論文

How Might the Neuroscientific Study of Reading Inform Us About Effective Methods for Teaching Reading to the Wide Variety of Readers in Today's Classrooms?

Aki Omote

The paper briefly reviews the theoretical background of literacy building (nature-nurture debate, theory, and methodology) by adopting Hulme and Snowling's (2009) framework for developmental disorders of language learning and cognition. The discussion develops a variation of reading that gives a clue to understanding why and how children's reading experiences engender typical and atypical development of the brain (Fern-Pollak & Masterson, 2014; Knowland & Donlan, 2014). It then helps answer the research questions: (1) What neural basis engenders typical versus atypical variations of reading? (2) How should we identify children at risk of having difficulty learning to read? And (3) what is the most effective method for teaching reading? The finding identifies how the neuroscientific study of reading might inform us to improve teaching reading to the wide variety of readers in today's classrooms.

Keywords: literacy building, variation of reading, typical and atypical development, educational neurocience

1. Introduction

Educational neuroscience has an increased focus on children's development of high-level cognition such as language learning, which in turn allows us a new perspective on an interaction between literacy building and environmental factors (ambient language by care givers, education, etc.). This perspective, neuroconstructivism (Mareschal et al., 2007), argues that the human brain and developmental cognition (e.g., learning to read) is best understood as an emergence from the context-dependent interaction of biological (genetic and brain) mechanisms, and that this interaction engenders children's typical and atypical reading processes at all different levels of granularity: gene level, brain level, and cognitive level (see Figure 1) (Knowland & Donlan, 2014; Hulme, & Snowling, 2009; Marshall, 2014).

Whilst a majority of children in their early stage of life can acquire speech naturally, learning how to read requires later systematic instruction and effortful studying (Fern-Pollak, & Masterson, 2014). Unlike speaking, therefore, the skill of reading (and spelling) is an important outcome of literacy building in conjunction with developmental speech functions, such as auditory discrimination, or phonological awareness. Moreover, studies of developmental literacy and developmental disorder have revealed that not only does an individual variation affect the act of reading (accuracy, fluency, and comprehension) but learning to read also affects the structure of

the brain, suggesting that both development and the act of reading are deeply intertwined if they share a relevant context (Karmiloff-Smith, 1998; Fern-Pollak, & Masterson, 2014; Hruby & Goswami, 2011).

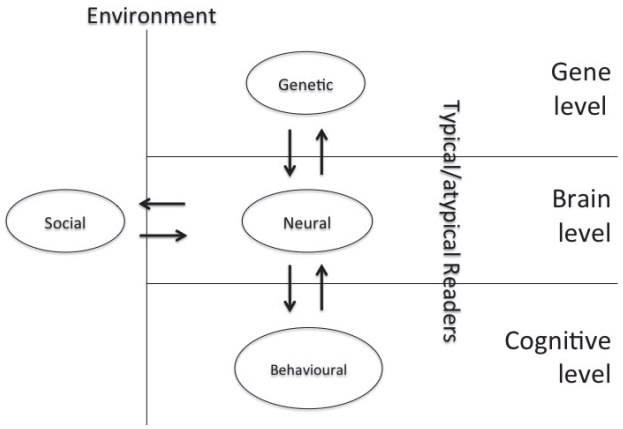

Figure 1 A multi-directional model of the potential gene-brain-cognition pathways. Modified from Hulme and Snowling (2009).

2. Theoretical Background of Literacy Building

2-1 Nature-Nurture Debate

There has been a long-standing nature-nurture debate between nativist and empiricists about how we should view a variation that emerges in children's speech and language acquisition (Behme, & Deacon, 2008; Kuhl, 2007, 2011). Nativists hypothesized that language is innate provided with a so-called universal grammar—a domain specific device in the brain—to allow us auditory input and phonological output (e.g., Pinker, 1999). However, this was derived from the nativist's assumption that babies are born to have a command of their mother tongue (the first language) and that this inherent ability operates various aspects of brain cognition that brings about individual variation (Behme, & Deacon, 2008). Such a view has a polemic advantage to debate but only gives us a perspective of the typically speaking population, not providing us with much insight into atypical readers' development and variation.

