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Verbal deficits in Down's syndrome and specific language impairment: a comparison

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Verbal deficits in Down’s syndrome and specific language impairment: a comparison

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Abstract

Background: Down’s syndrome is a chromosome disorder characterized by a range of physical and psychological conditions, including language impairment. The severity of impairment is variable, and some components of the language system appear to be more affected than others. This description could also be applied to typically developing children diagnosed with specific language impairment.

Aims: To compare findings from the largely separate research literatures on these conditions, and to address the questions about whether the language pathology associated with Down’s syndrome could be the same as that described as specific language impairment in typical development, and whether the two conditions could have similar causes.

Main Contribution: Research studies suggest similar patterns of language impairment in the two populations, and some similarities in underlying processing deficits.

Conclusions: Future research should consider whether similarities in the language behaviours associated with Down’s syndrome and specific language impairment could be related to similarities at other levels of analysis, including neurological development and genetics.

Keywords: Down’s syndrome, specific language impairment, language disorders.

Introduction

Down’s syndrome (DS) is the most common biological cause of developmental delay with an incidence rate of around 1:1000 live births (Steele 1996). In 95–98% of cases, DS is caused by a trisomy resulting from non-disjunction of chromosome 21 at meiosis. More rarely, it is the result of the translocation of chromosome material, or is due to mosaicism where an error has occurred during cell division in embryogenesis so that only a proportion of an individual’s cells is affected by the trisomy. As well as various medical problems and moderate-to-severe levels of learning disability, most people with DS face the challenge of language impairment. The present review explores the notion that this impairment mirrors that...
encountered in otherwise typically developing individuals diagnosed with specific language impairment (SLI), and considers whether the language disorders could have similar causes.

At first glance, the differences between SLI and DS are far more evident than the similarities. Children with SLI, by definition, have no general cognitive delay and no obvious signs of neurological impairment. The diagnosis is made when a child has selective difficulties with language development that cannot be accounted for in terms of low intelligence, hearing impairment, physical disabilities or abnormalities of the language-learning environment. Two-to-three times as many males than females appear to be affected (Robinson 1991). Although SLI can occur in the absence of speech impairment, a proportion of children have both speech and language difficulties (e.g. Newcomer and Hammill 1988, Bishop et al. 1995, Conti-Ramsden and Botting 1999, Shriberg et al. 1999, Tomblin and Zhang 1999).

SLI has been regarded as evidence for the modularity of the developing cognitive system, as it demonstrates that one can have selective and severe impairments in the domain of language despite apparently adequate non-verbal intellectual development. A great deal of research effort has focused on documenting the underlying nature of language deficits in SLI, with researchers divided between those who attribute the problems to impairment of a specialized language-learning module, and those who propose impairments of more general perceptual or learning systems as the primary cause (Bishop 1997, Leonard 1998). In contrast, language impairment in DS has attracted less research attention. It might be speculated that it was assumed that the verbal difficulties are an inevitable consequence of the physical characteristics and cognitive delay associated with this syndrome. However, relatively small contributions to language variation in DS have been attributed to cognitive ability, hearing status, speech difficulties, home environment or socio-economic factors (Wulpert et al. 1975, Miller 1988, Chapman et al. 1991, Rondal 1995). Also, other syndromes that lead to equivalent levels of non-verbal cognitive delay are not characterized by such severe language deficits—Williams syndrome is the clearest example, with several studies drawing explicit comparisons with DS (e.g. Harris et al. 1997, Mervis and Bertrand 1997, Klein and Mervis 1999, Mervis and Robinson 2000, also Lenneberg 1967, Tew 1979, Bellugi et al. 1988, Rondal 1995, Rondal and Edwards 1997, Abbeduto et al. 2001b). It is reasonable to conclude that language deficits in DS are not attributable to the learning disability.

In explaining the reasons for language problems in DS, it is important to consider the range as well as the average level of language skills. There is substantial unexplained variation in language development in DS, so that the chromosome abnormality is insufficient in itself to account for language impairment. It is necessary to look beyond the diagnosis of DS to establish the biological and environmental determinants of language difficulties in this population.

The review begins by presenting evidence for parallels in the language deficits seen in DS and SLI. Many researchers have argued that the study of individuals following atypical pathways of development can inform one’s understanding of normal development (e.g. Lenneberg 1967, Cicchetti 1984, Rondal and Edwards 1997, Nadel 1999, Abbeduto et al. 2001a). The usual approach is to focus on the dissociation of functions in groups with different genetic syndromes (Tager-Flusberg 1999, Dykens and Hodapp 2001). It is argued that it could be informative to extend this approach to a comparison of language pathology in otherwise normal development with what could be the same pathology combined with a known
genetic disorder such as DS. Observation of connections between different aspects of the language system, or between language and other domains, where these are developing under different conditions, could help clarify the nature of functional relationships. It could also provide more compelling evidence for causal connections between language impairment and underlying capacities or processes than can be provided by observations under just one condition.

A further motivation for comparing the nature of language impairment in these two populations is the question of whether phenotypic similarities between DS and SLI could reflect neurological and/or genotypic commonalities between these disorders. Over the past decade there has been mounting evidence for a substantial genetic contribution to SLI (for a review, see Bishop 2001a), but little is known about which genes are implicated and how they influence brain development. It is plausible that different genetic mechanisms in DS and SLI lead to similar neurological consequences, and hence affect language learning. A more radical possibility is that trisomy in DS affects the expression of genes that normally influence rate of language acquisition, and thus by studying genetic variation associated with language ability in DS one might gain understanding of genetic influence on SLI. This is an important question for the future, but before it can be addressed, it is one that first requires careful comparison of the language phenotypes associated with these conditions.

Language in DS and SLI: a comparison

Before embarking on a comparative review of these disorders, it is necessary to grapple with the issue of heterogeneity in SLI. Unlike DS, which can be defined in terms of objective physical criteria, SLI is diagnosed purely in terms of behavioural characteristics. Furthermore, this is largely a diagnosis by exclusion: many of the defining characteristics state what is not wrong with the child, rather than describing positive diagnostic features. It is perhaps not surprising to find that children who are included under the umbrella of SLI can be quite diverse in presentation. Before one can ask whether the language characteristics of SLI are similar to those in DS, it needs to be stated what kind of SLI is of concern. This is complicated by the fact that although most experts agree that SLI is heterogeneous, there is little consensus about how it should be subclassified. In general, in arguing for similarities between SLI and DS, focus will be on what might be regarded as ‘typical’ SLI, in which the most obvious difficulties experienced by the child are with structural aspects of language, i.e. phonology and syntax (Conti-Ramsden and Botting 1999). While accepting that boundaries between subtypes of SLI are difficult to draw, and most research samples contain a mixture of children, conclusions are not expected to apply to those children who have disproportionate difficulties with social use of language (so-called pragmatic language impairment; Bishop 2000).

