellation units involved with language. Thus, they also found the unexpected abnormal exaggerated L > R in both groups in the perisylvian area. Herbert and Kenet (2007) have concluded from these findings that autism and SLI share many, although not all, neurological features and thus could be viewed as overlapping conditions. I would also conclude that in studies where children with ALI were directly compared with children with SLI there is good evidence of similarity in brain morphology. It would appear that this pattern is more specific to poor language, possibly SLI, than to autism. It remains unclear as to why in these studies they found L > R in the perisylvian region where other studies have usually found the opposite. One likely reason is that both the De Fosse et al. and Herbert studies focused their analysis on a much smaller area in this region that the other studies. A question that I do not believe has been resolved is whether reversed hemispheric asymmetry in one form or the other serve as neurological markers for a special kind of poor language such as SLI or whether this is a brain feature of individuals with poor language.
Evidence for reversed asymmetry has been reported for children who are dyslexic (Eckert 2004). Thus, a conservative statement might be that children with and without ASD who have poor language also have similar brain morphology.

Familial patterns of SLI in ASD families

Recall that within the concept of the BAP was the notion that milder variants of the key features of ASD could be seen in near relatives. In fact, several of the studies that lead to the notion of the BAP reported on elevated language and language-related deficits in the parents and siblings of children with ASD. These studies were generally done in the 1990s and therefore the idea that SLI was running in these families was not broached. Recall that Kjelgaard and Tager-Flusberg did propose that these elevated rates of language problems in these BAP families might be reflective of SLI. The Tager-Flusberg research group in which I was participating (Ruser et al. 2007) compared parents of ASD, SLI or Down syndrome on communication and language from spontaneous language samples. The Down syndrome sample was used as a genetic transmission control since Down syndrome does not run in families. This study provided support for the notion that poor language skills ran in the families of ASD children just as it did in the families of children with SLI. However, when a distinction was made between the pragmatic skills and the structural language skills of the parents, it was shown that structural deficits were more common in the parents of children with SLI, whereas pragmatic problems were prominent in the parents of the children with ASD. Overall, we took these data as support for a possible genetic overlap between ASD and SLI. One thing to note in this study is that the ASD children were not differentiated into ALI and ALN subgroups. This distinction was made in a subsequent study by Lindgren et al. (2009) that compared language, phonological processing, and reading test performance in sibs and parents of children with SLI, ALI and ALN. The particular expectation was that within the ALI relatives we would see similar deficits to the relatives of the children with SLI given that the areas being measured are all language skills that are troublesome for children with SLI. Instead, we found that relatives of SLI children were generally poorer than relatives of the ALI or ALN. Thus, these data did not provide support that a particular kind of language impairment—SLI—was running in both the SLI and ALI families.

ASD and SLI co-morbidity questioned

What I have done so far has been to present the evidence that has motivated the Tager-Flusberg group to hypothesize that there is a subgroup of children with ASD who also have SLI. As this hypothesis was published, some raised questions as to whether these children with ALI were indeed SLI. Recently, Whitehouse et al. (2007, 2008) have argued against this notion of an overlap between SLI and ASD, claiming that SLI and ASD although similar in some surface manifestations of language deficits, differ with regard to the underlying bases of these deficits. For instance, the non-word repetition deficits in the ALI children were qualitatively different from children with SLI. They suggest that the apparent signs of SLI among children with ASD do not hold on closer inspection and that the overlap in familial risk is likewise weak and not well founded. These authors have concluded that there is no basis for claiming that SLI and ASD are related at the behavioural symptom or aetiological level. They concluded that children with SLI present with a disorder that involves deficits in phonological working memory and grammatical development whereas children with autism have impairments in language use and social interactions as well as restricted interests and behaviours. Even in cases where there appears to be symptom overlap such as with non-word repetition, the underlying reasons for poor repetition differ between the two groups. Thus, these authors have voiced doubt that there is a sizable subgroup of children with ASD who are also presenting co-morbid SLI.

Recently Williams et al. (2008) reached a similar conclusion based on a detailed analysis of the data found in the literature. One point that was emphasized in this review was the contrast between the profiles of children with ALI and SLI reported in both Kjelgaard and Tager-Flusberg (2001) and Lloyd et al. (2006). In both cases, the ALI children had greater rates of poorer receptive than expressive language problems and better speech sound abilities than the children with SLI. I do not believe that either of these are in fact core features of an SLI profile. Most studies of SLI recruit children who are receiving clinical services. In an epidemiological study of children with SLI that I conducted (Tomblin et al. 1997) most of the children with SLI were not and often had never been identified. In this study we learned that the rate of speech sound disorder was much lower than had been thought but was high in children who were receiving clinical services. Likewise, we found many more children with receptive problems in the children who had not been identified compared with those who had been identified. What we had discovered is a well known problem in medical research. Those people who seek and receive clinical help will present a different profile of an illness than those who are also affected but do not receive care. That is, there are built in biases in our clinical process that influences the clinical profiles of children in those services. We see very good evidence...
of this with respect to children with receptive language problems. We have found that these children often are not identified until their poor language comprehension results in poor reading comprehension. It should be of no surprise that the poor comprehenders described by Nation et al. (2004) are similar to children with SLI except that they have greater receptive problems. If we return to the notion of a three-dimensional space containing individual differences language, these biases mean that children in different parts of this space have different probabilities of getting clinically served by speech–language clinicians or reading specialist etc. The fact that children with SLI who enter clinical speech–language service centres are going to be those who show greater problems in speech and expressive language simply is due to the fact that these forms of communication disorder are more obvious and teachers are more likely to send children to a speech–language clinician if they don’t talk well. The children who comprise the ALI group get referred for their clinical services because of other troubling behaviours and therefore even those with good speech and poor receptive problems still are identified. The lesson we learn from this is that we cannot expect that the profiles for children who have been identified with ASD will perfectly match the children who have been identified with SLI.

