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Lexical Processing Deficits in Children with Developmental Language Disorder: An Event-Related Potentials Study

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Abstract

Lexical processing deficits in children with developmental language disorder (DLD) have been postulated to arise as sequelae of their grammatical deficits (either directly or via compensatory mechanisms) and vice versa. We examined event-related potential indices of lexical processing in children with DLD (n = 23) and their typically developing peers (n = 16) using a picture–word matching paradigm. We found that children with DLD showed markedly reduced N400 amplitudes in response both to auditorily presented words that had initial phonological overlap with the name of the pictured object and to words that were not semantically or phonologically related to the pictured object. Moreover, this reduction was related to behavioral indices of phonological and lexical but not grammatical development. We also found that children with DLD showed a depressed phonological mapping negativity component in the early time window, suggesting deficits in phonological processing or early lexical access. The results are partially consistent with the overactivation account of lexical processing deficits in DLD and point to the relative functional independence of lexical/phonological and grammatical deficits in DLD, supporting a multidimensional view of the disorder. The results also, although indirectly, support the neuroplasticity account of DLD, according to which language impairment affects brain development and shapes the specific patterns of brain responses to language stimuli.

For the majority of children, language acquisition is an early, rapid, and seemingly effortless process. However, a sizable group of children struggles to acquire their native language despite the absence of apparent sensory (e.g., hearing) deficits, general cognitive (e.g., nonverbal intelligence) impairments, and other known psychiatric, genetic, and neurodevelopmental conditions (e.g., autism spectrum disorder or epilepsy). The prevalence of this communication disorder, termed developmental language disorder (DLD), has been estimated to be around 7% among preschoolers (Tomblin et al., 1997), thus rendering it one of the most prevalent neurodevelopmental disorders.¹ Despite the relatively high prevalence of DLD in the general population, its developmental continuity into adolescence and adulthood (Poll, Betz, & Miller, 2010; Stothard, Snowling, Bishop, Chipchase, & Kaplan, 1998), and its significant negative impact on children's academic, socioemotional, and

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occupational outcomes (Conti-Ramsden & Botting, 2008; Conti-Ramsden & Durkin, 2008; Durkin & Conti-Ramsden, 2007; Durkin, Conti-Ramsden, & Simkin, 2012; Wadman, Botting, Durkin, & Conti-Ramsden, 2011; Wadman, Durkin, & Conti-Ramsden, 2008), little is known about the cognitive, neural, and genetic etiologies of DLD.

Children with DLD are a heterogeneous population, and they show deficits in the development and functioning of multiple domains of spoken language, in both production and comprehension. Morphosyntactic deficits, manifested by failure to acquire and/or efficiently use grammar, have been proposed as a hallmark of the disorder. Children with DLD have documented deficits in expressive morphology (e.g., omissions or incorrect use of morphological forms in a sentence; Bedore & Leonard, 2001; Dromi, Leonard, Adam, & Zadunaisky-Ehrlich, 1999; Leonard & Eyer, 1996) and comprehension and production of complex syntactic structures such as wh-questions (Friedmann & Novogrodsky, 2011), verbal passives (Marshall, Marinis, & van der Lely, 2007), and relative clauses (Stavrakaki, 2001).

Lexical and phonological development deficits in DLD are frequently less severe than morphosyntactic deficits and, arguably, have been less frequently studied than the latter. Thus, children with DLD have been shown to have atypical or less detailed phonological representations and abnormal phonological processing compared to their typically developing (TD) peers (Claessen, Leitao, Kane, & Williams, 2013; Gray, Reiser, & Brinkley, 2012; Haake, Kob, Wilmes, & Domahs, 2013), as well as a markedly reduced phonological working memory capacity (for a meta-analysis, see Estes, Evans, & Else-Quest, 2007). In the lexical–semantic domain, empirical studies found deficits in learning and retaining new lexical items by children with DLD (Ellis Weismer & Hesketh, 1996), deficits in the size and depth of their vocabularies and semantic knowledge (Brackenbury & Pye, 2005; McGregor, Oleson, Bahnsen, & Duff, 2013; Sheng, Pena, Bedore, & Fiestas, 2013), and abnormal dynamics of spoken-word recognition (McMurray, Samelson, Lee, & Tomblin, 2010).

Atypicalities in the domains of lexical and phonological development in DLD contribute to the behavioral heterogeneity of the disorder but also have important implications for our understanding of both typical and atypical language development, especially in the context of developmental neuroplasticity. Because deficits in these domains are frequently less severe than grammatical deficits in DLD, they have been conceptualized as representing relatively spared domains of language development and functioning in DLD, or domains in which deficits are secondary to deficits in other linguistic and general cognitive systems, or both.

¹Although the term most commonly used in the literature to refer to a developmental (rather than acquired) disorder of language development in the absence of obvious explanatory factors is specific language impairment, we will use the DLD label when referring to this condition, consistent with our published empirical reports on the unique population we sampled from (Kornilov, Rakhlin, & Grigorenko, 2012; Rakhlin, Cardoso-Martins, et al., 2013; Rakhlin et al., 2011; Rakhlin, Kornilov, et al., 2013; Rakhlin, Kornilov, & Grigorenko, 2014) with an understanding that it is similar to the categories of expressive and mixed expressive–receptive language disorders in the DSM-IV-TR (American Psychiatric Association, 2001). We emphasize that all of the children classified as DLD in this study would satisfy the conventionally used inclusion and exclusion criteria for specific language impairment (i.e., below normal limits for language, but within normal limits for other cognitive domains).

In general, as the development of language unfolds in time and its facets (i.e., phonological, lexical, and morphosyntactic) do not mature simultaneously, it is plausible that they do not manifest similar degrees of deficits and that their brain correlates also do not manifest similar degrees of atypicalities. This reasoning corresponds to the general hypothesis of neuroplasticity, where the development of language has a significant impact on the development of the brain (Bishop, 2013). Thus, those facets of language development that are impaired lead to more, rather than fewer, pronounced atypicalities in the brain response to language stimuli.

Specifically, under the residual normality view, language development in children with DLD is relatively selectively affected in the domain of morphosyntax. For example, Fonteneau and van der Lely (2008) investigated neural responses of children with grammatical DLD (grammatical specific language impairment) to both syntactic and semantic violations. They showed that semantic violations (i.e., a noun that violated the verb's semantic [animacy] selection restrictions in auditorily presented sentences) produced a predicted robust electrophysiological response (N400; see below) in children with DLD, as well as TD children. Violations that relied on structural syntactic dependencies produced a robust early left anterior negativity (ELAN) component in TD children, postulated to index early automatic processing of structural dependencies. The ELAN component was not present in the data obtained from children with DLD, who instead displayed a later N400 in response to these violations (the absence of the ELAN nearly perfectly classified individual children as having DLD). Fonteneau and van der Lely (2008) suggested that these results support the presence of selective grammatical deficits in children with grammatical DLD with the appearance of the N400 indexing "a relative strength in semantic processing" (p. 4). Note that under this view, children's morphosyntactic deficits are functionally decoupled from their language ability in other (i.e., lexical) domains.

Moreover, the emergence of the N400 in children with DLD in response to syntactic violations can be viewed as the result of a neuroplastic change of the language-processing system in DLD, instead of (or in addition to) being indicative of residual normality of lexical-semantic processing in DLD. In this case, the apparent application of semantic processing strategies to syntactic violations indexes the developmental coupling of syntactic and lexical-semantic processing in the context of disordered language development. Although the disorder might seemingly selectively affect children's grammar, it has profound effects on the efficiency of the language system as a whole, resulting in plastic changes to its organization. For example, it has been proposed that children with DLD have deficits in the neural systems underlying procedural memory, whereas the declarative memory system (supporting the development of the lexical and semantic knowledge) is relatively intact (Ullman & Pierpont, 2005). According to this procedural deficit hypothesis account of DLD, some observed declarative deficits in DLD may be largely attributed to high demands placed on cognitive systems that partially overlap with the procedural memory system, such as working memory. Although specifying a relatively selective impairment in the procedural memory (and grammar) in DLD, this account also suggests that declarative memory might at least partially compensate for functions that rely on procedural memory (Lum, Conti-Ramsden, Page, & Ullman, 2012). This compensatory view is partially supported by a recent behavioral study that showed that children with DLD

are more susceptible to lexical–semantic priming than are their TD peers (Pizzioli & Schelstraete, 2011) and suggested that the overactivation of semantic associates by children with DLD is due to compensatory reliance on lexical semantics in the face of grammatical deficits. 2

The complex pattern of postulated developmental links between different domains of language impairments in DLD is likely driven by the extreme heterogeneity of DLD as a population that exists at least at three different yet related levels: behavioral, cognitive, and etiological. In her recent review of the limited number of studies of lexical learning and processing in DLD, Nation (2014) emphasized the importance of studying the role of lexical deficits in the etiology of DLD, for example, establishing whether lexical deficits are a consequence of deficits in other domains of linguistic and cognitive development. From our perspective, at the current stage of the development of the field, a prerequisite task is establishing the place of lexical-semantic deficits in the overall syndromic DLD profile and providing a detailed characterization of these deficits, that is, describing them at the level of the underlying neurobiology and relating them to deficits in other domains both correlationally and in the context of experimental manipulations that explicate interplays between different domains of language development (e.g., lexical and phonological). The study reported in this paper capitalizes on the multidimensional approaches to studying DLD while using neural endophenotypes of lexical processing and maintaining the neuroplasticity assumption that specifics of this processing characteristic of children with DLD shape the specifics of the brain's response to the corresponding stimuli. We will now touch upon both of these general ideas prior to reviewing psychophysiological studies of lexical processing in DLD published to date and describing the goals of the current study.

First, we note that the field of DLD is slowly transitioning toward much-needed dimensional approaches to understanding the nature of the disorder. Thus, the focus is shifting from trying to uncover the single "core" deficit that would explain the multitude of behavioral manifestations of DLD toward providing a functional characterization of the cognitive systems underlying different deficit domains and the relationships between them. For example, a recent study by Ramus, Marshall, Rosen, and van der Lely (2013) established that in a mixed sample of children with DLD and reading disability, phonological skills (i.e., phonological awareness, rapid naming, and phonological memory) were partially independent of nonphonological language skills (i.e., lexical and morphosyntactic). This study demonstrated that among children with DLD, only a subset of children had phonological deficits, and their profile of phonological deficits was different from that of children with reading disability. It also showed that language skills in these subgroups were likely dissociable given the absence of grammatical but not lexical deficits in children with reading disability. Together, these results support a multidimensional model for both the relationship between different components of phonological and nonphonological skills and the overlap between DLD and reading disability. These conclusions were also corroborated

²However, deficits in morphosyntactic development have been suggested to negatively impact lexical learning in children with DLD because of their compromised ability to use syntactic cues in word learning (Rice, Cleave, & Oetting, 2000). It is also noteworthy that deficits in working memory itself, in particular phonological working memory, have been postulated to drive lexical (Baddeley, Gathercole, & Papagno, 1998; Gathercole, 2006) and, at least partially, grammatical deficits in DLD (Montgomery, 2003; Montgomery & Evans, 2009).

in a recent study by Rakhlin, Cardoso-Martins, Kornilov, and Grigorenko (2013), who suggested the co-occurrence of grammatical and phonological deficits in DLD is partial and probabilistic rather than deterministic, as suggested by accounts that attribute the former to the latter. Of note is that the conclusions of both studies were based on a careful examination of sources of variance in the performance of groups of children with language-based neurodevelopmental disorders and the acknowledgment of the interactive nature of language as a developmental system (Karmiloff-Smith, 1998). Such examinations permit inference regarding the degree of the overlap between multiple domains of language development and functioning, highlighting the constellations of more and less interdependent deficits.