Table 1 lists four characteristics of typical SLI. The following sections describe each of these and consider how far each applies also to DS.

Language delay in relation to non-verbal ability

Poor language skill in relation to non-verbal ability is a defining feature of SLI. Because children with DS function at an overall level comparable with that of
a much younger child, it was for many years not appreciated that they usually had disproportionate problems with language. However, several studies clearly demonstrate that when abilities are considered in terms of developmental level (i.e. the age at which a normally developing child would achieve that skill), spoken language lags behind non-verbal ability (e.g. Chapman et al. 1991, 1998). Expressive language is commonly assessed by mean length of utterance (MLU), a measure based on the total number of words, or number of words plus additional morphemes (e.g. verb inflections), produced in a conversational speech sample or narrative sample (Brown 1973). Expressive language deficits are indicated in DS and in SLI by lower MLUs than would be expected based on chronological age (CA) or mental age (MA). The MLUs of individuals with DS are also significantly lower than those of MA-matched controls with other intellectual impairments (Rosin et al. 1988).

Implicit in the term 'language delay' is the notion that language acquisition is on a normal developmental trajectory, but proceeding at an unusually slow rate. If one takes ‘delay’ literally, it might be expected that the same endpoint of competence would be reached, but at a later age. Although some language experts have cited DS as exemplifying delayed rather than deviant language development (e.g. Lenneberg 1967), systematic studies suggest that neither SLI nor DS can be so simply conceptualized. Delayed onset of language is a consistent feature of SLI (Leonard 1998), but outcomes for children are variable. In some cases, language does seem to be simply delayed, and problems resolve over time (Bishop and Edmundson 1987a, Bishop and Adams 1990, Thal et al. 1991, Whitehurst et al. 1991b, Paul 1993), particularly those predominantly affecting speech (Griffiths 1969, Hall and Tomblin 1978, Bishop and Adams 1990). However, there are poorer outcomes for children with more severe problems, especially when these include comprehension difficulties (Bishop and Adams 1990), or where there is a family history of language impairment (Whitehurst et al. 1991a). These difficulties can continue into teenage years (Aram et al. 1984, Johnson et al. 1999) and early adulthood (Tomblin et al. 1992). Even when overt language problems have resolved, residual evidence for them can persist as phonological memory deficits (Bishop et al. 1996a), poor phonological awareness (Goulandris et al. 2000) or reading disability (Stothard et al. 1998, Bishop 2001b).

There is also delayed onset of language for many children with DS (Fowler 1990), although some children start to talk at the time expected for typical children (Chapman 1995, Berglund et al. 2001). Some research shows that rates of progress are uneven and often interrupted in middle childhood by developmental plateaux (Fowler 1988). These might apply more to grammar than to vocabulary acquisition (e.g. Laws and Gunn 2004). However, although language status can remain fixed for some time, other evidence points to further progress in language development in late adolescence or early adulthood (Chapman et al. 2002, Fowler 1995). There is

Table 1. General descriptions applicable to SLI

<table>
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<th>Description</th>
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<td>Language acquisition is delayed in relation to non-verbal ability</td>
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<td>Language impairment cannot be accounted for by known medical or environmental causes</td>
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<tr>
<td>Expressive language is more severely affected than receptive language</td>
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<td>Grammatical components of the language system tend to be most vulnerable to delays and difficulties</td>
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wide variation in individual language progress and the levels of language skill eventually reached. Some exceptional individuals with DS achieve almost normal language (Rondal 1995), but Fowler (1990) reports that the majority achieve levels of language production that would be expected of children under three years. There is a somewhat brighter picture for language comprehension, which is less severely affected than expressive language (see below). However, although receptive vocabulary is often commensurate with, or in excess of non-verbal abilities, grammar understanding is below the level that would be expected based on non-verbal ability.

Another implication of ‘delay’ is that the profile of language skills is equivalent to that of a younger, normally developing child. In other words, the onset of language may be delayed but thereafter follows the same developmental pathway. Although many linguistic features of the language produced by children with SLI are consistent with MLU (Leonard 1998), some aspects of language differ in comparison to MLU-matched controls. For example, a more limited range of verbs may be used (Watkins et al. 1993), and there may be significant differences in the correct use of certain grammatical morphemes (Rice and Wexler 1996). These linguistic features illustrate an essential difference between children with SLI and younger children with the same MLU, suggesting that the former are not simply following a slower version of the normal developmental pathway. A similar picture emerges from research on DS, with limited use of verbs (Hesketh and Chapman 2000), and evidence for specific morphological deficits (e.g. Chapman et al. 1998, Eadie et al. 2002, Laws and Bishop 2003). The acquisition of morphological features in SLI and DS will be described further below.

Lack of association with medical or environmental causes

When diagnosing SLI, one excludes children for whom there are medical or environmental factors that could account for the language impairment. In contrast, there is usually a host of risk factors that could plausibly affect the language development of children with DS, including hearing loss (both conductive and sensorineural), and structural and neurological impairments of the speech apparatus. In addition, some people with DS are raised in relatively unstimulating institutional environments; perhaps, more so in the past. Given these factors, it could be concluded that it is hardly surprising that language is poor in DS. However, this conclusion does appear misguided, given research that shows that medical and environmental risk factors cannot account for all the variance in language skills in DS.

Hearing loss is perhaps the most obvious potential cause of language impairment in DS. Between 40 and 85% or more of individuals have impaired hearing (Dahle and McCollister 1986, Davies 1996, Roizen 1997), with severe impairments in 10–15% (Marcell and Cohen 1992, Marcell 1995, Davies 1996). In younger children, the problem is generally attributed to conductive hearing loss associated with glue ear and frequent middle ear infections (e.g. Cunningham and McArthur 1981). However, using auditory brainstem responses, Roizen et al. (1993) recorded nearly as many children with sensorineural losses as with conductive losses, and a similar number presented a combination of these problems. Other reports suggest that sensorineural losses appear in late adolescence (e.g. Widen et al. 1987). These losses may become worse in middle age (Evenhuis et al. 1992).
Evidence for the effect of hearing loss on language development in DS is inconsistent. Some research finds no significant correlation between these functions (e.g. Miller 1988, Marcell 1995, Jarrold and Baddeley 1997). Other research reports that only a small percentage of the variability in language performance can be attributed to hearing loss. For example, Chapman has reported hearing loss to account for between 4 and 7% of the variance in adolescents’ grammar comprehension scores (Chapman et al. 1991, 1998). However, individuals with more than mild hearing loss had been excluded from this sample. Laws (2004) found that although there was no significant correlation between average hearing threshold and MLU in a group of adolescents with DS, hearing did discriminate between participants who produced an intelligible narrative from which MLU could be derived and those excluded from the analysis because narratives were unintelligible. In the same sample, hearing threshold was also related to variation in receptive and expressive vocabulary scores, but not to grammar comprehension (Laws and Gunn 2004). Hearing loss clearly contributes to language outcomes in DS but, since individuals with good hearing nevertheless have language impairment, it is fair to say that language deficits in DS cannot be attributed to poor hearing.

