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Hypotheses for the 'Dyslexia Signal': Biomarkers in the Left and Right Brain of Developmental Dyslexia

Yujia He

Zhejiang Normal University, Zhejiang, China, 321004

heyujia_zjnu@163.com

Abstract. Dyslexia is one of the most common learning disabilities, yet its brain basis and core causes are not yet fully understood. MRI studies commonly demonstrate hypoactivation in left-hemispheric temporo-parietal, occipito-temporal, and inferior frontal networks, along with hyperactivation in corresponding right-hemispheric regions. Based on the recent 15 years of brain imaging researches, this paper proposes three hypotheses about the causes of the activation difference in Dyslexia, analyzes how recent researches support these hypotheses, and draws a conclusion that the three hypotheses have a fusion trend to a certain extent, which can provide possible directions for future studies.

Keywords: Dyslexia, activation pattern, compensatory hypothesis, etiological hypothesis, potential hypothesis.

1. Introduction

Developmental dyslexia is an unexplained inability to acquire accurate or fluent reading that affects approximately 5–17% of children. Dyslexia is associated with structural and functional alterations in various brain regions that support reading.

Meta-analyses of primary research findings have identified broad patterns of functional and structural differences between typical and dyslexic readers. The most common functional brain differences, in children and adults, are reduced activations in the left temporal, parietal, and fusiform (VWFA) regions[1]. In most cases, these hypo activations arise from comparisons between two tasks or conditions, and thus reflect a lack of differential sensitivity to reading demands rather than a broader dysfunction of those brain regions.

In some cases, individuals with dyslexia also show increased activation in corresponding right-hemispheric regions. It is not clear whether increased right-hemispheric activation is a compensatory mechanism underlying remediation or a brain characteristic of dyslexia reflecting failed left-hemispheric lateralization for language. Three hypotheses and their supportive studies are introduced in this article.

The compensation hypothesis is a frequently mentioned hypothesis, which believes that the abnormal activation of brain regions in the reading process of dyslexics is not innate, but is the result of acquired exercise. Due to some unknown shortcomings in the structure and function of the brain of dyslexic patients, they cannot activate the brain regions that should be involved in reading during the reading process. However, in the process of learning to read, they have developed a set of compensation mechanisms to replace the work of those brain regions that cannot be normally activated by other brain

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regions, to achieve the reading activities of dyslexics. After reading intervention, the dyslexic patients should show that the activation level of the brain area in the compensation area is enhanced. The intervention will not make the brain activation mode of the dyslexic patients approach that of the normal readers. They always work in an activation mode different from that of normal readers and continue to strengthen this unusual activation mode.

The potential hypothesis is a variant of the compensation hypothesis. The potential hypothesis believes that the abnormal activation of the right hemisphere of dyslexic patients is a deviation that some children have in the process of reading development. It is not a pathological feature, but a manifestation of delayed maturity. Through reading intervention, the brain activation pattern of dyslexic patients will be close to that of normal readers, thus achieving the role of correction.

The pathological hypothesis holds that the activation mode of the brain of dyslexia patients different from that of ordinary people is not the result of acquired exercise, but is innate and is the cause of dyslexia.

2. Theoretical basist

2.1. Study on the permeability of corpus callosum

Compared with normal readers, the unilateralization of dyslexics does not seem to have developed enough. The activation regions of two hemispheres have a certain tendency to be symmetrical for atypical readers, which might be caused by the inefficiency of the communication between the left and right hemispheres.

Effective communication across dispersed brain regions is necessary for the development of proficient reading. In a recent study, interhemispheric connections were measured using diffusion tensor imaging in a group of kids with various reading levels [2]. They discovered a favorable correlation between diffusivity perpendicular to the main axis of the callosal fibers connecting the temporal lobes and phonological awareness, a crucial component in learning to read. It's possible that those who are strong readers have fewer total axons traveling through this region of the callosum and a higher proportion of large-diameter axons. Poor readers had higher functional symmetry, which is consistent with this explanation because poor readers have denser interhemispheric connection. Poor readers appear to have overactive regions in the right hemisphere to make up for the ineffective communication between the two hemispheres.

2.2. Research on Predicting the Development of Reading

A longitudinal study gives us a different perspective, indicating that overactivation in the right hemisphere can be a positive sign for remediation. The study discovered that Greater right prefrontal activation during a reading task that demanded phonological awareness and right superior longitudinal fasciculus white-matter organization significantly predicted future reading gains in dyslexia [3]. These findings identify right prefrontal brain mechanisms that may be critical for reading improvement in dyslexia and that may differ from typical reading development. In regards to reading pathways, it appears that dyslexic readers who showed gains in reading did so by depending on a right-hemisphere pathway, in contrast to the left-hemisphere pathway that characterizes typical reading.

This study cannot determine whether the right activation pattern for dyslexic readers is a compensatory or etiological mechanism, but the indistinct right activation of the intervention-resistant readers seems to suggest that overactivation in the right side of the brain is not an underlying etiological feature.