Second, the acknowledgment of the behavioral heterogeneity in DLD, coupled with inconsistent findings from studies of lexical-semantic development and processing that employ offline methods (Pizzioli & Schelstraete, 2011), suggest that using online methods of studying language and cognitive processing, in particular neuroimaging and psychophysiological (i.e., electroencephalography [EEG] and event-related potentials [ERPs]) methods, might be a productive approach to investigating the cognitive profiles of DLD and their brain correlates. Neurophysiological methods provide online measures of processing with high temporal resolution (up to 1 ms). In addition, neurophysiological measures have been proposed as endophenotypes (Cannon & Keller, 2006; Roeske et al., 2011) of developmental disorders, that is, intermediate phenotypes between the levels of observable phenotypic deficits and genetic etiology. Thus, endophenotypes provide a more sensitive and detailed characterization of representational and processing deficits at the level likely more proximal to the etiology of the disorder (i.e., neurocognitive) than end-point behavioral performance measures. To date, this approach has been mostly applied to studying neurophysiological endophenotypes of auditory and phonological processing in DLD (Addis et al., 2010; Bishop, 2007). However, obtaining a more comprehensive picture of linguistic and cognitive deficits in DLD requires expanding the spectrum of linguistic endophenotypes of the disorder. Lexical processing is an especially interesting candidate for psychophysiological studies of DLD for several reasons. First, as noted above, lexicalsemantic deficits have been proposed as a cause and a consequence of deficits in other linguistic and cognitive domains in DLD. Second, they have been postulated to represent both the area of residual normality and the result of compensatory plastic changes in language development, subserved by the corresponding neuroplastic changes in the brain. Third, behavioral studies have yielded a mixed pattern of results with respect to the specifics of the atypicalities in lexical-semantic processing in DLD.

ERP studies of lexical–semantic processing primarily logically focus on the N400 component, a negative deflection in the scalp-recorded electric activity of the brain that occurs approximately 400 ms after the presentation of the stimulus and has a prominent centro-parietal topographic distribution. The N400 component is reliably elicited by semantic violations and other expectation violations at the word level and is sensitive to a number of factors, including word frequency, predictability, and expectancy (Kutas & Federmeier, 2000, 2011).³ Broadly, it is thought to index stimulus-induced semantic activity that occurs against the background of both long-term knowledge and recent experiences (e.g., experimentally generated top-down expectations) that influence the activation of lexical and semantic categories. The unique sensitivity of the N400 to the interplay between

different cognitive systems and processes make it both one of the most studied components in cognitive psychophysiology and potentially an interesting target for examining the dynamics of lexical processing in DLD.

To date, only four studies (excluding Fonteneau and van der Lely's study described above) have examined lexical-semantic processing in DLD using neurophysiological methods. Using visually presented sentences that varied with respect to the semantic appropriateness of the final word (anomalous vs. nonanomalous), Neville, Coffey, Holcomb, and Tallal (1993) found that children with combined DLD and reading disability showed a larger N400 in response to both anomalous and nonanomalous sentence-final words; moreover, the amplitude of the difference waveform (anomalous-nonanomalous) was larger in children with DLD compared to TD children. The findings were interpreted by the authors as indicative of greater compensatory effort required by children with DLD for successful integration of words with context. A similar pattern of results was observed for parents (especially fathers) of children with DLD, who also showed an abnormally large N400 in response to sentence-final semantically anomalous, as well as nonanomalous words (Ors et al., 2001). In that study, the amplitude of the difference N400 waveform was smaller in parents of children with DLD than in parents of TD children, suggesting a lesser degree of differentiation between congruous and incongruous sentential endings by parents of children with DLD potentially driven by their need to engage in a significant thematic (re)integration in both situations. Ors et al. (2001) suggested that this need for additional thematic integration in the congruous condition might be driven by the abnormal activation of semantic categories (via associative links) in long-term memory during reading in parents of children with DLD. The presence of N400 abnormalities in parents of children with DLD also tentatively supports the idea that the N400 amplitude might be a heritable endophenotype for the disorder and a marker for residual DLD in adults (although no behavioral language functioning data was reported in the manuscript for the adults).

Two more recently published studies are not fully consistent with the results reported by Ors et al. (2001) and Neville et al. (1993). Friedrich and Friederici (2006) investigated the N400 effect at 19 months in two groups of children: TD and children classified as being at risk for expressive DLD based on behavioral measures of language development at 30 months of age. They found that children not at risk for DLD showed a significant N400 effect in response to auditorily presented words that were incongruous with respect to the visually presented picture in a cross-modal picture–word paradigm and to phonotactically legal pseudowords (compared to a baseline when the pictured object's name was presented). In contrast, the group of children at risk for DLD did not display the N400 in either of these conditions. However, both groups showed a significant phonological–lexical priming effect manifested in an enhanced early negativity in response to congruous compared to incongruous words, although in children at risk for DLD this effect was more broadly temporally and spatially distributed. Because this early effect requires the activation of corresponding lexical elements to be robustly elicited, the reduced N400 in children at risk

³Note that the N400 can be elicited in response to violations presented in the context of a sentential frame, as well as in other paradigms (e.g., lexical priming, repetition, and picture–word matching paradigms) and other modalities (Kutas & Federmeier, 2000, 2011).

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for DLD could not be attributed to their insufficient lexical knowledge per se. Thus, the N400 deficits in this group were likely tapping into the delayed maturation of processes of lexical activation; the altered dynamics of the lexical activation, in turn, could be driven by a number of deficits, most likely in the degree of specification and robustness of phonological and semantic features in lexical knowledge, and the extent of their integration or coherence (thus indexing the quality of their lexical representations; see Perfetti & Hart, 2002).Consistent with this hypothesis, Friedrich and Friederici (2006) also suggested that the reduced N400 effect in children at risk for DLD might be related not to the delayed maturation of the processes involved in the generation of theN400 per se, but rather to their ability to apply these processes to weak or unstable semantic representations.

Finally, Malins et al. (2013) investigated ERP responses to words presented in a cross-modal picture-word paradigm to children with and without DLD. Their study involved an experimental manipulation not only of the degree of the semantic congruency of the presented word but also of the degree of the phonological overlap between the match and the mismatch words. In their study, both groups of children displayed significant N400 effects in response to words that were both semantically and phonologically unrelated to the target match word (e.g., see SHELL, hear "mug"), and an enhanced N400 effect to cohort mismatches (e.g., see DOLL, hear "dog") that overlapped with the target word initially. In addition, both groups showed a similar earlier phonological mapping negativity (PMN) effect, suggesting that children with DLD are capable of developing online phonological expectations (congruent with the priming effect observed by Friederich and Friederici, 2006) and detecting violations of these expectations. However, only TD children displayed a significant attenuation of the N400 effect in response to rhyme mismatches (e.g., see CONE, hear "bone"). The lack of this rhyme attenuation effect in the DLD children led the authors to suggest that children with DLD are either not as sensitive to rhyming as TD children (potentially due to problems with establishing robust phonological representations) or are not efficient at suppressing lexical alternatives during spoken-word recognition.

In sum, ERP studies of lexical and semantic processing in children with DLD have resulted in a complex landscape of findings that suggests, at the minimum, atypical organization of lexical–semantic processing in DLD. Indirect evidence also suggests that these atypicalities might be related to deficits in other domains of language development in DLD (e.g., given reduced rhyme sensitivity in the phonological domain; e.g., Desroches, Newman, & Joanisse, 2009: Malins et al., 2013; or by the apparent compensatory reorganization of the system to rely more heavily on semantic associations in the context of morphosyntactic deficits; Pizzioli & Schelstraete, 2011).

The goals of the current study were twofold. First, we examined the dynamics of lexical processing in children with DLD in a cross-modal picture–word matching paradigm that manipulated the degree of the phonological and semantic similarities between the word corresponding to the pictured object and words presented in other conditions (similar to that employed by Desroches et al., 2009; and Malins et al., 2013). Specifically, we investigated the neural responses of children with and without DLD to words that were related to the pictured word phonologically (i.e., phonologically overlapped with it initially or finally) or semantically (i.e., as semantic associates), or were semantically and phonologically

unrelated to the pictured word. Note that although it is difficult to draw predictions regarding the relative size of the N400 effect in these conditions, given Friedrich and Friderici's (2006) findings for infants at risk for DLD, one can speculate that for older children, the semantically associated condition might better reflect difficulties in processing because their lexical development might be sufficient for eliciting the basic N400 in response to words that are neither phonologically nor semantically related to the target word.

Second, to the best of our knowledge, no study has directly related behavioral indices of language development and neurophysiological indices of lexical processing in DLD. ⁴ Therefore, we took an individual differences approach to examine the links between phonological, morphosyntactic, lexical, and semantic/pragmatic development, on one hand, and phonological and lexical processing ERPs, on the other hand. Thus, if lexical deficits in DLD are related to deficits in phonological development, we would expect behavioral indices of phonological development to be related to the amplitudes of the PMN and the N400 in all experimental conditions, and particularly phonologically sensitive conditions (e.g., the final phonological overlap condition). Similarly, if lexical processing abnormalities in DLD are compensatory in nature, the N400 amplitudes should be related to the degree of the grammatical impairment in DLD.

METHOD

Participants

The participants came from a small geographically isolated Russian-speaking population (AZ; Rakhlin, Kornilov, et al., 2013) characterized by an unusually high prevalence of DLD. On average, 23% to 40% of children and adults exhibit atypical language development despite the absence of apparent neurobiological or sensory pathology, compared to 9% in the comparison rural population. Children with DLD from the AZ population have been characterized behaviorally as having expressive deficits (see Rakhlin, Kornilov, et al., 2013, for a detailed description of the procedure), receptive language deficits (Kornilov, Rakhlin, & Grigorenko, 2012; Rakhlin, Kornilov, & Grigorenko, 2014), and deficits in phonological working-memory capacity (Kavitskaya, Babyonyshev, Walls, & Grigorenko, 2011) and the development of literacy (Rakhlin, Cardoso-Martins, et al., 2013). Together, these characterizations of the population are consistent with the behavioral profile of children with DLD documented in other languages (Ebbels, Dockrell, & van der Lely, 2012; Graf Estes, Evans, & Else-Quest, 2007; Hick, Joseph, Conti-Ramsden, Serratrice, & Faragher, 2002; Marton, Abramoff, & Rosenzweig, 2005; Robertson & Joanisse, 2010).

