

Relations Among Speech, Language, and Reading Disorders

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Key Words

comorbidity, speech sound disorder, specific language impairment, developmental dyslexia, genetics

Abstract

In this article, we critically review the evidence for overlap among three developmental disorders, namely speech sound disorder (SSD), language impairment (LI), and reading disability (RD), at three levels of analysis: diagnostic, cognitive, and etiological. We find that while overlap exists at all three levels, it varies by comorbidity subtype, and the relations among these three disorders are complex and not fully understood. We evaluate which comorbidity models can be rejected or supported as explanations for why and how these three disorders overlap and what new data are needed to better define their relations.

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INTRODUCTION

A fundamental question to be addressed by psychologists is how atypical development relates to typical development. An adequate theory must account for both human universals and individual differences, preferably with the same underlying mechanisms. Every example of atypical development poses both a challenge and an opportunity for developmental theory. In this review, we focus on a pervasive characteristic of atypical development, co-occurrence or comorbidity among behavioral disorders, specifically speech, language, and literacy disorders. Understanding this comorbidity has implications for the genetics, neuropsychology, prevention, and treatment of these disorders as well as for our understanding of the development of language. Historically, theorists have focused on explaining individual disorders (e.g., Morton & Frith 1995) and have viewed their co-occurrence as a peripheral issue. However, in recent years theorists have increasingly recognized that comorbidity is of interest in its own right. We argue that placing comorbidity at the

center of inquiry leads to a new perspective on theoretical models of disorders.

In this review, we first define speech, language, and reading disorders and critically review the evidence for their overlap at three levels of analysis: (a) diagnostic, (b) cognitive or neuropsychological, and (c) etiological. Because overlap exists at all three levels of analysis, we consider and evaluate which models of comorbidity can be rejected by current evidence, which are supported, and what new data are needed to distinguish among currently supported models. We conclude with a discussion of broader implications and future directions for research.

DIAGNOSTIC DEFINITIONS AND EPIDEMIOLOGY

The essential defining characteristics of the three disorders are summarized in **Table 1**. In each case, the disorder involves an unexpected difficulty in one aspect of development that cannot readily be explained by such factors as low intelligence or sensorimotor impairment. All three disorders lack a sharp dividing line between impairment and normality; thus, diagnosis involves setting an arbitrary threshold on what are essentially continua.

It has been customary in language impairment (LI) and reading disability (RD) to focus on specific developmental disorders, i.e., those where a significant discrepancy exists between language or literacy and general intelligence. However, such definitions have been criticized on both logical and practical grounds. In the field of reading disability, it is now broadly accepted that it is not valid to distinguish between children who have a large discrepancy between poor reading and IQ, and those who do not. Both types of poor reader have similar underlying deficits in phonological processing and both respond to similar kinds of treatment (see review in Fletcher et al. 1999). Analogous arguments have been advanced for LI (e.g., Bishop & Snowling 2004). Although the focus here is on children of broadly normal nonverbal ability, we use the terms RD

Table 1 Basic characteristics of language impairment, reading disability, and speech sound disorder

	Language impairment (LI)	Reading disability (RD)	Speech sound disorder (SSD)
Synonyms	Developmental dysphasia Developmental language disorder	Developmental dyslexia	Phonological disorder Articulation disorder
Defining characteristics	Expressive and/or receptive language development is impaired in the context of otherwise normal development (i.e., nonverbal IQ and self-help skills) Language impairment interferes with activities of daily living and/or academic achievement	Child has significant difficulty learning to read accurately and fluently despite intelligence within normal limits and adequate opportunity to learn	Child substitutes or omits sounds from words more than do same-aged peers; speech production errors interfere with intelligibility of speech
Exclusionary criteria	Severe neglect Acquired brain damage Significant hearing impairment Known syndrome, such as autistic disorder	Inadequate educational opportunity Acquired brain damage Significant hearing impairment Known syndrome, such as autistic disorder	Structural or neurological abnormality of articulators Significant hearing impairment IQ < 70
Prevalence	Depends on cutoff used Epidemiological study (Tomblin et al. 1997): 7.4% (CI 6.3–8.5%) of 6-year-olds met psychometric criterion	Depends on cutoff used; typical value is around 9%	2%–13% (mean = 8.2%) (Shriberg et al. 1999)
Odds ratio M:F	3 in referred sample (Broomfield & Dodd 2004); 1.5 in epidemiological sample (Tomblin et al. 1997)	1.9 to 3.3 in 4 epidemiological studies (reviewed by Rutter et al. 2004)	1.5 to 2.4 (mean = 1.8) (Shriberg et al. 1999)
Risk factors	Family history has significant effect No effect of parental education; slight effect of birth order (later-born at more risk) (Tomblin et al. 1991)	Parental education Home literacy environment Bioenvironmental risk factors, such as lead poisoning and head injuries	No effect of race, SES, or otitis media history, but significant effects of gender, family history, and low maternal education (Campbell et al. 2003)

and LI without the “specific” prefix rather than adopt a discrepancy-based definition of these disorders.

When dealing in particular with LI, the question arises as to whether the child’s socio-cultural background might affect performance on language measures and hence liability to be diagnosed with disorder. In general, that should not be the case, provided one is sensitive to the possibility that some nonstandard dialects may have grammatical and vocabulary differences from the standard. Tests of language processing, rather than linguistic knowledge, are largely immune to cultural influence and are sensitive indicators of LI (Campbell et al. 1997).

EVIDENCE FOR COMORBIDITY

Any attempt to summarize the evidence for comorbidity between speech sound disorder (SSD), LI, and RD will be tentative, not least because the prevalence of these disorders is age dependent. Clearly, RD cannot be identified until children have been exposed to reading instruction. On the other hand, SSD is typically most apparent in the preschool years, and it often resolves by the time the child starts to learn to read. LI also declines in prevalence with age, with many children having an early delay in language development that subsequently resolves (Bishop & Edmundson 1986). Another problem for this field is that no study has evaluated

prevalence of SSD, LI, and RD in the same children; most comorbidity studies examine only two disorders. A third point is that sampling bias in clinical samples may artifactually inflate comorbidity. Berkson (1946) showed that apparent comorbidities between otherwise independent disorders will arise in referred samples if the probability for referral of either or both disorders is less than one. In this case, comorbid individuals will be overrepresented because their probability of being referred is a combined function of the referral rates of each of their disorders. Berkson's bias does not imply any overt bias to select comorbid cases; the bias is simply the result of the compounding of independent probabilities. The effect would be magnified if the concerns of parents or teachers resulted in an additional bias to refer comorbid cases. Epidemiological data are needed for unbiased estimates of comorbidity rates. However, few investigators have published epidemiological studies of the comorbidity among pairs of these disorders, and their three-way morbidity is touched upon only in unpublished epidemiological data.

