Examining the Comorbidity of Language Impairment and Attention-Deficit/Hyperactivity Disorder

Kathryn L. Mueller and J. Bruce Tomblin

Language impairment (LI) and attention-deficit/hyperactivity disorder (ADHD) are two relatively common developmental disorders that have important consequences in the lives of the affected individuals. Within clinical settings, many children present with both conditions. Individuals who are comorbid for disorders generally experience poorer social and academic outcomes than those with either one or other of the disorders (e.g., see Berkson, 1946; Caron & Rutter, 1991; Neale & Kendler, 1995). In addition, there is some concern that the expressive and receptive language problems of children with behavior and socioemotional disorders, such as ADHD, are not adequately addressed in current clinical assessment and therapeutic practices (e.g., see Redmond, 2002; Tannock, 2005). It is of interest, therefore, to clinicians and researchers alike to understand more about this phenomenon.

Research on the comorbidity of LI and ADHD can be divided between studies that apply research protocols to samples that are population based and studies that are based on children who have been clinically identified, with the majority of data derived from the latter group (Table 1).

COMORBIDITY IN POPULATION-BASED SAMPLES

Population-based studies on the comorbidity of LI and ADHD are surprisingly limited. Beitchman and colleagues (Beitchman et al., 1996, 2001; Beitchman, Hood, Rochon, &
Table 1. Summary of clinical studies on comorbidity

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample: Size, Source, Comparison Group</th>
<th>Age, years</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alloway, Elliott, &amp; Holmes, 2010</td>
<td>964 children from community sample, United Kingdom</td>
<td>10</td>
<td>8% community sample had ADHD symptomatology; ~50% of these were receiving additional classroom support for learning difficulties</td>
</tr>
<tr>
<td>Benasich et al., 1993</td>
<td>56 children with LI and 43 controls followed longitudinally from age 4–8, United States</td>
<td>4–8</td>
<td>18% of children with LI scored in the clinical range on the CBCL hyperactivity subscale vs. 0% controls</td>
</tr>
<tr>
<td>Baker &amp; Cantwell, 1987</td>
<td>Subset of 300 children from the sample of Cantwell &amp; Baker (1987)</td>
<td>Ascertained ages 2–16, followed for 5 years: 6–20 at follow-up</td>
<td>16% of children with S/LI had ADD at the time of the initial study; 37% of children with S/LI had ADD at follow-up</td>
</tr>
<tr>
<td>Baker &amp; Cantwell, 1992</td>
<td>Subset of 65 children from the sample of Cantwell &amp; Baker (1987), all comorbid for ADD</td>
<td>6–15</td>
<td>62% of children with ADD had S/LI; 22% had language only</td>
</tr>
<tr>
<td>Berry, Shaywitz, &amp; Shaywitz, 1985</td>
<td>134 ADD from clinic; 94 school-based controls, United States</td>
<td>6–13</td>
<td>26% of ADD had expressive language problems; 8% had receptive problems</td>
</tr>
<tr>
<td>Camarata et al., 1988</td>
<td>38 children with mild to moderately behavior disorder from public schools, United States</td>
<td>8–12</td>
<td>37 children (97%) fell at least 1 SD below the normative mean on one of more of the TOLD-I subtests</td>
</tr>
<tr>
<td>Cantwell &amp; Baker, 1987</td>
<td>600 children from community speech and hearing clinics, United States</td>
<td>2–16</td>
<td>23% of children in the S/LI group had ADD; 33% of children in the language group had ADD</td>
</tr>
<tr>
<td>Chess &amp; Rosenberg, 1974</td>
<td>563 referrals from psychiatric outpatient clinic, United States</td>
<td>Preschool</td>
<td>24% had LI, most of which were ADD symptomatology</td>
</tr>
<tr>
<td>Cohen, 1996; Cohen et al., 1989, 1993, 1998, 2000</td>
<td>399 consecutive referrals to psychiatric outpatient clinic, Canada</td>
<td>4–12</td>
<td>Children with SLI were more likely to have ADD symptomatology</td>
</tr>
</tbody>
</table>

(continues)
Table 1. Summary of clinical studies on comorbidity (Continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample: Size, Source, Comparison Group</th>
<th>Age, years</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gualtieri et al., 1983</td>
<td>26 consecutive admissions to psychiatric in-patient care, United States</td>
<td>5–13</td>
<td>90% of children with ADD had SLI</td>
</tr>
<tr>
<td>Love &amp; Thompson, 1988</td>
<td>200 referrals to child psychiatric outpatient clinic, Canada</td>
<td>Preschool</td>
<td>66% of children with ADD had SLI</td>
</tr>
<tr>
<td>Redmond &amp; Rice, 1998</td>
<td>17 LI, 20 age-matched controls</td>
<td>Kindergarten and first grade</td>
<td>Teachers endorsed symptomatology of ADHD in children with LI more frequently than parents, and the rate at which they did this was significantly different to how they rated control</td>
</tr>
<tr>
<td>Redmond &amp; Rice, 2002</td>
<td>12 LI, 19 age-matched controls</td>
<td>8–9</td>
<td>No significant difference in rates of ADHD among LI cases and controls</td>
</tr>
<tr>
<td>Snowling et al., 2006</td>
<td>71 adolescents with a preschool history of S/LI, 49 age-matched controls</td>
<td>15–16</td>
<td>3.3% of individuals with persistent LI had ADHD compared with 4% of individuals in the group with preschool S/LI that had resolved compared with 2% controls</td>
</tr>
<tr>
<td>Tannock, Ickowicz, Oram, &amp; Fine, 1995</td>
<td>20 ADHD referred for medication trial, Canada</td>
<td>7–11</td>
<td>60% children with ADHD had S/LI. All but one previously undiagnosed</td>
</tr>
<tr>
<td>Tirosh &amp; Cohen, 1998</td>
<td>120 children ascertained by teacher report</td>
<td>6–11</td>
<td>45% of children with ADHD were impaired in at least one language domain</td>
</tr>
<tr>
<td>Trautman et al., 1990</td>
<td>67 psychiatric referrals to day treatment program, United States</td>
<td>6–13</td>
<td>63% of S/LI had ADD; 27% of ADD had S/LI</td>
</tr>
</tbody>
</table>

Note: ADD = attention deficit disorder; ADHD = attention-deficit/hyperactivity disorder; CBCL = Child Behavior Checklist; LI = language impairments; SLI = specific language impairment; S/LI = speech and/or language impairment; TOLD = Test of Language Development-First Edition.
Peterson, 1989; Beitchman, Hood, Rochon, Peterson, Mantini, et al., 1989; Beitchman, Nair, Clegg, Ferguson, & Patel, 1986) ascertained 142 children with speech and/or language impairments (S/LI) in kindergarten and matched them with controls on age, gender, and classroom/school. Teacher report at this time showed elevated rates of hyperactivity among the S/LI group compared with controls on the Conners Teacher Rating Scale (Conners, 1969), although this difference was not statistically significant ($p = .12$); (Beitchman et al., 1986). Further psychiatric evaluation of a subset from this sample, which was then extrapolated to give an estimate for the total sample population, indicated that 30.4% of children in the S/LI group were estimated to have attention deficit disorder on the basis of the Diagnostic and Statistical Manual of Mental Disorders (DSM), Third Edition (DSM–III, 1980) definition compared with 4.5% of controls.