Thirty-nine children from the AZ population aged 7.17 to 15.83 years (M = 10.54, SD = 2.34; 23 boys) participated in the study. Of these, 23 were classified as DLD (M = 10.12, SD = 2.40; 16 boys) and 16 were classified as typically developing (TD; M = 11.14, SD = 2.18; 7 boys). The language status classification was based on a set of expressive and receptive language indices obtained using two standardized language development measures (the

⁴Friedrich and Friederici (2006) reported a significant correlation between children's scores on the test of expressive language and the phonotactic familiarity effect (i.e., the negative difference in response to phonotactically legal vs. illegal pseudowords); they did not report correlations for the N400 effect.

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classification scheme is described below). The two groups did not differ significantly with respect to either gender distribution, $\chi^2(1) = 2.60$, p = .107, or age, t(37) = 1.35, p = .186.

The Yale University Internal Review Board and the appropriate institutions in Russia approved the study protocol. Informed consents were obtained from the children's parents and oral assents were obtained from the children at the time of the data collection. In addition to the 39 children who participated in the study, 3 children also provided EEG data but did not provide behavioral data due to logistical reasons and were excluded from the analysis.

Language and cognitive development measures

Children's language development was assessed using two diagnostic tools: a standardized elicited narrative task developed for establishing the language development status in rural Russian populations (Rakhlin, Kornilov, et al., 2013) and the Assessment of the Development of Russian (Kornilov et al., 2012).

Narrative task—Expressive language development was assessed using two wordless storybooks: for children under 13, these were Frog, Where Are You? and One Frog Too Many (Meyer, 1969); for those over 13, these were Free Fall (Wiesner, 2008) and Tuesday (Wiesner, 1997). The audio and the transcripts of the elicited speech samples were analyzed by two native-Russian linguists and rated on a number of characteristics in phonological, syntactic, and semantic/pragmatic domains, combined to form the following measures: phonetic/prosodic development (i.e., phonological simplifications and omissions, misarticulations and prosodic abnormality); wellformedness (frequency of lexical and grammatical errors and false starts adjusted for the length of the narrative); number of complex structures (e.g., embedded and conjoined clauses, passives, and participial constructions); mean length of utterance in words (MLU_w); number of semantic/pragmatic errors; and lexical richness (i.e., number of distinct lexemes/100 words). Robust ageadjusted Z scores were calculated for each measure using data from a comparison rural population that resides in the same administrative region and is similar to the AZ population on a set of demographic and socioeconomic variables (population size, rate of unemployment, occupational structure, income, etc.; see Rakhlin, Kornilov, et al., 2013).

Assessment of the development of the Russian language—Children were also administered ORRIA (Kornilov et al., 2012), a standardized Russian language development test comparable to the Clinical Evaluation of Language Fundamentals (Semel, Wiig, & Secord, 1995), the Test of Language Development (Newcomer & Hammill, 1982), and the Comprehensive Assessment of Spoken Language (Carrow Woolfolk, 1999). ORRIA is aimed at comprehensively assessing language development in the areas of morphology, syntax, compositional semantics, and lexicon in both receptive and expressive domains. Standardized age-adjusted scores for overall language development (M = 100, SD = 15) were calculated using an external sample (n = 484) representative of the general population of Russian children using five ORRIA subtests (passive vocabulary, linguistic operators, sentence structure, and word structure).

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Diagnostic criteria for establishing language group status—Language impairment status (DLD vs. TD) was determined by using the cutoff criterion of a *Z* score at or below -1.25 on at least two of the six narrative scales listed above or an overall ORRIA score corresponding to this criterion (i.e., a score below 82, roughly corresponding to the epiSLI criterion; Tomblin et al., 1997). Predictably, given that these measures were used for grouping purposes, children with DLD significantly underperformed on almost all of the language measures, with the effect sizes (Cohen d) for the significant differences ranging from moderate (0.62) to large (1.29).

Nonverbal intelligence—Scale 2 of the Culture–Fair Intelligence Test (Cattell & Cattell, 1973), a standardized measure of nonverbal intelligence, was used to assess nonverbal cognitive functioning of the sample. All children scored above the cutoff score for intellectual disability (IQ > 70). Children with DLD did not significantly differ from TD children with respect to nonverbal intelligence, t (37) = 1.47, p = .150.

Verbal short-term and working memory—We also administered a 32-item digit span task (backward and forward) modeled after the Wechsler Intelligence Scale for Children— Fourth Edition (Wechsler, 2003) and a 21-item word span task to measure verbal short-term and working-memory capacity. Children with DLD displayed significantly lower scores on digit span, t(37) = 2.35, p = .024, d = 0.77, and word span, t(37) = 2.66, p = .011, d = 0.87, consistent with the established pattern of short-term memory deficits in DLD (Estes et al., 2007).

Hearing screening

All children were administered a bilateral hearing screening with a Beltone 119 (Beltone New England) portable audiometer at 500, 1000, 2000, and 4000 Hz. All children demonstrated normal hearing acuity by passing the screening at 25 dB.

Experimental stimuli and procedure

We used a set of high-frequency mono- and polysyllabic Russian words paired with color stock photographs in a picture–word matching paradigm to elicit the PMN and the N400 components. In this paradigm, modeled after Desroches, Newman, and Joanisse (2009), participants are presented with a picture and a set of spoken words; they are then asked to judge whether the word matches the picture.

The experiment consisted of 40 blocks with 8 trials per block. At the beginning of each block, a fixation cross was presented on the screen for 1000 ms. After that, the fixation cross was replaced by a picture, which remained on the screen throughout the whole block. After 1500 ms of preview time, a set of spoken words were presented with stimulus onset asynchrony of 2000 ms. A total of eight words were presented with each picture, split into five conditions. In the match condition, the word matched the picture (e.g., hear /tort/ "cake") — see tort "cake"). There were also four mismatch conditions: in the initial phonological overlap (IPO) condition, the (semantically unrelated) word matched the beginning of the name of the pictured object (e.g., hear /tors/ "trunk"—see tort "cake"); in the final phonological overlap (FPO) condition, the word matched the ending of the name of the

pictured object (e.g., hear /bort/ "board"—see tort "cake"); in the semantically associated (SA) condition, the word did not overlap with the name of the pictured object phonologically but instead was semantically associated with it (e.g., hear /t ai / "tea"—see tort "cake"); in the phonologically and semantically unrelated (UR) condition, the word was not related to the name of the pictured object phonologically or semantically (e.g., hear /sat/ "garden"—see tort "cake"). For each picture, the match word was presented four times, and each of the mismatching words was presented once in a randomized order. The order of blocks was randomized across participants.

The children sat in front of a PC laptop and were instructed to listen carefully to the words and indicate whether each word matched the picture by either pressing a mouse button (for "yes") or providing no response (for "no"). Prior to testing, all children were familiarized with the visual stimuli and were asked to name each picture. In two instances, when a child provided an incorrect name for the picture, the response was corrected and the picture was presented again after the rest of the stimuli.

The words were recorded by an adult male native Russian speaker using PRAAT audio software (Boersma & Weenink, 2009) with 16-bit resolution at a sampling rate of 44100 Hz and presented binaurally at 70 dB (SPL) via Etymotic insert headphones (Etymotic Research, Inc.). Forty photographs were selected from a commercial stock photo database. Two hundred highly imageable and frequent words were selected from a frequency dictionary of Russian (Sharoff, 2001). Most (63%) of the verbal stimuli were disyllabic (15% were monosyllabic, 19% were trisyllabic, and 3% contained four syllables). The words across five experimental conditions did not differ in either frequency or length (*ps* > .05). A panel of native Russian speakers (S.K., N.R., and E.L.G.) reviewed the verbal and the visual stimuli to ensure their appropriateness for the experiment (including but not limited to child appropriateness, semantic and phonological relatedness, and imageability for verbal stimuli, and visual clarity and concept relevance for visual stimuli). For the IPO condition, the onset of the mismatch relative to the target word happened on average at 324 ms poststimulus onset. For the FPO condition, the onset of the final phonological overlap with the match word had an average latency of 232 ms.

EEG recording, processing, and analysis

The EEG signal was recorded using a BioSemi Active Two system (BioSemi, Inc.) with 64 sintered Ag/AgCl electrodes mounted using electrolyte gel (SignaGel, Parker Laboratories, Inc.) in an elastic cap approximating the standard 10-20 system. An additional 7 electrodes were used to record the electric activity at the two mastoids and nose tip (data from these 3 electrodes were not used in the analyses reported in this manuscript), and to record the vertical electrooculogram (electrodes placed above and below the left eye) and the horizontal electrooculogram (electrodes positioned lateral to the outer canthi of both eyes). All impedances were kept below 25 kV.

The EEG signal was sampled at 1024 Hz and average referenced offline. The preprocessing of the data and the averaging were carried out using EMSE Suite 5.5 (Source Signal Imaging, Inc.). For each participant, EEG channels identified as containing a high amount of technical artifacts (i.e., excessive AC power line noise and/or loss of contact) on the basis of

visual inspection were reconstructed using a spline interpolation procedure. Then, the signal was filtered using a digital IIR bandpass filter of 0.50 to 30 Hz. To correct for eye movement artifacts and blinks, a data-driven spatial ocular artifact correction algorithm (Pflieger, 2001) was applied to the signal.

The EEG was epoched from -200 to 700 ms relative to stimulus onset using a 200-ms prestimulus baseline correction. We only analyzed correct trials (i.e., hits in the match condition and correct rejections in the mismatch conditions) in which the EEG activity did not exceed +115 mV. On average, the analyses included 127/160 match, 33/40 IPO, 31/40 FPO, 33/40 SA, and 33/40 UR trials. There were no significant differences in the number of trials included for TD and the DLD groups or between mismatch conditions (all *ps* > .05).

The wave forms were averaged separately for each condition. The conventional peak identification analysis was guided by a combination of visual inspection of averaged waveforms and prior literature regarding the timing and spatial topography of the potentials. The N400 component typically has a pronounced centroparietal scalp distribution (Kutas & Federmeier, 2000), while the PMN shows a more central scalp distribution (Desroches et al., 2009). However, in our preliminary analysis, both components showed a prominent parietal scalp distribution. Therefore, in our main analyses, we chose to focus on the parietal electrode clusters. Given that children with DLD have been shown to display atypical lateralization of auditory ERPs (Bishop, 2007; Friedrich & Friederici, 2006), we examined three clusters of electrodes to account for potential group differences in the lateralization of the components of interest: left parietal: P5, P3, and PO7; midline parietal: Pz, PO3, PO4, POz, and Oz; right parietal: P4, P6, and PO8.

Following the procedures used by Desroches et al. (2009) and Malins et al. (2013), the time windows were defined as 210–310 ms poststimulus for PMN, 310–410 ms for early N400, and 410–600 ms for late N400 based on the visual analyses of the raw waveforms for all five conditions, as well as difference waveforms for the four mismatch conditions. The N400 time window was split into two to account for the absence of the early N400 in the IPO condition (given the timing of the onset of the mismatch) and for the analyses to be comparable to those reported by Malins et al. (2013). The ERPs' amplitudes were quantified as average amplitudes in these time windows, and we also obtained fractional (50%) latency estimates for each of the potentials. In addition to this conventional peak-identification based analysis, to ensure that our analyses focused on the parietal region in specific time windows, we performed an exploratory temporospatial principal component analysis (PCA) following Dien's (2010, 2012) guidelines.

