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Developmental Language Disorders: Challenges and Implications of Cross-Group Comparisons

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Abstract

Historically, specific language impairment (SLI) and language deficits associated with autism spectrum disorders (ASD) have been viewed as distinct developmental language disorders. However, over the last decade or so a considerable amount of research has explored general similarities or specific areas of overlap between children with SLI and ASD based on language and cognitive profiles, neuroimaging findings, and genetic research. The clinical classification schemes that are used to identify the children necessarily influence the extent to which SLI and ASD are viewed as overlapping or distinct conditions. Yet, the criteria used to diagnosis these two populations vary across countries and even across investigators within a given country. This necessarily impacts the findings from comparative investigations of these groups. With these challenges in mind, clinical implications of evidence for similarities and distinctions between children with SLI and ASD will be discussed with respect to differential diagnosis and treatment.

Introduction

There has been an increased interest recently in the extent to which specific language impairment (SLI) overlaps with autism spectrum disorders (ASD), even though historically these two neurodevelopmental disorders were seen as quite distinct. SLI is typically defined as a developmental language disorder that is associated with no known sensory, neurological, intellectual, or emotional deficits [1-3]. Autism involves deficits in social interaction, communication, and restricted and repetitive behaviors and interests, with a spectrum of conditions ranging from autistic disorder to Asperger's syndrome [4]. Both of these disorders are extremely heterogeneous in nature. Most children with SLI have deficits in structural language (phonology, semantics, morphology, syntax) but others have relatively more difficulty with pragmatic aspects (social use) of language [5]. Children whose primary difficulties involve pragmatic aspects of language and communication rather than structural language have been referred to as having Pragmatic Language Impairment (PLI) [6-7]. Similarly, in addition to variability in other defining features of autism and co-morbid conditions (e.g., intellectual disability), children on the autism spectrum display considerable variation in language and communication skills [8-9]. Deficits in pragmatics are closely linked to difficulties in social interaction and are often viewed as a universal aspect of ASD. However, some individuals with ASD display structural language deficits that mimic those of SLI while others do not, leading to designations of ASD-LI (language impairment) and ASD-LN (normal language). Thus, although core defining criteria of SLI and autism differ it is at these intersections of subgroups within the larger populations that

questions of overlap arise – that is, PLI overlaps with autism spectrum features and ASD-LI overlaps with SLI.

Examination of SLI-ASD overlap

The overlap between children with SLI and ASD will be considered based on their phenotypes (i.e., language and cognitive profiles), neuroimaging findings, and genetic research. Claims regarding language overlaps for a subset of children with SLI and ASD have focused on similarities in social communication or pragmatic aspects of language as well as on structural language components. Cognitive profiles have been compared at a broad level in terms of the discrepancy between nonverbal cognition and language levels observed in SLI and a subset of children with ASD. Specific aspects of cognitive functioning (e.g., theory of mind, executive function) have also been compared in the two diagnostic groups. As reviewed below, comparisons have been made from the perspective of clinical markers of SLI (e.g., nonword repetition deficits) in children with ASD, and conversely from the viewpoint of defining features of autism (e.g., performance on autism diagnostic measures) in children with SLI.

Evidence regarding Phenotypes

Although the question of possible overlap between SLI and autism has a long history [10-11], this issue has sparked a vigorous debate in the literature that has escalated within the last 10 to 15 years. One proposed source of phenotypic overlap between SLI and ASD is in the area of pragmatics or social use of language. As noted previously, a subset of children with SLI has been identified as having Pragmatic Language Impairment (PLI) whose primary difficulties are with the use of language in context rather than core structural impairments in grammar, phonology or vocabulary [12-13]. Children with PLI have been characterized as displaying difficulties with topic maintenance, discourse comprehension, abstract language, semantic specificity, and sensitivity to the needs of their conversational partners [14-15]. While Bishop and Norbury [6] demonstrated that children with PLI did not meet the more stringent criteria for *autistic disorder* there has been a good deal of discussion about how best to conceptualize this subgroup of children and the relation between SLI and the broader autism spectrum [16-17]. Complicating the investigation of this issue are the shifting diagnostic boundaries between SLI and pervasive developmental disorders [18], as well as reports of actual evolution of social communication deficits and autistic features over development in children with a history of SLI [19].

