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Syndromic Analysis of the Comorbidity of Reading Disorders and Neurodevelopmental Disorders in Children with Preserved Intellectual Functioning

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Abstract

The comorbidity of reading disorders and neurodevelopmental disorders is a persistent phenomenon with high prevalence rates in the pediatric population; however, its neuropsychological structure remains insufficiently conceptualized. The present study provides a systematic review of theoretical and empirical research aimed at identifying the mechanisms underlying the overlap between reading disorders (particularly dyslexia) and attention-deficit/hyperactivity disorder (ADHD), as well as autism spectrum disorders (ASD), in the context of preserved intellectual functioning. An analysis of publications from the past five years, together with foundational works in neuropsychology and developmental psychology, indicates that the frequency of comorbidity significantly exceeds the level expected by chance co-occurrence, suggesting the presence of shared genetic, cognitive, and neurobiological mechanisms. The review outlines the principal models of intersyndromic profiles, compares findings related to cognitive deficits (regulatory, semantic-integrative, and phonological), and systematizes evidence from contemporary neuropsychological and neuroimaging studies. Based on the synthesis of empirical data, an integrative conceptual model is proposed that reflects the dynamic interaction between a core reading deficit and modifying neuropsychological profiles in comorbid conditions. The study emphasizes the necessity of a syndromic approach to enhance diagnostic precision and to inform the development of differentiated intervention strategies.

Introduction

Contemporary developmental psychology and cognitive neuroscience increasingly conceptualize learning disorders as the result of interactions among multiple neurocognitive factors rather than as the consequence of an isolated deficit. The shift from monocausal explanatory models to multifactorial frameworks has been driven by accumulating evidence concerning the heterogeneity of cognitive profiles and the high rate of co-occurrence among various developmental disorders. Pennington's multiple deficit model (Pennington, 2006) posits the overlap of deficits across different functional systems-including phonological processing, attentional regulation, executive functions, and working memory-which creates the conditions for the formation of stable comorbid profiles. Within this framework, comorbidity is understood not as the accidental coexistence of disorders but as a systematic outcome of partial overlap in underlying neurocognitive mechanisms.

Reading disorders have traditionally been primarily associated with a phonological deficit, manifested in difficulties with phonemic analysis and synthesis, decoding, and the automatization of reading skills (Snowling, 2000). However, empirical findings from recent decades indicate that the phonological model does not fully capture the complexity of the phenomenon. The high frequency of co-occurrence between reading disorders and attention-deficit/hyperactivity disorder, as well as autism spectrum disorder, suggests a more multilayered structure of impairment that includes regulatory, semantic, and integrative components. In many such cases, intellectual functioning remains within the normative range, which allows the observed difficulties to be interpreted as reflecting a specific organization of higher mental functions rather than as a consequence of general cognitive decline.

From the perspective of A. R. Luria's neuropsychology (Luria, 1973), reading constitutes a complex functional system that develops through instruction and is based on the interaction of several functional brain blocks. Its structure includes a regulatory component (programming and control of activity), an acoustic-gnostic component (processing of speech information), visuospatial analysis, and semantic-integrative processes that ensure text comprehension. Impairment of any of these components may lead to specific difficulties in the acquisition of reading skills, while their combination may result in the formation of variable syndromic profiles.

Within the context of comorbidity, particular attention is drawn to the superimposition of a core phonological deficit on additional impairments in regulatory or semantic-integrative mechanisms. In cases of comorbidity with attention-deficit/hyperactivity disorder, difficulties in voluntary regulation, sustained attention, and skill automatization

tend to become more pronounced. In autism spectrum disorder, even when decoding skills remain relatively preserved, distinctive features of semantic integration, contextual interpretation, and pragmatic text comprehension may be observed. Thus, comorbidity may be conceptualized as a dynamic system of overlapping deficits within the structure of higher mental functions.

Despite the substantial body of empirical research, findings on the comorbidity of reading disorders and neurodevelopmental disorders remain fragmented, and integrative explanatory models are insufficiently developed. The absence of a unified conceptual framework complicates the development of differentiated diagnostic and educational strategies.