The amplitude and latency estimates for each of the components were subjected to a set of mixed 5 (condition: match, IPO, FPO, SA, or UR) \times 3 (electrode cluster: left, midline, or right) \times 2 (language group: DLD or TD) analyses of variance (ANOVAs) with Greenhouse–Geisser corrections applied to the *p* values whenever appropriate.

RESULTS

Behavioral data

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We did not find statistically significant differences between children with DLD and TD children in either number of hits in the match condition or number of correct rejections in any of the four mismatch conditions (for a set of independent t tests, all ps > .27). In the match condition, children with DLD also showed (log-transformed) reaction times similar to those of TD children, Welch t (29.08) = -1.60, p = .12. Thus, overall, children with DLD displayed behavioral performance on the picture–word matching task similar to that of TD children. This result was expected due to the easy nature of the mismatch detection task (also manifested in the ceiling performance displayed by both groups of children, as is evident from Table 1).

Group differences in lexical-semantic and phonological processing ERP indices

The ERP waveforms and the topographic distributions for the components of interest for the two groups of children are plotted in Figures 1 and 2, respectively. Children with DLD displayed a trend toward significantly less negative amplitudes in the PMN time window across the five conditions, F(1, 37) = 3.97, p = .054, $\eta_p^2 = 0.097$. No group-related interactions were significant (all ps > .159). Thus, this borderline-significant main effect of language group suggests that children with DLD display deficits not in the processes involved in the detection of violations of phonological expectations per se, but rather in relatively early neural responses to auditorily presented words in general. Across the two groups of children, we found a significant main effect of condition, F(4, 148) = 10.07, p <.

001, $\eta_p^2 = 0.214$. Post hoc analyses with Bonferroni corrections revealed significantly more negative amplitudes in the FPO (p = .006), SA, and UR (both ps < .001) conditions, compared to the match condition, indicating the presence of robust negativity in the 210 to 310-ms time window for these conditions. Predictably, given the initial phonological overlap between the IPO and the match condition, no significant PMN effect was observed for the IPO condition (p = 1.00). The effect of electrode cluster was also significant, F(2, 74) =5.77, p = .006, $\eta_p^2 = 0.135$, with more positive amplitudes observed in the right parietal cluster compared to the midline (p = .020) and the right parietal (p = .004) clusters. However, this effect did not differ with respect to experimental conditions, as indicated by the nonsignificant interaction between electrode cluster and condition, F(8, 296) = 0.72, p= .640, $\eta_p^2 = 0.019$. No other effects reached statistical significance (all ps > .160). We did not

find any significant effects for the analyses of latency estimates in the PMN time window (all ps > .060).

Overall, in the early N400 time window, children with DLD displayed N400 amplitudes similar to those of TD children, F(1, 37) = 0.30, p = .590, $\eta_p^2 = 0.008$. The main effect of condition was significant, F(4, 148) = 18.36, p < .001, $\eta_p^2 = 0.332$, with the pattern of differences resembling that for the PMN: markedly more negative amplitudes were observed for the FPO, SA, and UR conditions (all *ps* < .001), but not the IPO condition (*p* = 1.00). These results indicate the presence of a robust N400 effect in all conditions but the with the

initial phonological overlap with the match word (likely because the response in the early window is generated within the period where the IPO items overlap with the expected word, that is, during the period prior to expectation violation). The early N400 amplitude was similar in the FPO, SA, and UR conditions (all ps = 1.00). We also obtained a significant main effect of electrode cluster, F(2, 74) = 11.71, p < .001, $\eta_p^2 = 0.240$; post hoc pairwise comparisons indicated that the early N400 was more prominent in the midline compared to the right parietal (p < .001) cluster (no other pairwise comparisons were significant). The analysis of early N400 latency also revealed that the N400 had an earlier latency in the right parietal compared to the left parietal cluster, F(2, 74) = 5.09, p = .009, $\eta_p^2 = 0.121$ (p = .022 for the pairwise comparison); no other effects were significant (all ps > .133).

The analysis of the amplitudes in the late N400 time window, however, revealed statistically significant differences between children with DLD and TD children. Across the two groups, there was a main effect of condition, $F(4, 148) = 15.69, p < .001, \eta_p^2 = 0.298$, with significantly more negative amplitudes in the four mismatch conditions compared to the match condition (all ps < .001) but no significant differences between the four mismatch conditions. Although the main effect of language group was not significant, F(1, 37) = 1.39, $p = .246, \eta_p^2 = 0.036$, the interaction between group and condition was significant, F (4, 148) = 3.04, p = .026, $\eta_p^2 = 0.076$, indicating that the two groups of children demonstrated different late N400 patterns across conditions. We investigated this interaction by comparing the average amplitudes of the difference waveforms (mismatch-match) separately for each condition in a set of 2 (language group) \times 3 (electrode cluster) mixed ANOVAs. These analyses revealed that the interaction between language group and experimental condition is likely driven by marked differences between children with DLD and TD children in the size of the late N400 in the IPO condition, F(1, 37) = 12.31, p = .001, $\eta_p^2 = 0.247$, and the UR condition, F(1, 37) = 5.50, p = .024, $\eta_p^2 = 0.129$, but not the FPO or the SA conditions (all *ps*) > .408; see Figure 1b). Additional analyses also revealed that these effects are unlikely to be driven by an enhanced N400 in the match condition, because the two groups did not differ with respect to the amplitude of the N400 when this condition was analyzed separately, F(1,

37) = 2.21, p = .145, $\eta_p^2 = 0.056$. We discuss possible explanations for these differences in the Discussion.

In addition, we found tentative evidence for atypical topographic distribution of the late N400 in children with DLD, F(2, 74) = 8.72, p = .051, $\eta_p^2 = 0.078$, for the two-way interaction between cluster and group; while the TD children showed a late N400 that was larger in the left and midline clusters compared to the right parietal cluster, F(2, 30) = 9.26, p = .001, $\eta_p^2 = 0.382$ (both pairwise ps < .024), children with DLD did not show this effect, displaying similar N400 responses in all three clusters, F(2, 44) = 0.89, p = .423, $\eta_p^2 = 0.038$. No significant effects were found for late N400 latencies (all ps > .057).

In retrospect, it might not be surprising that we did not find any N400 diminishment in the SA condition. The picture–word matching paradigm does not require semantic activation for

any item aside from the pictured object (and there, shallow activation sufficient to retrieve phonological form is all that is required); rather, comparison of just the first expected and heard phoneme (or first few phonemes, for IPO) would suffice. The absence of an FPO effect (predicted diminished late N400, as in Desroches et al., 2009; and Malins et al., 2013) may reflect differences in materials; true rhyme pairs conforming to the constraints of the experiment (concrete, imageable, roughly matched on frequency, etc.) are harder to find in Russian than in English, and so we relied upon items matching in two to six final phonemes (M = 3.20). It may be that observing a diminished late N400 depends upon overlap from syllable nucleus onward.

In addition to the amplitude and latency analyses, we analyzed the data using the full set of electrodes and a bottom-up approach via temporospatial PCA (Dien, 2012) as implemented in the MATLAB ERP PCA Toolkit (Dien, 2010). At the first step, the individual participants' average waveforms for the match and four mismatch conditions were subjected to a temporal PCA with Promax rotation. The scree test suggested that 25 temporal factors (explaining 96% of variance) should be retained. The factor scores from the first step were then subjected to a spatial ICA with Infomax rotation. This analysis identified 7 spatial factors that explained 73% of variance. Thus, the temporospatial PCA revealed a total of 25 (temporal) \times 7 (spatial) = 175 temporospatial factors. Only 14 of these 175 factors explained more than 1% of variance and were further analyzed using a set of 5 (condition) \times 2 (group) ANOVAs applied to the factor scores converted back into micro volts. These analyses revealed several effects, overall corroborating and even strengthening the pattern of findings presented above for the conventional peak identification analysis.

The first temporospatial factor for which the group differences were obtained was represented by the combination of the first temporal factor (TF1; peaking at 454 ms poststimulus) and the first spatial factor (SF1; characterized by the prominent parietal topography with the most negative amplitudes observed in the Oz electrode; see the onlineonly supplemental materials for the topographic maps of each of the temporospatial factors). Thus, this temporospatial factor, TF1SF1, can be thought of as overall corresponding to the N400 potential. For TF1SF1 (average factor score differences for the two groups are plotted in Figure 3), we found a statistically significant interaction between language group and condition, F (4, 148) = 2.71, p = .043, $\eta_p^2 = 0.068$. The follow-up analysis of this interaction revealed that whereas the TD children displayed robust N400 responses in all four mismatch conditions, F(4, 60) = 11.81, $p, .001, \eta_p^2 = 0.440$ (for pairwise comparisons between the match and the four mismatch conditions, all ps < .008), children with DLD showed a different pattern of results. Specifically, while they also displayed a significant main effect of condition, F(4, 88) = 8.61, p < .001, $\eta_p^2 = 0.281$, the N400 effect was only robust for the FPO, SA, and UR conditions (all ps < .005), but not the IPO condition (p = .260). Difference scores also revealed that, in addition to a markedly reduced IPO-match N400, t(37) = -2.75, p = .009, d = -0.86, children with DLD showed a reduced N400 in the UR condition, t (37) = -2.16, p = .038, d = -0.72 (all other ps > .472).

The second temporospatial factor for which group differences were observed was TF2SF1, which represents a component characterized by a relatively early latency (196 ms

poststimulus) and a parieto-occipital distribution similar to that of TF1SF1. This factor corresponds to the PMN/N200 component in the early time window. We did not find significant differences in TF2SF1 amplitudes across the five experimental conditions, F(4, 4)148) = 2.37, p = .067, $\eta_p^2 = 0.060$. However, children with DLD displayed significantly smaller TF2SF1 amplitudes than did children with TD overall, F(1, 37) = 10.83, p = .002, $\eta_p^2 = 0.226$. As Figure 3 demonstrates, children with DLD essentially showed TF2SF1 amplitudes close to zero; this result corresponds to the finding of less negative amplitudes in the conventional peak identification analysis of the PMN time window reported above. Finally, we found additional evidence for atypical neural processing responses between DLD and TD children in the IPO condition manifested in the amplitudes of the TF4SF1, a temporospatial factor characterized by a parietal positivity peaking at 325 ms poststimulus, a latency that corresponds to the onset of the phonological mismatch in the IPO condition. Specifically, we found a significant interaction between condition and group, F(4, 148) =3.44, p = .019, $\eta_p^2 = 0.085$; the follow-up investigation of this interaction revealed that TD children displayed a strong and significant effect of condition for TF4SF1 amplitude, F (4, $(60) = 12.01, p < .0001, \eta_p^2 = 0.445$, driven by the markedly more positive amplitudes for the IPO compared to the match condition (p = .009; other *ps* for the match vs. mismatch comparisons > .076). Children with DLD, however, did not display such an effect, F (4, 88)

= 2.41, p = .077, $\eta_p^2 = 0.009$ (all ps > .345 for the pairwise comparisons between match and mismatch conditions). Visual inspection of the IPO time course plotted in Figure 1c suggests that the mismatch response in DLD children was delayed until the late N400 window in this condition; again, we discuss possible explanations in the Discussion.