Research has also explored the extent to which structural language deficits in a subset of children with ASD are similar to those seen in SLI, with a number of studies providing evidence of phenotypic overlap [20-24]. For example, both children with SLI and children with ASD-LI often omit tense marking morphemes (especially in English speakers) and display deficits in phonological processing/memory span as indexed by nonword repetition tasks [21,23]. Deficits in marking tense have been proposed as a clinical marker of SLI [25], as have deficits in nonword repetition [26]. That is, both of these patterns of difficulties are central to the characterization of SLI, so it is particularly notable for these areas to also be problematic in ASD-LI. Further evidence of overlap in the language phenotype comes from an investigation by McGregor and colleagues [27] in which they found similar patterns of

cross-domain (i.e., lexical-syntactic) associations for children with SLI and children with ASD and language impairment (ASD-LI). Similar patterns of lexical-grammatical development have also been observed in toddlers with ASD and late talking toddlers without autism (who may be at risk for SLI) [28].

On the other hand, it is definitely not the case that all results are consistent in supporting the claim of structural language similarities in children with SLI and children on the autism spectrum who have language impairment. Williams and colleagues [29] have argued that the research evidence does not support commonalities in language phenotypes across these groups, noting that even though behavioral similarities might be observed at a given point in development it is important to consider differences in the overall trajectories in language development for these populations (also see Charman 2010 [30] regarding individual growth trajectories across development in autism). With respect to particular aspects of structural language, both groups have deficits in phonological processing/short-term memory as indexed by nonword repetition; however, they have been shown to display different error patterns [31-32]. In a recent study, nonword repetition abilities were compared in children with SLI, children with autism spectrum disorder who had language impairment (ASD-LI), and typical controls matched on verbal mental age [33]. Nonword stimuli were manipulated with respect to stimulus length, consonant cluster position, and wordlikeness. Findings revealed significant differences between the SLI and ASD-LI groups in terms of level of accuracy and error types, while the ASD-LI group performed similarly to the language-matched children with typical development. Williams and colleagues argue that these findings indicate that different cognitive factors underlie difficulties in nonword repetition in SLI and ASDLI. Demouy and colleagues [34] examined language characteristics of French-speaking children with autistic disorder (AD), pervasive developmental disorder – not otherwise specified (PDD-NOS), and SLI (mean age of 9 years) who were matched for chronological age, sex, and academic abilities in terms of various components of linguistic functioning including prosody. Vocabulary and phonology did not differentiate the groups. However, expressive syntax, pragmatics, and certain features of prosody did distinguish these groups. These investigators reported that more of a developmental delay profile was observed for SLI than the other groups in that the SLI group exhibited strong correlations between raw scores on the language measures and chronological age whereas the AD and PDD-NOS groups did not (except for pragmatic errors for the PDD-NOS group). Demouy et al. interpreted their findings as being indicative of different underlying mechanisms in AD and SLI, with PDD-NOS having an intermediate profile with some characteristics of each of the other two conditions.

Most of the research comparing SLI and ASD has examined language phenotypes, but there has also been some interest in more global comparisons of autistic features and of specific cognitive profiles. Two recent investigations have examined overlap between children with ASD relative to children with SLI or PLI based on their performance on autism diagnostic measures [35-36]. Reisinger and colleagues compared school-aged children with ASD and PLI in terms of ‘autism triad’ impairments on the Autism Observational Schedule (ADOS) [37] and the Social Communication Questionnaire [38]. The children with ASD had more severe impairments in communication and social interaction than children with PLI as well as a higher number of restricted and repetitive behaviors and interests (RRBIs); however,

these investigators emphasized the commonalities in the ASD and PLI groups based on the fact that both groups presented impairment in all three of the 'autism triad' areas. Similarly, Leyfer et al. [35] reported evidence for overlap in social and communication deficits in SLI and ASD based on a comparison of performance on the ADOS and Autism Diagnostic Interview-Revised (ADI-R) [39]. Specifically, when groups of children with SLI or ASD were matched on nonverbal IQ, results indicated that 41% of the SLI group met criteria for autism or autism spectrum on the social or communication domains of the ADOS, ADI-R, or both. Taylor and colleagues [40] conducted a review of recent literature to determine whether there is evidence that cognitive profiles of children with SLI and ASD overlap. The aspects of cognitive functioning that were explored were ones that have been identified as deficit areas for children with autism, namely theory of mind, emotion recognition, executive functions, and central coherence. Taylor et al. concluded that the results across studies are mixed and that there is not substantial support for the claim that SLI and ASD have overlapping cognitive phenotypes. Readers are also referred to a recent discussion regarding the relation between language and executive functions in children with ASD as compared to SLI [41].