Table 2 summarizes existing data on comorbidity among SSD, LI, and RD expressed as relative risks (i.e., rate in the group with the risk diagnosis/rate in the group without the risk diagnosis; for studies that did not have a control group, we used the population rate as denominator). So a relative risk of 2.0 means that those in the risk group (e.g., with SSD) are twice as likely to have a comorbid diagnosis (e.g., LI) as those without the risk diagnosis (e.g., non-SSD controls or the general population). For a common multifactorial disorder, these two methods of calculating relative risk should yield roughly similar estimates. The table is divided into data from epidemiological versus referred samples, and it illustrates that more variation exists among individual studies within each type of sample than across sample types. Thus, Berkson's and other referral biases do not appear to play a major role. Genuine comorbidity exists among these conditions because the pairwise comorbidities are significantly greater than chance in both types of sam-

ples. However, except for the Iowa sample (1*d*), the risk for later RD in SSD and LI is almost entirely restricted to SSD+LI (RR = 4.6–8.9), whereas the rate of later RD in SSD without LI is negligible (RR = 0.9–1.6, all ns). More data are needed to specify the risk for later RD in LI without SSD because the two values in the table (3.2–3.6 for Iowa and 0.5 for the Longitudinal Twin Study) do not agree.

The convergent results for elevated rates of later RD in SSD+LI could reflect that SSD+LI is an etiological and/or cognitive subtype, or that etiological and/or cognitive risk factors in SSD and LI interact synergistically to greatly increase risk for later RD. If the latter is true, it implies that the developing reader may compensate for the cognitive risk factors posed by SSD or even LI alone by using alternative strategies, but the combination of their cognitive risk factors makes compensation much more difficult.

In studies specifically concerned with SSD and LI, comorbidity varies with age. Broomfield & Dodd (2004) categorized all new referrals to pediatric speech and language therapy services in a British Primary Care Trust and found robust bidirectional comorbidity between SSD and LI (entry 6 in **Table 2**). In contrast, Shriberg et al. (1999) found a lower comorbidity rate in an epidemiological study of 6-year-olds in the United States (entry 1*a* in **Table 2**). Although different modes of sampling may have caused the differences in the two studies (clinical sample in the U.K. study and an epidemiological sample in the U.S. study), it is also likely that the degree of comorbidity varies with age: Only 15% of children in the study by Broomfield & Dodd (2004) were over age 6. Bishop & Edmundson (1987) noted that although preschool children appeared to be more vulnerable to speech than language problems, speech problems resolved more readily. In their clinically identified sample, many children who presented with both speech and language problems at age 4 had only residual language difficulties when seen 18 months later.

The best evidence for the comorbidity of LI and RD comes from the same Iowa

Table 2 Comorbidity rates among speech sound disorder (SSD), language impairment (LI), and reading disability (RD) (relative risks)

	Sample sizes		SSD in LI	LI in SSD	RD in LI	RD in SSD	RD in SSD+LI	RD in SSD-LI	RD in LI-SSD
Epidemiological	1328	1a ^r	3.3	2.3	—	—	—	—	—
	570*	1b ^r	—	—	6.2 (second), 6.9 (fourth)	—	—	—	—
	527	1c ^p	—	—	1.9–2.2 (second, fourth, eighth)	—	—	—	—
	604	1d ^r	—	—	2.8 (second), 3.1 (eighth)	—	3.9 (second), 4.9 (eighth)	2.2 (second), 2.3 (eighth)	3.2 (second), 3.6 (eighth)
	453	2 ^r	2.2	2.3	1.9	2.6	6.0	1.6+	0.5+
	925–955	3 ^r	—	—	4.4 (7y), 4.9 (9y), 5.1(11y)	—	—	—	—
	1655	4 ^r	6.1	6.1	—	—	—	—	—
Referred	277	5 ^p	—	9.1	—	1.6	8.1	1.6+	—
	936	6 ^p	5.7	6.9	—	—	—	—	—
	123	7 ^p	—	4.0	—	2.5	7.4	1.1+	—
	110	8 ^p	—	—	5.7	—	—	—	—
	82	9 ^p	—	—	2.3	—	—	—	—

+ = NS.
 * = defined RD as 1 SD below weighted mean on reading comprehension composite.
 Key: relative risk = rate in risk group/population rate (p) or rate in risk + group/rate in risk – group (r).
 1: Iowa sample. 1a, Shriberg et al. (1999); 1b, Catts et al. (2002); 1c, Catts et al. (2005); 1d, Tomblin (unpublished).
 2: Colorado Longitudinal Twin Sample, R.L. Peterson, B.F. Pennington, L.D. Shriberg, & R. Boada (manuscript under review).
 3: Silva et al. (1987).
 4: Beitchman et al. (1986).
 5: Cleveland SSD sample, B.A. Lewis & L.A. Freebairn (unpublished).
 6: Broomfield & Dodd (2004).
 7: Denver SSD sample, Raitano et al. (2004), R.L. Peterson, B.F. Pennington, L.D. Shriberg, & R. Boada (manuscript under review).
 8: McArthur et al. (2000).
 9: Bishop & Adams (1990).

epidemiological sample that was studied by Shriberg et al. (1999). Catts et al. (2002) followed up children who had been identified with specific language impairment (SLI) at 6 years of age and found a relative risk of about 3. In a later follow-up of the Iowa sample to eighth grade (Catts et al. 2005), the comorbidity rates between LI and RD were lower but still significant (Table 2). Interestingly, this study also found that the LI without RD group performed significantly better than either the RD or the comorbid groups on both phonological awareness and nonword repetition. Of these two phono-

logical measures, the LI without RD group was worse than controls only on nonword repetition, not on phonological awareness. Whether a similar cognitive profile would be found in LI without SSD is not known.

However, because LI and SSD are commonly comorbid, it can be difficult to establish whether associations with RD are specific to one of these disorders. Several prospective studies of smaller referred samples of children with SSD have found an increased rate of later RD (Bishop & Adams 1990, Catts 1993, Nathan et al. 2004, Snowling et al. 2000), although

Connectionist Model

Speech Development

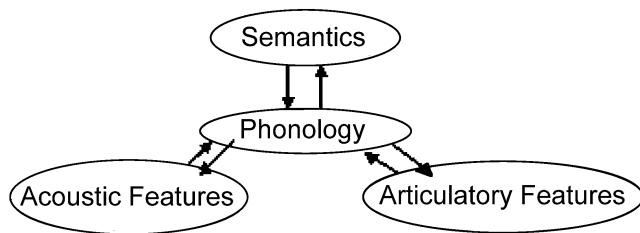


Figure 1

Connectionist models of speech development. Adapted from Guenther (1995), Joanisse (2000), Westerman & Miranda (2004).

several studies have found that the association is most robust for children with SSD+LI and is not found for children with isolated SSD (Bird et al. 1995, Larrivee & Catts 1999, Leitaio & Fletcher 2004, Lewis & Freebairn 1992). New unpublished data from three prospective studies with large samples of children with SSD have resolved this issue. As mentioned above, in all three studies the risk for later RD in SSD is mediated by comorbid LI and this risk is substantial (4.3–8.9). In contrast, the risk is negligible for later RD in SSD without LI or, in one case, LI without SSD. This pattern for RD risk in SSD+/-LI was found across referred and epidemiological samples; thus, it is not due to a referral artifact. But more research is needed to understand the conflicting findings for RD risk in LI without SSD.