Relating individual differences in ERPs and behavioral measures of linguistic and cognitive development

To further investigate which facets of DLD might be driving the group differences reported above, we performed a correlational analysis. Specifically, we related the ERPs shown to differentiate between groups (i.e., the amplitude of the N400 difference for the IPO and UR conditions from the conventional peak identification analyses, and the estimated difference factor scores for TF1SF1 in the IPO and UR conditions, TF2SF1 across all five conditions, and TF4SF1 in the IPO condition) and children's behavioral indices of verbal memory capacity and language development. To account for the potential effects of demographic characteristics (i.e., age and gender) and nonverbal intelligence, these variables were entered as covariates in the partial correlation analysis.

The correlations between assessment measures and ERP indices of lexical–semantic and phonological processing are presented in Table 2. We found an association between the N400 amplitude in the IPO condition and phonetic/prosodic characteristics (r = -.485, p < .05 for the amplitude difference and r = -.363, p < .05 for TF1SF1 factor score difference); we also found that the TF1SF1 IPO difference was associated with MLU_w (r = -.484, p < .05), lexical richness (r = -.480, p < .05), and verbal memory as indexed by Word Span (r = -.346, p < .05). Thus, the size of the TF1SF1/N400 in this condition was related to indices of phonological, grammatical, and lexical development, and the development of verbal

short-term memory. When controlled for verbal short-term memory, the pattern of correlations remained essentially the same, suggesting that the amplitude of the N400 in this condition is independently affected by language development and verbal memory. However, the association of the TF1SF1/N400 amplitude with an index of grammatical development, MLU_w, lost statistical significance when phonological and lexical development were controlled for (r = -.266, p > .05). In contrast to the TF1SF1 scores in the IPO condition, the TF1SF1 scores for the UR condition only correlated with lexical richness (r = -.331, p < . 05) but not measures of development in other linguistic domains or verbal memory. In sum, we found that the reduced amplitude of the N400 in the IPO and UR conditions was associated with lower lexical and phonological development, as well as verbal memory, but not grammatical development.

In the early PMN window, TF2SF1 amplitude negatively correlated with lexical richness (r = -.363, p < .05). We also found marginally significant correlations between the scores for the TF4SF1, the temporospatial factor characterized by a parietal positivity in the IPO condition for the TD children, and phonetic/prosodic characteristics (r = .312, p = .06).

In sum, we found that although children with DLD were just as accurate and fast on the picture–word matching task as TD children, their neural responses to auditory words presented in this paradigm were markedly different. Specifically, children with DLD displayed atypical ERPs in both the mid- and late latency range. Children with DLD showed significantly reduced amplitudes in the PMN time window across conditions; the PCA estimates of this component were linked to the lexical development of children in the sample. They also did not display a positive component in response to the phonological mismatch in the IPO condition; the absence of this component could be also tentatively linked to their deficits in phonological development. Finally, we found that children with DLD displayed markedly reduced late N400 amplitudes in response to words that acoustically and phonologically initially overlapped with the target word (i.e., in the IPO condition). However, the results of the correlational analysis suggested that while the latter was associated with lexical development, the former is linked to both lexical and phonological development.

DISCUSSION

Lexical-semantic and phonological processing deficits in DLD

We investigated lexical and phonological processing in children with DLD using a crossmodal picture–word matching paradigm similar to the one used by Desroches et al. (2009) and Malins et al. (2013).

We found that children with DLD displayed significantly attenuated N400 amplitudes in the late time window compared to TD children in the UR and the IPO conditions. Given the absence of significant differences between the magnitudes of the N400 effects for the four mismatch condition within groups, we must be cautious with any explanations of effects that manifest as Condition \times Group interactions. The difference in the IPO condition (similar timing, but reduced amplitude for DLD) could result from reduced activation for the

expected (pictured) word. Such a reduction could follow from sluggish lexical activation (consistent with accounts that postulate the presence of generalized processing speed deficits in DLD; Miller, Kail, & Leonard, 2001), which in turn could result from overactivation (Pizzioli & Schelstraete, 2011) or reduced phonological or lexical inhibition (Magnuson et al., 2011); either overactivation or reduced inhibition would result in more words being activated in the early time course, and if we assume word recognition involves competition among activated lexical representations (McClelland & Elman, 1986), lexical activations will increase more slowly when more words are activated.

The relative depression of the N400 response for children with DLD in the UR condition is more challenging to explain. First, we must assume that any differential activation for the SA and UR conditions (again, relative to TD N400s) indicates that children with DLD do not focus attention at the phonological level, contrary to TD children. This would be testable in future work using other paradigms that normally shift attention away from semantics (e.g., semantic priming is normally reduced in a lexical decision task compared to a task like artifact judgment). Second, we must account for why the N400 is depressed in children with DLD in the UR condition but not in the SA and FPO conditions. We offer two explanations for this pattern of findings. First, it could result from abnormal effects of overlap for SA and FPO, whereby overlap has opposite effects than are normally found. That is, rather than semantic or phonetic relatedness diminishing the N400 response, overlap somehow enhances the N400, perhaps because abnormal connectivity in the DLD lexicon initially leads to increased activation of the expected word and thus to a larger mismatch effect when the heard word finally overcomes the expected word in lexical competition. Second, the other possibility is that the unrelated word has functionally greater consistency with the expected word for children with DLD. This could follow from the overactivation hypothesis (Pizzioli & Schelstraete, 2011), which postulates that items in the mental lexicon of children with DLD have developed stronger associative links and, overall, the lexicosemantic network is activated more strongly but diffusely in children with DLD than in TD children (due to compensatory reliance on such associations in the face of difficulties at morphological and syntactic levels of processing). On this account, one might speculate that target word activation spreads over more levels of linkage in the lexicon in DLD children during the picture preview period (e.g., rather than activating just directly associated items, e.g., CAKE \rightarrow TEA, stronger associative links could permit more diffuse activation, e.g., CAKE \rightarrow PARTY \rightarrow GARDEN PARTY \rightarrow GARDEN).⁵

Again, though, we offer these speculative explanations for effects that must be addressed with caution (there were relatively weaker N400 responses for the IPO and UR conditions for DLD children relative to TD children, in the absence of reliable differences between these conditions within the sample). These effects are intriguing and, as we have just discussed, can motivate several testable hypotheses as to differences in language processing in DLD that could be explored in future work. In contrast, we can have much greater confidence in the patterns that emerged over the full sample of children (TD and DLD) in

⁵One might still have to make the same assumption as above regarding the SA and FPO items, that is, that more closely related items boost the expected word's activation. To our knowledge, the resources needed to explore these hypotheses in detail (semantic association norms for Russian) are not available.

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our individual differences analyses, and as we discuss next, our correlational findings are consistent with the Condition \times Group interactions.

Once more, children with DLD displayed a significantly attenuated N400 in the IPO condition compared to TD children. In this condition, the bottom-up acoustic/phonetic information is consistent with the match word for a period of approximately 324 ms. Desroches et al. (2009) and Malins et al. (2013) found that this condition elicited the largest N400 in the picture–word priming paradigm in both children and adult samples. This enhancement is driven by the bottom-up reinforcement (from the overlapping onset) of the top-down expectations regarding the identity of the upcoming stimulus. The initial phonological overlap thus strengthens the activation of the target lexical item, and the wordmedial mismatch produces a large N400 because the expected target has been so strongly activated; it must be deactivated as the actual auditory target activates. In our data the amplitude of the N400 effect was not statistically different across the four mismatch conditions (although in the combined sample, a trend was observed for larger N400 amplitudes in the IPO compared to the UR and SA conditions with p = .06), the visual analysis of the waveforms presented in Figure 1 suggests that the IPO condition produced the largest N400 in the group of TD children but not children with DLD. Unlike the reduction of the N400 in the UR condition, this reduction of the N400 in the IPO condition correlated with behavioral indices of both lexical and phonological development. Although TF1SF1/N400 amplitude in the IPO condition correlated with MLU_w, a measure of grammatical development, in our sample MLUw correlated with both lexical and phonological development. When both lexical and phonological development were controlled for, the TF1SF1 amplitude in the IPO condition was no longer significantly related to MLU_w, suggesting that it indexes deficits in lexical and phonological processing and is relatively independent from grammatical or memory deficits in DLD.

We hypothesize that this reduction of the N400 in the IPO condition is thus due to the combination of lexical and phonological processing deficits in children with DLD. One possible explanation we have already discussed is that lexical activation is slower in children with DLD; thus, even given initial phonological overlap, the expected word may not be substantially activated by the time mismatch is encountered word medially. Slower activation could be attributed to less specified phonological features of lexical representations or otherwise altered dynamics of lexical activation (e.g., overactivation or reduced phonological or lexical inhibition, as discussed above), or both. Consistent with this hypothesis, children with DLD have been shown to require more acoustic/phonological information to recognize spoken words than their do TD peers (Dollaghan, 1998) and show lower performance on tasks aimed at measuring the precision of phonological representation, as well as nonword repetition; Ramus et al., 2013).

Alternatively, one could speculate that the N400 is not enhanced in the IPO condition for children with DLD, not because lexical activation is sluggish, but rather because they are less capable of detecting the phonological mismatch that occurs in the middle of the word. This would lead them to essentially accept lexical items in the IPO condition as corresponding to the target match words. Consistent with this hypothesis, children with

DLD did not show the same parietal PCA component in response to the onset of the mismatch in the IPO condition as was observed in the TD group. Although the amplitude of the PMN effect (match-mismatch) in other conditions was similar in the two groups, suggesting that children with DLD are capable of detecting violations of phonological expectations per se, it is possible that in the IPO condition, this detection (picked up by the PCA analysis as a separate temporospatial factor) is less robust because it happens further along in the processing stream than in other conditions. However, this hypothesis predicts that the reduction of the N400 in the IPO condition should be linked to the reduction of the TF4SF1 amplitude. Additional analyses revealed that the amplitude of TF4SF1 did not correlate with the TF1SF1/N400 difference amplitude in the IPO condition (r = -.06, p = .725), suggesting that this explanation is not likely. This hypothesis makes two additional predictions not supported by the data. First, it predicts a significant increase in the rate of false alarms in the IPO condition in the group of children with DLD compared to TD children, which was not the case. Second, it predicts that the timing of the N400 in the IPO condition would be atypical/delayed in children with DLD compared to TD children, which was also not the case.

We also found that children with DLD showed overall reduced amplitude of the N200/PMN response in the early time window across conditions. This potential has been linked to both phonological processing (Connolly & Phillips, 1994; Lee, Harkrider, & Hedrick, 2012) and early semantic processing following the initial phonological analysis of the available (incomplete) information about the word and early lexical selection (van den Brink, Brown, & Hagoort, 2001). In our data, the PCA-derived amplitude of this component was positively related to the PCA-derived amplitudes of the N400 effect in both the IPO condition (r = .432, p < .01) and the UR condition (r = .341, p < .05), and also to the behavioral measure of lexical development (r = -.363, p < .05) but not the measure of phonological development (r= .126, p > .05). This pattern of correlations suggests that this component is more likely to represent aspects of lexical rather than phonological processing, and that children with DLD might have deficits in processes involved in early lexical selection, consistent with the general pattern of atypical dynamics of lexical activation in DLD. However, we would like to emphasize that these data should be interpreted cautiously given that the topographic characteristics of this component were more similar to the N400 than in the previously published studies mentioned above.