Neuroimaging Results

Researchers have debated whether the apparent language similarities between SLI and a subgroup of children with autism who have language impairment actually reflect a common etiology [29,33,42-44]. Findings from neuroimaging studies as well as genetic investigations have attempted to shed light on this issue.

In two separate studies using magnetic resonance imaging (MRI), Herbert and colleagues demonstrated enlargement of cerebral white matter in children with autism [45] and children with 'developmental language delay' (this term is used in manner that is generally consistent with the term 'SLI') [46]. In subsequent investigations that directly compared these clinical groups Herbert et al. have reported similarities with respect to enlargement of radiate white matter (corona radiata) [47] and rightward cerebral cortical asymmetry [48]. Several neuroimaging studies have compared the same set of boys with autism with language impairment (ALI), autism but normal language, SLI, and typical development [49-50]. De Fossé and colleagues [49] found that the SLI and ALI boys displayed significant reversal of volumetric asymmetry in frontal language association cortex such that both groups with language impairment had a rightward bias whereas the groups without language impairment had a leftward bias. These findings indicate that reversal asymmetry in Broca's area is related to language impairment rather than autism diagnosis. Hodge and colleagues [50] reported finding reversed asymmetry in posterior cerebellar lobule VIIIA in boys with language impairment (with and without autism), but not in normal language boys (with or without autism). Further, they found that scores on language tests correlated with lobule VIIIA asymmetry and with anterior vermis volume. When combined with this lab's prior findings of reversed asymmetry in Broca's area these results provide evidence that ALI and SLI groups display abnormalities relative to controls (a typically developing group and autism group without language impairment) in fronto-cortiocerebellar circuits implicated in motor control and processing of language, working memory, and attention; this pattern of findings suggests that these neural anomalies are associated with language and cognitive

deficits rather than autism per se. However, Hodge and colleagues point to genetic evidence for overlap in ASD and SLI and suggest that genetic factors which influence the development of cerebral asymmetry may impact the atypical patterns seen in both SLI and ASD.

An international team of investigators (from Belgium, The Netherlands, Italy and the USA) addressed the question of whether there is a common neuroanatomical substrate for the overlapping language phenotypes seen in SLI and ASD-LI using diffusion tensor imaging (DTI) techniques [51]. These researchers studied the white matter microstructural properties of the superior longitudinal fascicle (SLF), one of the critical language tracts connecting Broca's area and Wernicke's area. Children with ASD-LI (mean age of 13 years) and age-matched typically developing controls were compared to children with SLI (mean age of 10 year) and their age-matched controls. Results from standardized language tests confirmed that both clinical groups displayed mixed receptive-expressive language deficits, demonstrating similar linguistic phenotypes at least at a broad level. DTI findings revealed no significant differences in mean SLF fractional anisotropy (FA) and mean SLF apparent diffusion coefficient values for the ASD-LI and their control group. Conversely, the mean SLF FA for the SLI group was significantly reduced relative to their controls. These results indicate structural differences in SLI that were not seen in ASD-LI, suggesting different neuroanatomical bases for these disorders.

Genetic Findings

Behavioral genetic studies examining the overlap between SLI and ASD have yielded mixed results (for a review of potential genetic influences on developmental language disorders see [52]). Familial aggregation studies have demonstrated an increased risk of ASD to siblings of children with SLI [53-54] and an increased risk of SLI to siblings of children with ASD [55]. Despite the fact that the findings of Pickles and colleagues [53] replicated earlier reports of an elevated risk of ASD in families of children with SLI, their overall results did not support a common etiology for these disorders. Using the Family History Interview [56], Pickles et al. examined communication and social deficits in relatives of SLI, ASD and Down syndrome (DS) probands (a non-heritable disorder that served as a control for the other clinical groups). Findings indicated that relatives of SLI probands had more communication deficits than social difficulties compared to ASD and DS relatives. Conversely, relatives of ASD probands displayed elevated levels of social deficits when compared to SLI-only and DS relatives. A subgroup of the SLI probands were identified as SLI+ASD; that is, children with SLI who later developed autistic symptomology [57-58]. The relatives of the SLI+ASD subgroup had a higher rate of social deficits than the SLI-only subgroup relatives, much like the relatives of the ASD probands. Other genetic research has focused on clinical markers for SLI, with a comparison to ASD. High heritability of phonological short-term memory deficits as measured by nonword repetition tasks has been clearly demonstrated for SLI [26,59]. Although children with SLI and ASD both exhibit deficits in nonword repetition, there is evidence from genetic studies that these deficits are not familial in autism [60-61]. A series of studies have indicated that the CNTNAP2 gene is associated with both SLI and autism, suggesting shared etiology of these conditions or language impairment more broadly (see review by Tomblin [44]). Some quantitative

