A neuro-constructivistic research strategy to study the underlying causes of dyslexia

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The present article suggests an extended neuroconstructivistic research strategy for assessing the causes of dyslexia. Instead of following one line of argumentation, such as the influence of auditory perception on the development of phonological awareness, a systematic facet model is suggested to identify the development of singular, bi-, and cross-modal perception, attention, and higher cognition in dyslexia over time. In addition, such a study should also include etiological measures such as genetic and family risk factors. This developmental approach is necessary to suggest a pragmatic concept for investigating all processing types of dyslexia under several methodological aspects.

Keywords: dyslexia; cause; modality; etiology; development; perception; cognition

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Dyslexia is a specific developmental disorder of scholastic skills, the symptoms of which vary in degrees of reading and spelling difficulties. Depending on the criteria and sample sizes used, the prevalence rate of dyslexia among children ranges from 2 to 10%, and one third of the dyslexic population is male (1–3). Dyslexia can lead to intellectual and emotional issues and, consequently, to severe problems in academic and economic development (4, 5). Intervention strategies that prevent and treat dyslexia primarily focus on two approaches: cognitive-oriented interventions that are related to the training of phonological awareness or speech sound perception and symptomatic approaches that directly address reading and spelling. Results from long-term studies have shown that implementing a combination of cognitive skills training and a symptom-oriented approach leads to success on both the preventive and therapeutic levels (6, 7).

Currently, different models are used to account for the causes of dyslexia. These models operate on neurological, cognitive, and behavioral levels and are used to explain the disorder-related observable indicators in a theoretical causal framework. To illustrate the interplay of functionality in dyslexia, Frith (8, 9) developed a model of the interactions among these neurological, cognitive, and behavioral levels (8, 9). Over time, the model has been extended to include the results of genetic, neurological, neurocognitive, and behavioral studies (10–13).

Probabilistic multiple cognitive models give an understanding of how developmental disorders can be explained by multiple or single factors. In addition, these models also consider the etiology such as the genetic determination as well as family risk factors. These models also explain the comorbidity between different developmental disorders such as attentional disorder, autism, and dyslexia (14).

One essential line of reasoning contained within this model is a genetically determined abnormality in the left perisylvian speech area that is thought to be the fundamental cause of the phonological deficit. Supporters of this cognitive-oriented, phonological hypothesis claim that a deficit in the representation of phonemes plays a central role in dyslexia and explains the difficulty dyslexic people have with the development of reading and spelling skills (15–18). The key problem is thought to be a failure in phonological awareness, and, consequently, a failure to learn the ability to assign graphemes to phonemes (19). Klicpera and Gasteiger-Klicpera (20) and Franke (21) refer to these mappings as grapheme-phoneme correspondences, a set of assignments that, as a system, regulates the representation of the sounds of letters, and a dysfunction in the acquisition of grapheme-phoneme correspondence rules has a direct impact on the ability to learn how to read and write (20, 21). Furthermore, visual, auditory, and fine motor problems can occur as a consequence of failing to acquire grapheme-phoneme correspondence rules. It is thought that, under certain hormonal conditions during pregnancy, the developmental anomalies that underlie dyslexia can also induce secondary disturbances in the sensory routes emanating from the thalamus (22).
Another important hypothesis regarding the mechanisms underlying dyslexia is neurobiologically oriented and assumes a structural deficit in the magnocellular system in dyslexic people that leads to a decreased ability to detect rapidly presented visual and auditory stimuli. Proponents of this hypothesis originally assumed that a disturbance in dynamic vision and, thus, an inability to fixate letters, might be responsible for dyslexia (23, 24). Later, some authors also subsumed specific aspects of auditory processing disorders, referring in particular to the perception of melodies and sound transitions into syllables. As proposed, these sound transitions are necessary for the differentiation of consonants within a consonant category.

Consequently, this hypothesis claims that the magnocellular deficit may hinder the development of phonological awareness, which makes grapheme-phoneme correspondences more difficult to acquire (25, 26).

There are a considerable number of studies in the field of dyslexia research, each addressing visual (27), auditory (28), or phonological deficits separately (29). In these studies, the phonological deficit was found to be a cardinal symptom in all subjects, but specific neurocognitive profiles have also been identified in dyslexia. Ramus and his colleagues identified subgroups with auditory, visual, and fine motor deficits, but all of the subjects showed a disorder of phonological processing (11). Additionally, cluster analyses designed to identify subtypes of dyslexia found a failure in phonological processing to be the primary symptom in dyslexic people (30).

Problems in research on dyslexia
To form conclusions regarding the causes of dyslexia and to prove the persistence of neurocognitive profiles, it is necessary to observe participants over time (31, 32). Thus, metrologically and statistically, it is only possible to determine the causes of dyslexia when data acquisition can be repeated over several developmental stages (16, 33). However, most studies operate only on a cross-sectional level within specific age groups, mostly using adults (11, 34–36). One major issue facing the study of developmental disorders is that adults have acquired strategies to compensate for their reading and writing problems (37). These compensations are related to plastic changes in the brain and lead to problems interpreting neurocognitive outcomes (38). In particular, the results of neurophysiological or neurofunctional studies in adults have limited validity in regard to drawing conclusions about the neurocognitive causes of dyslexia. The results of studies using adults have limited validity, and cross-sectional studies using children only present snapshots of the complexity of dyslexia. These studies provide no insight regarding how these deficits have occurred or changed over time and provide no satisfactory explanations regarding the causes of the disorder.