Implications for understanding neuroplasticity in the context of atypical language development in DLD

Whereas the primary meaning of neuroplasticity, at least in studies of development and psychopathology, relates to the reorganization of the brain in response to implicit learning or targeted intervention, whether medical (Lonka et al., 2013) or behavioral (Eldar & Bar-Haim, 2010; Seppänen, Hämäläinen, Pesonen, & Tervaniemi, 2013; Song et al., 2010; Wild-Wall, Falkenstein, & Gajewski, 2012), another important aspect of neuroplasticity is how the system develops in the context of both typical (Brandwein et al., 2011) and atypical/disordered development (Föcker, Best, Hölig, & Röder, 2012; Mills et al., 2013; Spironelli, Bergamaschi, Mondini, Villani, & Angrilli, 2013). In the case of DLD, several promising attempts at finding a specific, single core deficit (e.g., such as generalized slowing,

phonological working memory, procedural memory, or deficits circumscribed to grammar) have proved untenable when all of the affected children are considered. The neuroconstructivist perspective of Karmiloff-Smith and colleagues (Elman et al., 1996; Karmiloff-Smith, 1998) provides a more nuanced but theoretically challenging possibility: subtle differences or deficits in one developmental cognitive domain (e.g., memory) can have pervasive effects on distal domains such as language, and the same is true for deficits in different subdomains within the language system. That is, the normal developmental trajectory in a domain that can be descriptively isolated (e.g., morphosyntax or lexical knowledge) depends crucially on its relation to and interactions with disparate domains throughout the course of development.

For children with DLD, word-level strengths and weaknesses have been alternatively attributed to lower level causes (auditory, phonological, or memory; e.g., Ellis Weismer & Hesketh, 1996; Gathercole, 2006) or higher level causes such as atypical development of grammar (e.g., Pizzioli & Schelstraete, 2011). The attraction of such explanations is clear; if the constellation of symptoms that emerge in DLD were fully attributable to a single cause, the search for the etiology of DLD would presumably be greatly simplified. Single-cause or core-deficit hypotheses might at first seem consistent with a neuroconstructivist perspective; however, even if there were originally a very specific deficit underlying DLD, the neuroconstructivist perspective predicts a recursive impact on closely and possibly distantly related domains. That is, grammatical deficits could impair word learning, which could in turn lead to additional compromise of grammatical abilities (e.g., grammatical deficits could increase memory load by requiring a child to hold an utterance in memory in a holistic form rather than in a more compact, syntactically recoded form, impairing the child's ability to encode grammatical or even phonetic details of new words when they are encountered). Thus, even a very specific deficit in one subdomain would be expected to lead to altered development across a wide variety of domains. All the same, we can assess which domains covary, and attempt to rule out hypothesized primary causes of specific deficits.

Along with the accumulation of data on the multidimensionality of DLD, its faceted nature, and the relative concurrent and developmental dissociation of its facets, there has been an increase in the evidence substantiating the remarkable amount of neuroplasticity demonstrated by the brain as it learns to deal with linguistic stimuli experimentally (Chen et al., 2012; Kung et al., 2014; Spironelli, Galfano, Umiltà, & Angrilli, 2011) and developmentally by children (Kuhl, 2010) and adults (Batterink & Neville, 2013; Fernandez, Tartar, Padron, & Acosta, 2013). This growing literature substantiates the hypothesis that the brain does not shape language unilaterally; rather, language development also structures the brain, so that impaired language development is traceable in the brain's response to linguistic stimuli.

As such, the current study has important implications for understanding the complex pattern of linguistic deficits in DLD and their correlates in the brain. We found that abnormal N400 amplitudes demonstrated by children with DLD could be partially attributed to their lexical abilities (for the IPO and UR conditions) and to their phonological abilities (for the IPO condition). Although the precise mechanisms responsible for the generation of the N400are not known (but see Lau, Phillips, & Poeppel, 2008), it is nevertheless reasonable to surmise

that if the N400 indexes at least in part the efficiency of lexical–semantic processing, its amplitude should be related to the development of language in domains most proximal to lexical–semantic processing (i.e., such as vocabulary size and ease of lexical access as measured by lexical richness or quality of phonological representations, mostly word level, as measured by phonetic/prosodic characteristics).

Recall that several accounts view the absence of lexical-semantic processing deficits in DLD within the frameworks of residual normality (Fonteneau & van der Lely, 2008), and their presence, within the framework of compensatory reorganization of processing (Pizzioli & Schelstraete, 2011), while others attribute evidence for lexical processing difficulties to relatively distal domains (lower or higher level; see above). Crucially, the neurobiological indices of atypical lexical and phonological development were not related to the levels of basic morphosyntactic and complex syntactic development in our sample, suggesting relative independence of lexical/phonological and morphosyntactic development in the context of language disorder. Therefore, instead of being viewed as compensatory, lexical and phonological deficits represent a relatively independent locus of language disorder in DLD, which is best viewed as syndromic and dimensional. We would like to emphasize that deficits in different subdomains of language in DLD are not necessarily fully functionally isolated. For example, it is possible that typical and atypical development in the domains of lexicon and morphosyntax are related, but the close link between the two can only be detected when examined within the critical periods for the acquisition of grammar, lexical knowledge, or both. Correspondingly, a neuroplastic change of the language system that either accommodated morphosyntacitc processing difficulties in a compensatory way or detrimentally affected other language domains could have happened earlier in the development than we were able to capture in this study. In this case, the seeming independence of lexical-semantic, phonological, and grammatical deficits in DLD could be viewed as emergent (Karmiloff-Smith, 2009), mirroring findings from adult patients with acquired language disorders. If this is the case, our findings nevertheless suggest that either this neuroplastic relationship is moderate in strength and does not persist into school age or some other factors play a larger role in the development of either of these domains beyond the "critical overlap" period.

Alternatively, our findings might suggest the presence of multiple loci rather than a single locus of deficits in DLD. Our results suggest certain constraints on the dynamic interplay between the (atypical) development of different facets of language. While such interactions, including the use of compensatory processing strategies capitalizing on developmental plasticity, might occur for certain domains of language development, certain processing deficits (e.g., in lexical processing) might be at least partially separable from other deficits (i.e., grammatical deficits).

Overall, the results of our study suggest that children with DLD have deficits in lexical processing that are related to their levels of lexical and phonological but not grammatical development. These results, in addition to providing further evidence for the separation of grammatical and lexical domains in typical development, indicate that these domains also represent relatively independent domains of deficits in DLD, while highlighting the links between phonological and lexical development abnormalities in DLD. We found atypical

amplitudes of ERPs in children with DLD at different stages of processing, implicating initial phonological analysis, early lexical access, and, presumably, semantic processing in the lexical processing deficits in DLD. Further studies should focus more closely on the interplay between phonological and lexical development and processing in DLD, for example, by interrelating neurophysiological (rather than behavioral) indices of language development or by examining which specific characteristics of lexical and phonological development might be driving deficits in lexical processing in DLD.

Finally, note that the ERP study reported in this manuscript is part of a larger epidemiological study of the genetic and neurocognitive bases of DLD in a geographically isolated Russian-speaking population (described by Rakhlin, Kornilov, et al., 2013). The population is characterized by a high prevalence of DLD, a high degree of environmental homogeneity, and reduced genetic variability. Thus, the sample recruited for this study is inherently more homogenous than typical referral-based samples of children with DLD. As part of the larger study, we have been examining various ERP indices of cognitive and language processing in the same sample that we report on in this manuscript. Two other ERP studies (Kornilov et al., 2014) found that children with DLD from the same population displayed atypical attentional auditory processing (manifested in a decreased P3b amplitude) but not preattentive auditory processing (manifested in intact mismatch negativity amplitudes and latencies). In individual differences analyses, deficits in attentional auditory processing were linked to the development of complex syntax, vocabulary, and verbal working memory. Thus, contrary to the hypotheses that the locus of deficits in DLD lies in low level phonological representations and/or processing, our work so far suggests that in our sample, the deficits are related to more complex aspects of processing. Our goal in this study was to explore to what degree ERP measures indicate that lexical representations and/or processing are atypical in this sample, and whether atypical lexical processing could be related to individual differences in specific aspects of linguistic or cognitive ability or, even broader, viewed as an indicator of a particular neuroplastic trajectory, capturing the dynamic relationships between the development of language and that of its key substrate: the brain.

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References

- Addis L, Friederici AD, Kotz SA, Sabisch B, Barry J, Richter N, et al. A locus for an auditory processing deficit and language impairment in an extended pedigree maps to 12p13.31–q14.3. Genes, Brain and Behavior. 2010; 9:545–561.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2001. text revision

- Baddeley A, Gathercole S, Papagno C. The phonological loop as a language learning device. Psychological Review. 1998; 105:158–173. [PubMed: 9450375]
- Batterink L, Neville H. Implicit and explicit second language training recruit common neural mechanisms for syntactic processing. Journal of Cognitive Neuroscience. 2013; 25:936–951.[PubMed: 23631551]
- Bedore LM, Leonard LB. Grammatical morphology deficits in Spanish-speaking children with specific language impairment. Journal of Speech, Language, and Hearing Research. 2001; 44:905–924.
- Bishop DVM. Using mismatch negativity to study central auditory processing in developmental language and literacy impairments: Where are we, and where should we be going? Psychological Bulletin. 2007; 133:651–672. [PubMed: 17592960]
- Bishop DVM. Cerebral asymmetry and language development: Cause, correlate, or consequence? Science. 2013; 340:1230531. [PubMed: 23766329]
- Boersma, P.; Weenink, D. Praat: Doing phonetics by computer (Version 5.1.05). Amsterdam: Institute of Phonetic Sciences; 2009.
- Brackenbury T, Pye C. Semantic deficits in children with language impairments: Issues for clinical assessment. Language, Speech, and Hearing Services in School. 2005; 36:5–16.
- Brandwein AB, Foxe JJ, Russo NN, Altschuler TS, Gomes H, Molholm S. The development of audiovisual multisensory integration across childhood and early adolescence: A high-density electrical mapping study. Cerebral Cortex. 2011; 21:1042–1055. [PubMed: 20847153]
- Cannon TD, Keller MC. Endophenotypes in the genetic analyses of mental disorders. Annual Review of Clinical Psychology. 2006; 2:267–290.
- Carrow-Woolfolk, E. Comprehensive assessment of spoken language. Circle Pines, MN: American Guidance Service; 1999.
- Cattell, RB.; Cattell, AKS. A culture-fair intelligence test. Champaign, IL: Institute for Personality and Ability Testing; 1973.
- Chen Z, Liu P, Wang EQ, Larson CR, Huang D, Liu H. ERP correlates of language-specific processing of auditory pitch feedback during self-vocalization. Brain and Language. 2012; 121:25–34. [PubMed: 22377260]
- Claessen M, Leitao S, Kane R, Williams C. Phonological processing skills in specific language impairment. International Journal of Speech–Language Pathology. 2013; 15:471–483. [PubMed: 23327452]
- Connolly JF, Phillips NA. Event-related potential components reflect phonological and semantic processing of the terminal word of spoken sentences. Journal of Cognitive Neuroscience. 1994; 6:256–266. [PubMed: 23964975]
- Conti-Ramsden G, Botting N. Emotional health in adolescents with and without a history of specific language impairment (SLI). Journal of Child Psychology and Psychiatry and Allied Disciplines. 2008; 49:516–525.
- Conti-Ramsden G, Durkin K. Language and independence in adolescents with and without a history of specific language impairment (SLI). Journal of Speech, Language, and Hearing Research. 2008; 51:70–83.
- Desroches AS, Newman RL, Joanisse MF. Investigating the time course of spoken word recognition: Electrophysiological evidence for the influences of phonological similarity. Journal of Cognitive Neuroscience. 2009; 21:1893–1906. [PubMed: 18855555]
- Dien J. The ERP PCA Toolkit: An open source program for advanced statistical analysis of eventrelated potential data. Journal of Neuroscience Methods. 2010; 187:138–145. [PubMed: 20035787]
- Dien J. Applying principal components analysis to event-related potentials: A tutorial. Developmental Neuropsychology. 2012; 7:497–517. [PubMed: 22889342]
- Dollaghan C. Spoken word recognition in children with and without specific language impairment. Applied Psycholinguistics. 1998; 19:193–207.
- Dromi E, Leonard LB, Adam G, Zadunaisky-Ehrlich S. Verb agreement morphology in Hebrewspeaking children with specific language impairment. Journal of Speech, Language, and Hearing Research. 1999; 42:1414.