genetics findings have suggested a locus of susceptibility common to ASD and SLI [62-63] but other genetic linkage studies have reported different linkage signals [64].

Several comprehensive review articles have addressed the question of etiological overlap in ASD and SLI [29,44]. Based on a summary of language, neurobiological, and genetic data, Williams and colleagues [29] acknowledge certain dimensions of language overlap for children with SLI and ASD at some points in development, but argue that surface similarities do not mean that the language profiles stem from the same underlying causes. With respect to both neurobiological and genetic data they contend that the evidence does not support the claim that SLI and ASD are overlapping disorders. Whitehouse and colleagues [32,61] have expressed this same view concerning the relationship between these disorders. A review paper by Tomblin [44] considers the relationship between SLI and ASD based on language data, brain imaging findings, and genetic research. Although he contends that partial etiological overlap appears likely, he qualifies this conclusion by pointing out the complexities inherent in this debate, including the heterogeneous nature of SLI and ASD, the fact that features of SLI also occur in other neurodevelopmental disorders, and that the clinical classification schemes that are used necessarily influence the features of the conditions under investigation. Focusing on genetic evidence, Bishop [43] compared etiological models of ASD and SLI. Based on her findings she rejected a 'phenomimicry' account which claims that similarities in these clinical phenotypes are superficial in nature and actually have different underlying causes. Instead, she claimed that results from family studies and molecular genetic research were best explained by a simulated model that postulates overlapping etiology for SLI and ASD through the incorporation of gene-gene interaction. A recent study by Barlett and colleagues [65] provides empirical support for Bishop's [43] hypothesis and computer simulation, suggesting that gene-gene interaction can account for common molecular genetic findings across SLI and ASD while allowing for phenotypic differences related to each diagnosis.

Summary of SLI-ASD Comparisons

After more than a decade of investigation, the empirical findings do not offer a clear answer regarding the exact nature of the relationship between ASD and SLI. Certainly, both disorders entail language and communication problems (as do a number of other neurodevelopmental disorders) yet each is defined by other distinct diagnostic criteria. Unlike disorders that have an identified genetic basis (e.g., Fragile X syndrome, Down syndrome) or an obvious sensory component (e.g., language impairment associated with hearing loss), both ASD and SLI have unknown etiology. When subgroups of children from these two different diagnostic categories appear to display intriguingly similar behavior in certain areas of language and/or cognitive functioning researchers have attempted to ascertain the boundaries of the overlap with respect to the behavioral phenotypes and follow these clues in search of shared etiology. Whether or not the bulk of the evidence supports the notion of shared etiology between SLI and ASD is a matter of considerable debate [29,43,44], as is the question of whether investigation of this particular cross-group comparison is even a productive line of research [29].

Terminology issues

Adding to the complexity of this area of research is the fact that the classification schemes used to identify SLI and ASD necessarily impact the extent to which these disorders are viewed as overlapping [44], yet these schemes are not entirely consistent. In fact, based on a large scale three-year investigation sponsored by the British government, referred to as the Better Communication Research Programme (BCRP), a group of researchers in England noted that the lack of clear and consistent terminology regarding language and communication disorders across research, clinical, and educational settings within and across different countries is very problematic [66].