If speech–language impairment is treated as a risk factor for ADHD, then the relationship between these conditions can be expressed in terms of relative risk, which reflects the degree to which the rate of a disorder changes as a result of a particular exposure or condition (i.e., risk factor), with a test statistic centered around 1. In this instance, a relative risk of 1 would indicate that LI does not affect the rate of ADHD; relative risk greater than 1 would indicate LI is a risk factor for ADHD; relative risk less than 1 would indicate that LI protects against ADHD. Here, relative risk is calculated by dividing the rate of ADHD in children with S/LI by the rate of ADHD in children without S/LI. Within the Beitchman et al. study, the data yielded a RR of 6.75. This means that if a child has LI, his or her risk for having ADHD increases by more than a factor of 6.

Children in the study of Beitchman et al. (1986) were then followed longitudinally. At age 12, no significant difference was found between the S/LI group and controls on measures of ADHD (Beitchman et al., 1996). However, when the S/LI group was broken apart by disorder, the rate of ADHD among children with LI (23.4%) was significantly different from controls (6.2%; $p < .01$). Here, the relative risk of ADHD conditioned upon LI is slightly smaller than it was in kindergarten, at 3.7. However, this still shows that if a child has LI, it greatly increases his or her chances of also having ADHD. Comorbidity rates for the speech-only group were not reported, but from the available data, we may surmise that LI, but not speech disorder, was related to ADHD in this sample. This finding is of interest because it is consistent with two previous reports that some forms of communication disorders carry greater risk for ADHD than others (e.g., see Beitchman, Hood, Rochon, & Peterson, 1989; Beitchman, Hood, Rochon, Peterson, Mantini, et al., 1989; Cantwell & Baker, 1987, discussed later).

The rate at which comorbidity occurs in the sample of Beitchman et al. (1986) is also far greater than expected by chance, given the rate at which the two disorders occur in the general population. This can be estimated by multiplying the base rates of each of the disorders. Using a prevalence rate of 7% for S/LI and a mid-range estimate of 10% for ADHD, the expected rate of comorbidity between S/LI and ADHD, by chance, should be approximately 0.7% (7% x 10%). This is far lower than the comorbidity observed in the sample of Beitchman et al.

It should also be noted that Beitchman et al. (1986) did not compute relative risks in their original studies, although, as we have just shown, it would have been possible to do so. Because they were working with a population sample, there were other ways in which these authors could have mined their data. For example, if they had reported the rate of unaffected children in their sample (i.e., the number of children with neither LI nor ADHD), it would have been possible to derive an estimate for the rate of comorbidity between LI and ADHD in the general population. They should also have been able to compute the relative risk for LI conditioned upon ADHD, which might have helped in characterizing the nature of the relationship between LI and
ADHD. Comparing this with the rate of ADHD conditioned upon LI would have served as an indicator as to whether there is any directionality to the relationship between the two disorders. In the subsequent text, we apply these kinds of analyses to a data set of our own.

A second study, by Tirosh and Cohen (1998), also used epidemiological sampling methods to study the comorbidity between LI and ADHD in a large cohort of children between 6 and 11 years of age \( (N = 3,208) \). A teacher questionnaire, based on the \textit{DSM-III}, Revised (\textit{DSM-III-R}) identified 5.2\% of the sample as having symptomatology of ADHD that would be classified as clinical (i.e., if every child had a formal diagnosis). This is on the low side in terms of the reports on the population prevalence of ADHD and reflects the fact that researchers in this study chose to exclude children who were comorbid for other behavioral disorders (e.g., conduct disorder) from the final data analysis. Other exclusionary criteria resulted in a study sample of 101 children with ADHD, who were then administered a more comprehensive set of tests measuring attention, IQ, and language. About half of these children (45\%) also demonstrated moderate-to-severe levels of LI, as measured on standardized assessments.

Some researchers have raised concerns over IQ as a potential confound in the discussion of the comorbidity between LI and ADHD (e.g., see Redmond & Rice, 1998). Most studies, however, have not controlled for IQ as a factor, either in their study design or in their analyses. When Tirosh and Cohen (1998) compared children with ADHD with children comorbid for ADHD and LI, they found no significant difference between the two groups on measures of performance IQ. Differences were seen on full-scale and verbal IQ measures, with the comorbid group scoring significantly lower than the group with ADHD, but these are in the direction expected given the poor language abilities of the comorbid group. This study is interesting because it suggests that the association between LI and ADHD is not necessarily mediated by IQ.

**COMORBIDITY IN CLINICALLY IDENTIFIED SAMPLES**

As noted at the outset to this discussion, the majority of research on the comorbidity of LI and ADHD has been based on samples that are clinically ascertained; within this set, the majority of LI and ADHD have been concerned with language abilities of children in psychiatric samples (see Table 1).

Findings from clinically referred samples are more difficult to interpret because, by definition, they are ascertained on the basis of one or other of the disorders. Because the total sample space is now defined by the presence of the first disorder (as opposed to a general population parameter), overlap between the disorders is artificially increased. Berkson (1946) suggested that the relative frequency of disease in a population sampled from the hospital or clinical setting is inherently biased when compared with the whole population because sampling in this way likely leads to an oversampling of comorbid cases. As a consequence, the comorbid group will represent a phenotypically “enriched” sample, that is, the sample is more likely to comprise individuals who exhibit either a greater number or greater severity of symptomatology than those from a population-based sample. A byproduct of this is that sampling comorbidity in clinical populations will alter the strength and profile of the observed association between disorders.