The purpose of the present article is to conduct a review-analytical synthesis of contemporary research and to develop a conceptual model of the comorbidity of reading disorders and neurodevelopmental disorders in children with preserved intellectual functioning, based on a syndromic neuropsychological approach.

Methods

The methodological foundation of the study was based on the following scientific approaches:

1. Systems Approach

Reading disorders and neurodevelopmental disorders were conceptualized as elements of a complex functional system that develops throughout ontogenesis and includes interrelated cognitive, neuropsychological, and behavioral components. This approach corresponds to contemporary perspectives on the heterogeneity of developmental disorders (Pennington, 2006).

1. Cultural-Historical Theory of Higher Mental Functions

The analysis was grounded in the conceptualization of higher mental functions as systemic formations that emerge through activity and are mediated by social interaction (Luria, 1973). Reading was considered a complex functional system comprising regulatory, acoustic-gnostic, and semantic-integrative components.

1. Syndromic Neuropsychological Analysis

Comorbidity was interpreted not as the mechanical coexistence of diagnoses but as the result of the superimposition of deficits across different components of the functional system. The methodological basis was the logic of syndromic analysis, which involves identifying the leading factor and the structure of secondary impairments (Luria, 1973).

1. Multiple Deficit Model of Developmental Disorders

In interpreting comorbidity, the concept of overlapping deficits was taken into account, according to which different developmental disorders may share partially common cognitive and genetic foundations (Pennington, 2006). This model is widely applied in analyses of the co-occurrence of dyslexia and attention-deficit/hyperactivity disorder (Willcutt et al., 2005), as well as in studies examining the overlap of symptomatology among neurodevelopmental disorders (American Psychiatric Association, 2013).

Analytical Procedure

The literature analysis was conducted in several stages:

- Thematic coding - identification of key cognitive mechanisms (phonological deficit, regulatory deficit, semantic-integrative difficulties).
- Comparative model analysis - examination of differences among the phonological model of dyslexia (Snowling, 2000), the regulatory model of attention-deficit/hyperactivity disorder (Willcutt et al., 2005), and the multiple deficit model of development (Pennington, 2006).
- Identification of overlapping mechanisms - analysis of data concerning the role of executive functions, working memory, and attention in the formation of comorbid profiles (McGrath et al., 2011; Snowling & Hulme, 2021).
- Systematization according to functional components of reading - classification of the identified deficits in relation to the components of the functional system (regulatory, acoustic-gnostic, and semantic-integrative).

Thus, the methodological design made it possible to move from a descriptive review toward a conceptual synthesis and the development of an integrative model of comorbidity.

Results

The analysis of theoretical and empirical studies revealed consistent patterns that allow for the systematization of data concerning the comorbidity of reading disorders and neurodevelopmental disorders in children with preserved intellectual functioning. The results are presented within a framework that compares cognitive mechanisms and functional profiles.

1. The Multifactorial Nature of Comorbidity

Empirical studies indicate that the comorbidity of reading disorders and other neurodevelopmental disorders occurs significantly more frequently than would be expected under independent diagnostic distributions. Within the multiple deficit model of development, it is assumed that different disorders may partially share cognitive and neurobiological mechanisms, which explains their high rate of co-occurrence (Pennington, 2006).

Willcutt et al. (2005) demonstrated that children with attention-deficit/hyperactivity disorder and children with reading disorders exhibit overlapping deficits in executive functions, including working memory and attentional control. In a subsequent study, McGrath et al. (2011) showed that phonological processing, processing speed, and executive functions serve as independent but partially overlapping predictors of difficulties in both reading and attentional regulation.

Moll, Snowling, and Hulme (2020), in their analysis of empirical data on the comorbidity of dyslexia with other developmental disorders, emphasize that the overlap among disorders reflects a variable combination of shared and disorder-specific cognitive deficits. The authors demonstrate that the heterogeneity of profiles is determined by different configurations of phonological, regulatory, and language-related risk factors.

Thus, findings from specific empirical studies support the interpretation of comorbidity as the result of the superimposition of partially overlapping cognitive mechanisms rather than as the accidental coexistence of independent disorders.