On the contrary, empiricists, who postulated social aspects of language acquisition in a more complex situation (bilingualism, learning difficulties, etc.), claimed that language is a cultural artifact and babies should learn spoken and written language primarily through experience (e.g., Kuhl, 2007, 2011). Empiricists admitted that specific aspects of human cognition are due to a dynamic and continuous mechanism of development that involves ontogenetic (neural) growth from the brain modalities. Knowland and Donlan (2014), for example, pointed out the importance of exploring why a substantial degree of variability in reading (word recognition) is seen during

infancy and early childhood (0–5 years). They contend that we should "consider the progressive maturation of the language system at the neural level, and the gradual building of language behavior over developmental time" (p. 135). This is a notable point given that a majority of children seem to be able to build up speech successfully in their early years and that variations might increase in future as children become literate through nursery and educational (thus not inherent but social) interventions.

Furthermore, studies of developmental disorders together with neuroimaging techniques provide with a broader framework of typical and atypical development which allows us to assess the development of brain function and social cognition in children. For example, Karmiloff-Smith (2009), using computational modeling, claimed that the dominant nativist approach that the developmental states in the brain are structured, hard wired, and unaffected by environmental factors (e.g., Piattelli-Palmarini, 2001) should be replaced by a more dynamic approach in which genes, brain, cognition, and environment interact multi-directionally (see *Figure 1*), and presented the evidence using a neuroconstructivist approach (Mareschal et al., 2007; Sirois et al., 2008). This claim seems particularly valid for the neural basis of mechanisms in atypical readers, such as dyslexics, who have been observed and documented as having a different pattern of brain activation showing compensation for their difficulty (Fern-Pollak, & Masterson, 2014; Shaywitz et al., 2003). Methodologically, dyslexia studies have indeed contributed to the elucidation of reading and reading impairment, aided by a large number of brain-image technology (e.g., Leonard et al., 2006; Shaywitz et al., 2004) and brain damage studies (e.g., Geschwind, 1970; Temple, 1997).

2-2 Theory of Development in Reading

From an information-processing view, reading refers to transforming print to speech, or print to meaning (Coltheart, 2005). Specifically, reading requires decoding and the subsequent interpretation (comprehension). Gough and Tunmer (1986) formalized the idea in the Simple View of Reading model where reading (R) is the product of decoding (D) and linguistic comprehension (C). The formula was expressed as follows:

$$R = D \times C \quad (1)$$

Notably, this formalization denotes that, while reading is in process, decoding is necessary since no reading occurs without decoding in itself—if D is equal to zero, then the result is null R, as in the situation where we see foreign passages right in front of us. How beginners learn to decode printed words should determine a stage in the development of reading. In this regard, Ehri's (2005) theory of learning to read (alphabetic) language has emphasized different decoding phases (i.e., pre-alphabetic, partial alphabetic, and full alphabetic) prior to the developed stage of reading with complete comprehension (automaticity). Hence, the Simple View model further predicts three types of different reading impairments in children: (1) atypical (undeveloped)

decoders—children with dyslexia, who have adequate linguistic comprehension and intelligence; (2) atypical (undeveloped) comprehenders—children with reading comprehension impairment, who have adequate reading accuracy; and (3) other "garden variety" readers, who demonstrate deficient adequacy in both comprehension and accuracy (Gough & Tunmer, 1986, p. 8).