Diagnostic criteria for autism and language disorders have been set forth by the ICD-10 [4], the tenth revision of the International Statistical Classification of Diseases and Related Health Problems developed by the World Health Organization (WHO). Alternately, these disorders are identified within the United States and certain other countries using the Diagnostic and Statistical Manual of Mental Disorders (DSM) published by the American Psychiatric Association (APA). The diagnostic criteria for language impairment and autism provided by the fourth edition of the DSM (DSM-IV) [67] and ICD-10 were fairly compatible with respect to autism and language disorders. Both the ICD-10 and DSM-IV classifications include subgroup distinctions based on language modality, i.e., receptive language disorder and expressive language disorder, and provide similar diagnostic classifications for autistic disorder, Rett's syndrome, other childhood disintegrative disorder, Asperger's syndrome, and pervasive developmental disorder – unspecified. However, there is considerably less compatibility across ICD-10 and the new DSM-5 classifications [68] for these two disorders. The category of Communication Disorders in the DSM-5 includes classifications of 'language disorder' (without separate codes to distinguish between receptive or expressive subtypes) and 'social communication disorder' which appears to be equivalent to the term pragmatic language impairment (PLI) in the research literature. More substantive changes have occurred in the DSM-5 guidelines for autism diagnosis such that Rett's syndrome has been removed and the remaining classifications have been merged under the single term 'autism spectrum disorder', with further distinctions being made on the basis of severity.

In addition to inconsistencies in these two sets of official diagnostic criteria, varying terminology is used by researchers and clinicians. Conti-Ramsden and Botting [18] noted that a shift has occurred over time in the boundary between SLI and pervasive developmental disorder within the UK. Children identified as having PLI appear to fall between typical SLI and autism classifications. The PLI classification has never been widely used in the US among researchers or clinical practitioners. Now that this classification, labeled 'social communication disorder', is identified in the new DSM-5 it will be interesting to see how this unfolds and whether children meeting these criteria will ultimately be identified and receive services. Although the term SLI is commonly used internationally within the research literature, it is not used in clinical or educational settings – at least not within the US. In fact, when there was consideration of including SLI in the DSM-5 as a separate classification of communication disorder, the American Speech-Language-Hearing Association strongly argued against this terminology saying that it is not

used clinically and that most SLTs do not have cognitive/IQ data on which to make this diagnosis [69]. There were a series of contentious debates among researchers and clinicians from the US, Australia, France, and elsewhere regarding the revisions to the autism diagnostic scheme adopted by the DSM-5 [34,70-73]. Interestingly, Dr. Thomas Insel, Director of the National Institute of Mental Health, a division within the National Institutes of Health in the US, came out with a strong criticism of DSM criteria claiming that while providing reliability in use of terminology, the DSM diagnostic categories lacked validity. Dr. Insel stated, "Indeed, symptom-based diagnosis, once common in other areas of medicine, has been largely replaced in the past half century as we have understood that symptoms alone rarely indicate the best choice of treatment" [74]. As an alternative, the NIMH has launched a 10-year project referred to as the Research Domain Criteria Project in which researchers funded by this agency will be required to include patients across DSM categories or subdivide current categories and to collect cognitive, imaging, genetic, or physiologic data to determine how these cluster with respect to response to treatment [75].

Clinical Implications for Diagnosis and Treatment

Diagnosis

Even if there were complete agreement with respect to classification schemes, the task of implementing classifications in clinical practice for the purpose of diagnosis is not always straightforward. This is not only true for differential diagnosis at very young ages, but also at various points in development. For example, Tierney and colleagues [76] present a discussion between physicians regarding two case examples of 7-year-old boys to illustrate difficulties in distinguishing borderline cases of SLI and ASD. This can be a problem for physicians, psychologists, and speech-language therapists (SLTs).

One approach that may be useful for the purpose of differential diagnosis is to focus on the points of distinction that have been noted in the research literature comparing SLI and ASD, as a supplement to the symptoms and descriptions detailed by the ICD-10 or DSM-5. Studies have found that severe receptive language deficits (as opposed to primarily expressive language deficits) are typically indicative of ASD rather than SLI or early language delay [10,77-78]. Prosodic deficits have been reported in ASD across various languages [34,79-81] and have been shown to distinguish between children with ASD and SLI [34]. In this regard, Demouy and colleagues [34] noted the usefulness of automated approaches to assessing prosodic/vocal characteristics of children in naturalistic settings [82-83]. Error patterns on nonword repetition tasks, rather than overall accuracy levels, have been shown to differentiate ASD and SLI groups [32,34]; thus, error analysis or use of a nonword repetition task recently developed by Williams et al. [33] which manipulates nonword length, cluster position, and presence or absence of a suffix could be a helpful means of distinguishing between these disorders. Manolitsi and Botting [77] assessed language abilities in Greek-speaking children with SLI and ASD using standardized tests and narrative language samples. Their findings suggest that narrative assessment is a useful tool for identifying qualitative differences in language abilities between these groups that may not be revealed on standardized measures. A large-scale study of Italian-speaking children also found that narrative language samples were useful in distinguishing