In summary, population samples have established that SSD and LI are comorbid, although the rates vary with age, and that LI and later RD are comorbid, but we lack published population

data on the comorbidity between SSD and later RD. However, SSD+LI carries most of the risk for later RD, and the risk posed by SSD only, or sometimes LI only, appears to be negligible. Overlap among pairs of these three disorders sometimes varies as a function of comorbidity with the third disorder. This complex pattern of comorbidity makes it unlikely that all three disorders are pleiotropic manifestations of the same underlying cognitive or genetic liability (e.g., generalist genes for a general verbal trait; Plomin & Kovas 2005). This raises the question of whether the cognitive overlap of the three disorders mirrors the diagnostic overlap. That is, is the cognitive profile in SSD+LI (and later RD) distinct from that in either SSD without LI or LI without SSD?

COGNITIVE MODELS OF LANGUAGE IMPAIRMENT, READING DISABILITY, AND SPEECH SOUND DISORDER

It is useful to consider cognitive models of LI, RD, and SSD in the context of typical development and in relation to each other. **Figures 1** and **2** depict typical development of speech, language, and literacy, with written language skills building on earlier developing oral language skills. In developing an oral language, one of the first tasks an infant must master is the perception and production of the speech sounds specific to the native language (Kuhl 2004). Although innate constraints influence some aspects of human language acquisition (e.g., Pinker 1991), mastering a particular oral, and especially written, language requires extensive learning, much of it implicit statistical learning (Saffran et al. 1996). Consequently, connectionist models, which implement statistical learning of speech, language, and reading, provide a useful framework for thinking about relations among LI, RD, and SSD at the cognitive level.

Figure 1 depicts a simplified connectionist model of speech development (adapted from Guenther 1995, Joanisse 2000, Westermann & Miranda 2004) and **Figure 2** depicts a

Connectionist Model

Reading Development

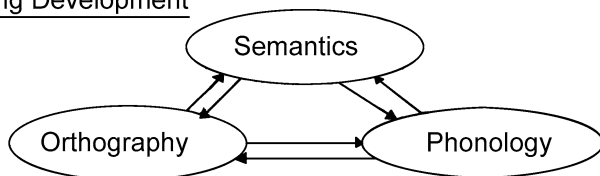


Figure 2

Connectionist model of reading development. From Harm & Seidenberg (1999).

connectionist model of single-word reading development (Harm & Seidenberg 1999). Two key components are shared by both models: phonology and semantics. These models illustrate that a problem in developing phonological representations could affect speech, language, and reading development, and indeed, as reviewed below, evidence does exist for phonological impairment in SSD, LI, and RD. However, these models also illustrate that development in each of these three domains is affected in multiple ways, and some of these ways have not been considered in cognitive models of these disorders.

The development of speech production has been depicted in several computational models (Guenther 1995; Joanisse 2000, 2004, 2007; Joanisse & Seidenberg 2003; Markey 1994; Menn et al. 1993; Plaut & Kello 1999; Westermann & Miranda 2004). The difficult developmental task these models address is how young children learn the complex mapping between acoustic features and articulatory gestures, particularly when important aspects of the articulatory gestures made by adult models are not observable. Babbling almost certainly supplements imitation in learning these mappings. In these models, hidden units learn the particular abstract mapping between acoustic features and articulatory gestures that signal meaning differences for that child, and the representations of these abstract mappings are phonological representations. However, these models do not explain clinical cases where phonological representations develop without speech (e.g., anarthric children with oral reading skills; Bishop 1985).

Lexical semantics is intimately involved in the determination of which acoustic and articulatory features are counted as relevant for a young child's particular lexicon in a particular language. As the child's vocabulary increases, the nature of her phonological representations also changes. Consequently, the development of phonological representations is protracted, and the weighting given to different acoustic features in speech perception changes with development (Nitttrouer 1999).

These computational models demonstrate that a problem in speech production such as that found in SSD could have several causes, including a bottom-up problem in processing acoustic features, a motor problem in planning and producing articulatory gestures, a problem learning the mapping between the two, a problem identifying which phonetic differences signal differences in meaning and which are equivalent (i.e., in learning phonological representations, a top-down problem in learning semantic representations that impedes the differentiation of phonological representations), or some combination of these problems. Similarly, these models suggest that there could be bottom-up auditory, representational (phonology in RD and syntax in LI), top-down semantic, learning, and multiple deficit theories of RD and LI. As we discuss below, existing cognitive models of these disorders have focused on single cognitive deficits and have tended to be static rather than developmental. That is, they have posited a congenital deficit in either a bottom-up auditory skill or a particular kind of representation (phonology or syntax) and have not considered the possibilities of how deficits might emerge from a developmental process or how deficient learning of new mappings between representations could cause disorders.

Language Impairment

A broad distinction can be drawn between two classes of LI model: those that regard the language difficulties as secondary to more general nonlinguistic deficits, and those that postulate a specifically linguistic deficit. The best-known example of the first type of model is the rapid temporal processing (RTP) theory of Tallal and colleagues, which maintains that language learning is handicapped because of poor temporal resolution of perceptual systems. This bottom-up auditory model of LI has also been applied to RD and SSD.

The first evidence for the RTP theory came from a study where children were required to match the order of two tones (Tallal & Piercy

1973). When tones were rapid or brief, children with LI had problems in correctly identifying them, even though they were readily discriminable at slow presentation rates. The theory has continued to develop over the years, and Tallal (2004) proposed a neural basis in the form of spike-timing-dependent learning. Tallal argued that although the underlying mechanism affected all auditory stimuli, its effects were particularly detrimental to language learning because development of neural representations of phonemes depends on fine-grained temporal analysis. Children who have poor temporal resolution will chunk incoming speech in blocks of hundreds of milliseconds rather than tens of milliseconds, and this will affect speech perception and hence on aspects of language learning.

Another theoretical account that stresses nonlinguistic temporal processing has been proposed by Miller et al. (2001), who showed that children with LI had slower reaction times than did control children matched on nonverbal IQ on a range of cognitive tasks, including some, such as mental rotation, that involved no language. Unlike the RTP theory, this account focuses on slowing of cognition rather than perception.

A more specialized theory is the phonological short-term memory deficit account of SLI by Gathercole & Baddeley (1990a). These authors noted that many children with SLI are poor at repeating polysyllabic nonwords, a deficit that has been confirmed in many subsequent studies (Graf Estes et al. 2007). This deficit has been interpreted as indicating a limitation in a phonological short-term memory system that is important for learning new vocabulary (Gathercole & Baddeley 1990b) and syntax. This theory, like the more specifically linguistic theories, places the core deficit in a system that is specialized for language processing, but the system is for memory and learning rather than for linguistic representations per se.

More recently, Ullman & Pierpont (2005) proposed a theory that encompasses both short-term memory and syntactic deficits under the umbrella of "procedural learning," which is

contrasted with a declarative learning system that is involved in learning new verbal information. They argue that LI is not a specifically linguistic disorder but is rather the consequence of an impaired system that will also affect learning of other procedural operations, such as motor skills.

Many authors, such as Bates (2004), have argued that domain-general deficits in cognitive and perceptual systems are sufficient to account for LI. This position differs radically from linguistic accounts of LI, which maintain that humans have evolved specialized language-learning mechanisms and that LI results when these fail to develop on the normal schedule. Thus, language-specific representational theories of LI coexist with similar linguistic theories of RD and SSD that focus on phonological representations. A range of theories of this type for LI focus on the syntactic difficulties that are a core feature of many children with LI. Children with LI tend to have problems in using verb inflections that mark tense, so they might say "yesterday I walk to school" rather than "yesterday I walked to school." Different linguistic accounts of the specific nature of such problems all maintain that the deficit is located in a domain-specific system that handles syntactic operations and is not a secondary consequence of a more general cognitive processing deficit (see, e.g., Rice & Wexler 1996, van der Lely 1994).