More recently, the dual-route (DR) model (Castles, 2006; Ziegler, Perry, & Coltheart, 2000) contributed much to the identification and understanding of different varieties of developmental dyslexia because it explained the problem of accuracy and fluency that dyslexics in early childhood (developmental dyslexia), as well as adults with reading disability (acquired dyslexia), face. For example, the two routes of the DR model are helpful for predicting dyslexic children's error-prone reading when they read printed words (Fern-Pollak & Masterson, 2014). Namely, if the lexical/semantic route that allows meaning retrieval (lexical access) is impaired, children may have more difficulty learning irregularly spelled words due to regularization errors, which is quite similar to the symptomatic dissociation called *surface dyslexia* in adult acquired dyslexia (Marshal & Newcomb, 1973). Alternatively, if the sub-lexical/phonological route that involves analytic, one-by-one parsing of the letter string (grapheme-to-phoneme conversion) is impaired, children may have more difficulty in correctly pronouncing novel words and nonwords, although they can recognize familiar words irrespective of spelling, which is also similar to the adult situation called *phonological dyslexia* (Funnell, 1983).

Notably, however, despite the similarity between child developmental dyslexia and adult acquired dyslexia predicted by the DR model (i.e., phonological causality to the reading difficulties), developmental dyslexia is characterized not only by the absence of brain damage but also by the absence of intellectual disorders (Snowling, 2000). Therefore, children with dyslexia have a distinct reading impairment of accuracy, if not in fluency, which appears different from poor comprehenders (postulated as such in Simple View model) as poor comprehenders have a primary difficulty not with accuracy but with comprehension (Hulme & Snowling, 2009). Poor comprehenders frequently results in a diagnosis of specific language impairment (SLI), which will not be addressed further in this essay.

2-3 Methodology Underpinning Studies of Development

According to the framework of Hulme and Snowling (2009), two contrasting views—typical versus atypical pattern, and slow versus deviant timing—are of great importance for designing the study of literacy development in typical and atypical readers from babies through adults. First, it is important to note that reading engenders both typical and atypical neural patterns seen in different trajectories through the development of the brain (Knowland & Donlan, 2014; Mareschal et al., 2007). Despite a large number of studies that contrasted children's reading impairment with the impairment seen in adults following brain damage (e.g., Temple, 1997), Hulme and Snowling rejected the adult versus child approach as theoretically invalid because those studies were based on a view that cognition seen in adults should be essentially innate and

competent. They postulated instead that neural circuits are being modified by the action of impaired reading so that the reading impairment might be compensated for as the reader matures and follows his or her own developmental trajectory (e.g., Shaywitz et al., 2003). They outlined this typical versus atypical continuum approach as follows:

> A theory of reading development needs to specify how typically developing children learn to read. A theory of developmental dyslexia would then specify how and why the processes that are involved in typical reading development are impaired in children with developmental dyslexia (pp. 19–20).

According to Hulme and Snowling (2009), the timing of development is also important, particularly for the design of dyslexia studies. Since development involves change over time, the complexity of development gave researchers a hypothesis about the extent to which different developmental disorders reflect delay (maturation speed) or deviance (abnormal maturation). Hulme and Snowling identified delay in development as a distinguishing characteristic, illustrating that patterns of reading seen in dyslexic children, at least early in development, "usually resemble those seen in younger typically developing children: such patterns are described as delayed rather than deviant," albeit in some cases deviant in later developing processes (p. 23). This view underlies the methodology used in numerous research studies (Snowling, 2000), which have demonstrated a majority of dyslexic children have more difficulties in reading than normally developing children (a chronological-age [CA] matching) but comparable to younger children of roughly similar reading skill (a reading-age [RA] matching).

3. The Neural Basis of Reading in Typical and Atypical Readers

3-1 Typical Readers

Research can trace back to studies of aphasia that made an inference from aphasic deficit models to intact language processing (Bear et al., 2015). Classical neurolinguistic models emphasized the fixed roles of three neural bases (Broca's area in the inferior frontal lobe, Wernicke's area between the superior temporal and the parietal lobes, and the angular gyrus). This model, the Wernicke-Geschwind Model (Geschwind, 1970), posited left-cortical localization that subserves processing of speech and becoming verbally competent. However, since this model was based on post mortem examinations of cortical lesions, it lacked generalizability and replicability.