As can be seen in Table 1, most clinically ascertained studies of comorbidity have not included a control sample. The exception to this is a study by Benasich, Curtiss, and Tallal (1993), who reported the rate of psychopathology in children with LI versus a typically developing (TD) control group. The most prominent psychopathology was ADHD, which occurred in 18\% of the children with specific language impairment (SLI) and was not observed in the controls. Because the rate of ADHD in the controls was 0\%, we could not compute relative risk in this sample. Even in cases where clinically ascertained samples
do include a separate control group, cases and controls are not necessarily ascertained in the same way. For example, “controls” may, in fact, be patients with another diagnosis (e.g., children with asthma). This is often the case when studies are carried out via hospital or inpatient clinics. In the absence of a randomized control group, it becomes impossible to discuss comorbidity in terms of population prevalence or relative risk. Furthermore, because the sample is drawn from a larger clinical population, children with the disorder of interest may be over- or underrepresented in that sample. For example, children with ADHD tend to be studied as part of a larger group of children with socioemotional problems under the umbrella of psychiatric disorders. The possibility of related disabilities is thus harder to sort out in studies that are clinically ascertained. It therefore follows that findings from these studies should be interpreted with some caution and be viewed only in terms of the sample population on which they are based.

LANGUAGE IMPAIRMENT IN CHILDREN WITH CLINICALLY IDENTIFIED ADHD

From Table 1, we can see that rates of LI reported in children with ADHD vary widely across studies that have been clinically ascertained. This is most likely for reasons discussed earlier (e.g., Berkson’s bias, differences in ascertainment). Despite this variation, most studies report a rate of LI that is considerably higher than prevalence estimates in the population, with estimates frequently exceeding 50%, and as many as 90% of children with ADHD suggested to have coexisting language problems (Camarata, Hughes, & Ruhl, 1988; Gualtieri, Kouriath, van Bourgondien, & Saleebey, 1983; Love & Tompson, 1988; Tirosh & Cohen, 1998; Trautman, Giddan, & Jurs, 1990). Moreover, a sizeable proportion of these children with LI were previously undiagnosed. That is, there were many instances of LI among children with ADHD that remained unidentified until a (standardized) language assessment was carried out (i.e., as part of a research study (Cohen, Barwick, Horodezky, Vallance, & Im, 1998; Cohen, Davine, Horodezky, Lipsett, & Isaacson, 1993; Cohen, Davine, & Meloche-Kelly, 1989). In explaining this, Tannock and Schachar (1996) have suggested that children with unidentified language problems likely exhibit fewer difficulties with the expressive and social aspects of language, which is why a diagnosis of LI is overlooked. In addition, poor comprehension abilities may be misattributed by parents and teachers as inattention and/or oppositional behavior (Howlin & Rutter, 1987). Although receptive and expressive language skills are primary behavioral categories considered in the diagnosis of ADHD, standardized language assessments are rarely a routine part of a psychiatric evaluation. Moreover, speech–language pathologists are typically not included in the multidisciplinary team that makes the diagnosis. Potential confounds arising from linguistic biases in the assessment of ADHD are discussed in more detail later.

ADHD IN CHILDREN WITH CLINICALLY IDENTIFIED LI

Evidence from both cross-sectional and prospective longitudinal studies of children with S/LI also suggests there is heightened risk for ADHD among children with S/LI (Baker & Cantwell, 1987; Cantwell & Baker, 1987; Beitchman et al., 1996; Beitchman, Hood, Rochon, & Peterson, 1989; Beitchman, Hood, Rochon, Peterson, Mantini, et al., 1989; 1996; Benasich et al., 1993; Redmond & Rice, 1998, 2002; Snowling, Bishop, Stothard, Chipchase, & Kaplan, 2006). The largest study of this kind by Baker and Cantwell performed a prospective follow-up on the psychiatric status of 600 children ascertained on the basis of

*As a reminder, population prevalence of LI and ADHD has been estimated to be 7%–12% and 8%–12%, respectively (Faraone, Sergeant, Gillberg, & Biederman, 2003; Tomblin, Records, & Zhang, 1996).
receiving speech and language services (Baker & Cantwell, 1987; Cantwell & Baker, 1987). They reported the rate of ADHD in children with LI to be 16% at the time of the initial study. When this sample was reviewed 5 years later, this figure had more than doubled, rising to 37%. Language, but not speech, in the initial study was predictive of psychiatric status at follow-up. It, therefore, appears that some forms of communication disorders carry greater risk for ADHD than others. In their sample, Beitchman, Hood, Rochon, & Peterson (1989); Beitchman, Hood, Rochon, Peterson, Mantini, et al. (1989), and Beitchman et al. (1996) noted a particularly poor prognosis for children with receptive language problems.

There is also some evidence for an association between lack of improvement in speech-language functioning and ADHD (Beitchman et al., 1996; Benasich et al., 1993; Snowling et al., 2006). Snowling et al. (2006) looked at the psychosocial outcomes of children with a preschool history of S/LI when they were 15 years of age. Only children whose language delay had resolved by school age had a good outcome. For those whose language difficulties persisted into the school years, there was a raised incidence of attention and social difficulties. These difficulties were partially associated with different language profiles. When the group was subdivided, the group with attention problems showed a profile of specific expressive language difficulties. In comparison, the group with both attention and social difficulties showed global language difficulties and low IQ.

Redmond and Rice (1998, 2002) examined ADHD symptomatology in children with and without SLI at kindergarten and first grade. Although they found means in the SLI groups on the Attention subscales of the Child Behavior Checklist (CBCL) and the Teacher Report Form (TRF) to be significantly higher than controls, these means were still within the normal range for standardized norms (Achenbach, 1991a, 1991b). One possible explanation for this finding is that children with poor nonverbal IQ were excluded from this sample (i.e., children in the group with SLI were selected on the basis of poor language abilities and performance IQ ≥85). This constraint may have limited the sample to include only children with mild levels of LI and ADHD. Recently, Dennis et al. (2009) have provided a strong case against controlling IQ in studies of neurodevelopmental disorders. Also, these studies were based on very small samples, and lack of power could account for the null findings.