In summary, I believe that the debate over an overlap between SLI and ASD rests on shaky ground. It assumes that SLI defines a natural kind of language learner and that this distinctive quality is shown in characteristic markers and profiles. Instead I have argued that many of these features can be seen in other groups of children with poor language and that some of these features are likely to arise from systematic biases in the referral and case selection process. Since I do not accept that SLI represents a distinct type of language learner, then I must conclude that children who are ALI are not likely to be a simple mixture of SLI and ASD. But this still leaves us with the important observation that Kjelgaard and Tager-Flusberg raised. Why is it that we see so many children with ASD who also have poor structural language? Below I will propose three reasons that this may be so.

Shared aetiology
Recall that when I was talking about the three principal dimensions of communication, I noted that these dimensions could quite reasonably share some common mechanisms. In this regard the independence is not complete. This may hold for the relationship between poor structural language and ASD. We found evidence of just this type of aetiological overlap in the structural brain imaging and recently another example has come from molecular genetics.

CNTNAP2
The CNTNAP2 gene is in the 7q35 region and has been associated with both autism and SLI. Bakkaloglu et al. (2008), Alarcon et al. (2008) and Arking et al. (2008) simultaneously published papers showing that common and rare genetic polymorphisms within CNTNAP2 were associated with autism. In both the Bakkaloglu et al. and the Arking et al. studies the phenotype was based on autism diagnosis. In the Alarcon et al. study the phenotype most strongly associated with CNTNAP2 was a quantitative trait concerned with the age at which the parents reported the child with ASD acquired the first word and therefore these findings implicated CNTNAP2 as a gene that might be associated with language problems with or without autism. Indeed, Vernes et al. (2008) reported that the same mutation in CNTNAP2 that was associated with autism was also associated with phonological short term memory in a sample of children with SLI. These findings have provided the first strong evidence supporting a shared aetiology between autism and SLI; however, we could just as easily speculate that this gene influences individual differences in language in general.

Direct interactions
While partial aetiological overlaps in language and ASD seem quite likely; I cannot believe that the ALI children will be identical to children with poor language but who are not ASD. In this regard, I return to the notion of local warping of relationships by the direct influence of the developmental status of one dimension onto another. By definition, children with ASD have a range of cognitive and social difficulties as well as many behavioural difficulties that come from their rigidity, highly focused interests and preference for repetition. Not only are these behaviours likely to interact within the child, they also will have dramatic effects on the manner in which others around them interact with them. It is hard to believe that language, even the structural aspects, is so modularized that these features do not have an impact on their language development. I believe that this is the point raised by Williams et al. and Whitehouse et al. (2007, 2008). The fact that the background risk factors for poor language interact with the features of autism will make it very difficult to tease these apart in the language behaviours of these ASD children.

Aspects of diagnosis
Finally, there is a fairly obvious explanation for the excess in structural language problems in ASD. Early poor language and communication are a part of the
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conditions that must be met for a diagnosis of autism. If a child developed language normally from early on, it is likely the child would be considered as having Asperger syndrome. One of the principal early signs of ASD is late onset of speech and language. Thus, simply on the basis of how we sort children into diagnostic classes, children with poor language will have a greater probably of receiving the diagnosis.

Conclusions

I began this paper by pointing out that our clinical classification systems reflect beliefs about the processes that give rise to these clinical conditions. If we distinguish between ASD and SLI, do we assume that each of these is a unified distinct clinical entity with its own distinct causal system? Furthermore, if we observe an excess of SLI in the population of children with ASD, should we assume that this points to an overlap in the aetiologies of these two? This appears to be the nature of the debate that has been carried on during the past decade. What I have attempted to do has been to redefine our view of SLI (and perhaps ASD) such that they are not distinct kinds of disorders. Instead the children with SLI and ASD are indeed children first. They, like all children, develop from very complex developmental systems. Some of these systems when perturbed in particular ways may tip the developmental trajectories of a child toward ASD or SLI (poor structural language learners), thus there are likely to be some unique factors associated with each, but also there are likely to be shared or common aetiological factors as well. Additionally, to make things more complex, we can expect to find local causal relationships that warp the overall independence of subsystems or dimensions. Furthermore, we cannot ignore that our clinical services and diagnostic schemes are actually carving up a multidimensional space that houses individual differences. These systems reflect value systems and professional politics, but ultimately shape the similarities and differences of the clinical groups.

In conclusion, ASD and what has been known as SLI very likely overlap. However, I doubt that they overlap in the form of overlapping natural kinds. Instead, the overlap is reflective of deep complexity that probably produces a complex mixture of similarities and differences just as children with both SLI and ASD overlap in many ways with typically developing children.

Note

1. Structural language in this case includes both grammar and vocabulary.

References


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