Although these theories focus on different perceptual, cognitive, and linguistic deficits, they are nonetheless hard to choose between for several reasons. First, the theories do not necessarily predict pure deficits in just one area—for instance, the RTP theory predicts that children with SLI will have phonological and syntactic problems, but the theory regards these as secondary to the basic perceptual deficit. Even where a domain-specific linguistic deficit is postulated, it could be argued that other more general deficits may coexist, perhaps because of pleiotropic effects of genes. Second, it is often easy to explain away a failure to find a predicted deficit on the grounds that the child has grown out of the deficit (which nevertheless has

affected language acquisition) or that the deficit applies only to a subgroup of children with SLI.

Studies that examine deficits predicted by different theories in the same children aid in disentangling different theoretical accounts. The results of such studies can be illuminating. Bishop et al. (1999) studied a sample of twin children, many of whom met criteria for LI. These children were given a battery of tests, including a measure of auditory processing, derived from the RTP theory, and a measure of phonological short-term memory, nonword repetition. Children with LI did worse than controls did on both measures, but some children have normal language despite poor scores on the tests of RTP or nonword repetition. The intercorrelation between the measures, though significant, was low (around 0.3). Furthermore, genetic analysis suggested different etiologies for the auditory deficit, which appeared environmental in origin, and the nonword repetition deficit, which was heritable. One might wonder whether these deficits identify different subgroups of children with LI, but the results indicated rather that the two deficits interacted and that children with a double deficit were the most severely affected. A similar pattern of results was obtained in a later twin study in which children were assessed on a test of nonword repetition and on a test of productive verb morphology (Bishop et al. 2006). Both measures revealed deficits in children with LI, and in this case, both were heritable, yet the intercorrelation between these measures was low (though significant) and there was no evidence that common genes were implicated. Once again, children with both deficits had the most severe problems, and some children with normal language scored in the impaired range on one of the measures of underlying deficit.

These two studies raise some general points that also apply to other developmental disorders:

- a) Any theory that postulates a single underlying deficit is inadequate to account for the disorder: several distinct deficits seem

implicated, none of which is necessary or sufficient on its own for causing LI.

- b) Although the different deficits can be dissociated and appear to have distinct etiologies, they tend to co-occur at above-chance levels.
- c) It is possible to have a single deficit—e.g., in auditory processing or phonological short-term memory—without necessarily showing LI.
- d) Children with LI typically show more than one deficit.

Reading Disability

A cognitive model of RD has greater consensus than does a model of LI. **Figure 3** depicts the processes involved in extracting meaning from written text. This figure shows that reading comprehension can be first broken down into cognitive components and then into developmental precursors of these cognitive components. One key component is fluent printed word recognition, which is highly predictive of reading comprehension, especially in the early years of reading instruction (Curtis 1980). The other key component is listening comprehension, that is, oral language comprehension (Hoover & Gough 1990).

The terms “developmental dyslexia” or “reading disability” have traditionally been reserved for children who have difficulties with basic printed word recognition. It is possible for a child to have reading comprehension problems despite adequate printed word recognition, but this is not counted as dyslexia. Instead, individuals with such problems are described as poor comprehenders, and the cognitive causes of their reading comprehension problems are distinct from those that interfere with word recognition (Nation 2005).

Printed word recognition can be broken into two component written language skills, phonological and orthographic coding (**Figure 3**). Phonological coding refers to the ability to use knowledge of rule-like letter-sound correspondences to pronounce words that have never

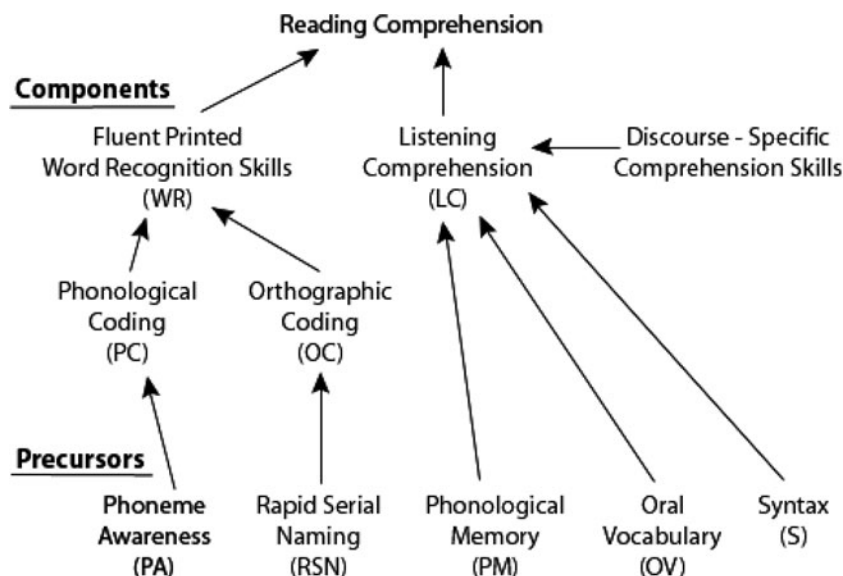


Figure 3

The processes involved in extracting meaning from written text.

been seen before (usually measured by pseudoword reading); orthographic coding refers to the use of word-specific patterns to aid in word recognition and pronunciation. Words that do not follow typical letter-sound correspondences (e.g., *have* or *yacht*) must rely, at least in part, on orthographic coding to be recognized, as do homophones (e.g., *rows* versus *rose*).

A large body of work has shown that most children with RD have disproportionate problems with pseudoword reading and that this deficit in phonological coding is related to poor phonological awareness. Phonological awareness is measured by tasks that require manipulation of the sound structure of spoken words (e.g., “what is cat without the /k/?”). Despite agreement about the importance of phoneme awareness deficits in RD, there is disagreement about whether these difficulties are themselves caused by lower-level processing deficits. Phoneme awareness is a complex metalinguistic skill that clearly involves multiple components. One argument is that phoneme awareness deficits arise from impaired phonological representations (Fowler 1991a, Swan & Goswami 1997a). Another argument postu-

lates that the central deficit is not specific to language, but rather is a consequence of the same RTP deficit proposed as an explanation for SLI (Tallal 1980). However, evidence for the RTP hypothesis for RD is patchy at best (see McArthur & Bishop 2001 for a review).

A parsimonious explanation for current data is that deficits in phonological representations lead to both phoneme awareness and phonological coding difficulties in RD. The phonological representations hypothesis is appealing because it helps explain why RD is associated not only with deficits in phoneme awareness, but also with impairments on a wide variety of phonological tasks, including phonological memory (Byrne & Shea 1979, Shankweiler et al. 1979) and picture naming (Fowler & Swainson 2004, Swan & Goswami 1997b).