**BASES FOR THE COMORBIDITY BETWEEN LI AND ADHD**

As outlined by Tomblin and Mueller (2012), multiple models have been proposed to explain the possible bases of comorbidity between two co-occurring conditions. One possibility is that biases in raters and measurement scales affect assessment of the disorders (e.g., Model 2 as outlined in Tomblin & Mueller, 2012). At the outset, the assessment of LI and ADHD seem completely different: LI is measured by standardized language tests, whereas ADHD is defined by symptom checklists, behavior rating scales, and diagnostic interviews. Closer examination, however, reveals that many items on standardized ADHD assessment and rating scales rely on the rater making judgments of the child’s communication skills to make inferences about their underlying socioemotional competence (Table 2). Redmond (2002) has outlined cautions associated with using behavioral rating scales on children with LI, particularly for differentiating between receptive language difficulties and symptomatology of inattention (see also Tannock, 2005).

Charach, Chen, Hogg-Johnson, and Schachar (2009) showed that the risk for misdiagnosis raised by Redmond (2002) is more than just a theoretical concern. In a sample of children referred for clinical assessment of ADHD, children with LI represented 19% of the observed false positives associated with standardized teacher rating scales on the Conners Teacher Rating Scales-Revised (Conners, 2001; i.e., 19% of children...
Table 2. Examples of items in ADHD behavior rating scales that have a linguistic bias

<table>
<thead>
<tr>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Inattention</td>
<td>Competence items</td>
</tr>
<tr>
<td>Often does not seem to listen when</td>
<td>Performance in academic subjects</td>
</tr>
<tr>
<td>spoken directly</td>
<td>Is your child in a special class or school?</td>
</tr>
<tr>
<td>Often does not follow through on</td>
<td>Has your child repeated a grade?</td>
</tr>
<tr>
<td>instructions and fails to finish</td>
<td>Has your child had any academic problems</td>
</tr>
<tr>
<td>schoolwork, chores, or duties in the</td>
<td>or other in school?</td>
</tr>
<tr>
<td>workplace</td>
<td></td>
</tr>
<tr>
<td>Hyperactivity</td>
<td></td>
</tr>
<tr>
<td>Often has difficulty playing or engaging</td>
<td></td>
</tr>
<tr>
<td>in leisure activities quietly</td>
<td></td>
</tr>
<tr>
<td>Impulsivity</td>
<td></td>
</tr>
<tr>
<td>Often talks excessively</td>
<td></td>
</tr>
<tr>
<td>Often blurts out answers before questions</td>
<td></td>
</tr>
<tr>
<td>have been completed</td>
<td></td>
</tr>
<tr>
<td>Often interrupts or intrudes on others</td>
<td></td>
</tr>
<tr>
<td>(e.g., butts into conversations or games)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>(Conners, 2001)</td>
<td>Conners Comprehensive Behavior Rating Scales (Conners, 2001)</td>
</tr>
<tr>
<td>Does not seem to listen</td>
<td>Does not seem to listen</td>
</tr>
<tr>
<td>Difficulty doing homework</td>
<td></td>
</tr>
<tr>
<td>Does not seem to listen</td>
<td></td>
</tr>
</tbody>
</table>

Note. ADHD = attention-deficit/hyperactivity disorder;

incorrectly identified as having ADHD were children with LI). This contrasted with only 9.5% true positives (i.e., only 9.5% of children who were comorbid for LI and ADHD were correctly identified as such), which represents a statistically significant difference ($p = .009$).

Tallal, Dukette, and Curtiss. (1989) performed a discriminant analysis of the 1983 version of the CBCL (Achenbach & Edelbrock, 1983) and found that items concerned with judgments of children’s language skills loaded onto multiple subscales of the CBCL, indicating that speech-language problems are inherent to the symptomatology of many psychiatric disorders. Rice and Redmond (1998) compared parent and teacher ratings of children with and without LI on two assessments commonly used in the assessment of ADHD, the CBCL/4-18 and the TRF (Achenbach, 1991a, 1991b). They reported cross-informant ratings were only modestly associated. Teachers endorsed symptomatology of ADHD in children with LI more frequently than parents, and the rate at which they did this was significantly different to how they rated controls, $\chi^2(1) = 5.40, p < .05$. When Redmond and Rice (1998) removed the three items that are most obviously linguistic in nature from the TRF and reanalyzed the data, the apparent difference between parent and teacher ratings of ADHD symptomatology disappeared.

There are two explanations for this finding. The first is simply that teachers are misattributing language problems as attention problems. The second is that the classroom setting requires more from the child in terms of attention; thus, deficits are more likely to show up in this area. Empirical evidence suggests that children with LI may have deficits in selective attention (Stevens, Fanning, Coch, Sanders, & Neville, 2008) and sustained attention (Finneran, Francis, & Leonard, 2009; Spaulding, Plante, & Vance, 2008), which can be seen as important prerequisites for learning and perhaps explains why attention deficits are more likely to be reported by teachers than by parents.

Summary

Although there is considerable variation, studies examining rates of LI in children with ADHD, as well as those examining rates of ADHD in children with LI, indicate there is a high rate of co-occurrence between the two disorders. The overlap has been described
as asymmetrical, in that more children with ADHD have co-occurring S/LI, average 50%, than children with S/LI have ADHD, average 20% (Tannock & Schachar, 1996). As outlined by Tomblin and Mueller (2012), many possible models have been proposed to explain the phenomenon of comorbidity. If the assertion of Tannock and Schachar is correct, it would be compatible with a model in which ADHD increases the risk for LI but has a separate and distinct etiology of ADHD as a disorder occurring in isolation. This would be Model 4 in the Tomblin and Mueller taxonomy, “Three Independent Etiologies,” in which disorders A, B, and AB have independent risk factors but shared symptomatology.

At present, the literature is surprisingly limited with respect to well-designed population studies on the comorbidity of LI and ADHD and is therefore limited in empirical ways that make interpretation regarding the bases of comorbidity problematic. Nevertheless, we can reject some models on the basis of existing evidence. First, comorbidity is not likely due to the chance co-occurrence of two unrelated disorders. Both clinically ascertained and population-based samples demonstrate rates of co-occurrence between LI and ADHD that are well above the expected rates, given the population at large; neither is the degree of overlap so great that LI and ADHD can be assumed to be the same disorder (i.e., there is no one-to-one correlation). Evidence that co-morbidity of LI and ADHD is common to both community (Beitchman et al., 1986, 1996; Beitchman, Hood, Rochon, & Peterson, 1989; Camarata et al., 1988) and clinical samples (Baker & Cantwell, 1987; Cantwell & Baker, 1987; Cohen et al., 1989, 1993, 1998, 2000; Gualtieri et al., 1983; Love & Tompson, 1988) suggests that this phenomenon is not an artifact of sampling bias (Model 1, proposed by Tomblin & Mueller, 2012).