Poor language-learning environment is an exclusionary factor in the diagnosis of SLI, but it is not so easy to rule out in the case of DS. Historically, indirect evidence for environmental effects on language in DS comes from comparison of early studies of institutionalized children compared with later studies focusing on children raised at home. Not unexpectedly, developmental outcomes for earlier cohorts were less favourable than for later cohorts raised in families (e.g. Shotwell and Shipe 1964). More recently, improved health care and education mean that one’s expectations of the developmental potential of people with DS are now higher than in the past (Wishart 1998). Although it is not yet clear what effect this may have on language development, a selective approach to the literature has been adopted in this review, concentrating on studies of more recent cohorts (for reviews of language development in DS, see Gibson 1978, Gunn 1985, Miller 1987, 1988, 1999, Mervis 1988, Dodd and Leahy 1989, Fowler 1990, 1995, Chapman 1995, 1997, Chapman et al. 1991, Rondal 1995, Gunn and Crombie 1996, Stoel-Gammon 1997, Tager-Flusberg 1999).

Expressive versus receptive language

Although it is traditional to make a distinction between expressive and expressive–receptive forms of SLI (World Health Organisation 1992, American Psychiatric Association 1994), Bishop (1979) argued that most children with SLI can be shown to have some level of receptive impairment when appropriate tests are used. Nevertheless, it is usually the case that expressive problems are more marked than receptive difficulties. This is clearly shown when studies report composite measures based on standard scores (e.g. Montgomery 2000, McArthur and Hogben 2001). Early expressive language ability is also an important predictor of later language outcomes in children with SLI (Bishop and Edmondson 1987a, Davison and Howlin 1997, Botting et al. 2001).

The picture of disproportionately poor expressive language is mirrored in DS (Gibson 1978, Cunningham et al. 1985, Miller 1988, 1999, Mundy et al. 1988,
Beeghley et al. 1990, Chapman et al. 1991, 1998, Dykens et al. 1994, Fowler et al. 1994, Casselli et al. 1998, Laws and Bishop 2003). Since DS is recognized from birth, the divergence of expressive language from comprehension can be charted from an early age. Miller (1999) has studied the longitudinal development of language comprehension and production skills relative to other cognitive abilities for young children with DS using parent reports (MacArthur Communicative Development Inventories; Fenson et al. 1993). These studies show that language production fails to keep pace with increases in MA, but language comprehension is closer to the level expected for MA. After 37 months MA (about half CA), the language production skills of 92–100% of the children lagged general cognitive ability by at least 6 months.

The gap between production and comprehension continues to grow throughout childhood and adolescence (Chapman et al. 1991, 1998), and expressive language continues out of line with cognitive impairment. MLU is significantly lower than mental age-matched controls with other intellectual impairments (Rosin et al. 1988). As for children with SLI, there are also differences between individuals with DS and younger typically developing controls with the same MLU. However, unlike children with SLI, these comparisons are not all unfavourable since individuals with DS often have larger vocabularies than younger MLU-matched controls (e.g. Harris 1983). Laws and Bishop (2003) directly compared the language profiles of a group of adolescents with DS with those for MA-matched groups of younger children with SLI and typically developing children. The groups with DS and SLI had very similar language profiles. In both language-impaired groups, expressive language was more severely affected than language comprehension relative to the typically developing group.

### Grammatical difficulties

In both populations, asymmetries in the growth of different aspects of language are manifested as specific weakness in grammar combined with relative strength in vocabulary (SLI: Aram et al. 1984, Gopnik 1990, Tomblin and Zhang 1999, DS: Fowler 1990, 1995, Chapman et al. 1991, Chapman 1995, Rondal 1995, Gunn and Crombie 1996, Tager-Flusberg 1999, Laws and Bishop 2003). Tomblin and Zhang (1999) screened over 7000 US kindergarten children, classifying those scoring more than 1.25 SD below children of the same CA on at least two composite language scores as language impaired. These children had non-verbal IQs above 87 and no additional developmental problems. They were compared with control children who passed the language screening, and to another group that also had low language scores but were in addition more generally delayed. Tomblin and Zhang (1999) found a composite grammar score, based on tests of expression and comprehension, was most likely to be failed, and that very few children had unaffected grammar alongside low vocabulary and narrative scores. Similar patterns of performance in children with low IQ were obtained.

DS research indicates a similar pattern of specific strengths and weaknesses relative to general cognitive ability (Fowler 1990, 1995, Chapman 1995, Rondal 1995, Gunn and Crombie 1996), and relative to the performance of individuals with other syndromes (e.g. Harris et al. 1997, Tager-Flusberg 1999, Abbeduto et al. 2001b). Deficits in morphological development are indicated by significantly less
use of free grammatical morphemes than MLU matched controls, and less reliable use of bound morphemes (Chapman *et al.* 1998). The most severely disadvantaged aspect of language for people with DS seems to be the production of complex syntactic structures (Chapman *et al.* 1998), although recent research suggests that the content of sentences may be in line with MLU (Thordardottir *et al.* 2002). The acquisition and use of grammar is poorer than would be predicted from non-verbal abilities (Fowler 1990, Chapman 1995, Abbeduto *et al.* 2001b), but receptive vocabulary may be better than cognitive abilities would predict (e.g. Chapman 1995), and can exceed that of non-disabled children of comparable MA or MLU (Barrett and Diniz 1989). Although morphosyntactic development may reach a ceiling, continued vocabulary growth results in a gradual divergence of vocabulary from grammar knowledge with CA (Chapman *et al.* 1993, Fowler 1995, Laws and Gunn 2004). Fowler (1995) reported that her entire sample of 31 adolescents had higher levels of vocabulary than grammar understanding. Although few studies comment on individual profiles, group means usually indicate significant differences between levels of receptive vocabulary and grammar comprehension (Miller 1987, 1988, Rosin *et al.* 1988, Fowler 1990, Chapman *et al.* 1991, Chapman 1995, Laws 1998, Laws and Bishop 2003, Laws and Gunn 2004).