Later, Peterson, Fox, Posner, & Raichle (1988), examined healthy individuals' reading (single-word processing) by using positron emission tomography (PET) in both brain hemispheres to reveal that audio- and visually-induced brain activations occurred extendedly along the central fissures of both hemispheres. Their conclusion was a compelling one going beyond the Wernicke-Geschwind Model (superiority in left-localization) in so far as reading process (word

recognition) involves audio and visual areas across both hemispheres. Nevertheless, it was not until recently that a developmental design was adapted by studies and the relationship between age, education, and development, became empirically more noticeable in terms of educational neuroscience.

Recent advances in functional magnetic resonance imaging (fMRI) found that the neural basis of typical reading undergoes continued shifts and dynamic refinements as a consequence of both biological maturation and relevant experiences; the localization of language-related brain activations goes from the right to the left hemisphere as a function of age and education (Knowland & Donlan, 2014). McNealy, Mazziotta, and Dapretto (2011), for example, tested a total of 156 participants—from five years old to adulthood—and demonstrated that word segmentation (the detection of word boundaries in speech) is arguably based on the lateralization that takes place substantially during mid-childhood and through puberty. Similar results were also found by an electrophysiological study using an index of auditory discrimination called mismatch negativity (MMN, Näätänen, Paavilainen, Rinne, & Alho, 2007). Bishop, Hardinam, and Barry (2011), comparing electro-encephalographic (EEG) data with behavioural data to find a significant linear trend of increasing synchronization across different age groups (mid-childhood, puberty, and adulthood). To put these results together, brain lateralization as well as auditory processing might typically evolve through the accumulation of phonological (or sub-lexical) experiences in reading from childhood through adolescence.

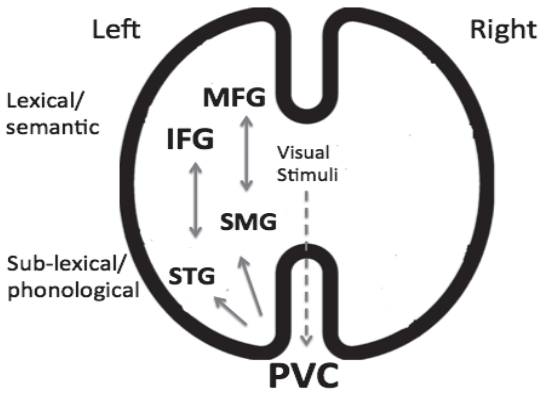

Figure 2 Schematic representation of neural connectivity in later reading. Red arrows represent connections between major regions, whilst dotted arrow represents visual stimuli. Bidirectional arrows represent competitive and cooperative relationships between lexical/semantic function and sub-lexical/phonological function in reading. IFG, left inferior frontal gyrus (Broca's area); MFG, left middle frontal gyrus; PVC, primary visual cortex; SMG, supramarginal gyrus; STG, left superior temoral gyrus (Wernicke's are). Modified from Fern-Pollak and Masterson (2014).

Synthesizing the results of neuroimaging studies in terms of a long-term emergence of complex neural activation in later reading, Fern-Pollak and Masterson (2014) summarized that "areas that act as mediators between orthographic and phonological information lie within the left association cortex" (p. 183). Simply put, reading begins with sub-lexical and phonological neural connectivity of letters (visual stimuli) in the left inferior temporal region (fusiform gyrus). This region activates equally for word and nonwords (Mareshal et al., 2007), culminating in extended lexical and semantic connections (supramarginal gyrus [SMG], superior temporal gyrus [STG, Wernicke's area]). Those connections emerge as comprehension increases in the left frontal cortices (middle frontal gyrus [MFG], inferior frontal gyrus [IFG, Broca's area]) (see *Figure 2*). Interestingly, the view ensures consistency with the DR model from a developmental point of view. That is, the act of reading normally activates areas within the left association cortex in a cooperative and competitive manner, and, therefore, the neural connectivity would in turn have a facilitative effect on children's later reading skills. For example, the act of reading high frequency words immediately induces fast lexical representations, whilst the act of reading low frequency words or nonwords increases time that is needed for detecting connection (Mareshal et al., 2007). In the latter case, as the DR model predicted, there would be an effect of conflict that factors such as lexicality (word versus nonword) and spelling consistency (i.e., word length or transparency of orthography that varies across language) would modulate the preference (phonological awareness) of readers (Hruby, & Goswami, 2011), as well as reading speed (Ziegler & Goswami, 2005).