- Durkin K, Conti-Ramsden G. Language, social behavior, and the quality of friendships in adolescents with and without a history of specific language impairment. Child Development. 2007; 78:1441–1457. [PubMed: 17883441]
- Durkin K, Conti-Ramsden G, Simkin Z. Functional outcomes of adolescents with a history of specific language impairment (SLI) with and without autistic symptomatology. Journal of Autism and Developmental Disorders. 2012; 42:123–138. [PubMed: 21424233]
- Ebbels SH, Dockrell JE, van der Lely HKJ. Non-word repetition in adolescents with specific language impairment (SLI). International Journal of Language and Communication Disorders. 2012; 47:257–273. [PubMed: 22512512]
- Eldar S, Bar-Haim Y. Neural plasticity in response to attention training in anxiety. Psychological Medicine. 2010; 40:667–677. [PubMed: 19627649]
- Ellis Weismer S, Hesketh LJ. Lexical learning by children with specific language impairment: Effects of linguistic input presented at varying speaking rates. Journal of Speech, Language, and Hearing Research. 1996; 39:177–190.
- Elman, JL.; Bates, E.; Johnson, MH.; Karmiloff-Smith, A.; Parisi, D.; Plunkett, K. Rethinking innateness: A connectionist perspective on development. Cambridge, MA: MIT Press; 1996.
- Estes KG, Evans JL, Else-Quest NM. Differences in the nonword repetition performance of children with and without specific language impairment: A meta-analysis. Journal of Speech, Language, and Hearing Research. 2007; 50:177–195.
- Fernandez M, Tartar JL, Padron D, Acosta J. Neurophysiological marker of inhibition distinguishes language groups on a non-linguistic executive function test. Brain and Cognition. 2013; 83:330– 336. [PubMed: 24141240]
- Föcker J, Best A, Hölig C, Röder B. The superiority in voice processing of the blind arises from neural plasticity at sensory processing stages. Neuropsychologia. 2012; 50:2056–2067. [PubMed: 22588063]
- Fonteneau E, van der Lely HKJ. Electrical brain responses in language-impaired children reveal grammar-specific deficits. PLOSONE. 2008:3.
- Friedmann N, Novogrodsky R. Which questions are most difficult to understand? The comprehension of Wh questions in three subtypes of SLI. Lingua. 2011; 121:367–382.
- Friedrich M, Friederici AD. Early N400 development and later language acquisition. Psychophysiology. 2006; 43:1–12. [PubMed: 16629680]
- Gathercole SE. Nonword repetition and word learning: The nature of the relationship. Applied Psycholinguistics. 2006; 27:513–543.
- Graf Estes K, Evans JL, Else-Quest NM. Differences in the nonword repetition performance of children with and without specific language impairment: A meta-analysis. Journal of Speech, Language, and Hearing Research. 2007; 50:177.
- Gray S, Reiser M, Brinkley S. Effect of onset and rhyme primes in preschoolers with typical development and specific language impairment. Journal of Speech, Language, and Hearing Research. 2012; 55:32–44.
- Haake C, Kob M, Wilmes K, Domahs F. Word stress processing in specific language impairment: Auditory or representational deficits? Clinical Linguistics & Phonetics. 2013; 27:594–615. [PubMed: 23806129]
- Hick RF, Joseph KL, Conti-Ramsden G, Serratrice L, Faragher B. Vocabulary profiles of children with specific language impairment. Child Language Teaching and Therapy. 2002; 18:165–180.
- Karmiloff-Smith A. Development itself is the key to understanding developmental disorders. Trends in Cognitive Sciences. 1998; 2:389–398. [PubMed: 21227254]
- Karmiloff-Smith A. Nativism versus neuroconstructivism: Rethinking the study of developmental disorders. Developmental Psychology. 2009; 45:56–63. [PubMed: 19209990]
- Kavitskaya D, Babyonyshev M, Walls T, Grigorenko EL. Investigating the effects of syllable complexity in Russian-speaking children with SLI. Journal of Child Language. 2011; 38:979–998. [PubMed: 21310096]
- Kornilov SA, Landi N, Rakhlin N, Fang SY, Grigorenko EL, Magnuson JS. Attentional but not preattentive neural measures of auditory discrimination are atypical in children with developmental language disorder. Developmental Neuropsychology. 2014; 39:543–567. [PubMed: 25350759]

- Kornilov, SA.; Rakhlin, N.; Grigorenko, EL. Morphology and developmental language disorders: New tools for Russian. In: Zinchenko, YP.; Petrenko, VF., editors. Psychology in Russia: State of the art. Moscow: Russian Psychological Society; 2012. p. 371-387.
- Kuhl PK. Brain mechanisms in early language acquisition. Neuron. 2010; 67:713–727. [PubMed: 20826304]
- Kung CC, Hsieh TH, Liou JY, Lin KJ, Shaw FZ, Liang SF. Musicians and non-musicians' different reliance of features in consonance perception: A behavioral and ERP study. Clinical Neurophysiology. 2014; 125:971–978. [PubMed: 24252396]
- Kutas M, Federmeier KD. Electrophysiology reveals semantic memory use in language comprehension. Trends in Cognitive Sciences. 2000; 4:463–470. [PubMed: 11115760]
- Kutas M, Federmeier KD. Thirty years and counting: Finding meaning in the N400 component of the event-related brain potential (ERP). Annual Review of Psychology. 2011; 62:621–647.
- Lau EF, Phillips C, Poeppel D. A cortical network for semantics: (De)constructing the N400. Nature Reviews Neuroscience. 2008; 9:920–933.
- Lee JY, Harkrider AW, Hedrick MS. Electrophysiological and behavioral measures of phonological processing of auditory nonsense V–CV–VCV stimuli. Neuropsychologia. 2012; 50:666–673. [PubMed: 22245012]
- Leonard, LB.; Eyer, JA. Deficits of grammatical morphology in children with specific language impairment and their implications for notions of bootstrapping. In: Morgan, JL.; Demuth, K., editors. Signal to syntax: Bootstrapping from speech to grammar in early acquisition. Mahwah, NJ: Erlbaum; 1996. p. 233-247.
- Lonka E, Relander-Syrjänen K, Johansson R, Näätänen R, Alho K, Kujala T. The mismatch negativity (MMN) brain response to sound frequency changes in adult cochlear implant recipients: A followup study. Acta Oto-laryngologica. 2013; 133:853–857. [PubMed: 23768012]
- Lum JAG, Conti-Ramsden G, Page D, Ullman MT. Working, declarative and procedural memory in specific language impairment. Cortex. 2012; 48:1138–1154. [PubMed: 21774923]
- Magnuson, JS.; Kukona, A.; Braze, D.; Johns, CL.; Van Dyke, JA.; Tabor, W., et al. Phonbiological instability in young adult poor readers. In: McCardle, P.; Miller, B.; Lee, JY.; Tzeng, OJL., editors. Dyslexia across languages: Orthography and brain–gene–behavior link. Baltimore, MD: Paul H. Brookes; 2011. p. 184-201.
- Malins JG, Desroches AS, Robertson EK, Newman RL, Archibald LMD, Joanisse MF. ERPs reveal the temporal dynamics of auditory word recognition in specific language impairment. Developmental Cognitive Neuroscience. 2013; 5:134–148. [PubMed: 23523986]
- Marshall C, Marinis T, van der Lely H. Passive verb morphology: The effect of phonotactics on passive comprehension in typically developing and grammatical-SLI children. Lingua. 2007; 117:1434–1447.
- Marton M, Abramoff B, Rosenzweig S. Social cognition and language in children with specific language impairment (SLI). Journal of Communication Disorders. 2005; 38:143–162. [PubMed: 15571714]
- McClelland JL, Elman JL. The TRACE model of speech perception. Cognitive Psychology. 1986; 18:1–86. [PubMed: 3753912]
- McGregor K, Oleson J, Bahnsen A, Duff D. Children with developmental language impairment have vocabulary deficits characterized by limited breadth and depth. International Journal of Language & Communication Disorders. 2013; 48:307–319. [PubMed: 23650887]
- McMurray B, Samelson VM, Lee SH, Tomblin BJ. Individual differences in online spoken word recognition: Implications for SLI. Cognitive Psychology. 2010; 60:1–39. [PubMed: 19836014]
- Meyer, M. Frog, where are you?. New York: Dial Books for Young Readers; 1969.
- Miller CA, Kail R, Leonard LB. Speed of processing in children with specific language impairment. Journal of Speech, Language, and Hearing Research. 2001; 44:416–433.
- Mills DL, Dai L, Fishman I, Yam A, Appelbaum LG, St George M, et al. Genetic mapping of brain plasticity across development in Williams syndrome: ERP markers of face and language processing. Developmental Neuropsychology. 2013; 38:613–642. [PubMed: 24219698]