In Laws and Bishop’s (2003) direct comparison of language profiles in DS and SLI, there was marked dissociation between grammar understanding and receptive vocabulary in both populations, relative to controls. Whereas grammar was poor, receptive vocabulary scores did not differ significantly from those of the typically developing comparison group. This was the pattern of results for expressive language scores as well as language comprehension scores. However, there is some question about whether grammar comprehension is as severely affected as its expression in other studies of SLI or DS. For example, although the composite grammar score reported in Tomblin and Zhang’s (1999) study of SLI indicated an overall weakness in grammar, separate test scores suggested that grammar comprehension as well as vocabulary comprehension was a relative strength for the language delayed children, and that any weakness in grammar was associated with expressed language. However, this may not be the case for all children with SLI. Other studies show that children can have difficulties in understanding grammatical constructions as well as in making correct use of them in speech (Bishop 1979, 1982, Adams 1990, Van der Lely and Stollwerck 1996). These differences between study findings could be related to different means of ascertainment or to different assessment instruments. The criteria employed by Tomblin and Zhang (1999) would have identified some children with simple language delay whose problems would resolve and who would not go on to develop SLI. Children with longer lasting problems of SLI do have grammar comprehension difficulties more often.

In DS, most studies report levels of grammar comprehension below those of non-verbal ability (Bridges and Smith 1984, Rosin *et al.* 1988, Buckley 1993, 1995, Laws 1998, Laws *et al.* 2000, Vicari *et al.* 2000, Laws and Gunn 2004). Chapman *et al.* (1991) reported grammar comprehension in line with non-verbal ability, but this was probably because the non-verbal measure incorporated a test of short-term memory, itself a core deficit associated with the syndrome. Chapman (1995) suggests that it is only in adolescence that syntax comprehension begins to fall behind that of MA controls. However, characteristics of study samples other than
CA may be important. Since there is a higher ceiling on non-verbal ability than on grammar comprehension, findings may depend on the proportion of a sample achieving high non-verbal scores. The higher the reported mean non-verbal score for a research sample, the less likely it is that the age equivalent score for grammar comprehension will equal it. It is true that this situation will more often apply to older study groups, but other factors such as school placement might also influence findings (e.g. Laws et al. 2000).

Morphological development

Rice and Wexler (1996) have argued that clinical observation of the use of grammatical morphemes provides a more sensitive test of SLI than a consideration of language profiles. Late acquisition of grammatical morphemes is characteristic of SLI (Johnston and Schery 1976, Khan and James 1983, Johnston and Khami 1984, Leonard et al. 1988, Bliss 1989, Marchman et al. 1999). Children with SLI do not find all grammatical morphemes difficult to acquire (Rice et al. 1998), but there is regularity in the morphemes that children do find difficult, and these tend to be the ones that young normally developing children are slow to use consistently. A period of inconsistent usage of correct grammatical forms in normal development is referred to as an ‘optional infinitive’ stage and, because children with SLI are so slow to move out of this stage, Rice and colleagues refer to an ‘extended optional infinitive’ stage. For example, there is little variability in the use of verb tense markers by normal 5-year-olds, but children with SLI struggle to acquire correct use of the past tense and third person singular ‘s’, and this difficulty can be persistent (Rice and Wexler 1996, Rice et al. 1998, Van der Lely and Ullman 2001, Conti-Ramsden and Windfuhr 2002). The problem does not appear to be due to poor perception of speech sounds for these morphemes. Some reports find that third person singular ‘s’ is affected, but children have no apparent difficulty in acquiring plural ‘s’s, which should be no more easily perceived (Oetting and Rice 1993).

The sensitivity and specificity of tense marking as a test to identify children with language impairments has been demonstrated in studies to compare the performance of 5-year-old children with SLI to CA-matched controls. When children who used correct tense markers less than 80% of the time were classified as disordered, 97% of cases of SLI were correctly identified, and 98% of controls (Rice and Wexler 1996). Poor performance by children with SLI persisted up to 8 years of age, and although there was some improvement, there was no evidence that they caught up controls (Rice et al. 1998). Further, children with SLI are poor at identifying, as well as using, correct tense markers (Rice et al. 1999b). Rice (2000) suggests that these properties of the tests for grammatical markers, together with the poor relationships found between them and environmental factors such as mother’s education (Rice et al. 1999a), make them strong candidates for identifying cases of SLI for genetic study. However, note that when past tense and noun plural tasks have been used with children with SLI under 5-years-old, they have not performed as satisfactory clinical risk markers (Conti-Ramsden and Hesketh 2003).

The acquisition and use of morphemes has been studied in DS. As in SLI, there is some evidence that particular morphemes may be more difficult to acquire than others (Rondal and Lambert 1983, Sabsay and Kernan 1993, Rutter and Buckley 1994, Chapman et al. 1998, Eadie et al. 2002, Laws and Bishop 2003). Rutter and
Buckley (1994) investigated the acquisition of fourteen morphemes by twelve young children with DS, by asking parents to record monthly diaries over two to three years. After taking into account the delayed production of first words by the children with DS, the children's acquisition of most of these morphemes was within range of the three typically developing children studied by Brown (1973), although they were not acquired in the same order. However, three rules were never grasped by any of the children: the uncontractable copula 'be' form (e.g. 'he is' in response to 'who's there?'), the uncontractable auxiliary 'be' form ('he is' in response to 'who's coming to the party?'), and third person singular 's' for present tense verbs. Irregular third person present tense (e.g. 'Mummy has' or 'Mummy does') was acquired by just one child. These rules are also reported as difficult for children with SLI.

Chapman et al. (1998) reported that regular past tense 'ed' as well as plural 's', noun possessive 's', third person singular 's', contractible auxiliaries and copulas, and present progressive 'ing' were frequently omitted from the language produced by their sample of 5–20-year-olds with DS. However, many of these individuals had MLUs below 4.5, the stage at which these bound morphemes are expected to appear in normal development. Some adolescents with DS with MLU above 4.5 may make more normal use of third person singular and past tense verbs (Laws and Bishop 2003). Other research involving adults with DS shows persistent use of telegraphic speech, with poor use of verb tense inflections, and few articles, prepositions, pronouns, copulas, or conjunctions (Rondal and Lambert 1983). Sabsay and Kernan (1993) found that, unlike adults with intellectual disability of unknown origin, adults with DS fail to master the use of auxiliary verbs, suggesting that the difficulties are a feature of DS rather than of intellectual impairment more generally. Eadie et al. (2002) directly compared the acquisition of morphemes by MLU-matched groups of children with SLI, DS and typically developing children. Although both language-impaired groups were limited in their acquisition of specific morphemes relative to the comparison group, they did not resemble each other in the patterns of morpheme use. Laws and Bishop (2003) noted that differences in the morphological difficulties of each group were possibly related to the stronger vocabulary skills of the DS group in the study.