3-2 Atypical Readers.

There has been agreement that some genetic origin with regard to atypical development in reading deficit should exist with evidence presented by twin studies or genetic linkage and association studies. For example, The Colorado Twin Study (DeFries, Fulker, & LaBuda, 1987) found that the concordance rates were 68% for identical (MZ) and 38% for fraternal (DZ) twins, which suggested that genetic influences are indispensable. More recently, Cope et al. (2005) detected the existence of chromosome 6p called KIAA0319, which seems to be a susceptibility locus for developmental dyslexia. However, studies that investigated the cognitive symptoms observed in dyslexia have presented different underlying causes that have been associated with error-prone reading performance in dyslexic children and adults: auditory processing deficit (Tallal, 1980), visual processing (magnocellular) deficit (Stein, 2001), visual and attentional processing deficit (Boss, Tainturier, & Valdois, 2007), and most compellingly, phonological processing deficit (Snowling, 2000).

Against this background, increased evidence has been presented from brain-image findings that developmental differentiation in patterns of neural activation exist between dyslexic pre-school readers and reading counterparts, accompanied by reduced specialization in the

cortices of the left hemisphere: e.g., IFG (production process), SMG (phonological process), angular gyrus (AG, visual-to-phonological process), and fusiform gyrus (visual encoding of written word forms)(Fern-Pollak & Masterson, 2014). In essence, the core deficit in dyslexia is phonological in nature (Hulme & Snowling, 2009; Shawywitz el al., 1998). In other words, developmental dysfunction of left-lateralization—the dysfunction of left frontoparietal regions followed by the poor development of occipitotemporal regions—seems probably a most compelling theory for the impairments in the observed behaviour of accuracy and fluency in dyslexic reading. Most importantly, however, as Fern-Pollak and Masterson pointed out, there are questions of whether or not the differentiation exists at earlier stages of development in the same situation: Do adult dyslexics have such patterns of neural activation as a consequence of a lifetime error-prone reading, or does abnormal neural processing characterize dyslexic children? These are pressing questions for researchers and practitioners to address from a *prognostic* perspective of education and educational interventions for children with reading difficulties (Bruer, 2014; Marshal, 2014).

4. The Early Identification of Children at Risk of Having Difficulty Learning to Read

For teachers to identify children at risk of developing reading difficulties is not an easy task due to the lack of consensus about the definition of reading developmental disorders (Bruer, 2014). Despite a large number of studies of dyslexia and associated areas (educational and clinical), issues of definition for dyslexia in the Diagnostic and Statistical Manual of Mental Disorders (DSM) have been controversial (Hulme & Snowling, 2009). From the educational point of view, for example, Snowling and Hulme (2012) claimed that DSM-5 (2010) does not detail inter-relationships between learning disorders (dyslexia, dyscalculia, and disorders of written expression) and communication disorders (language impairment, SLI, and speech-sound disorder), and that DSM-5 should reflect the "multiple risk factors and causal mechanisms" of comorbidity (on which dyslexia has an important effect)(p. 593). According to Snowling and Hulme, therefore, primary understanding of dyslexia as a core factor of reading disorders—difficulties with decoding followed by difficulties with comprehension—"is essential if practitioners are to ensure that children's reading difficulties are identified early, and timely interventions are put in place" (p. 593).