2.3. Research before and after intervention

Different imaging techniques have been used to investigate the effects of different reading impairment treatment strategies on brain function. By measuring the brain correlates of language performance before, after, and in some cases during training regimens, these research have specifically followed them longitudinally. Reading and reading-related skills have consistently improved in behavior according to

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studies, and there has been a large plastic rearrangement in the left temporoparietal-occipital regions that has moved from the right homologous areas.

To illustrate, the markers of cortical reconfiguration were monitored using one of the EEG components, N150, during the course of six months of phonological therapy for 14 dyslexic Italian children [4]. N150 is produced in the left occipito-temporal cortex (Brodmann areas 39, 37, and 19) in typical readers, but in previous research and this sample, it was observed in right homologous regions in children with reading problems. Following therapy, the primary N150 generator moved to the left occipito-infero-temporal cortex, specifically to Brodmann areas 37 and 19, with very minor task-dependent changes. Similarly, 80 hours of rigorous remedial coaching led to a noticeable shift from the right to the left hemisphere in a group of struggling readers [5].

The research has demonstrated that cortical plastic remodeling can occur in tandem with improvements in reading accuracy and fluency as a result of treatments. It is not yet apparent, though, whether the observed behavioral alterations are caused by cortical reconfiguration or whether they are just correlates of these behavioral changes, which may be caused by something else.

2.4. Study on the congenital activation pattern

Studies have indicated that the distinct patterns of activation(including overactivation in the right hemisphere) in response to print can be observed as early as kindergarten[6], [7].However, even before formal schooling, it is still difficult to determine whether the brain differences are associated with the underlying neurobiological etiology of dyslexia, or are instead the consequence of altered and often vastly reduced reading experience (including compensatory alterations in reading networks).

White matter anomalies in the left reading networks (but not the right) can be seen as early as infancy, according to a study of pre-reading kindergarteners who spoke Dutch[8]. Reduced FA was seen in the left hemisphere's ventral inferior fronto-occipital tract in FHD+ kindergarten kids, but not in the right, suggesting that white matter abnormalities in the left reading networks precede the initiation of reading. Additionally, a recent study found that these white matter atypicalities exist as early as infancy[9]. In this study, children with FHD+ exhibited lower FA in the central region of the arcuate fasciculus when compared to infants with FHD-. Furthermore, improved language development across all newborns was linked to increased FA in this location.

In a study with Finnish FHD+ infants, ERPs to consonant-vowel syllables (/ba/, /da/, /ga/) were recorded within 36 h after birth[10]. FHD+ newborns showed a bigger response between 50 and 170 ms and a delayed polarity shift from positive to negative in the right hemisphere in response to /ga/ than controls. This slower shift in polarity was associated with both groups' lower pre-literacy scores at 6.5 years and 2.5 years, as well as their worse verbal memory at 5 years [11]. As a result, changes in hemisphere lateralization in FHD+ children have been consistent across various ERP investigations and have been linked to a variety of reading outcomes. Such lateralization differences have either been interpreted as aberrant or delayed lateralization of language areas that causes perceptual deficits in dyslexia, or as compensatory mechanisms used by at-risk children during language processing to offset the improper function of the left-hemispheric language areas.

Another study[12] compared dyslexic children to both age- and ability-matched normally reading children who are years younger than the dyslexic children but read at the same level in an effort to separate the origin and effect of dyslexia in the brain [12]. The idea behind ability-matched children is that they have roughly the same amount of reading experience as older dyslexic children. In comparison to children of similar ages and abilities, dyslexic children in the research showed lower left parietal and occipito-temporal activations, suggesting that these hypoactivations were connected to the underlying causes of dyslexia. Areas of hyperactivation in dyslexia, on the other hand, revealed mechanisms independent of dyslexia associated to the degree of present reading ability.

3. Discussion & analysis

Some of the above studies support a certain hypothesis. For example, follow-up studies before and after the reading intervention show that the intervention makes the right hemisphere of the brain of the

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dyslexic turn to the left hemisphere, supporting the potential hypothesis; Some studies cannot directly support a certain hypothesis, and further research is needed.

3.1. How the potential hypothesis is supported

The study on the permeability of the corpus callosum does not support the pathological hypothesis. The potential hypothesis can be well explained by combining the follow-up study before and after the reading intervention.

(1) The study found that the permeability of the corpus callosum connecting the left and right hemispheres of dyslexic patients is lower than that of typical readers, and the reduction of the permeability of the corpus callosum will lead to poor communication between the left and right hemispheres, This is probably the result of abnormal activation of the right brain in the reading process of dyslexia patients (these abnormally activated regions of the right brain have a trend of symmetrical distribution with the activated regions of the left brain, that is, the unilateral advantage of the brain is not obvious or stunted). The causal relationship between the permeability of the corpus callosum and the abnormal activation mode is more likely to be that the former leads to the latter, rather than that the latter leads to the former. Therefore, the abnormal activation mode is not the cause of dyslexia, but the manifestation of dyslexia. This study does not support the pathological hypothesis.