1. The “Reading Disorders + ADHD” Profile: Regulatory Deficit

Empirical studies demonstrate that the co-occurrence of reading disorders and attention-deficit/hyperactivity disorder is associated with more pronounced impairments in executive functions compared to isolated forms of either condition. Willcutt et al. (2005) showed that deficits in executive functions, including inhibitory control and working memory, are characteristic of both children with attention-deficit/hyperactivity disorder and children with reading disorders, and that the severity of these impairments increases when the conditions co-occur.

McGrath et al. (2011), in examining the multiple deficit model in reading disorders and attention-deficit/hyperactivity disorder, identified phonological processing, processing speed, and executive functions as independent yet partially overlapping predictors of difficulties. In particular, working memory and processing speed were found to function as shared cognitive risk factors across both groups.

Genetic studies further support the partial overlap of mechanisms. Willcutt et al. (2010) reported a significant genetic correlation between reading disorders and attention-deficit/hyperactivity disorder, indicating the presence of shared heritable risk factors.

Taken together, these findings suggest that in the comorbidity of reading disorders and attention-deficit/hyperactivity disorder, the regulatory component-specifically deficits in programming, control, and sustained attention-emerges as the leading factor. The phonological deficit may remain the core mechanism underlying reading impairment; however, its manifestations are intensified due to insufficiencies in executive functioning.

1. The “Reading Disorders + Autism Spectrum Disorder” Profile: Semantic-Integrative Deficit

Studies examining reading in individuals with autism spectrum disorder without intellectual impairment demonstrate a specific cognitive profile. Nation, Clarke, Wright, and Williams (2006) showed that children with autism spectrum disorder may exhibit relatively preserved decoding skills alongside reduced text comprehension. This pattern is described as a discrepancy between technical reading ability and semantic processing.

Snowling and Hulme (2021) note that in autism spectrum disorder, difficulties in text comprehension are often associated with impairments in contextual integration and the interpretation of implicit information. These characteristics are consistent with findings on deficits in pragmatic language processing and challenges in constructing coherent cognitive representations of text.

The DSM-5 (American Psychiatric Association, 2013) identifies impairments in social communication and cognitive flexibility as core features of autism spectrum disorder. Such characteristics may manifest in difficulties establishing causal relationships, interpreting characters’ intentions, and understanding implicit meanings within texts.

Thus, in the comorbidity of reading disorders and autism spectrum disorder, the semantic-integrative deficit emerges as the leading factor, affecting deep meaning processing rather than being limited to the phonological level.Начало формыКонец формы

1. Overlapping Cognitive Mechanisms

A comparison of empirical findings makes it possible to identify several cognitive mechanisms involved in the formation of comorbid profiles:

- Phonological processing as the core factor underlying specific reading disorders (Snowling, 2000).
- Executive functions, including inhibitory control and working memory (Willcutt et al., 2005).
- Processing speed as a general cognitive predictor (McGrath et al., 2011).
- Semantic integration and comprehension of connected text (Nation et al., 2006; Snowling & Hulme, 2021).

These mechanisms partially overlap; however, their relative contribution varies depending on the co-occurring disorder. In attention-deficit/hyperactivity disorder, the regulatory component becomes more pronounced, whereas in autism spectrum disorder, the integrative-semantic component plays a more central role.

1. Systematization According to Functional Components of Reading

The synthesis of empirical evidence allows the identified deficits to be aligned with the functional components of the reading system:

- Acoustic-gnostic component - phonological processing and decoding (Snowling, 2000).

- Regulatory component - programming, control, and sustained attention (Willcutt et al., 2005).
- Semantic-integrative component - construction of coherent textual meaning (Nation et al., 2006; Snowling & Hulme, 2021).

Comorbidity emerges as the superimposition of a core phonological deficit with a modifying regulatory or semantic profile.

