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The Multiple Deficit Model: Progress, Problems, and Prospects

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Abstract

The multiple deficit model (MDM) was proposed because the prevailing single-deficit model provided an inadequate account of atypical neuropsychological development. Across methods and levels of analysis, there has been support for the two fundamental tenets of the MDM, that multiple predictors contribute probabilistically to neurodevelopmental disorders and shared risk factors contribute to comorbidity. Diagnostically, the multiplicity of factors means that no single cognitive deficit or combination of deficits can be used to rule in or out most neurodevelopmental disorders. Challenges for the MDM are that the theory is difficult to falsify and that current cross-sectional studies cannot establish causality. Prospects for further development of the MDM include incorporating an explicit focus on promotive and protective factors and pursuing mechanistic connections between multiple factors across levels of analysis.

The multiple deficit model (MDM) of neurodevelopmental disorders (Pennington, 2006) was proposed because the then prevailing single deficit model had failed. The single deficit model (Morton & Frith, 1995; Pennington & Ozonoff, 1996) held that each neurodevelopmental disorder, such as dyslexia, autism, or ADHD, was due to a single underlying cognitive deficit, such as a phonological deficit in dyslexia, a theory of mind deficit in autism, or an inhibition deficit in ADHD. The single cognitive deficit model failed for both theoretical and empirical reasons.

Regarding theory, the single deficit model failed because it took a static neuropsychological approach to understanding neurodevelopmental disorders (Oliver, Johnson, Karmiloff-Smith, & Pennington, 2000). According to this approach, there were innate, localized cognitive modules in the human brain, and each neurodevelopmental disorder was due to a deficit of its particular cognitive module. Considerable research has since contradicted this modular approach to understanding human typical and atypical cognitive development (as reviewed in Johnson & De Haan, 2011). Unlike cognitive modules, specialized processing areas of the human brain are not strictly localized, informationally encapsulated, or innate. Instead, these specializations emerge developmentally and interactively, and their brain substrates change as they develop.

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Empirically, the single deficit model failed because it was common to find children with neurodevelopmental disorders that lacked the key underlying cognitive deficit, as well as children without the disorder that nonetheless had the key underlying deficit. But, most importantly, it failed empirically because it could not account for the pervasive phenomenon of comorbidity among developmental disorders, which is the theme of this *Special Issue*.

In sum, the single deficit model failed because it espoused an inadequate theory of atypical neuropsychological development, and it could not account for key empirical findings about neurodevelopmental disorders, including pervasive comorbidity and inter-individual variability in neuropsychological profiles. The MDM provides a ready explanation of comorbidity by positing that there are multiple possible risk factors for each neurodevelopmental disorder and that some of these risk factors are shared by comorbid disorders. The MDM also accounts for inter-individual variability in profiles by proposing that each risk factor is probabilistically (rather than deterministically) related to neurodevelopmental disorders.

In what follows, we will discuss the advancement of the MDM since it was first proposed in 2006, including *progress, problems,* and *prospects*.

Progress

The MDM is a multi-level framework for understanding neurodevelopmental disorders, spanning etiology (genes, environments, and gene-environment interplay), brain mechanisms, neuropsychology, and behavioral symptoms. Several methods have been applied to test the MDM across these levels of analysis. The bulk of the work has occurred at the neuropsychological level of analysis where our group and others have used structural equation modeling (Christopher et al., 2012; McGrath et al., 2011; Peterson et al., 2017; Slot, van Viersen, de Bree, & Kroesbergen, 2016), clinical designs (Catts, McIlraith, Bridges, & Nielsen, 2017; Moll, Gobel, Gooch, Landerl, & Snowling, 2016; Moura et al., 2017; Ring & Black, 2018), and regression outlier approaches (Pennington et al., 2012). Beyond cognition, the MDM has been applied to behavioral genetic studies (Daucourt, Erbeli, Little, Haughbrook, & Hart, 2019 this issue; Willcutt et al., 2010) longitudinal family risk studies (van Bergen, van der Leij, & de Jong, 2014), and neuroimaging studies (McGrath & Stoodley, 2019; Peters, Bulthé, Daniels, Op de Beeck, & De Smedt, 2018). Across methods and levels of analysis, there has been consistent support for the fundamental tenets of the MDM that multiple probabilistic risk factors are associated with neurodevelopmental disorders and that shared risk factors contribute to comorbidity.