(2) As the permeability of the corpus callosum decreases, the communication efficiency between the two hemispheres of the brain decreases, and more overlapping parts appear in the work of the left and right hemispheres, such as the auditory area of the temporal lobe and the visual area of the prefrontal lobe. The activated areas of the two hemispheres tend to be symmetrically distributed, which can be seen as a compensation effect or a manifestation of functional disorder. Based on the follow-up study before and after the reading intervention, after intervention, the abnormal activation area on the right side of the dyslexic patients shifted to the left, that is, the brain activation mode of the dyslexic patients realized normalization, which can be explained as follows: after the reading intervention, the permeability of the corpus callosum of the dyslexic patients increased, the communication efficiency of the two hemispheres of the brain increased, the repetitive work of the right hemisphere decreased, and the advantages of the left hemisphere increased. The study of corpus callosum permeability combined with the follow-up study before and after reading intervention can well explain the potential hypothesis.

3.2. How the compensatory hypothesis is supported

The research on the prediction of reading gains by the activation level of the abnormal region on the right can provide some reference for the compensatory hypothesis, but cannot distinguish between the compensation hypothesis and the potential hypothesis.

(1) Research shows that the activation level of the right prefrontal lobe can predict long-term reading gains, and a stronger activation level of the right prefrontal lobe in early reading means better reading development. Therefore, the author speculates that dyslexics and typical readers have different brain activation patterns. Just like some people have right-hand advantages and some people have left-hand advantages, dyslexics gain progress in reading ability through the compensation of right brain function.

(2) The biggest difference between the compensation hypothesis and the potential hypothesis is that after the long-term reading intervention, the abnormal activation areas in the right hemisphere of the brain of dyslexic patients are enhanced or weakened, that is, whether the brain activation pattern of the dyslexic patients tends to normalize or intensify abnormality. Unfortunately, research on predicting reading gains has not fully proved this point. The early activation of the right prefrontal lobe seems to play a crucial role in future reading development, but this study did not prove whether the activation of the right prefrontal lobe, which is different from that of typical readers, has a direct role in the current reading of dyslexics. Specifically, this experiment only measured the initial activation level of the right prefrontal lobe of the dyslexic, and did not measure the activation level of the corresponding region during or after the intervention. In the long-term reading development, if the abnormal activation area on the right side is enhanced, then the compensation hypothesis is supported, otherwise, the potential hypothesis is supported.

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3.3. How the pathological hypothesis is supported

The pathological hypothesis is the most difficult hypothesis to prove for two reasons: (1) Because individuals have been exposed to language, hearing, and visual reading since birth, even preschool children cannot rule out exposure. When an individual can be measured, it is usually inevitable that he or she has read and practiced, and the measurement results cannot distinguish between the acquired or congenital differences. (2) If this difference in activation mode is congenital, can it be explained that this difference is pathological, or that this difference is the cause of dyslexia? Or, to some extent, it can be further verified that this congenital difference is the physiological basis for the occurrence of dyslexia. Is there no risk of dyslexia for newborn infants screened without this pathological feature?

There are two ways to study whether the abnormal activation mode is congenital. One is to study infants to eliminate the influence of the environment as much as possible; Another is to separate the cause and result through experimental design.

The research on infants shows that the lateralization difference of the cerebral hemisphere has appeared as early as infancy, the under-activation of the left hemisphere has been observed, and the overactivity of the right hemisphere has no obvious evidence. However, the abnormality observed in infancy does not seem to indicate that it must be the cause, because the hemispherical advantage of the baby's brain has not been formed, and the accuracy of the measurement results is relatively low; The experimental results of separating the causes and results show that under activation is related to the causes of dyslexia, and over activation is related to the results of dyslexia, which is usually interpreted as compensation. Hypoactivation of specific parts of the left hemisphere appears to be causative, but currently, there is a lack of evidence of whether hyperactivation of the right hemisphere is also causative.

4. Conclusion

The above three hypotheses can be merged to some extent. Atypical reading development was associated with reduced activation of left parietal and occipito-temporalregions and, after the intervention, with plastic reorganization in these regions or compensatory mechanism of frontal regions. Importantly, abnormal neural activation to print preceded reading onset but was malleable to intervention.

The above three hypotheses, especially the first two, have obvious distinctions. According to the above analysis, the biggest difference between the potential hypothesis and the compensatory hypothesis is whether the abnormal activation of the right hemisphere contributes to the reading function of the dyslexic, which is what is lacking in the current research. Future research will focus on testing whether the activation pattern of individuals with dyslexia has a trend of normalization before and after the intervention, to distinguish or modify the potential hypothesis and compensation hypothesis, and to further explore the causes of the activation difference in Dyslexia.

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