Thus, existing empirical studies allow the following conclusions to be drawn:

- In the comorbidity of reading disorders and attention-deficit/hyperactivity disorder, the regulatory deficit associated with executive functions emerges as the leading factor.
- In the comorbidity of reading disorders and autism spectrum disorder, difficulties in semantic integration predominate despite relatively preserved decoding skills.
- Intellectual functioning may remain within the normative range in such cases.
- Comorbidity represents a structural modification of the functional system of reading, resulting from the superimposition of overlapping cognitive mechanisms.

The comparative characteristics of the identified syndromic profiles are summarized in Table 1.

Table 1. Comparative Syndromic Profiles

Component	Reading + ADHD	Reading + ASD
Core deficit	Phonological	Phonological
Modifying factor	Regulatory deficit	Semantic integration deficit
Decoding	Impaired	Relatively preserved
Comprehension	Variable	Significantly impaired
Primary mechanism	Regulatory	Integrative-semantic

Discussion

The synthesized findings of empirical research allow the comorbidity of reading disorders and neurodevelopmental disorders in the context of preserved intellectual functioning to be conceptualized as a systemic phenomenon. The identified overlapping cognitive mechanisms are consistent with the multiple deficit model of development, according to which different disorders partially share common neurocognitive foundations (Pennington, 2006). Such an interpretation excludes an understanding of comorbidity as the mechanical summation of symptoms and instead requires an analysis of the structure of the functional system.

1. Comorbidity as a Systematic Configuration of Risk Factors

The high rate of co-occurrence between dyslexia and attention-deficit/hyperactivity disorder, documented in the studies by Willcutt et al. (2005) and McGrath et al. (2011), indicates the presence of shared regulatory mechanisms involved in the development of both disorders. Genetic evidence (Willcutt et al., 2010) further supports the partial overlap of risk factors.

In the case of the co-occurrence of reading disorders and autism spectrum disorder, empirical studies (Nation et al., 2006; Snowling & Hulme, 2021) point to a different pattern of overlap-not at the level of decoding, but at the level of semantic integration of text. These findings are consistent with the description of cognitive characteristics of autism spectrum disorder in the DSM-5 (American Psychiatric Association, 2013), which emphasizes difficulties in cognitive flexibility and the interpretation of social context.

Thus, comorbidity emerges as a stable configuration of overlapping cognitive risk factors rather than as an exceptional instance of the coexistence of independent disorders.

1. Differentiation of Syndromic Profiles

From the perspective of the syndromic neuropsychological approach (Luria, 1973), the identified differences may be interpreted as the superimposition of deficits across distinct functional components.

In the comorbidity of reading disorders and attention-deficit/hyperactivity disorder, the regulatory component-programming, control, and sustained attention—functions as a modifying factor that amplifies the manifestations of the core phonological deficit (Willcutt et al., 2005; McGrath et al., 2011).

In contrast, in the co-occurrence of reading disorders and autism spectrum disorder, the semantic-integrative component predominates, characterized by difficulties in constructing a coherent semantic representation of text despite relatively preserved decoding abilities (Nation et al., 2006; Snowling & Hulme, 2021).

Thus, the findings of empirical research support the existence of at least two qualitatively distinct modifying profiles that influence the structure of reading impairments.

1. Theoretical Implications

The synthesized findings extend the traditional phonological model of dyslexia (Snowling, 2000) by demonstrating that the structure of reading impairments in the context of preserved intellectual functioning may vary depending on the accompanying cognitive profile.

These findings are consistent with the concept of overlapping deficits (Pennington, 2006) and underscore the necessity of shifting from categorical diagnosis toward a functional analysis of underlying cognitive mechanisms.

