Report

Reduced Neural Integration of Letters and Speech Sounds Links Phonological and Reading Deficits in Adult Dyslexia

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Summary

Developmental dyslexia is a specific reading and spelling deficit [1] affecting 4% to 10% of the population [2, 3]. Advances in understanding its origin support a core deficit in phonological processing [4-6] characterized by difficulties in segmenting spoken words into their minimally discernable speech segments (speech sounds, or phonemes) [7, 8] and underactivation of left superior temporal cortex [9, 10]. A suggested but unproven hypothesis is that this phonological deficit impairs the ability to map speech sounds onto their homologous visual letters, which in turn prevents the attainment of fluent reading levels [7, 11]. The present functional magnetic resonance imaging (fMRI) study investigated the neural processing of letters and speech sounds in unisensory (visual, auditory) and multisensory (audiovisual congruent, audiovisual incongruent) conditions as a function of reading ability. Our data reveal that adult dyslexic readers underactivate superior temporal cortex for the integration of letters and speech sounds. This reduced audiovisual integration is directly associated with a more fundamental deficit in auditory processing of speech sounds, which in turn predicts performance on phonological tasks. The data provide a neurofunctional account of developmental dyslexia, in which phonological processing deficits are linked to reading failure through a deficit in neural integration of letters and speech sounds.

Results and Discussion

Successful acquisition of basic letter-speech-sound (LS) mappings is crucial for attaining fluent reading skills [12]. Functional magnetic resonance imaging (fMRI) in nonimpaired readers has identified heteromodal superior temporal sulcus and gyrus (STS and STG) as well as auditory cortex (heschl sulcus [HS] and planum temporale [PT]) as integration sites

for letters and speech sounds [13, 14]. Reading problems in dyslexia have been primarily associated with a deficit in adequately representing the smallest speech segments (speech sounds, or phonemes) [7, 8], which in turn has been suggested to interfere with the acquisition of LS mappings and hence with the progression from letter-by-letter to fluent, automated reading [7]. The present fMRI study examined the neurofunctional correlates of LS integration as a function of reading ability. Thirteen nonimpaired readers and 13 dyslexic readers, matched for educational level, age, handedness, and IQ (Wechsler Intelligence Scale for Adults, standard scores for nonimpaired = 11.15, for dyslexic = 10.42), were tested on a battery of measures for reading status. All dyslexic readers showed impaired reading (within the lower tenth percentile on a standardized test of word reading) and poor performance on subtests involving phonological awareness, phonological decoding, and spelling (see Supplemental Data available online). Letters and speech sounds were presented during scanning in four experimental conditions: visual, auditory, audiovisual congruent, and audiovisual incongruent.

In the first step of the fMRI analysis, we assessed the relative contribution of unisensory auditory and visual conditions against the baseline by using a multisubject general linear model (GLM 1) for each reading group.

Figure 1 demonstrates that dyslexic and nonimpaired readers activated a comparable network of brain regions in response to unisensory presented letters (occipito-temporal cortex and inferior-parietal lobule, shown in green) and unisensory speech sounds (HS, PT, and STG, shown in red). Furthermore, cortical sites that were activated for both unisensory stimuli in fluent and dyslexic readers were found in the lower bank of STG and STS, structures previously implicated in LS convergence and integration [14] (see Supplemental Data).

In the second step, we examined potential group differences in LS processing between dyslexic and fluent readers by computing the interaction between "reading status" and "experimental condition" with a mixed 2 × 4 factorial model (GLM2). No main effect of reading status was found, but the interaction with condition revealed an STG bilateral cluster anterior-lateral to primary auditory cortex (Figure 2A; F_{3, 72} = 14.3, p = .000 left, $F_{3.72}$ = 7.3, p = .000 right; Talairach coordinates, x = -46, y = -26, z = 6 (left) and x = 45, y = -22, z = 7(right)). Here, the BOLD responses in the dyslexic group were reduced for unisensory presentations of speech sounds $(t_{24} = 4.99, p = .000 [left]$ and $t_{24} = 3.79, p = .001 [right])$ and congruent LS pairs ($t_{24} = 3.85$, p = .001 [left] and $t_{24} = 2.59$, p = .016 [right]) (Figure 2B). Although these differential effects seemed slightly lateralized to the left hemisphere, the statistical interaction with the hemisphere in STG did not reach significance.