A second important issue is the investigation of sensory, cognitive, and motor deficit profiles in dyslexia and whether they are additive or conjunctive on the behavioral and neurocognitive levels (11, 14). Sensory processing includes all modalities that are necessary to process written language, depending on salience, different levels of perceptual load, and lingual content. Cognitive processes include the mental effort and attention required to identify acoustically presented sounds and phonemes and to select visually presented letters, syllables, words, and whole sentences (39). Additionally, short-term, working, and long-term memories with different levels of lingual information are necessary to keep information in mind and to combine and segregate the necessary elements to produce and retrieve lingual information (40). In addition, it seems that a systematic latency effect of letters on speech sound processing reflects the association of letters and speech sounds that is the basic foundation to build grapheme phoneme correspondences (41). A systematic description of how to measure this deficit profile is still lacking.

Third, the question still remains for operational findings instead of theoretical or probabilistic approaches. Probabilistic multiple cognitive models can describe the potential of connections between developmental disorders and their determination, but an operational model is necessary to investigate mutual causes and different levels of severity of these disorders, to understand the complexity of these, and to develop what strategies are helpful and in which state of development they have to be applied.

Taken together, these points lead to the questions of how sensory and cognitive deficit profiles in developmental dyslexia occur and how they change over a lifetime on a behavioral as well as a neurocognitive level.

A proposal for neuroconstructivistic research
To investigate deficit profiles in dyslexia, a systematic facet model is required that includes modality-specific attention and perception and higher cognitive functions, such as the representation of words in working memory and the language-specific knowledge stored in long-term memory, which are necessary to perform complex linguistic tasks, such as reading and writing (see Table 1 and Fig. 1) (42). First, a detailed analysis of auditory and visual processing will be necessary to investigate the ability of a dyslexic person to perceive auditory frequencies and frequency sequences, as well as letters, words, and sentences, containing different grades of complexity. Furthermore, it will be necessary to investigate temporal aspects, such as the duration of rising and declining lingual and non-lingual sound information as a possible link between lingual and non-lingual processing in the auditory domain (43). In the visual domain, it may also be necessary to understand the mechanisms of purely
visual processing, such as coherent motion paradigms and language-related visual processing (44).

Second, regarding the measure of working memory as a time consuming process, it will be necessary to utilize tests that have been developed to examine different modalities and information processing types to assess response times, hits, and false alarms for each operation type. Other aspects of working memory, such as the coordination and integration of information during storage, are also necessary to process written words (42). Thus, it will be useful to measure the span of working memory and the systematic investigation of the latency of letters and speech sound processing to have a clear understanding of to which time point and how much information dyslexic people are able to process (41).

Third, comparable to the trajectories of knowledge and cognitive speed, the different processing types might have an influence on reading and spelling (31, 45–47). An examination of the development of these different processing types from early childhood through adulthood will provide insight regarding the genuine development of reading and spelling skills, sensitive phases of attention, perception and higher cognitive abilities, and the influence of external factors, such as specific or nonspecific training or the education level of the parents. In other words, it will be possible to explore additional causes for dyslexia.

Fourth, the identification of etiological risk factors, such as genetics and family, are necessary to get a deep understanding of the influence on cognitive and behavioral outcomes in dyslexia (14).

Fifth, with a multi-methodic approach, it will be possible to investigate several perspectives of neurocognitive processing. With electroencephalogram, functional magnetic resonance imaging, and systematic behavioral testing batteries, it will be possible to examine profiles of neurocognitive processes that are associated with dyslexia and that are related to behavioral data.

The combination of cross-sectional and longitudinal studies with at least two dyslexic groups and chronologically, intellectually, and reading age-matched control groups would allow for the investigation of the causes of dyslexia using a developmental design to control variables such as the mental effort used by dyslexic people, biological age, or cognitive developmental stage (48, 49). To achieve insight into the causes of dyslexia, it will be necessary to use a developmental design with several repeated measures from early childhood until adulthood including genetic analyses to identify the specific and nonspecific influences of systematic reading and spelling instruction over time (see Fig. 2) (50).

In addition, different types of dyslexia related to reading or spelling problems could be identified. Furthermore, on the basis of the concept of the speed-accuracy trade-off, it will also be necessary to identify groups with different processing styles, such as being faster and making more mistakes instead of processing at a slower rate but more correctly. Furthermore, a distinction should be made between dyseidetics (51) and dysphonetics (52).

The present article suggests an extended neuroconstructivistic research strategy to study underlying causes of dyslexia (32). Instead of following one line of argumentation, such as the influence of auditory perception...
on the development of phonological awareness, a systemic facet model must be used to identify the development of bi-, cross- and transmodal perception, attention, and cognition in dyslexia over time (31).

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