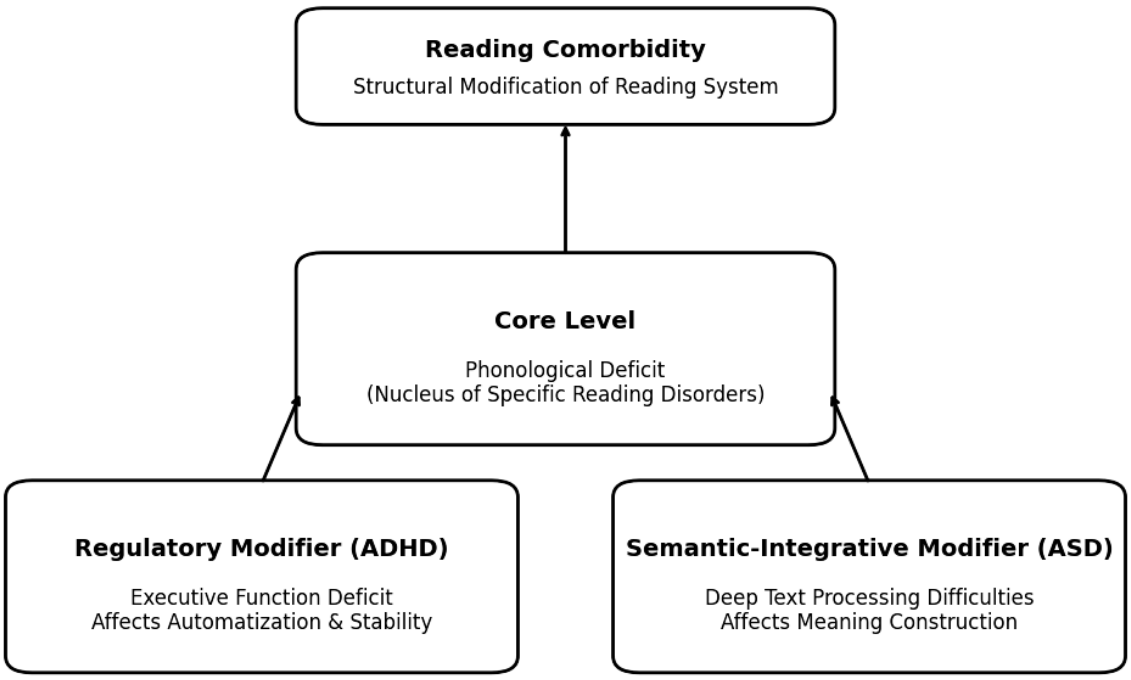
1. Author's Conceptual Model

Based on the synthesis of empirical evidence, a three-level model of comorbidity is proposed:

- Core level - a phonological deficit as the nucleus of specific reading disorders (Snowling, 2000).
- Regulatory modifier - a deficit in executive functions that intensifies difficulties in the automatization and stability of reading in attention-deficit/hyperactivity disorder (Willcutt et al., 2005; McGrath et al., 2011).
- Semantic-integrative modifier - difficulties in deep text processing in autism spectrum disorder (Nation et al., 2006; Snowling & Hulme, 2021).

The theoretical interpretation of the structural interaction among cognitive mechanisms is visualized in the three-level conceptual model (Figure 1).

Figure 1. Three-Level Conceptual Model of Reading Comorbidity



Within this model, comorbidity is conceptualized as the result of the interaction between a core phonological deficit and one of the modifying cognitive profiles. Intellectual functioning may remain within the normative range, underscoring the specific functional nature of the impairments.

1. Practical Implications

A systemic understanding of comorbidity makes it possible to differentiate directions for educational support.

In the “reading disorders + attention-deficit/hyperactivity disorder” profile, emphasis should be placed on the development of regulatory mechanisms—voluntary attention, planning, and control.

In the “reading disorders + autism spectrum disorder” profile, priority should be given to the development of semantic integration, contextual interpretation, and the understanding of narrative structure.

1. Limitations and Future Directions

The present analysis is theoretical and analytical in nature and does not include a quantitative meta-analysis. A promising direction for future research is the empirical validation of the proposed model through the use of neuropsychological assessment and neuroimaging methods. Further studies are required to clarify the role of genetic and neural mechanisms underlying the overlap of disorders (Pennington, 2006; Willcutt et al., 2010).

Conclusion

A comparison of empirical findings indicates that the comorbidity of reading disorders and neurodevelopmental disorders in the context of preserved intellectual functioning reflects a structural modification of the functional system of reading. The syndromic neuropsychological approach provides a theoretical foundation for the differentiated interpretation of cognitive profiles and for the development of targeted educational strategies.

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