An important caveat is that the relationship between phoneme awareness and reading is bidirectional, so that over time, poor reading also causes poor phoneme awareness (Morais et al. 1979, Perfetti et al. 1987, Wagner et al. 1994). Another caveat is that the evidence for the emergence of phoneme awareness being a necessary precursor for reading

development is not airtight (Castles & Coltheart 2004) because longitudinal studies supporting this claim have not completely eliminated the confound of preschoolers already having some reading skill at time one. A recent study found that children with chance-level performance on phoneme awareness tasks could nonetheless use letter names to learn letter sounds and thus begin to decode printed words (Treiman et al. 2008). So explicit phoneme awareness may not be necessary for learning to read, but it seems clear that appropriately structured phonological representations are needed. The exact meaning of “appropriately structured” is still unclear in the context of reading development. As discussed above, phonological representations develop and can develop atypically in a variety of ways. The nature of the underlying phonological deficit in RD is the subject of several hypotheses, including that children with RD (*a*) lack segmental phonological representations (Boada & Pennington 2006, Fowler 1991), (*b*) have problems detecting suprasegmental information in phonological representations (Goswami et al. 2002), (*c*) retain allophonic representations (Serniclaes et al. 2004), or (*d*) have less-distinct phonological representations (Elbro et al. 1998). More research is needed to test these hypotheses not only in RD, but also across all three disorders considered here (e.g., Corriveau et al. 2007 have recently extended Goswami’s suprasegmented theory to LI). These three disorders possibly share deficits on broad phonological measures, such as phonological awareness or nonword repetition, but they differ in how their phonological representations are deficient.

Because RD is often comorbid with LI, the question is raised as to the extent to which RD is associated with broader language deficits, e.g., as measured by tests of vocabulary and syntax. Although the phonological deficit hypothesis stresses children’s difficulties in learning letter-sound mappings, poor general language skills could also handicap reading acquisition because one can use linguistic context to infer meaning of a novel word (e.g., Cain et al. 2004). On IQ tests, children with RD tend to

underperform relative to their typically developing counterparts not only on phonological tasks, such as digit span, but also on all verbal subtests (D’Angiulli & Siegel 2003). Some of this performance deficit likely resulted from RD, since children with reading difficulties have impoverished opportunities to learn from print (e.g., Stanovich 1986; cf. Scarborough & Parker 2003), but some may well reflect subtle, wide-ranging language impairments. The evidence on this point from predyslexic children is particularly compelling, because deficits on a wide range of language skills are evident before they learn to read (Pennington & Lefly 2001, Scarborough 1990).

The presence of comorbid language problems in RD raises doubts that a phonological deficit is sufficient to cause RD. Moreover, as is discussed below, children with SSD have phonological deficits similar to those found in RD, but they usually do not develop RD unless they have comorbid LI. It appears that normal performance on rapid serial naming (RSN) tasks is a protective factor (R.L. Peterson, B.F. Pennington, L.D. Shriberg, & R. Boada, manuscript under review; Raitano et al. 2004). RSN is impaired in both RD and attention deficit/hyperactivity disorder (ADHD) (Shanahan et al. 2008), so RSN appears to be a cognitive risk factor shared by RD and ADHD. Moreover, because nonlinguistic processing-speed measures such as perceptual speed tasks (Wechsler Symbol Search) played a role similar to that of RSN as a cognitive risk factor for both RD and ADHD, it does not appear that the RSN problem is just a by-product of phonological or name-retrieval problems. We recently tested the hypothesis that processing or perceptual speed is a shared risk factor for RD and ADHD using structural equation modeling (L. McGrath, B.K. Pennington, R.K. Olson, & E.G. Willcutt, manuscript under review). We found that processing speed (i.e., latent traits composed of RSN and nonlinguistic perceptual speed tasks) was a unique predictor of both RD and ADHD symptoms and reduced the correlation between them to a nonsignificant value. Phoneme awareness and language skill were

unique predictors of RD symptoms, and inhibition was a unique predictor of ADHD symptoms. These results support a multiple-deficit model of both RD and ADHD. The total variance explained in RD symptoms by phoneme awareness, language skill, and processing speed was more than 80%. Thus, the best current understanding of the neuropsychology of RD indicates that at least three cognitive risk factors are involved, which is consistent with a multiple-deficit model.

At least one of these underlying deficits, deficient phonological representations, overlaps with deficits that are found in studies of LI. However, rather surprisingly, a deficit in RSN is not characteristic of children with LI unless they also have reading impairment (D.V.M. Bishop, D. McDonald, & S. Bird, manuscript under review). Furthermore, although children with RD tend to have lower scores on language tests, they typically do not show the kinds of grammatical limitation seen in LI (Bishop & Snowling 2004). Overall, multiple underlying deficits appear to exist in RD, as in LI, with the most serious problems being found in children who have two or more of the disorders. Of particular interest is the indication that at least one underlying deficit, poor phonological processing, is common to both RD and LI.

The procedural learning account of LI is relatively new, and few studies have tested its predictions. Nevertheless, it is noteworthy that it overlaps with the automatization deficit account of dyslexia (Nicolson & Fawcett 1990). Both theories maintain that specific brain circuitry involving the cerebellum is involved in the poor learning of reading or rule-governed aspects of language, especially phonology and syntax, and in both cases it is argued that associated motor impairments are another symptom of this neurobiological deficit (Nicolson et al. 2001, Ullman & Pierpont 2005). The automatization deficit account of dyslexia has been challenged as a general account of this disorder by findings that motor impairments are seen in only a subset of cases (e.g., Ramus 2003). Nevertheless, as we have argued for LI, this does not necessarily mean that these deficits are ir-

relevant to the causation of the disorder; they may have their effect only when in combination with other deficits.

Speech Sound Disorder

SSD was originally considered a disorder of generating oral-motor programs, and children with speech sound impairments were said to have functional articulation disorder (Bishop 1997). However, a careful analysis of error patterns has rendered a pure motor deficit unlikely as a full explanation for the disorder. For example, children with SSD sometimes produce a sound correctly in one context but incorrectly in another. If children were unable to execute particular motor programs, then we might expect that most of their errors would take the form of phonetic distortions arising from an approximation of that motor program. However, the most common errors in children with SSD are substitutions of phonemes, not distortions (Leonard 1995). Moreover, a growing body of research demonstrates that individuals with SSD often show deficits on a range of phonological tasks, including speech perception, phoneme awareness, and phonological memory (Bird & Bishop 1992, Kenney et al. 2006, Leitaio et al. 1997, Raitano et al. 2004). Though it remains possible that a subgroup of children have SSD primarily because of motor impairments, it now seems likely that the majority of children with SSD have a type of language disorder that primarily affects phonological development. Interestingly, RSN is not impaired in SSD (Raitano et al. 2004), and SSD children can have persisting phoneme awareness problems but normal reading development (R.L. Peterson, B.F. Pennington, L.D. Shriberg, & R. Boada, manuscript under review). Thus, intact RSN appears to be a protective factor in these children.