Table 1 described some general features of SLI. The above sections expanded on these descriptions for SLI and investigated the extent to which they apply also to DS. It is argued that these comparisons show that the language characteristics of these conditions are very similar in terms of surface descriptions. Of course, similar surface descriptions do not necessarily mean that the impairments arise from the same underlying causes. It is possible that common profiles of language development occur because the affected components are simply those most vulnerable to any sort of risk factor, and that DS is just an additional, if powerful, risk factor. Such an account would not necessarily mean that people with DS should also resemble those with SLI in terms of deficits to underlying capacities or processes. An alternative account could be developed around the idea that people with DS have language profiles similar to SLI because they are prone to the same underlying difficulties hypothesized to cause SLI, whether these are biological or environmental in origin. The next step in this enquiry is to consider whether the similarities observed are superficial or whether they could be explained by similar deficiencies in underlying capacities or processes.
Implications for understanding of processes involved in language acquisition

Consideration of underlying causes bears on the more fundamental question of which theoretical account can best explain language impairment. An emphasis on poor grammatical development in SLI, and the background to the tests of morphology described above, arises from a linguistic deficit theory. Given the importance of difficulties in acquiring specific grammatical morphemes in this account of SLI, it could be important to establish that the same morphemes pose difficulties for people with DS if the pathology is to be shown to mirror SLI. A more general difficulty with the acquisition of morphology could indicate a different type of disorder, or perhaps one that arises from one or more different underlying problems, such as hearing impairment. Despite the evidence for morphological deficits described above, more direct comparisons of the linguistic features of DS and SLI may be necessary to be confident about a correspondence between them at this level.

The linguistic-deficit account contrasts with more psychologically based accounts which view language impairment as secondary to underlying processing deficits. Table 2 lists hypotheses that have been advanced to explain SLI in terms of underlying processes, including auditory processing deficits, poor phonological memory skills, motor development delays and atypical cerebral specialization. Given heterogeneity in the manifestation of SLI it seems unlikely that a single explanation will suffice, and it is also likely that deficits in underlying processes differentially affect the various components of language. The differential effects of multiple causes on multiple components of a language system could explain the heterogeneity evident in the language profiles of people with SLI (Bishop 2001a).

Modular approaches to understanding language treat subdomains of language as separate components, depending on separate neurobiological substrates. This approach distinguishes between computational and conceptual aspects of language (e.g. Chomsky 1980, Tager-Flusberg 1997). The computational component includes the hierarchical processes associated with phonological and syntactic development, and is distinguished from a conceptual component on which depend lexical and semantic aspects of language. These components may be differentially affected by deficits in underlying capacities, and be more or less vulnerable to genetic and environmental risks. Chomsky (1986) suggested that biological constraints are more important in limiting syntactic development, but that environment may be more influential in determining other aspects of language such as the lexicon. For example, increases in vocabulary depend on exposure to experiences provided by the environment. Given intact biological functions to support vocabulary learning, variation in the numbers of words acquired is likely to depend on experience. In contrast, if biological deficits impose the major limitation on syntactic development.

### Table 2. Underlying processes associated with SLI

| Deficits in auditory processing ability |
| Poor phonological short-term memory |
| Deficits in motor development |
| Atypical patterns of cerebral specialization |
then poor syntactic development could be the outcome whatever the environment provides.

In normal development, language acquisition proceeds in an integrated fashion, and in line with non-verbal cognitive abilities, resulting in flat developmental profiles. When something goes wrong, the dislocation of functions can result in the growth of uneven profiles. Uneven language profiles are a feature of many genetic syndromes and disorders, and contrasting profiles based on dissociations of functions have been established between conditions (e.g. Tager-Flusberg et al. 1990). As argued herein, the language profiles in DS and in typical SLI show marked impairment of computational processes alongside relatively intact conceptual aspects of language. If there should be similar patterns of strength and weakness in terms of underlying capacities or processes, and these can be associated with the same aspects of language development in both conditions, this would provide further evidence for a common language impairment.

**Auditory processing deficits**

Clearly, any disruption of normal processing of language sounds could be expected to affect language development. One major difference between SLI and DS is that whereas hearing problems are excluded as a cause of SLI, hearing losses are common in children with DS (see above). However, defective peripheral hearing is not the only way in which auditory processing can be disrupted and major efforts have been devoted to identifying defects in higher level auditory processes to account for SLI (e.g. Tallal and Piercy 1973a, Tallal 1976, Tallal et al. 1981, Neville et al. 1993, Wright et al. 1997, McArthur and Hogben 2001). Whether difficulties lie in the perception of sounds by the ear or in the processing of speech sounds by the brain, the effects on language outcomes could be similar. Despite different histories in terms of hearing loss, it is reasonable to compare the two populations in terms of possible relationships between auditory processing and language development.

An auditory temporal processing deficit in SLI is said to result in impaired discrimination of rapid or briefly presented non-speech sounds, even when there is no problem differentiating the same stimuli presented more slowly or for longer duration (e.g. Tallal and Piercy 1973a, b). It is argued that this deficit makes it difficult for children to discriminate between consonants that differ by just a brief part of the acoustic signal, and leads to problems in acquiring the phonology essential to the development of spoken language (Tallal 2000). Despite extensive experimental evidence to support this hypothesis, there are many contrary reports and conflicting evidence arising from methodological problems associated with the experiments and from individual differences within samples of people with SLI (McArthur and Bishop 2001).

While it is possible to speculate that similar problems could account for slow acquisition of phonology in DS, attempts to study higher level auditory perception in this population are problematic due to difficulties in explaining the tasks to participants, and the fact that the tasks can impose a memory load beyond the capacity of many people with DS. Further, since hearing losses are common in DS, it could be difficult to differentiate high level processing effects from peripheral deficits. However, Marcell and Cohen (1992) and Marcell (1995) used a procedure involving backward masking to assess the identification of words by people with DS. Presentation of stimulus words was followed by a burst of white noise.
presented after intervals of varying duration. The task is analogous to one used to detect deficits in auditory temporal processing in SLI where backward masking is used to manipulate the time available for the detection of a non-verbal stimulus tone (e.g. Wright et al. 1997). Children with SLI have higher thresholds for detecting a tone than control children, i.e. to make a correct response, they require to hear more of the tone before the masking noise appears. Marcell (1995) found that adolescents with DS identified fewer words than adolescents from a matched group with other intellectual impairments when the words were followed by a masking noise after 40 ms. They did not differ from the comparison group when the masking noise appeared after 320 ms, giving them longer to identify the word. However, much of the difficulty with the 40 ms condition could be attributed to hearing losses. Individuals with bilateral hearing losses identified significantly fewer words than a subgroup of adolescents with DS with normal hearing in one or both ears. Poor hearing was not a factor in the identification of words in the 320 ms condition. Marcell and Cohen (1992) suggest that the poor performance of the hearing impaired group in the 40 ms condition might be attributed to the combined effects of low auditory acuity and slow processing speed. Clearly, further experiments would be required to establish whether people with DS have the same kind of auditory temporal processing deficit that is thought to be a feature of SLI.