We acknowledge that the assessment of ADHD and, therefore, assessing its comorbidity with LI, is difficult because certain items in ADHD rating scales are linguistically biased (i.e., Model 2, proposed by Tomblin & Mueller, 2012). Studies that have controlled for cognitive abilities in the study of LI and ADHD are rare, and we must continue to entertain the possibility that comorbidity is mediated by this separate, though not unrelated, “third party” factor (Model 4). We also have seen that there are few studies that are well designed to examine comorbidity. The scarcity of population-based samples means it is hard to gauge the relative magnitude of the relationship between LI and ADHD in the forms of prevalence estimates and relative risks (i.e., Model 3).

In the current study, we draw data from a large population-based sample of children with the aim of addressing some of these issues. We also examine whether there is evidence for shared liability between these disorders in the form of familiality, either cultural or genetic (Models 5 and 6 proposed by Tomblin & Mueller, 2012). Specifically, we are interested in whether a family history of communication disorders is associated with either ADHD or comorbidity of LI and ADHD.

**COMORBIDITY OF LI AND ADHD IN A POPULATION SAMPLE: THE IOWA COHORT**

Children in this study were originally ascertained as part of a larger epidemiological study on the prevalence of language disorders in kindergarten (Tomblin, Records, et al., 1997; Tomblin et al., 1996; Tomblin, Smith, & Zhang, 1997). A subset of these children was followed throughout their school years into adulthood. The data for the present analysis were obtained when the children were around 10 years old and typically in fourth grade (Table 3).

**METHODS**

**Sample and participant selection**

**Screening phase**

The original pool of participants (N = 7,218) was screened in kindergarten as part of a large cross-sectional epidemiological study on the prevalence of language disorders.
Table 3. Cohort characteristics and sample size across the waves of the study

<table>
<thead>
<tr>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
<th>Wave 4</th>
<th>Wave 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nominal grade</td>
<td>Kindergarten</td>
<td>2nd</td>
<td>4th</td>
<td>8th</td>
</tr>
<tr>
<td>Mean age, years</td>
<td>5.9</td>
<td>8.0</td>
<td>9.9</td>
<td>13.9</td>
</tr>
<tr>
<td>Range, years</td>
<td>5.3–6.7</td>
<td>7.1–9.0</td>
<td>9.1–10.9</td>
<td>13.1–15.4</td>
</tr>
<tr>
<td>Total, N</td>
<td>604</td>
<td>604</td>
<td>570</td>
<td>527</td>
</tr>
</tbody>
</table>

in childhood (Tomblin, Records, et al., 1997). The screening tool, which was composed of 40 items from the Picture Vocabulary, Sentence Imitation, and Grammatic Completion subtests of the Test of Language Development-Primary: Second Edition (TOLD-P:2; Newcomer & Hammill, 1988) was developed especially for this purpose and had a very high predictive accuracy for diagnostic outcome. A more detailed description of these methods is available elsewhere (Tomblin, 2010; Tomblin et al., 1996; Tomblin, Records, et al., 1997; Tomblin, Smith, et al., 1997).

Diagnostic phase

Following the screening phase, a stratified-cluster sampling method was used to invite schools and individuals to participate in the diagnostic phase of the epidemiological study (Tomblin, Records, et al., 1997). Children who failed the language screening test, plus a random sample who passed (approximately 33%), were administered a more comprehensive set of language and cognitive assessments as a diagnostic battery for LI (n = 2,009). These are described in detail elsewhere (Tomblin, Records, et al., 1997; Tomblin et al., 1996). Briefly, children completed five language subtests from the TOLD-P:2: Picture Vocabulary, Oral Vocabulary, Grammatic Understanding, Sentence Imitation, and Grammatic Completion, as well as the test of Word Articulation (Newcomer & Hammill, 1988). These scores were converted into composite language ability scores and language diagnoses, which were used to estimate the prevalence of language disorder in kindergarten (Tomblin, Records, et al., 1997). The Block Design and Picture Completion subtests of the Wechsler Preschool and Primary Scale of Intelligence-Revised (Wechsler, 1989) were also used to obtain estimates of performance IQ. All children included in the present analysis were required to have a performance IQ greater than 85 and normal hearing. To minimize potential confounds, children who did not have English as their primary

Table 4. Characteristics of the children with language impairment and normal language status at Wave 3 (fourth grade) by gender, kindergarten language ability, performance IQ, and mother's education

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>% Males</th>
<th>Composite Language Score (z-Score)</th>
<th>Performance IQ</th>
<th>Mother's Education, years</th>
</tr>
</thead>
<tbody>
<tr>
<td>TD</td>
<td>385</td>
<td>57.7</td>
<td>0.03</td>
<td>98.0</td>
<td>13</td>
</tr>
<tr>
<td>LI</td>
<td>187</td>
<td>53.81</td>
<td>−1.67</td>
<td>88.1</td>
<td>12</td>
</tr>
</tbody>
</table>

Note. LI = language impairment (≥2 language composite scores below −1.25 SD for age, normal hearing, absence of other developmental disorders such as autism, cerebral palsy, or mental retardation); TD = typically developing (no more than 1 language composite score out of 5 possible below −1.25 SD for age).
language were excluded from the study. Table 4 provides a description of the sample in kindergarten with regard to gender, language status, nonverbal IQ, and parental education.

**Longitudinal follow-up Wave 3**

From the cross-sectional sample, a subgroup of 604 children went on to participate in the longitudinal study. In Wave 3 of the study, teachers were asked to complete a questionnaire with items from the *DSM, Fourth Edition (DSM-IV)* checklist for ADHD as part of an assessment of children’s psychosocial development (see Mueller & Tomblin, 2012). This provided an overall severity rating of ADHD symptomatology on a scale of 0–18, as well as information on the ADHD subtypes. Teachers also completed the *Teacher's Report Form* (Achenbach, 1991b). Parents were asked to complete the companion to the TRF, the *CBCL/4-18* (Achenbach, 1991a). Both the TRF and the CBCL have a quantitative Attention subscale. This is commensurate with the *DSM–III* definition of ADHD and includes questions on both inattentive and hyperactive behaviors. In addition, parents were asked whether their child had ever received a clinical diagnosis or medication for the treatment of ADHD (Table 5).