Studies of dyslexia have presented early individual differences in an attempt to distinguish between different types of activation for various poor readers, with an assumption that learning to read arguably changes the structure of individual brain with divergent effects on neural outcomes (Leonard et al., 2006; Shaywitz et al., 2003, 2004). There is increased evidence that older dyslexic readers learn to compensate for the disruption in posterior brain (visual-to-auditory) regions by recruiting different (right inferior frontal cortex, Shaywitz et al., 1998), and more extended regions (right prefrontal areas, Shaywitz et al., 2003). Using fMRI, Shaywitz et al. (2003), for example, studied three groups of young adults (18.5–22.5 years: compensated dyslexics, persistent

dyslexics, and good readers), as they read nonwords (phonological task) and real words (semantic task). The compensation was identified as being poor in primary but not in secondary school, whilst the persistence was identified as being poor throughout primary and secondary school.

The result revealed task-dependent, compensated neural engagement and corresponding performances that varied between each group. Compensated readers' reading was more accurate and fluent than persistent readers. Furthermore, there were different activation patterns between the former (right IFG, right middle temporal gyri, and left anterior cingulate gyrus) and the latter (only right IFG), although both groups of dyslexic readers and both children and adults had reduced activation in left hemisphere posterior brain systems (sub-lexical/phonological regions in *Figure 2*). The authors suggested that despite similar socioeconomic risk factors early in life (controlling variables), the emergent activations might have allowed compensated readers to minimize the consequences of their phonological deficit.

The other findings indicated subtle differences in brain activation but significant variations in performance. There was no significant difference between persistent readers and reading controls' activation in the temporoparietal area. However, an increase in the occipitotemporal region in the persistent readers' activation compared to reading controls was unexpectedly observed, despite the better performance of real-word reading in reading controls (96% accuracy) than in the compensated (92% accuracy) and in the persistent (92% accuracy), and the significantly better performance of nonword reading in the reading (94%) and in the compensated (92%) than in the persistent (83%). Hence, the findings supported their hypothesis that when children with persistent dyslexia tackle real word reading, the occipitotemporal region should function as a component of a mnemonic network that is connected to right prefrontal areas often associated with memory retrieval. In short, a variation seen in the development of children with dyslexia is, presumably, due to a compensatory mechanism for the phonological deficit, in the connectivity of the visual system with higher-order cognitive systems in the right frontal cortex, which would lead to the divergent trajectories between compensated readers and persistent readers (Fern-Pollak & Masterson, 2014; Shaywitz et al., 2003).

5. Effective Methods for Teaching Reading

We have confirmed that variation between compensated and persistent readers emerges according to the task type (real words reading versus nonwords reading) and the cognitive load (high working memory load versus low working memory load), accompanied by the specific patterns of neural activation (Shaywitz et al., 2003). Of great value is the educational implication; that is, tailoring task focus (type and workload) depending on children's need. This is a cornerstone of accommodation and the quality of intervention for children with dyslexia so that they might be able to attain compensatory strategies for their reduced phonological processing. With instructional focus on the phonologically mediated intervention in an attempt to improve reading fluency in dyslexic children, Shaywitz et al. (2004), using fMRI, studied the effects of a

year-long intervention on children aged 6.1–9.4 years.

Shaywitz and colleagues (2004) examined whether the effects on automatic, instant word recognition can be linked to changes in patterns of activation in occipitotemporal regions that were required for younger dyslexic readers to develop later phonological awareness and then to become skilled in fast-paced reading (Shaywitz et al., 1998; Shaywitz et al., 2003). The study provided evidence that a pinpointed—what was called "evidence-based" (p. 926)—intervention could engender significant accommodation by creating neural connections that was similar to the connectivity that had emerged in reading controls. The findings, therefore, strongly indicated that the nature of the early remedial, educational intervention is critical to later success in dyslexic children's reading (Hulme & Snowling, 2009).