Phonological memory deficits

A more established similarity between DS and SLI is the occurrence of poor phonological memory, or memory for speech sounds. Phonological memory is thought to be functionally important for the normal development of language (Gathercole and Baddeley 1993, Baddeley et al. 1998), and language development is slower in children with poor phonological memory (e.g. Gathercole et al. 1992). This relationship has been studied extensively by Gathercole and her colleagues using non-word repetition, a task which assesses the accuracy with which unfamiliar but word-like phoneme sequences such as ballop or blonterstaping are repeated. The task may reflect the role of phonological memory in mediating the development of long-term representations as the lexicon grows. Phonological memory may also be important for the acquisition of grammar by allowing multiword utterances to be maintained while the long term representations of abstract syntactic rules are constructed (Speidel 1989, 1993, Baddeley et al. 1998). This process allows children to acquire templates of syntactic constructions used in adult speech that can be drawn on to support their own spoken output.

Children with SLI find the non-word repetition task difficult, especially when asked to repeat long non-words (Kamhi and Catts 1986, Gathercole and Baddeley 1990, Bishop et al. 1995, 1996a, Montgomery 1995, 2000, Dollaghan and Campbell 1998, Edwards and Lahey 1998, Ellis Weismer et al. 2000, Conti-Ramsden and Hesketh 2003). Bishop et al. (1996a) administered the task to twins, at least one of whom had experienced speech or language disorder in the past. Non-word repetition was impaired even for individuals whose language problems had resolved. This could indicate that these individuals had been able to compensate in some way for poor phonological memory, and so achieve normal language skills, but that the underlying phonological memory problem remained. In this interpretation of results, poor phonological memory is the cause of poor language,
rather than a consequence of language problems. Conti-Ramsden and Hesketh (2003) found that poor non-word repetition was an accurate risk marker for SLI in young children, but cautioned that more research was necessary before any causal relationship between poor phonological memory and language impairment could be established.

Phonological memory has also been described as a selective deficit in DS (Varnhagen et al. 1987, Hulme and Mackenzie 1992, Kay-Raining Bird and Chapman 1994, Wang and Bellugi 1994, Fowler et al. 1995, Vicari et al. 1995, Jarrold and Baddeley 1997), and there is some evidence of a relationship between this deficit and language. Vallar and Papagno (1993) described a case of a young Italian woman with DS with good vocabulary who, unusually for a person with this condition, also recorded a digit span within the normal range. Although this might indicate that her good language was a consequence of spared short term memory, other studies report only a weak correlation between phonological memory and receptive vocabulary, compared with that found in normal development (Hulme and Mackenzie 1992), and some studies report no relationship (Marcell 1995, Jarrold and Baddeley 1997, Chapman et al. 2002). However, these researchers assessed phonological memory using digit span. When non-word repetition has been used, significant correlations have been established between phonological memory and language development (Laws 1998, 2004, Laws and Gunn 2004). In a 5-year follow up study of 10–24-year-olds with DS, earlier phonological memory scores predicted later receptive vocabulary and grammar comprehension (Laws and Gunn 2004). The expressive language abilities of this sample were also assessed at the end of this study. Fifty per cent of the variation in MLU and sentence recall was attributed to non-word repetition scores, even after allowance was made for the effects of CA, MA and word repetition (Laws 2004). Some researchers have argued that this relationship is due to the common demands placed on output processes by non-word repetition and expressive language tasks (e.g. Snowling et al. 1991, Van der Lely and Howard 1993). If this was the case, the correlation between verbal memory and expressive language should be abolished if a memory task is used which has no spoken language requirement (Adams and Gathercole 2000). However, just as Adams and Gathercole reported for typically developing children, Laws found that a verbal memory span task which required a pointing response was as strongly related to MLU and sentence recall as non-word repetition.

Interestingly, phonological memory has been linked not only to the language phenotypes of SLI and DS but also to a genetic background in SLI. Bishop et al.'s (1995) analysis of twin data described earlier showed non-word repetition to be a useful phenotypic marker for inherited language impairment; MZ (identical) co-twins resembled each other more closely than DZ (non-identical) co-twins. A more recent twin study replicated this finding of high heritability for non-word repetition (Bishop et al. 1999). In this study, Tallal's repetition test was also administered (Tallal and Piercy 1973a), so that memory for speech sounds could be compared with memory for non-verbal tones. The results showed that although phonological memory was under strong genetic influence there was no genetic influence on non-verbal auditory deficits. Instead, deficits in memory for tones were more subject to environmental influence. Since the children most affected by SLI in this study were those who showed evidence of both deficits, Bishop et al. (1999) concluded that the deficits act as independent risk factors and are most likely to cause SLI when they occur together. This could explain the high levels of language impairment in the
DS population where probably a large majority of individuals have co-occurring auditory processing deficits and phonological memory deficits.

**Association with deficits in motor development**

Another factor worth exploring as a potential link between language impairment in SLI and DS is motor delay. Poor motor skills are often a problem for children with SLI (Hill 2001), and several studies have identified relationships between motor delay and language (e.g. Johnston et al. 1981, Bishop and Edmundson 1987b, Moore and Law 1990, Schwartz and Regan 1996, Owen and McKinlay 1997, Bishop 2002). Although some relationship between fine motor skill and speech might be expected, on the basis that both depend on systems for motor programming and control, the interesting thing about some of these studies is that motor skills have been related to language comprehension. For example, Schwartz and Regan (1996) found that fine motor tasks requiring rapid timing and repetitive sequencing were strongly related to language comprehension measures in 4–8-year-olds with SLI. Some evidence that this association can be linked to heritable factors was provided by Bishop (2002) who studied motor skill in the sample of twins described earlier (Bishop et al. 1995, 1996a). Motor skill, assessed using a simple tapping task, was significantly related to speech impairment but children with pure language impairments and unaffected speech also had motor deficits. There was evidence for strong genetic effects on speech and language impairments and on motor skill, and evidence that motor skill and language impairment (but not speech problems) shared a common heritable factor.