We did not include data from the TRF in the present analysis because many teachers failed to complete this form, and doing so would have severely limited the size of the sample. It must be emphasized that the CBCL and the DSM–IV checklist were not intended to replace a clinical diagnosis; as shown in Table 5, there were questions in this study that asked specifically about whether the child had been clinically identified with ADHD. However, these measures are frequently used as part of the clinical diagnostic process for ADHD and have also been used extensively in research. In the present study, we used them to assign a risk likelihood for ADHD, as described later.

**Establishing risk for ADHD**

In accordance with *DSM–IV* diagnostic standards on the cross-situational nature of ADHD, our analyses included only children for whom both parent CBCL and teacher *DSM–IV* data were available. This resulted in a loss of 42 children from the study, which, combined with overall attrition of the sample between kindergarten and fourth grade, resulted in a sample of 530. Of these children, 73 had LI. A risk for ADHD was then calculated for each child on the basis of (1) teacher endorsement of six or more symptomatology of inattention or hyperactivity/impulsivity on the subscales of the *DSM–IV* checklist or (2) a T-score greater than 60 (< -1 SD) on the CBCL Attention subscale. Children were then divided into groups depending on whether (1) both parent and teacher had endorsed symptomatology of ADHD (high risk for ADHD; ADHD-HR), (2) either teacher or parent had endorsed symptomatology of ADHD (moderate risk for ADHD; ADHD-MR), and (3) neither parent nor teacher had endorsed symptomatology of ADHD (low risk for ADHD; ADHD-LR). Because children with LI had been oversampled as part of the original longitudinal study, a weighting scheme was used to adjust the current sample to represent the original study population from which it was drawn (see Tomblin, Records, et al., 1997). All further analyses are subject to this weighting scheme. The CBCL does not differentiate between ADHD subtypes, so teacher report on the *DSM–IV* checklist was used to further subdivide children in the ADHD-HR and ADHD-MR groups into ADHD-Inattentive (ADHD-IA), ADHD-Hyperactive/Impulsive (ADHD-H/I), and ADHD-Combined (ADHD-C) subtypes.

**Table 5. Questions concerning history of ADHD diagnosis and treatment**

<table>
<thead>
<tr>
<th>Question</th>
</tr>
</thead>
<tbody>
<tr>
<td>Has doctor ever told you that your child has ADD?</td>
</tr>
<tr>
<td>Has a doctor ever told you that your child has ADHD?</td>
</tr>
</tbody>
</table>

*Note. ADD = attention-deficit disorder; ADHD = attention-deficit/hyperactivity disorder.*
Family history of communication disorders

As a part of a questionnaire regarding the child’s developmental history, parents were asked to indicate whether either biological parent had a history of speech, language, or reading impairments. Previously, Tomblin (1989) has shown that although a family history of S/LI does not in itself predict whether a child is at risk for developing these disorders, a family history of reading impairment does. This is likely because speech and language problems are less well recognized than reading problems. Given that reading impairment is often grounded in LI, a family history in which either biological parent had a history of speech, language, and/or reading impairment (collectively termed communication disorders) was considered as a positive family history for the purposes of this study.

RESULTS

As already described, risk for ADHD in this sample was attributed on the basis of parent and teacher report of ADHD symptomatology. Although the CBCL and DSM–IV checklist are routinely used in the diagnosis of ADHD, independent validation was possible in this study by examining the extent to which these measures correlate with formal diagnoses of attention deficit disorder or ADHD. These data are presented in Table 6. As noted earlier, this sample contained an excess of children with LI (35.85%) because of an intentional oversampling of children with this disorder as part of the original longitudinal study. We therefore weighted the sample so that it was representative of the population from which it was drawn before performing further analyses (see Tomblin, Records, et al., 1997).

A chi-square test confirmed that parental and teacher report were significantly associated with a clinical diagnosis of ADHD, \( \chi^2(2, N = 517.6) = 68.02, p < .0001 \). If a clinical diagnosis is the gold standard, our data show that the CBCL and DSM–IV rating scales are fairly sensitive as proxy measures for a formal diagnosis and support current research practices of using these as proxy measures. Most children with a formal diagnosis of ADHD (81.2%) were assigned to one of the two ADHD risk groups (ADHD-MR or ADHD-HR) on the basis of parent and teacher report. These tests were also found to be moderately specific, that is, most of the children without a history of ADHD (70.8%) were correctly assigned to the ADHD-LR group. The majority of classification errors were in the direction of children being classed as at risk for ADHD on the basis of parent and teacher report but without a formal diagnosis. Intuitively this makes sense; it is unlikely that all children with ADHD symptomatology have a diagnosis because not all will have access to clinical services.

Prevalence of ADHD and LI

After weighting, the rate of LI in this sample was 14%. The theoretical rate of LI, based on the diagnostic scheme outlined by Tomblin et al. (1996), has also been estimated as 14%. From this, we can see that weighting the sam-

| Table 6. Correspondence between parent and teacher assignment of risk for ADHD and formal diagnosis |
|----------------------------------|----------------|----------------|
| ADHD-HR\(^a\) | ADHD-MR\(^b\) | ADHD-LR\(^c\) |
| Positive history | 3.9 | 4.5 | 1.95 |
| Negative history | 7.5 | 18.9 | 65.46 |

Note. ADHD = attention-deficit/hyperactivity disorder, HR = high risk, MR = moderate risk, LR = low risk.

\(^a\)High risk for ADHD: both parent and teacher had endorsed symptomatology of ADHD.

\(^b\)Moderate risk for ADHD: either teacher or parent had endorsed symptomatology of ADHD.

\(^c\)Low risk for ADHD: neither parent nor teacher had endorsed symptomatology of ADHD.
ple provides a good approximation of population prevalence. The rate of ADHD, before weighting, was 30% for ADHD-MR and 16% for ADHD-HR (46% total). After weighting, this fell to 24% for ADHD-MR and 11% for ADHD-HR (35% total). The prevalence of ADHD in the general population is estimated to be in the range of 8%–12%. This is very similar to the rate we found in the ADHD-HR group, which is our most conservative estimate of risk likelihood.

**Prevalence of ADHD subtypes**

Using items from the *DSM-IV* checklist, we computed prevalence rates for each of the three ADHD subtypes. After weighting, the prevalence of ADHD-IA was 13%, ADHD-H/I was 2%, and ADHD-C was 6.1%. There were only 10 children in the ADHD-H/I subtype, so the confidence interval for this prevalence estimate was quite high. This is consistent with prior literature showing that ADHD-H/I is the least commonly occurring of the subtypes. These combine to give an overall prevalence of 21%, which is similar to the rate for ADHD-MR group. This is as expected, given that information for subtyping comes from the *DSM-IV* checklist, which was completed only by teachers but not by parents.