EVIDENCE FOR COGNITIVE OVERLAP

This brief review of cognitive models of disorders indicates that some close similarities exist in the theories that have been advanced to

account for LI, RD, and SSD. For all three disorders, phonological deficits, possibly due to auditory perceptual problems, have been proposed as a core underlying cause. Although this overlap may help explain why the disorders are often comorbid, it leaves us with the puzzle of phenotypic variation between disorders. In short, if the same theoretical account applies to all disorders, why do they involve different behavioral deficits? And why, for instance, do we find children with SSD who have poor phonological skills yet do not have reading problems? An answer is suggested by our analysis of RD and LI as disorders that involve multiple cognitive deficits. A phonological deficit may be a key feature of all three disorders, yet its specific manifestation will depend on the presence of other deficits. This kind of model is implicit in the analysis by Bishop & Snowling (2004), who argue that LI is not just a more severe form of RD—rather, RD and LI both usually involve poor phonological processing, but LI is seen when this deficit is accompanied by broader difficulties affecting aspects of language such as syntax. The finding (by R.L. Peterson, B.F. Pennington, L.D. Shriberg, & R. Boada, manuscript under review) that children with SSD often read well despite poor phonological skills indicates that phonological deficit alone will not usually lead to later reading problems—it does so when it is accompanied by poor RSN. A closely similar conclusion was reached (by D.V.M. Bishop, D. McDonald, & S. Bird, manuscript under review) in a study of comorbidity between RD and LI. The study found that children who had LI without RD performed normally on tests of RSN. Both these studies suggest that although phonological deficit is a risk factor for RD, good RSN can act as a protective factor. This evidence for interaction between deficits has implications for how we model comorbidity between disorders (discussed below).

In summary, although cognitive overlap exists among these three disorders (i.e., phonological deficits), the cognitive profile varies as a function of comorbidity. Moreover, these cognitive profile differences appear to map onto the

comorbidity patterns reviewed above. That is, the presence or absence of RSN deficits in SSD and LI relates to their comorbidity with later RD. But more systematic research is needed to test how cognitive profiles vary by comorbidity subtypes. This research will require large samples that have been followed longitudinally.

EVIDENCE FOR ETIOLOGICAL OVERLAP

Strong evidence demonstrates that LI, RD, and SSD are genetically influenced. That evidence is summarized in **Table 3**, which shows that each disorder is familial, moderately heritable, and has several replicated linkages to specific chromosome locations (for reviews, see Fisher & Francks 2006, Lewis et al. 2000, McGrath et al. 2006, Paracchini et al. 2007). **Table 3** also contains a footnote that explains the nomenclature for chromosome locations and loci associated with disorders. The RD loci have replicated across languages and cultures, including Swahili-speaking children in Tanzania (Grigorenko et al. 2007). For both RD and SSD, several candidate genes have been identified, and several of these are candidates for both disorders.

Twin studies have also been used to examine relations among these disorders. Most twin studies have found high h^2_g (0.6 or above)¹ for LI. However, a recent analysis by Bishop & Hayiou-Thomas (2008) found that this depended on whether children with SSD were included in the sample. For 4-year-olds who had LI without SSD, genes did not seem implicated in the etiology. Few twin studies have looked at both LI and RD in the same children, although in two separate samples Bishop and colleagues reported that RD was heritable only when

¹The term h^2_g refers to the heritability of the extreme group's deficit. Unlike h^2 , which estimates what proportion of the phenotypic variance across the whole distribution is attributable to genetic influence, h^2_g estimates the magnitude of genetic influences on the low (or high) tail of the distribution. Unless they are completely categorical, h^2_g is the appropriate heritability for disorders.

Table 3 Summary of genetic studies of speech sound disorder (SSD), language impairment (LI), and reading disability (RD)

	SSD	LI	RD
Familiality (relative risk)	~6 ^a	2–4 ^a	4–8 ^d
Heritability*	~0.80–1.00 ^{a,b}	0.36–0.96 ^{b,c}	0.58
Chromosome regions ^{c,g}	1p34–36 (DYX8) 3p12–q13 (DYX5) 6p22 (DYX2) 15q21 (DYX1)	13q21 (SLI3) 16q24 (SLI1) 19q13 (SLI2)	1p34–36 (DYX8)** 2p15–16 (DYX3) 3p12–q13 (DYX5) 6p22 (DYX2) 15q21 (DYX1) 18p11 (DYX6) Xq27.3 (DYX9)
Candidate genes ^{c,f}	FOXP2 ROBO1 DCDC2 KIAA0319 DYXC1		ROBO1 DCDC2 KIAA0319 DYX1C1 MRPL19 C20RF3

*Estimates based on twin concordance data (double difference between monozygotic and dizygotic) or from group heritability computed using DeFries-Fulker method (DeFries & Fulker 1985).

**Nonsex chromosomes are numbered according to their size, so chromosome 1 is the largest and chromosome 22 (or possibly 21) is the smallest. Each chromosome has two arms, one short (p) and one long (q). Morphologically defined regions within each arm are denoted by a number, counting outward from the centromere that lies between the two arms (e.g., p1, p2, p3, and q1, q2, q3), and these regions are subdivided into bands (p11) and subbands (p11.1) and sub-subbands (p11.11). So the term “1p34–p36” means a location on the short arm of chromosome 1 including regions 34, 35, and 36. The names of loci associated with a disorder are capitalized and numbered according to order of discovery (DYX1 means the first dyslexia locus discovered, DYX2 means the second, and so on).

^aLewis et al. (2006).

^bViding et al. (2004).

^cBishop & Hayiou-Thomas (2008).

^dPennington & Olson (2005).

^eMcGrath et al. (2006).

^fAnthoni et al. (2007).

^gNewbury & Monaco (2008).

accompanied by poor nonword repetition (Bishop 2001, Bishop et al. 2004).

A multivariate analysis of reading skill in twins (Tiu et al. 2004) supported the multiple-deficit model (L. McGrath, B.K. Pennington, R.K. Olson, & E.G. Willcutt, manuscript under review) discussed above, in which phoneme awareness, RSN, and language skill independently contributed to predicting reading skill. Tiu et al. (2004) found that phoneme awareness, RSN, and full-scale IQ each made independent phenotypic contributions to reading skill and, in the etiological model based on the twin design, each construct had both shared

and independent genetic relations to reading skills. More work of this kind is needed to test multiple-deficit models of each disorder and how both familiality and heritability vary by comorbidity subtypes.

Family and twin studies can provide evidence for genetic influences on disorders and their relations, but to identify the genes involved we need to use methods of molecular genetic analysis. Linkage analysis identifies chromosomal regions that are likely to harbor genes involved in etiology of disorder. This method capitalizes on the fact that genes close together on a chromosome tend to be inherited together.

Thus, the method involves looking for chromosomal regions that are co-inherited at above-chance levels in affected family members. It is important to recognize that discovering linkage is not the same as finding a gene—there may be many genes in the linkage region, and it can be a painstaking task to identify which are implicated. Furthermore, it is unusual to find genetic variants that are perfectly associated with disorder; the famous case of a mutation in the FOXP2 gene that was found in all affected members and no unaffected members of a family with LI and SSD is the exception rather than the rule. In general, where a disorder has a complex, multifactorial etiology, the genes involved will have only a probabilistic influence on disorder—i.e., they act as quantitative trait loci (QTLs) rather than as major genes that cause disorder. Furthermore, any one linkage analysis is likely to turn up some spurious linkages that arise by chance, purely because many statistical comparisons are conducted. Molecular geneticists in this field are rightly cautious about interpreting linkages until they have been replicated in an independent sample (Newbury & Monaco 2008).

Initially, there was surprise that RD and LI linkages did not overlap, but it has become clear that the most powerful way to demonstrate common linkage is to study two disorders together; when this is done, there is evidence that some linkage regions affect more than one disorder (Monaco 2007). As shown in **Table 3**, SSD shows linkage to known RD risk loci (Smith et al. 2005, Stein et al. 2004). Recent attempts to replicate the 6p22 and 15q21 loci in an independent SSD sample have been partially successful (Stein et al. 2006).