To assess whether the activation differences in STG reflect discrepancies in multisensory LS integration between dyslexic and nonimpaired readers, we used two complementary statistical criteria. The congruency criterion, which indexes integration through stronger responses to congruent than to incongruent LS pairs (represented as AV congruent > AV incongruent) directly evaluated the processing of the learned

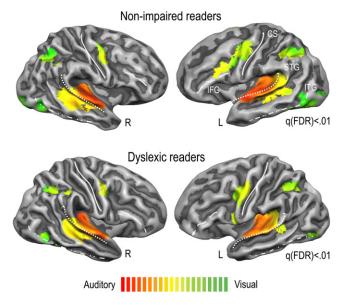


Figure 1. Activation for Letters and Speech Sounds
Response pattern for regions processing speech sounds (red), letters
(green) or both unisensory conditions (yellow) in non-impaired (upper panel)
and dyslexic readers (lower panel).

audiovisual relatedness between letters and speech sounds. Consistent with previous results [14, 15], we found that nonimpaired readers showed stronger activation for congruent than

incongruent LS pairs in bilateral STG ($t_{12} = 5.53$, p = .000 left, $t_{12} = 6.72$, p = .000 right). In contrast, dyslexic readers showed no such activation difference, indicating reduced LS integration. Importantly, this effect cannot be explained by dyslexic readers' insufficient knowledge about LS mappings because they were highly accurate in judging the congruency of LS pairs in offline behavioral tasks (see Supplemental Data). Yet, dyslexic readers were significantly slower than nonimpaired readers, indicating less automatic processing of LS mappings [15, 16].

The second criterion we used to determine LS integration in STG was the multisensory interaction index (MSI) [17]. The MSI represents the multisensory response (MS) relative to the maximally unisensory response (US $_{\text{max}}$), where positive MSI values indicate response enhancement and negative values indicate response suppression (MSI = ([MS - US_{max}]/US_{max}) \times 100). Using the MSI in addition to the congruency criterion is particularly useful in the present study, where dyslexic readers showed reduced activation for unisensory presentations of speech sounds, because it accounts for individual differences in unisensory response strength when one is classifying an area as an integration site. We found that nonimpaired readers exhibited response suppression in bilateral STG for incongruent LS pairs in comparison to the maximal unisensory response ($t_{12} = -3.92$, p = .002 [left] and $t_{12} =$ -4.09, p = .002 [right]), whereas dyslexic readers failed to show such a suppression effect (Figure 2C) (nonimpaired versus dyslexic readers: $t_{24} = -3.19$, p = .004 [left] and $t_{24} =$ -2.75, p = .011 [right]). In contrast, dyslexic and nonimpaired

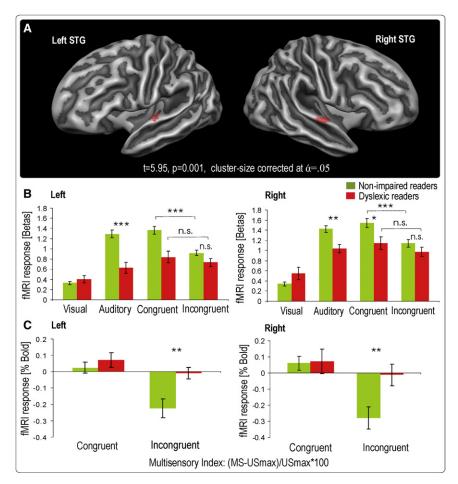


Figure 2. Interaction between Reading Ability and Condition

Group results for the "reading status*condition" interaction analysis (corrected for cluster-size at alpha = .05) projected on inflated cortex-based aligned group map showing clusters in bilateral STG (A). Mean BOLD response and standard error of the mean (SEM) for both reading groups indicates a reduced response to speech sounds and congruent LS pairs in dyslexia (B) and a reduced suppression of incongruent LS pairs relative to the maximal unisensory response (USmax) (C).

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