morphological and syntactic deficits in SLI compared to typically developing children [84]. Norbury and Bishop [85] examined narratives produced by English-speaking children with SLI, PLI, and high functioning autism. They found that the diagnostic groups were differentiated by their use of referencing skills such that children with autism produced more ambiguous nouns and pronouns than the other groups; however, overall, there was a good deal of overlap across groups especially with respect to structural language abilities. Of course, when attempting to assess language disorders it is important to keep in mind that language-specific features will impact how disorders are manifested across different languages [86].

Treatment

When considering interventions, it is important to be concerned with etiology even though there is a complex dynamic between genes, brain, and behavior. Etiologic factors are especially important when considering pharmaceutical treatments; however, nearly all of the interventions for language and communication deficits involve behavioral approaches. Can different types of interventions be used effectively with different diagnostic groups? Perhaps this is where research findings regarding points of overlap or similarity across the children with ASD, SLI, and PLI might be instructive. Based on the national study undertaken by investigators in England as part of the Better Communication Research Programme, it was reported that children's individual characteristics, rather than their classification, predicted their learning needs. However, it was classification (Language Impairment vs. ASD) that determined the amount of resources they received [66]. Reisinger and colleagues [17] have stressed the importance of similarities between ASD and PLI groups with respect to intervention. They concluded that sorting out diagnostic distinctions is important from a “neurodevelopmental perspective” but suggest that it may be equally important in certain cases to disregard categorical boundaries and instead focus on individual areas of strengths and deficits. They conclude that the commonalities across these groups may be more important than their differences from an intervention standpoint. Reisinger et al. suggest that children with PLI, like children with ASD, could benefit from treatment focused on social skills development and may be at risk of being bullied, with associated mental health problems. It could similarly be argued that children with ASD-LI who have deficits in grammatical aspects of language [87] like those seen in SLI may respond to the same types of interventions focused on facilitating grammatical acquisition through intensive modeling, sentence recasting and expansions. Consider for a moment cases in which the etiology of language disorders is known to be different, such as Down syndrome and SLI. There is a clear genetic basis for Down syndrome in which language impairment is associated with cognitive delay whereas SLI has no general cognitive delay but also presents with an overall pattern of delayed language. Caselli and colleagues [88] compared language abilities of Italian-speaking children with Down syndrome and SLI who were matched on mental age. Their findings revealed a number of commonalities across these groups in lexical and morphosyntactic areas, including striking similarities in morphosyntactic error patterns. Research has also demonstrated that both of these groups exhibit difficulties in auditory processing and working memory thought to be related to their linguistic difficulties [89-90]; therefore, they may benefit from similar intervention targeted on this area. As suggested by the new initiative proposed by the National Institutes of Health, it may be fruitful to cross

diagnostic categories to focus on commonalities in specific cognitive underpinnings of language and communication deficits as they relate to treatment response.

Concluding Thoughts

Despite the fact that some researchers have questioned the utility of studies comparing ASD and SLI [29], it can be argued that information gained from these investigations has important clinical implications with respect to differential diagnosis and intervention. Even though the findings concerning overlap between these disorders are decidedly mixed, they do point to particular areas of commonality and distinction. Given the lack of consistent terminology and diagnostic criteria, results from studies that have directly contrasted these diagnostic groups provide an additional evidence base on which to draw. From a global perspective, it is critical to understand the extent to which cross-cultural issues impact the identification - and subsequent treatment - of developmental disorders [91-93]. Norbury and Sparks [92] provide an in-depth and thought-provoking discussion of how cultural issues influence whether individual variations in the behavioral symptoms manifested in SLI and ASD are viewed simply as differences or disorders and emphasize the need for cross-cultural studies to extend the largely North American and European view of these disorders. Whether or not ASD, SLI, and PLI are ultimately found to have shared etiology, evidence should be gathered to determine if common interventions geared towards points of language, cognitive, or social overlaps among these groups yield similar response to treatment.

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