Unfortunately, although delayed motor development is an invariable feature of DS, there is little research to investigate relationships between motor skill and language development. Studies of children with DS show that motor development follows a normal sequence but remains below the level expected for MA (e.g. Frith and Frith 1974, Connolly et al. 1993, Reid and Block 1996, Jobling 1998, Torres and Buceta 1998). There is some evidence to link motor skill with vocal features. As in typical development, there is synchrony in the onset of canonical babbling, that is infants’ repetition of well-formed syllables such as ba, da and ma, and rhythmic hand banging. A similar association in seen in infants with DS (Cobo-Lewis et al. 1996). Cobo-Lewis et al. suggest that these rhythmic behaviours may be underpinned by common neuromuscular systems. In adulthood, there is poor motor performance in those who stutter compared with those with fluent speech (Devenny et al. 1990), and greater speech dysfluency in left- or mixed-handed adults (Devenny and Silverman 1990). However, there is limited information about whether individual variation in the degree of motor difficulties accounts for variation in language competence throughout childhood. Given evidence for a common genetic basis to motor immaturity and language impairment in SLI (Bishop 2002), further exploration of the part played by motor deficits in the language development of individuals with DS would be worthwhile.

**Association with atypical patterns of cerebral specialization**

In most people, it is usual for the left cerebral hemisphere to be specialized for processing language. This arrangement is reflected in a right ear advantage for the perception of speech sounds in dichotic listening tasks. In this paradigm, different
sounds or words are presented to each ear simultaneously, but the listener generally reports only the stimulus received by the advantaged ear (e.g. Kimura 1967). Lateralization of motor function is also indicated indirectly, by the preference for using the right hand demonstrated by about 90% of the population. It has been argued that if the process of lateralization is incomplete, or if functions are organized differently, the cost could be developmental disabilities such as SLI or dyslexia (e.g. Geschwind and Galaburda 1987, Gauger et al. 1997).

Dichotic listening performance by children with SLI varies across studies, but some research finds qualitative differences in responses. Children with SLI may more often show a left ear advantage or weak right ear preference compared with typical children (e.g. Cohen et al. 1999). Although most children with SLI are right handed (e.g. Bishop 1990), quantitative assessment suggests this is only a weak preference (Bishop et al. 1996b, Hill and Bishop 1998, Bishop 2001c). This feature could reflect atypical morphological asymmetries (e.g. Jernigan et al. 1991, Plante et al. 1991) or could reflect immature neuromotor development rather than a radical difference in the brains of these children (Hill and Bishop 1998).

In DS, extensive research has been directed to understanding whether atypical organization of motor and language functions limits language development (Chua et al. 1996). These researchers have developed a model of cerebral organization that features the separation of speech and motor functions into different cerebral hemispheres. In this model, the centre for motor control is in the left hemisphere, as evidenced by a normal pattern of finger tapping performance favouring the right hand (Elliott 1985, Elliott et al. 1986). However, unlike the general population, children and adults with DS tend to display a left ear advantage in dichotic listening studies, indicating right hemisphere processing of language (Elliott et al. 1994). Separation of motor and language functions could reduce the effectiveness of communication between these systems. There is some evidence for this in that performance on tasks which require either oral or limb movements is poorer in response to verbal instruction than to visual demonstration (Elliott and Weeks 1993). Elliott and Weeks found that performance was poorest for participants who had shown a left ear advantage in dichotic listening tasks.

**Summary and implications**

The review has addressed the question of whether the language impairment in DS could be the same impairment as that found in otherwise typically developing children diagnosed with SLI. The question has been approached in terms of (1) a comparison of language profiles in the two groups and (2) a discussion of underlying deficits that could be implicated in the language impairments. Conclusions are summarized in table 3.

In terms of the phenotypes of language disorder, there is considerable evidence for similarity between the language profiles of individuals with DS and those of the majority group of children with SLI. Despite very different backgrounds to language development in terms of general cognitive ability, the main features of impairment in both populations are more severe expressive language deficits relative to levels of language comprehension, dissociation between grammatical and lexical components of the language system, and difficulties in the acquisition of morphology. Research studies which have investigated the effects that various underlying deficits might have on language impairment paint similar pictures in
Table 3. Summary of conclusions

<table>
<thead>
<tr>
<th></th>
<th>SLI</th>
<th>Down's syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Language in relation</td>
<td>Poor language relative to cognitive ability is a diagnostic criterion</td>
<td>Although vocabulary can be equal to or in advance of cognitive ability, phonology and grammar are usually poorer. Also, language is poor relative to that of people with other learning disabilities</td>
</tr>
<tr>
<td>to general cognitive</td>
<td></td>
<td></td>
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<tr>
<td>ability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Association with other</td>
<td>Hearing problems, neurological impairment and psychiatric disorder</td>
<td>Multiple potential causes, but these do not explain the substantial variation in language skill. Further research is needed to understand the effects of mild-to-moderate hearing loss</td>
</tr>
<tr>
<td>problems</td>
<td>are ruled out as a cause of language problems</td>
<td></td>
</tr>
<tr>
<td>Expressive versus</td>
<td>Expressive problems &gt; receptive problems. Early expressive abilities</td>
<td>Expressive problems &gt; receptive problems</td>
</tr>
<tr>
<td>receptive language</td>
<td>predict later language outcomes</td>
<td></td>
</tr>
<tr>
<td>Co-morbid speech</td>
<td>Proportion of children have speech difficulties</td>
<td>Speech impairment is often a feature. It is not clear how this affects language development</td>
</tr>
<tr>
<td>impairment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grammatical difficulties</td>
<td>Grammar is the most frequently affected component. Difficulties in understanding and in producing complex sentences</td>
<td>Possible ceiling on grammatical development. Difficulties in understanding and in producing complex sentences</td>
</tr>
<tr>
<td>Morphology</td>
<td>Late acquisition of grammatical morphemes. Research on whether verb tense marking is a specific deficit gives inconsistent results</td>
<td>Poor use of grammatical morphemes, but may be more normal for individuals with longer mean lengths of utterances. Patterns of morpheme use differ from specific language impairment, but only two comparative studies have been reported so far</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>Proportion of children have word-learning and word-finding difficulties.</td>
<td>Often, but not always, a strength. Evidence for continued vocabulary growth into young adulthood</td>
</tr>
<tr>
<td></td>
<td>For others, vocabulary is in line with cognitive abilities</td>
<td></td>
</tr>
<tr>
<td>Higher level auditory</td>
<td>Some evidence for deficits in processing brief or rapid sounds, and also conflicting evidence and contrary reports. Further research is necessary</td>
<td>Little available research</td>
</tr>
<tr>
<td>processing</td>
<td></td>
<td></td>
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<tr>
<td>Phonological memory</td>
<td>Evidence for difficulties in non-word repetition. Poor scores predict language impairment. Less evidence for direct correlation with language measures</td>
<td>Dissociation between phonological memory and visuospatial memory. Non-word repetition is variable, and variation is related to language abilities</td>
</tr>
<tr>
<td>deficits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor development</td>
<td>Motor delays related to language impairment</td>
<td>Evidence for motor delay. Little research to investigate an association with language development</td>
</tr>
</tbody>
</table>
both populations. This is particularly so with respect to phonological memory deficits. In terms of the effects of higher level auditory processing deficits, the impact of motor delay, and the part that atypical cerebral specialization might play in language development, there is less parallel research available from which to draw conclusions about common causes and more direct comparisons would be worthwhile in future research.