Once linkage has been established, the next step is to identify a specific gene and understand its mode of action. So far, the greatest success for this approach in speech and language disorders, or even behaviorally defined disorders generally, has been with the FOXP2 gene, whose effects in brain and whose roles in the evolution of vocal communication across species have been studied extensively (Fisher 2007).

As shown in **Table 3**, the linkage studies of RD have been followed by the initial identification of six candidate genes in four of these linkage regions. The names of genes can be acronyms for the gene product (DCDC2 means “doublecortin doublecortin 2”) but can also refer to the disorder they help cause (in the name DYX1C1, C1 means the first candidate for the first dyslexia risk locus, DYX1) or to other things. All these candidate genes for RD are involved in brain development, either in neuronal migration or in axon guidance. Their role in neuronal migration is consistent with that found in the pioneering work of Galaburda et al. (1985), who discovered ectopias (i.e., neurons that end up in the wrong location, such as in white instead of gray matter, because of migrational errors) in the brains of deceased dyslexics (most of whom had comorbid LI).

Although considerable progress has been made in specifying the genetic causes of these disorders, we should not forget that the heritability of these disorders is generally significantly less than 100%, so environmental variables must play a role in their development (see risk factors in **Table 1**). Even when heritabilities are higher, as they are for SSD, the environment can affect the phenotype. Such environmental variables are likely to include the home language environment and instructional quality (especially for RD), as well as environmental events that have a more direct effect on biology (e.g., maternal health during pregnancy, lead poisoning, or head injury). Unfortunately, few studies investigating main effects of such environmental variables on language development have used genetically sensitive designs.

In addition to main effects of environment, it is likely that the disorders considered here are influenced by gene-by-environment ($G \times E$) interactions. A $G \times E$ interaction is identified when the impact of a given environment depends on the genotype of the individual. $G \times E$ interaction has been clearly demonstrated in selective breeding experiments with animals and plants, but is much harder to demonstrate in humans, where control over genotype is not possible. If candidate genes are

identified, one can then see whether a measured environmental factor interacts with genotype in determining the phenotype (Rutter 2006).

A recent study investigated $G \times E$ interaction using SSD/RD linkage peaks with the strongest evidence of linkage to speech phenotypes, 6p22 and 15q21, and measures of the home language/literacy environment in a sample of children with SSD and their siblings. Results showed four significant and trend-level $G \times E$ interactions at both the 6p22 and 15q21 locations across several phenotypes and home environmental measures (McGrath et al. 2007). The direction of the interactions was such that, in relatively enriched environments, genetic risk factors substantially influenced the phenotype, whereas in less-optimal environments, genetic risk factors had less influence on phenotype. This directionality of the interactions is consistent with the bioecological model of $G \times E$ (Bronfenbrenner & Ceci 1994). This work is preliminary because these linkage-based methods are a step away from the ideal of using identified risk alleles to test for $G \times E$ (Rutter 2006).

In summary, evidence indicates that LI, RD, and SSD are familial, heritable, and linked to QTLs in certain chromosomal regions. One genetic mutation in FOXP2 has been shown to have a clear role in causing a rare form of speech dyspraxia associated with LI, and several candidate genes have been identified that act as QTLs for RD. Preliminary evidence suggests that some of these QTLs are pleiotropic and affect both RD and SSD, whereas less evidence exists for QTLs shared by LI and RD. Since the heritability of these disorders is less than 100%, environmental factors also play a role in their etiology, and it is likely that $G \times E$ correlations and $G \times E$ interactions also exist in their etiology.

WHICH COMORBIDITY MODELS ARE SUPPORTED?

Models of comorbidity have been proposed by Klein & Riso (1993), quantified by Neale & Kendler (1995), and applied to the disor-

ders considered here by D.V.M. Bishop, D. McDonald, & S. Bird (manuscript under review) and Pennington et al. (2005). Space does not permit a full description of these comorbidity models; instead, we focus on which comorbidity models can be rejected as explanations for the comorbidity among SSD, LI, and RD based on the data reviewed in the previous sections. Summarizing these data briefly, we found robust comorbidity among pairs of the three disorders (Table 2) but not perfect overlap. Cognitive and genetic risk factors (Table 3) are also shared by LI, RD, and SSD.

First, we can conclude that the comorbidity between SSD and LI and LI and RD is not an artifact of selection, population stratification, definitional overlap, or rater biases. These comorbidities have been found in population samples (Table 2). Their diagnostic definitions do not overlap (Table 1) and depend on objective tests, not raters.

Turning to nonartifactual explanations, Neale & Kendler (1995) differentiated three broad types: alternate forms, multiformity, and correlated liabilities. All three types of comorbidity models are versions of the continuous liability threshold model, which assumes that there is a continuous liability distribution of multifactorial causes (genetic and/or environmental causes) for a disorder, and that the sum total of their influences can be represented as a continuous liability distribution; a disorder occurs if an individual crosses a particular threshold on that liability distribution (see Figure 4).

The simplest kind of model proposes a single underlying liability distribution—in effect, the risk factors for all three comorbid disorders are seen as identical, but the manifestation of that risk can vary. This kind of model is suggested by research by Lewis and colleagues, who in a series of studies have noted that relatives of children with SSD are at increased risk of LI and RD as well as SSD (Lewis et al. 2007). This finding is compatible with the notion that the same heritable liability increases risk for all three disorders, although these authors hypothesized disorder-specific genes as well. So the single-liability model suggests that “generalist

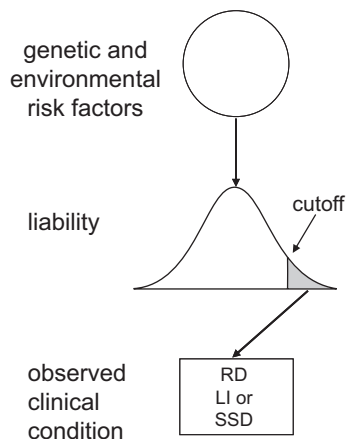


Figure 4

Continuous liability distribution. RD, reading disability; LI, language impairment; SSD, speech sound disorder.

genes” (Plomin & Kovas 2005) may exist for a general verbal trait, the low extreme of which underlies these disorders. In its simplest form, this corresponds to Neale & Kendler’s (1995) alternate forms model, which hypothesized that individuals who cross a particular threshold on a liability distribution have the probability of p of having a disorder A and the probability r of having a disorder B. This means that both disorders share a single liability, yet whether the person manifests disorder A or B depends on chance or risk factors that vary across individuals. This model raises the question of why the same liability should manifest differently in different people: It seems unlikely that this would be entirely due to chance; rather, it seems plausible that different environmental or genetic risks in the individual child interact with the underlying liability to determine the outcome.

Age is one systematic factor that has been proposed as influencing outcome. Scarborough & Dobrich (1990) suggested that the same risk factors that led to LI in a preschool child could lead to RD in a school-aged child. They talked of “illusory recovery,” in which the problems of a child with LI appeared to resolve only to be replaced later by literacy problems. Subsequent research, however, suggests that LI does not disappear in children with LI who then develop

RD, although LI may become less overt (Bishop & Adams 1990, Catts et al. 1999). A variant of this model treats RD as a condition that is both later in onset and less severe than SSD or LI; according to such a severity model, a child with a moderate liability may present only with RD in middle childhood, whereas one with a higher liability will be identified with SSD and/or LI in preschool and with RD a few years later.