6. Conclusion

This essay reviewed the background, theories, and methodology of developmental disorders of language learning and cognition in order to explore how children's reading experiences in conjunction with neural engagement engender typical and atypical development of the brain. This helped understand reading to the wide variety of readers in today's classrooms. The DR model demonstrated reading difficulties in children with less accuracy and fluency, predicting that the core deficit in dyslexia exists in a way such that visual encoding processes might weakly connect to phonological processes followed by a severe conflict and poor connectivity to lexical and semantic processes.

However, studies of neuroscience combined with cognitive studies also inform us how learning to read changes the structure of our brain. Studies suggested that dyslexia is open-ended; different connectivity and focal engagement (e.g., receiving a pinpointed intervention) can serve as an ameliorable remediation in children with dyslexia, although a limitation has been shown for children with persistent reading difficulty.

Hence, the answers for the research questions are as follows:

(1) What neural basis engenders typical versus atypical variation of reading?

Reading begins with sub-lexical and phonological neural connectivity of visual stimuli in the left inferior temporal region that activates for word and nonwords, and then culminates the lexical and semantic connections in the left frontal cortex. Typical reading activates areas within the left association cortex in a cooperative and competitive manner, and therefore, the neural connectivity would in turn have a facilitative effect on children's reading by later development of reading at the higher-order level. On the other hand, developmental differences in patterns of neural activation exist between dyslexic readers and reading controls, accompanied by reduced specialization in the cortices of left hemisphere. The core deficit in such poor readers is phonological in nature. Developmental dysfunction of left-lateralization seems probably an underlying cause that impedes accuracy and fluency in poor reading.

(2) How should we identify children at risk of having difficulty learning to read?

Although there is neither complete agreement on, nor established etiology for how we should define and treat developmental reading disorder, a considerable amount of data is available for researchers or practitioners to identify children at risk of having difficulty. Studies have indicated that dyslexic participants present a different pattern of activation than reading controls, and that older dyslexic participants tend to compensate for the disruption in posterior brain regions and this is correlated with phonological deficits. Studies also indicated a variation of children with difficulty learning to read (i.e. accuracy and fluency), revealing that due to the compensatory mechanism for this phonological deficit, the variation seen in dyslexia lies in the connectivity of the visual system with higher-order cognitive systems in the right frontal cortex, which leads to the divergent trajectories between compensated readers and persistent readers.

(3) What is the most effective method for teaching reading?

Early remediation is critical to later success in dyslexic children's reading, although children with persistent dyslexia still read with less reading accuracy and less fluency even after intervention. Effective methodology for (classroom) teaching should be considered in terms of timing and task. Research suggests that this should be at an early school age, probably when systematic experience of reading starts to burden brain activation. An early remedial education at school and clinic to improve phonological deficit can have the effect of boosting, or engendering significant accommodation by approximating neural connections that is distinct in structure but functionally similar to the typical connectivity.

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学校での読解教育における脳神経科学の影響とは何か？

表　昭浩

本論文では、ことばの学習と認知的な発達障害に関する Hulme and Snowling（2009）の枠組みを採用し、子どもの教育におけるリテラシー形成の理論的背景（Nature-Nurture 論争、理論、方法論）を概観し、子どもの読み手としての体験がなぜ・どのように脳の定型的・非定型的発達を生み出すのかという問いに対して、読み手が独自に発達させている様々なバリエーション（Fern-Pollak & Masterson, 2014; Knowland & Donlan, 2014）を特定する。これにより、(1) どのような神経基盤が、読み手の定型発達及び非定型発達の区別（バリエーション）を生み出すのか、(2) 読みの習得に困難を感じ，リスクを抱えている子どもをどのように見分けるべきか、(3) これらを踏まえた最も効果的な読みの指導法とは何か，という問いへの解答を試みる。得られた知見は学校教育における神経科学的研究の重要性を明らかにしており、それは今日学校に存在している多様な発達の子どもたちへの読解指導を改善する情報を提供している。

キーワード：リテラシー形成、読み手のバリエーション、定型発達と非定型発達、教育神経科学