The review has been restricted to behavioural data but the implications of similar language phenotypes are that they could be the expression of similar genotypes, and perhaps reflect similar atypical brain development. Although researchers are beginning to uncover some links between genetics and language impairment, the chain of events between genes (and the chemical and regulatory processes governed by them) and a phenotype of language impairment remains mysterious. However, somewhere between genes and expressed phenotypes there lie more proximal explanations for language impairment in the form of atypical patterns of brain development or defective underlying capacities or processes which restrict language development. If language impairments in DS and SLI are of the same nature then one might find the same cascades of causes and effects linking genes to language phenotypes.

In DS, there is the possibility that the severity of disorders associated with the condition, including language impairment, could be influenced by variation in specific genetic factors on chromosome 21. For example, if there is an allele of a gene on chromosome 21 that increases the risk of a particular disease, people with DS who have an extra copy of this allele may show unusually high rates of the disease, whereas those with a different allele do not show such vulnerability. This kind of pattern has been observed for heart disease, where genetic variability in a gene on chromosome 21 is associated with the congenital heart defects observed in many individuals with DS (e.g. Davies et al. 1995). A second possibility is that trisomy 21 produces a genetic background against which risk factors from genes on other chromosomes are more strongly expressed. This pattern has been observed in the development of Alzheimer’s disease in DS, where the expression of a gene associated with the disease on chromosome 21 is modified by allelic variation in genes encoded on chromosome 19 (Corder et al. 1993, Roses and Saunders 1994, Del-Bo et al. 1997, Schupf et al. 1998).

A similar mechanism might be implicated in SLI. Suppose, for instance, that there are several alleles that act as quantitative trait loci for language impairment, i.e. they raise the probability that language will be impaired but do not have a simple one-to-one relation with SLI. The likelihood of a child having SLI will be a function of the number of risk quantitative trait loci present. It is very likely that environmental factors will also play a part, so that SLI is only seen when a high-risk genotype is exposed to a non-optimal environment (Bishop 2001a). However, the same alleles may have a much lower threshold for expression when they occur in the context of trisomy 21 because of the general disruption to developmental processes that the chromosome abnormality entails.

At present, the suggestion of shared genetic factors as background to language impairment in the two populations is speculative but is worth considering in future research. Similarly, although consideration of the research on the complex neurobiology of these conditions is beyond the scope of this review, it is worth noting two features that they have in common. First, there is some evidence of
minor abnormalities of neuronal migration in DS (Coyle et al. 1986, Wisniewski 1990, Golden and Hyman 1994) and in SLI (Galaburda et al. 1985, Clark and Plante 1998). Second, there appears to be disproportionate reduction in the size of the cerebellum and frontal lobes in DS (Jernigan et al. 1993, Raz et al. 1995, Murphy et al. 2000, Capone 2001, Pinter et al. 2001). Eckert et al. (2003) have noted similar abnormalities in the brains of dyslexic individuals, and there is suggestive evidence that the same circuits may be implicated in SLI (Jernigan et al. 1991). Although there are also many points of difference between DS and SLI at this level, future research should identify whether shared atypical features of brain development contribute to language difficulties.

**Clinical implications**

The main implication for practitioners of a similarity between language profiles in DS and SLI is that it weakens any notion that language impairment in DS should be considered as an inevitable consequence of the learning disability. If language impairment is relatively independent of non-verbal ability then, logically, therapeutic goals should not be determined by the child’s non-verbal intelligence level. In both DS and SLI, understanding the problems posed by deficits in the processes underlying language development could provide targets for intervention. If these conditions do share the same language pathology, it might make sense to transfer successful interventions from one condition to the other.

To date, interventions at the level of underlying deficits have focused on different targets in each population. In SLI, intervention has targeted auditory processing deficits, based on the theory (described above) that language deficits can be attributed to difficulties with processing rapid, successive sounds. The aim of intervention is to provide intensive training exercises to improve the discrimination and representation of sounds. The most widely offered training programme is Fast ForWord (Scientific Learning Corporation 1996), and researchers claim remarkable language gains for children with SLI following the training (e.g. Merzenich et al. 1996, Tallal et al. 1996). However, there remains the need for considerable research effort to determine which children can benefit from auditory training, which aspects of language can be improved and what the precise mechanism is for any improvement (Gillam et al. 2001). If it can be established that higher level auditory processing deficits contribute to language impairment in DS, there is the hope that auditory training might also benefit these children. However, until one understands more about the processes involved, it would be premature to offer such hope.

The main focus of intervention at the level of underlying deficits in DS has been on training short-term memory (e.g. Hulme and Mackenzie 1992, Broadley and MacDonald 1993, Comblain 1994, Laws et al. 1996, Conners et al. 2001). The rationale for memory training is based on Baddeley’s working memory model (Baddeley 1986, Baddeley and Hitch 1974), which includes a phonological loop component specialized for the short term memory of speech based material. A range of clinical and experimental evidence points to the dissociation of processes within the phonological loop so that deficits can be associated either with the storage of phonological material or with the maintenance of inputs by articulatory rehearsal. Individuals with DS may not develop spontaneous rehearsal strategies (Hulme and Mackenzie 1992) and so have difficulty in maintaining inputs (e.g. new
vocabulary items), with adverse consequences for language development. The aim of memory training studies has been to teach a cumulative rehearsal strategy. Despite the short term success of programmes in increasing memory span (e.g. Broadley and MacDonald 1993), skills have not been maintained over the longer term (Laws et al. 1995) and the impact of increasing memory span on language development is unknown. Despite evidence for phonological memory problems in SLI, there appear to be no comparable studies. However, Montgomery (2002), in a recent review of the evidence for the role of phonological memory in the language difficulties of children with SLI, has suggested teaching cumulative rehearsal alongside a range of games aimed at increasing metaphonological awareness. Comparing the effects of training interventions based on auditory processing or memory training in both populations could further understanding of the complex relationships between these deficits and general language skills, as well as potentially benefiting affected children.

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Down's syndrome and SLI