Although a model with a single-liability dimension is parsimonious, most experts in this field would regard it as unlikely on the grounds that all three disorders—SSD, LI, and RD—appear heterogeneous. Although agreement is lacking about the best way of subtyping these disorders, quite marked phenotypic and etiologic differences can exist within each category, as we have shown. Furthermore, some children with severe LI do not have RD, which is inconsistent with a model that treats LI as indicative of a more severe liability. In addition, some children with SSD do not have RD, which also rejects this severity hypothesis for the relation between SSD and RD.

These results suggest that a better kind of model may be one that allows for separate liabilities for the different disorders, but where at least one of the liabilities can lead to a comorbid form. This corresponds to a multiformity model (Neal & Kendler 1995). Pennington et al. (1993) considered this kind of model when investigating comorbidity between RD and ADHD, and suggested that RD might lead to the phenotypic manifestation of ADHD in the absence of etiological influences typically associate with ADHD in isolation. One can readily imagine that a child might appear to be inattentive or hyperactive in the classroom because of the frustration elicited by difficulties with reading rather than as a consequence of the neurocognitive difficulties that are typically associated with ADHD in the absence of RD. The multiformity model usually describes each disorder as having its own distinct etiology, but there are cases where the etiology for disorder A can lead to comorbid A+B. However, if rates of comorbidity are high, the multiformity model can also be seen as an extension of the alternate

forms model, whereby A and B usually have the same underlying cause (but with different liability thresholds), but subtypes of either A or B exist with separate etiological pathways.

A more extreme view of subtypes is to treat the comorbid disorder as etiologically distinct from either disorder occurring alone. This model of three independent disorders might postulate, for instance, that SSD with LI is etiologically distinct from pure SSD or pure LI, requiring three separate liability distributions to account for the patterns of co-occurrence between these two disorders. The comorbidity and genetic results presented here are consistent with this model, but the cognitive results are not consistent because SSD+LI overlaps cognitively with both SD and LI.

Neale & Kendler (1995) also postulated correlated liabilities models. In these models, each disorder has its own liability, a continuous relation exists between the liability to one disorder and the liability to the second disorder. An increase in liability for one disorder is correlated with the increase in liability for the second disorder. The relationship between the liability of the two disorders occurs via a significant correlation between the risk factors (correlated liabilities) or a direct causal relationship between the manifest phenotypes of the two disorders (A causes B, B causes A, or reciprocal causation). If the relationship is between manifest phenotypes, this actually becomes the multiformity model. In contrast, if the liabilities themselves are correlated, even at subthreshold levels, then subclinical comorbidity will occur even if one diagnostic phenotype is not expressed.

Our review of the etiology of LI, RD, and SSD indicates that some support exists for the correlated liabilities model of the relation between SSD and RD because both disorders are linked to some of the same QTLs. But this finding is also somewhat puzzling because SSD without LI does not pose much risk for later RD. So this finding needs to be tested in larger samples to determine whether the linkage of SSD to RD loci is mainly due to SSD+LI. Another puzzle is the robust comorbidity between SSD and LI and between LI (even without

SSD) and RD in some studies, but so far, no QTLs have been discovered that are shared in either case. Clearly, more work is needed to test how well the correlated liabilities model (i.e., shared genetic and possibly environmental risk factors) accounts for the comorbidity among SSD, LI, and RD.

Although the Neale & Kendler (1995) models are a major contribution to the comorbidity literature because they are the most complete set of models yet proposed and because they are specified quantitatively, they nonetheless have some limitations. Specifically, they do not include either a neural or a cognitive level, they are not explicitly developmental, and they only deal with pairwise comorbidities. The main way of testing between them involves considering family or twin data to see how far a given disorder, or a comorbid form, breeds true, but even with ideal (simulated) data sets, some of the models can be difficult to distinguish empirically (Rhee et al. 2004).

The specific developmental disorders that we consider here, however, provide an alternative route to testing between models, provided one is willing to make the assumption that disorders caused by different liabilities might have different cognitive profiles. For instance, to test the three independent disorders model to explain comorbidity between RD and LI, we might predict that the cognitive profile in comorbid RD+LI would differ from that in pure RD or pure LI. Because the phenotype of each of the disorders considered here can be quite variable, and well-articulated models elucidate the underlying cognitive deficits associated with these disorders, this seems a promising approach. Contrary to the three independent disorders model, cognitive deficits characteristic of SSD, LI, and RD overlap to some extent.

In summary, the comorbidity observed among LI, RD, and SSD is not readily explained by any of the Neale and Kendler models but rather may require a model in which multiple cognitive deficits interact. Models of this type have been proposed by Bishop & Snowling (2004), Bishop (2006), and Pennington (2006), and a specific version to account for

comorbidity between LI and RD was formally specified and tested by D.V.M. Bishop, D. McDonald, & S. Bird (manuscript under review).

The multiple overlapping risk factors model of developmental disorders proposed by D.V.M. Bishop, D. McDonald, & S. Bird (manuscript under review) differs from the Neale and Kendler models in that it does not have a separate liability for each disorder, nor does it attempt to account for two disorders in terms of a single liability. Rather, it postulates several independent liability distributions, each of which determines a specific underlying deficit, and the disorder that is observed depends on the combination of liabilities that are suprathreshold. Some liabilities, such as that for phonological processing deficit, are implicated in SSD, LI, and RD. Others, such as liability for RSN deficit, appear to be specific to RD (although they might turn out to be implicated in other neurodevelopmental disorders such as ADHD; see Shanahan et al. 2008).

The multiple overlapping risk factors model can account for several features of the data reviewed here. In particular, it predicts that we should find some risk factors that are general to the three disorders of SSD, LI, and RD, and others that are specific. Furthermore, it predicts that a deficit in one underlying cognitive skill will not lead to overt disorder unless other deficits are also present.

CONCLUDING COMMENTS

LI, RD, and SSD are conditions that have traditionally been viewed as separate, and indeed,

they can occur in pure form. This has led researchers to look for a single underlying cause for each disorder, both at the cognitive level and at the etiological level. Insofar as single cause explanations do not work, the alternative approach has been to look for subtypes that have a single cause. The research reviewed here suggests that such an approach is doomed to failure. LI, RD, and SSD are complex multifactorial disorders, not only in terms of their genetic and environmental etiology, but also in terms of their cognitive underpinnings. Each disorder appears to arise as the consequence of a specific constellation of underlying deficits. Each individual deficit may be common in the general population and may only assume clinical significance when combined with another deficit. Some deficits, especially those affecting phonological processing, appear to play a part in all three disorders; others are specific to one of the disorders.

We have shown that some evidence exists for similar patterns of relations among the three disorders at the three levels of analysis considered here: diagnostic, cognitive, and etiological. But more systematic research is needed to test these patterns and define the relations among these three communication disorders.

In the past, researchers often either ignored comorbidity or strenuously attempted to avoid it by studying “pure” groups. We argue that this is the wrong approach, and that to understand these disorders fully, we need to consider the relationships between them, both cognitive and etiological.

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