Cognitive multiple deficit models have been most successful for dyslexia and dyscalculia. In latent models of single-word decoding and math calculation, multiple deficit models have accounted for as much as 75-85% of the variance in reading and math, which is impressive in relation to similar models for ADHD that have only reached 25-35% (McGrath et al., 2011; Peterson et al., 2017). The small amount of variance accounted for by cognitive models of ADHD is an ongoing challenge. Recent work has suggested that emotion regulation deficits might add to the predictive variance (Martel & Nigg, 2006; Shaw, Stringaris, Nigg, & Leibenluft, 2014; Sjöwall, Roth, Lindqvist, & Thorell, 2013), which is a

promising new direction. In comparison to learning disabilities and ADHD, the MDM has been less influential in the autism literature, though the "fractionable autism triad hypothesis" (Brunsdon & Happé, 2013; Happé & Ronald, 2008) aligns with the principles of the MDM.

The MDM framework has been useful for directing research toward shared risk factors between disorders that may contribute to comorbidity. For example, we have found that processing speed weaknesses partially explain the comorbidity between dyslexia, dyscalculia, and ADHD (McGrath et al., 2011; Peterson et al., 2017) and that oral language weaknesses further contribute to the comorbidity of dyslexia and dyscalculia (Peterson et al., 2017). Efforts to identify such shared cognitive deficits between neurodevelopmental disorders has aligned with the "Research Domain Criteria" (RDoC) framework to identify transdiagnostic risk factors in psychiatric disorders (Insel et al., 2010). As a result of common efforts across neurodevelopmental and psychiatric disorders, the far-reaching impact of cognitive correlates such as processing speed and executive functions across domains of learning and mental health is becoming apparent (Doyle et al., 2017; McGrath et al., 2016; Snyder, Miyake, & Hankin, 2015).

Our empirical work has led us to continue refining our understanding of MDM. One lesson from our dyslexia research is that some children (roughly 25%) can be adequately explained by single deficits even though multiple deficits contribute at the population level (Pennington et al., 2012). These "single deficit" children were a surprise to us and continue to represent one of the challenges to the MDM theory (discussed below). The second lesson is that all of the cognitive deficits identified so far are probabilistic predictors and cannot be considered "core." This is true even for the strong, causal link between phonological awareness and reading abilities (Hulme & Snowling, 2013). In our empirical work, approximately 50% of children with dyslexia *do not* have a phonological awareness deficit (Pennington et al., 2012). This finding is pushing research in dyslexia in innovative ways beyond the "classic" deficits and has important clinical implications for assessment and diagnosis.

Clinical Implications

The single deficit model held that cognitive or neuropsychological assessment was needed for diagnostic assessment of neurodevelopmental disorders in order to establish that the presumed causal deficit was present. As we have seen, however, research supporting the MDM makes clear that no single cognitive deficit can be used to rule in or out neurodevelopmental disorders at the individual level. Nevertheless, the remnants of the single deficit model continues to lurk in clinical care in some cases. For instance, the idea that all children with dyslexia must have a phonological deficit has been rejected by the MDM, but the notion continues to influence some in the assessment community.

What about cognitive profiles? A decade ago, we believed that the field could eventually identify performance patterns across neurocognitive domains that would be diagnostic. However, the dimensional and probabilistic nature of the deficits has precluded clear mappings of cognitive profiles to diagnoses. Even among individuals with multiple

neuropsychological deficits, various constellations are possible, and as discussed earlier, some individuals have the disorder but have only a single deficit or even no identifiable deficit. Fletcher and colleagues have extensively critiqued the related "patterns of strengths and weaknesses approach" to learning disability identification and reached the same conclusion (Miciak, Fletcher, Stuebing, Vaughn, & Tolar, 2014). Diagnosis based on cognitive profiles is ineffective because it produces unstable categories (small changes in diagnostic criteria lead to large changes in who is identified) and identifies a much lower base rate than symptom definitions (Stuebing, Fletcher, Branum-Martin, & Francis, 2012).

Taken together, this work leads to the conclusion that detailed neurocognitive evaluation is not necessary to diagnose behaviorally-defined neurodevelopmental disorders such as learning disabilities, ADHD, and autism spectrum disorder, although clinical neuropsychological evaluation can still be helpful for some individuals with these disorders to answer referral questions that go beyond diagnosis. Similar to the etiologic and brain levels, group research at the neuropsychological level is valuable to inform the scientific understanding of typical and disordered neurodevelopment, but findings at the group level do not currently have appropriate psychometric properties to be useful in individual diagnostic decision-making. Instead, the cornerstone of diagnostic assessment should be evaluation of the defining symptoms (based on clinical history, observations, and reliable, validated tests) as well as careful attention to functional impairment and possible comorbid conditions. While detailed neurocognitive evaluation may not be necessary for diagnostic decision-making, as discussed next, cognitive profiles might still be helpful for treatment planning and for helping families and educators adapt to the unique cognitive strengths and weaknesses of the child (e.g., accommodations for working memory or processing speed challenges).