**Comorbidity of ADHD and LI**

Using the three possible risk categories for ADHD (high, medium, and low) and the two possible categories of language status (LI and TD), we computed estimates of prevalence for each joint condition. These are shown in Table 7. A chi-square test showed significant association between language status and risk for ADHD, $\chi^2(2, N = 519.6) = 31.42, p < .0001$. When ADHD-HR is tested separately from ADHD-MR and then tested with ADHD-LR, both were significantly associated with LI, $\chi^2(1, N = 396.93) = 26.80, p < .0001$, and $\chi^2(1, N = 461.5) = 17.06, p < .0001$, respectively. As shown in Table 7, the comorbidity of LI with ADHD-HR is 3.5% compared with 5.2% for LI with ADHD-MR. We can estimate the rate at which we expect two disorders to co-occur by chance by multiplying the prevalence rates of each of the separate disorders. We have previously calculated these as follows: LI = 0.14; ADHD-MR = 0.23; and ADHD-HR = 0.16. Next, we tested whether rates of comorbidity in this sample were significantly different to that expected by chance alone. We found that both LI with ADHD-HR, $z(N = 532) = 6.59, p < .0001$, and LI with ADHD-MR, $z(N = 532) = 6.59, p < .0001$, exceeded chance expectations. Thus, we can conclude that the risk for ADHD and LI is significantly associated in this sample.

**Relative risk for LI and ADHD**

To test whether there is a greater level of risk for ADHD given LI, or vice versa, we can compute relative risks. As has been noted earlier in this article, relative risk reflects the degree to which the rate of a disorder changes as a result of a particular exposure or condition (i.e., risk factor), with a test statistic centered around 1 (i.e., null effect on risk).

**Table 7.** Population estimates of the prevalence rate (percentage of total population) of the joint occurrence of High, Medium or Low ADHD risk levels with LI or TD status

<table>
<thead>
<tr>
<th>LI status</th>
<th>% of Population</th>
<th>ADHD Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>LI</td>
<td>14</td>
<td>High risk</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3.52</td>
</tr>
<tr>
<td>TD (non-LI)</td>
<td>86</td>
<td>7.66</td>
</tr>
</tbody>
</table>

*Note. ADHD = attention-deficit/hyperactivity disorder; LI = language impairment; TD = typically developing.*

*aRates represent estimates of population prevalence based on weighted values using weights that adjust for oversampling of children with poor language in the study sample (Tomblin, Records, et al., 1997).*
**Relative risk for ADHD when LI is the risk factor**

First, we looked at the risk for ADHD as a function of language status. Figure 1 shows that the rates of ADHD-MR and ADHD-HR in children with LI are greater than those that of the TD controls (49% in the ADHD-MR group and 40% ADHD-HR group, contrasting with 23% and 8% in the TD group, respectively). These result in a relative risk of 3.52 for the ADHD-HR group, which is significantly greater than 1 ($z = 5.34, p < .0001$), indicating that LI does affect the risk for ADHD. The relative risk value for the ADHD-MR given LI = 2.77, which is also significant ($z = 4.07, p < .0001$). From this, we can conclude that, although the rate of comorbidity between LI and ADHD is higher in ADHD-MR group than in the ADHD-HR group, the relative risk for ADHD given LI is greater for the ADHD-HR group than for the ADHD-MR group.

**Relative risk for LI when ADHD is the risk factor**

Relative risk for LI was computed for each of the three ADHD risk groups, and results are displayed in Figure 2. From this, we can see that the risk for LI varies as a function of ADHD status. The base rate of LI is 8% in the ADHD-LR group, which is essentially the same as the population prevalence and is as we would expect, given that children in this group are essentially without ADHD symptomatology. This rate rises to 22% in the ADHD-MR group and is highest for the ADHD-HR group at 31%. The relative risk for LI conditioned upon ADHD was 3.9 for the ADHD-HR group, which is significantly greater than 1 ($z = 5.05, p < .0001$). The relative risk was 2.8 in the ADHD-MR group, which is also significantly greater than 1 ($z = 4.07, p < .0001$). Again, we see that the relative risk varies as a function of ADHD severity, with the more severe phenotype resulting in a higher risk for comorbidity.

These data further support the likelihood of an association between LI and ADHD. Although the rate of ADHD in children with LI is higher than the rate of LI in children with ADHD, this is likely because of the differences in the base rates for the two disorders. When relative risks are compared, the relative risk for ADHD given LI and the relative risk for LI given ADHD are quite similar: $LI \mid ADHD-HR = 3.9$, ADHD-HR $\mid LI = 3.5$; and $LI \mid ADHD-MR = 2.8$, ADHD-MR $\mid LI = 2.8$. The symmetry of these indicates that we cannot assume any directionality to relationship between LI and ADHD. We do not see that one disorder exerts a greater influence over the other or that one is nested within the other.

**Comorbidity of subtypes of ADHD**

Previous literature suggests that comorbidity of LI is highest in ADHD-IA, followed by ADHD-C, and is relatively low in ADHD-H/I.
Table 8. Prevalence rates (proportion of total sample) of the joint occurrence of ADHD and LI

<table>
<thead>
<tr>
<th>LI status</th>
<th>ADHD Subtype</th>
<th>Inattentive</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>LI</td>
<td>.036</td>
<td>.018</td>
<td></td>
</tr>
<tr>
<td>TD</td>
<td>.096</td>
<td>.043</td>
<td></td>
</tr>
</tbody>
</table>

Note. ADHD = attention-deficit/hyperactivity disorder; LI = language impaired; TD = typically developing.

Table 8 shows prevalence rates for the co-occurrence of LI by ADHD subtype in the current sample. There were not enough children in the ADHD-H/I group to examine comorbidity in this group. A chi-square test for the overall association of LI by ADHD subtype was, however, significant $\chi^2(3, N = 568.9) = 20.34, p < .0001$. Breaking this down by subtype, we see that, as expected, LI occurs most frequently with the ADHD-IA subtype (3.6%). When we test the strength of association between LI by ADHD subtype, LI is significantly associated with ADHD-IA $\chi^2(1, N = 522.8) = 13.34, p < .0001$, and ADHD-C $\chi^2(1, N = 481.9) = 9.8, p < .002$. It is not associated with ADHD-H/I $\chi^2(1, N = 458.5) = 0.39, p = .53$.