- Montgomery JW. Working memory and comprehension in children with specific language impairment: What we know so far. Journal of Communication Disorders. 2003; 36:221–231. [PubMed: 12742669]
- Montgomery JW, Evans JL. Complex sentence comprehension and working memory in children with specific language impairment. Journal of Speech, Language, and Hearing Research. 2009; 52:269–288.
- Nation K. Lexical learning and lexical processing in children with developmental language impairments. Philosophical Transactions of the Royal Society: Biological Sciences. 2014; 369:1471–2970.
- Neville HJ, Coffey SA, Holcomb PJ, Tallal P. The neurobiology of sensory and language processing in language-impaired children. Journal of Cognitive Neuroscience. 1993; 5:235–253. [PubMed: 23972156]
- Newcomer, P.; Hammill, D. Test of Language Development-Primary. Austin, TX: Pro-Ed; 1982.
- Ors M, Lindgren M, Berglund C, Hagglund K, Rosen I, Biennow G. The N400 component in parents of children with specific language impairment. Brain and Language. 2001; 77:60–71. [PubMed: 11247656]
- Perfetti, CA.; Hart, L. The lexical quality hypothesis. In: Verhoeven, L.; Elbro, C.; Reitsma, P., editors. Precursors of functional literacy. Vol. 11. Amsterdam: John Benjamins; 2002. p. 67-86.
- Pflieger, ME. Theory of a spatial filter for removing ocular artifacts with preservation of EEG. Paper presented at the EMSE Workshop; Princeton University; 2001.
- Pizzioli F, Schelstraete MA. Lexico-semantic processing in children with specific language impairment: The overactivation hypothesis. Journal of Communication Disorders. 2011; 44:75–90. [PubMed: 20739027]
- Poll G, Betz SK, Miller CA. Identification of clinical markers of specific language impairment in adults. Journal of Speech, Language, and Hearing Research. 2010; 53:414–429.
- Rakhlin N, Cardoso-Martins C, Kornilov SA, Grigorenko EL. Spelling well despite developmental language disorder: What makes it possible? Annals of Dyslexia. 2013; 63:253–273. [PubMed: 23860907]
- Rakhlin N, Kornilov SA, Grigorenko EL. Gender and gender agreement processing in children with developmental language disorder. Journal of Child Language. 2014; 41:241–274. [PubMed: 23390959]
- Rakhlin N, Kornilov SA, Palejev D, Koposov RA, Chang JT, Grigorenko EL. The language phenotype of a small geographically isolated Russian-speaking population: Implications for genetic and clinical studies of developmental language disorder. Applied Psycholinguistics. 2013; 34:971– 1003.
- Rakhlin N, Kornilov SA, Reich J, Babyonyshev M, Koposov RA, Grigorenko EL. The relationship between syntactic development and theory of mind: Evidence from a small-population study of a developmental language disorder. Journal of Neurolinguistics. 2011; 24:476–496. [PubMed: 21743776]
- Ramus F, Marshall CR, Rosen S, van der Lely HKJ. Phonological deficits in specific language impairment and developmental dyslexia: Towards a multidimensional model. Brain. 2013; 136:630–645. [PubMed: 23413264]
- Rice ML, Cleave PL, Oetting JB. The use of syntactic cues in lexical acquisition by children with SLI: Specific language impairment. Journal of Speech, Language, and Hearing Research. 2000; 43:582–594.
- Robertson EK, Joanisse MF. Spoken sentence comprehension in children with dyslexia and language impairment: The roles of syntax and working memory. Applied Psycholinguistics. 2010; 31:141– 165.
- Roeske D, Ludwig KU, Neuhoff N, Becker J, Bartling J, Bruder J, et al. First genome-wide association scan on neurophysiological endophenotypes points to trans-regulation effects on SLC2A3 in dyslexic children. Molecular Psychiatry. 2011; 16:107.
- Semel, E.; Wiig, E.; Secord, W. Clinical evaluation of language fundamentals. San Antonio, TX: Psychological Corporation; 1995.

- Seppänen M, Hämäläinen J, Pesonen AK, Tervaniemi M. Passive sound exposure induces rapid perceptual learning in musicians: Event-related potential evidence. Biological Psychology. 2013; 94:341–353. [PubMed: 23886959]
- Sharoff, S. The frequency dictionary for Russian. 2001. Retrieved May 10, 2010, from http:// bokrcorpora.narod.ru/frqlist/frqlist.html
- Sheng L, Pena ED, Bedore LM, Fiestas CF. Semantic deficits in Spanish–English bilingual children with language impairment. Journal of Speech, Language, and Hearing Research. 2013; 55:1–15.
- Song Y, Sun L, Wang Y, Zhang X, Kang J, Ma X, et al. The effect of short-term training on cardinal and oblique orientation discrimination: An ERP study. International Journal of Psychophysiology. 2010; 75:241–248. [PubMed: 19995581]
- Spironelli C, Bergamaschi S, Mondini S, Villani D, Angrilli A. Functional plasticity in Alzheimer's disease: Effect of cognitive training on language-related ERP components. Neuropsychologia. 2013; 51:1638–1648. [PubMed: 23685197]
- Spironelli C, Galfano G, Umiltà C, Angrilli A. Word position affects stimulus recognition: Evidence for early ERP short-term plastic modulation. International Journal of Psychophysiology. 2011; 82:217–224. [PubMed: 21924299]
- Stavrakaki S. Comprehension of reversible relative clauses in specifically language impaired and normally developing Greek children. Brain and Language. 2001; 77:419–431. [PubMed: 11386707]
- Stothard SE, Snowling ME, Bishop DVM, Chipchase BB, Kaplan CA. Language-impaired preschoolers: A follow-up into adolescence. Journal of Speech, Language, and Hearing Research. 1998; 41:407–418.
- Tomblin BJ, Records NL, Buckwalter P, Zhang X, Smith E, O'Brien M. Prevalence of specific language impairment in kindergarten children. Journal of Speech, Language, and Hearing Research. 1997; 40:1245–1260.
- Ullman MT, Pierpont EI. Specific language impairment is not specific to language: The procedural deficit hypothesis. Cortex. 2005; 41:399–433. [PubMed: 15871604]
- van den Brink D, Brown TT, Hagoort P. Electrophysiological evidence for early contextual influences during spoken-word recognition: N200 versus N400 effects. Journal of Cognitive Neuroscience. 2001; 13:967–985. [PubMed: 11595099]
- Wadman R, Botting N, Durkin K, Conti-Ramsden G. Changes in emotional health symptoms in adolescents with specific language impairment. International Journal of Language and Communication Disorders. 2011; 46:641–656. [PubMed: 22026566]
- Wadman R, Durkin K, Conti-Ramsden G. Self-esteem, shyness, and sociability in adolescents with specific language impairment (SLI). Journal of Speech, Language, and Hearing Research. 2008; 51:938–952.
- Wechsler, DA. Wechsler Intelligence Scale for Children. 4. San Antonio, TX: Psychological Corporation; 2003.
- Wiesner, D. Tuesday. New York: Sandpiper; 1997.
- Wiesner, D. Free fall. New York: HarperCollins; 2008.
- Wild-Wall N, Falkenstein M, Gajewski PD. Neural correlates of changes in a visual search task due to cognitive training in seniors. Neural Plasticity. 2012; 2012:529057. [PubMed: 23029625]

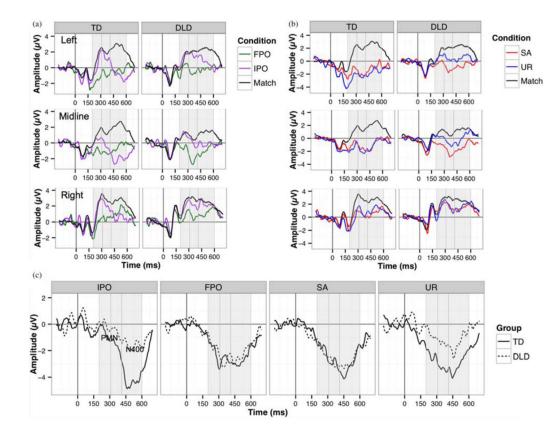


Figure 1.

(Color online) Sample average event-related potential (ERP) waveforms at three parietal electrode clusters (left, midline, right) in two groups of children, developmental language disorder (DLD) and typically developing (TD). (a) Sample waveforms for match, initial phonological overlap (IPO), and final phonological overlap (FPO) conditions; (b) sample waveforms for match, semantically associated (SA), and phonologically and semantically unrelated (UR) conditions; and (c) average difference waveforms for the four mismatch conditions (IPO, FPO, SR, UR) at the parietal midline. Negative is plotted downward.

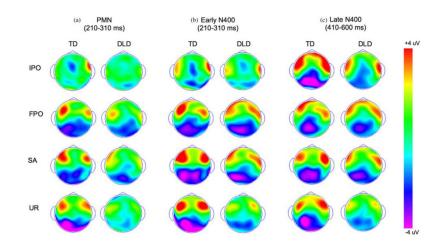


Figure 2.

(Color online) Topographic maps for components of interest (based on difference wave forms) in two groups of children, developmental language disorder (DLD) and typically developing (TD). (a) Average in the phonological mapping negativity (PMN) time window, (b) average in the early N400 time window, and (c) average in the late N400 window. IPO, Initial phonological overlap; FPO, final phonological overlap; SA, semantically associated; UR, phonologically and semantically unrelated.

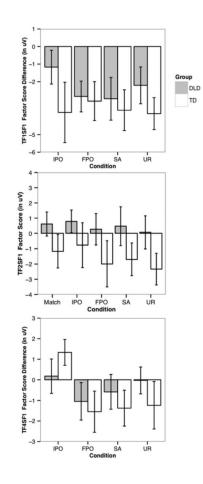


Figure 3.

Average principal component analysis (PCA) factor scores for three temporospatial factors (with 95% confidence intervals). Difference factor scores for each of the mismatch conditions are plotted for TF1SF1 (top row) and TF4SF1 (bottom row). Raw factor scores for each of the five experimental conditions are plotted for TF2SF1 (middle row). IPO, Initial phonological overlap; FPO, final phonological overlap; SA, semantically associated; UR, phonologically and semantically unrelated; DLD, developmental language disorder, TD, typically developing. Negative is plotted downward.

Table 1

Behavioral performance of children with DLD and TD children on the picture-word matching task

	T	D	DI	LD
	M	SD	M	SD
Match hits	0.92	0.13	0.86	0.19
Correct rejections				
IPO	0.86	0.18	0.87	0.11
FPO	0.92	0.13	0.93	0.08
SA	0.92	0.14	0.89	0.18
UR	0.93	0.13	0.93	0.06
Match reaction time (ms)	891	114	955	114

Note: DLD, Developmental language disorder; TD, typically developing; IPO, initial phonological overlap; FPO, final phonological overlap; SA, semantically associated; UR, semantically and phonologically unrelated.

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Table 2

Intercorrelations between ERP indices of lexical and phonological processing and behavioral indices of language and verbal memory development

	Late N400 IPO	Late N400 UR	TF1SF1 IPO	TF1SF1 UR	TF2SF1	TS4SF1 IPO
Verbal memory						
Digit span	196	216	308	249	131	.279
Word span	281	.206	346*	301	149	.051
Narrative scales						
Phonetic/prosodic characteristics	485*	114	363	058	.126	.312†
Well-formedness	.195	.072	.129	.081	.236	090
Complex structures	317^{+}	164	192	057	.068	.282
MLUw	453*	173	484	234	193	020
Semantic/pragmatic errors	.062	060	.006	.193	.268	.121
Lexical richness	476*	260	480*	331*	363*	223
ORRIA	.087	171	.169	092	199	.300

of Spoken Language (Carrow-Woolfolk, 1999). For the late N400 IPO and A, semantically related; UR, semantically and phonologically unrelated; imparable to the Clinical Evaluation of Language Fundamentals (Semel, TF1SF1, differences in factor scores (mismatch – match) were used. For TF2SF1, average factor scores across five conditions were used. For TF4SF1, the difference in factor scores (IPO – match) was the late N400 UR, amplitude difference measures (IPO/UR - match) at the midline parietal clusters were used. For the N200, the average amplitude across five conditions at the midline were used. For used. â

 $f_{p} = .06.$

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 $_{p < .05.}^{*}$