The MDM also has implications for treatment; to date, relevant work has focused primarily on reading disabilities. Existing research suggests that cognitive profiles are of limited benefit (after accounting for initial reading severity) in predicting which individuals with reading disabilities will respond adequately to intervention (Fletcher et al., 2011; Miciak, Stuebing, et al., 2014). However, there is some evidence that cognitive profiles are useful for individual treatment planning. For example, Connor and colleagues have shown that optimal reading instruction varies as a function of child characteristics, including vocabulary level (Connor et al., 2013; Connor, Morrison, Fishman, Schatschneider, & Underwood, 2007). A refined understanding of the multiple deficits associated with other neurodevelopmental disorders has the potential to support individually tailored interventions that may ultimately be more successful than "one-size fits all" approaches.

Research in the MDM framework could eventually lead to the development of new treatment approaches that are particularly suited to children with comorbid conditions, which is currently an area of significant clinical need (Larson, Russ, Kahn, & Halfon, 2011; Willcutt et al., 2013). For example, the finding that oral language weaknesses partially explain the overlap between dyslexia and dyscalculia (Peterson et al., 2017) suggests that interventions addressing academic vocabulary, listening comprehension, or other language skills might be especially valuable for students who struggle across the curriculum, although of course, this hypothesis will have to be empirically evaluated.

Problems

One theoretical challenge is determining how the MDM could be falsified. Single deficit models were much easier to reject because they made strong predictions. The probabilistic nature of MDM means that most outcomes can be accounted for by the model. As previously described, at the individual level, some children do fit a “single deficit” model. Does this falsify the model? We do not believe so because multiple predictors can be considered risk factors at the population level. However, this finding highlights the fundamental challenge of heterogeneity in neurodevelopmental disorders. Different cognitive profiles are seen in children with the same neurodevelopmental disorder and this makes the specification of universal models very challenging. We believe that more advanced testing of the MDM will require elaboration of the best-fitting population-based models as well as indicators of how well individuals conform to or deviate from the best-fitting MDM (e.g., Reise, Kim, Mansolf, & Widaman, 2016). We will then need to examine whether individuals who fit or do not fit the MDM differ at other levels of analysis, such as genetic and environmental risk factors, brain mechanisms, and response to treatment.

The bulk of the work on the MDM has been cross-sectional. Hence, an ongoing limitation of current MDM models is that we do not know if the associations between cognitive deficits and symptoms outcomes are cause, consequence, bidirectional, or attributable to third variables. Moreover, we cannot assume that the MDM will be static across age. Rather, future work should consider the trajectory of the MDM over time.

Prospects

The MDM has largely focused on identifying risk factors, but this neglects the potential role of protective and promotive factors across levels of analysis (Haft, Myers, & Hoeft, 2016; Slomowitz et al., submitted). Promotive factors are those that improve outcomes equally for all individuals, regardless of whether they are at high or low risk for a disorder (Masten, 2011). Protective factors are those that provide stronger buffering effects for individuals at high risk than those at low risk (Masten, 2011). The research literature on neurodevelopmental disorders would benefit from a more explicit focus on identifying promotive and protective factors. For this reason, the MDM might be renamed to the “Multiple Factors Model” to encourage research that spans the range of risk and promotive factors.

In the initial theoretical paper, Pennington (2006) laid out the 4-level framework (etiology, brain mechanisms, neuropsychology, behavior) and set an agenda for identifying risk factors at each level of analysis and importantly, the connections between levels of analysis. In the past 14 years, we have made progress on identifying risk factors within each level of analysis and confirmed their probabilistic nature and the existence of shared risk factors contributing to comorbidity. What remains elusive are the connections between levels of analysis, and yet, these connections will be the hallmark of a full explanatory theory. Progress in identifying the “connective marrow” of the MDM will require interdisciplinary collaboration that moves toward mechanistic explanations. For example, it is possible with current neuroimaging methods to connect the brain and neuropsychological levels of

analysis by localizing activity in brain areas that are associated with performance on a cognitive task. But, a deeper understanding of this connection will require a neurocomputational account of how the brain implements the cognitive performance. Similarly, the connection between genetics and brain mechanisms is possible with current imaging genetic methods, but a deeper understanding of this connection will require a more elaborated developmental biology of the brain to explain the nature and timing of specific genetic effects on brain development (Pennington, McGrath, & Peterson, 2019). These challenges are not unique to the MDM, but the multi-level framework of the MDM does not let us stay complacent in our home disciplines. Rather, the MDM framework encourages an interdisciplinary perspective on the important work ahead – elaborating a full explanatory theory of neurodevelopmental disorders.

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