Relative risks for LI by ADHD subtype

We also examined the relative risk for LI by ADHD subtype. Again, we were interested in the symmetry between this and ADHD conditioned on LI, which might suggest that one condition is antecedent to the other. The relative risk for LI conditioned on ADHD-IA was 2.34 ($z = 3.65, p = .0084$). When this relationship was reversed, and ADHD-IA was conditioned on LI, the relative risk was 2.36 ($z = 3.68, p = .008$). These rates are very similar. The relative risk for LI conditioned on ADHD-C was 2.60 ($z = 3.19, p = .0001$), whereas the relative risk for ADHD-C conditioned on LI was 2.91 ($z = 3.06, p = .00026$). Again, on the basis of the subtype, we cannot conclude that there is directionality to the relationship between LI and ADHD. The relative risks for ADHD-H/I and LI were not significant because of the low number of children in the ADHD-H/I group.

Family history of communication disorders as a risk factor for ADHD

Given that a family history of communication disorders is a predictor of LI, we hypothesized that it would also be a risk factor for ADHD. Figure 3 shows the rates of family history by the ADHD risk group. There is a clear trend to the data, with rates increasing as the risk for ADHD increases, and this trend is significant, Mantel-Hansel $\chi^2(2, N = 488) = 10.48, p = .0012$.

Returning to relative risks as a means to characterize the strength of this relationship, the relative risk for ADHD-HR given a positive family history of communication disorders was 1.81, which is significantly different from 1, $z = 2.5, p = .01$. In comparison, the relative risk for the ADHD-MR was 1.30, which is not significantly different, $z = 1.64, p = .10$. It therefore appears that a family history of communication disorders only increases the risk for ADHD when high levels of ADHD symptomatology are reported in the child.

Finally, we wanted to see whether any evidence of common etiology could be found in these data. If LI and ADHD share a common
Figure 4. Rates of a positive family history of communication disorders in children with ADHD and children comorbid for ADHD and LI. Note. ADHD = attention-deficit/hyperactivity disorder; ADHD-HR = high risk for ADHD; ADHD-MR = moderate risk for ADHD; LI = language impairment.

etiology, and this is familial, we would expect to see that children who are comorbid for the disorders would also show elevated rates of positive family histories for communication disorders. We compared rates of positive family history in children comorbid for LI and ADHD (LI and ADHD-HR or LI and ADHD-MR) with children without ADHD (ADHD-LR) irrespective of language ability. These data are shown in Figure 4. Again, we see that the rate of familial communication disorders increases as a function of comorbidity and ADHD severity (LI + ADHD-HR > LI + ADHD-MR > ADHD-LR). This overall relationship is significant, Mantel-Hansel $\chi^2(1, N = 522) = 6.55, p = .009$. The relative risk for comorbidity given a positive family history is 1.97 in the ADHD-HR and LI group, which is significantly different from 1, $\chi^2(1, N = 522) = 5.05, p = .02$. It is not, however, significant in the MR-ADHD group. Overall, we see that the risk for ADHD is positively associated with a family history of communication disorders, as is the comorbidity between ADHD and LI.

DISCUSSION

A review of the literature shows that LI and ADHD frequently co-occur and have, therefore, been said to be comorbid. Much of this research, however, has been carried out in clinical populations, which are influenced by factors, such as Berkson’s bias, that artificially inflate estimates. This is only the second study to examine the comorbidity of LI and ADHD in a sample that is population based. Children for this study were drawn from a larger cross-sectional study on the prevalence of LI in kindergarten. We, therefore, can say that the pattern of comorbidity observed in this study is an unbiased estimate of the general population from which these children were drawn.

Consistent with prior literature, we found that LI and ADHD were significantly associated, regardless of whether this was estimated conservatively, based on positive evidence from both parent and teacher (e.g., ADHD-HR), or less conservatively (ADHD-MR). We have estimated the comorbidity between LI and ADHD to be between 3% and 5%, depending on the ADHD risk group. These estimates are rather high for a chronic health condition.

Based solely on rates of co-occurrence, it appeared that children with LI were at greater risk for developing ADHD than children with ADHD were for LI. This pattern has been observed in previous studies (see Tannock & Schachar, 1996, p. 133). When comorbidity was computed in terms of relative risk, however, by controlling for differences in population base rates, we did not see this asymmetry. Both the risk for LI given ADHD and the risk for ADHD given LI are very similar when compared in terms of relative values, all in the range of 2–4. Relative risks reflect the degree of association between two disorders, rather like an effect size. Using these, we can see children with LI are 2–3 times more likely than children with typically developing language abilities to have ADHD. This effect is the same if LI is conditioned on ADHD.

Results from this analysis can be used to test various competing models of comorbidity (Neale & Kendler, 1995; Tomblin & Mueller, 2012), as they pertain to LI and ADHD. Tomblin and Mueller’s “alternate forms model” (Model 5), “correlated liabilities model” (Model 6), and “causal model” (Model 8) rely on the notion of shared, or
correlated etiologies, as the basis for comorbidity. These are compatible with the symmetrical pattern of overlap we see between LI and ADHD in the current sample. Thus, this pattern must be retained as possible explanations for the data. The “reciprocal causation” model (Model 7) also must be retained. In fact, we must be cautious in attempting to discern causality from these patterns of comorbidity, which are fundamentally correlational. We cannot distinguish further between these models, given the data in this study.

Family history of speech, language, or reading disorder and ADHD

Although we cannot completely rule out the “by-product” model of comorbidity (i.e., ADHD is a by-product of LI; LI is a by-product of ADHD), our data suggest that LI and ADHD share a common etiology. As noted by Mueller and Tomblin (2012), these disorders have been shown to be both familial and heritable, suggesting the possibility of a genetic basis. At present, however, we are unable to say how much of the phenotypic overlap between these disorders is due to shared genetic factors (i.e., how much of overlap is due to bivariate heritability). The strongest method for testing this is a twin design from which measures of genetic correlation can be calculated. Because this study comprised only unrelated probands, we were limited in investigating familiality by asking, “To what extent does a positive family history of speech, language, or reading problems confer risk for ADHD?” We found that a family history of communication disorders was positively associated both with ADHD and with comorbidity in LI and ADHD. Because the relationship between LI and ADHD appears to hold across generations, this points to cross-generational transmission of these disorders. This could be the result of shared environment, shared genes, or both.

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