Mini Review

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Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Task-General Efficiency Clinical Neuroscience Implications

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Abstract

A fundamental area of cognitive science, the ability to quantify individual differences in higher-order cognitive functions has significant repercussions for psychopathology research. In these fields, the predominant strategy has been to attempt to fractionate higher-order functions into hypothesized components through factor analysis and experimental manipulation. However, there has recently been a lot of theoretical and empirical criticism levelled at the putative constructs produced by this paradigm. In parallel, a new strategy centered on the parameters of mathematical psychology-developed formal computational models of cognition has emerged. These models can be used to measure the latent mechanisms that underpin performance because they offer explanations of the data-generating process for cognitive tasks that are both biologically plausible and experimentally validated. Recent applications of this method have revealed that efficiency of evidence accumulation, a computational mechanism defined by sequential sampling models, is largely responsible for individual and clinical differences in performance on a wide range of cognitive tasks, from simple choice tasks to complex executive paradigms. The hypothesis that efficiency of evidence accumulation is a central individual difference dimension that explains neurocognitive deficits in multiple clinical disorders is supported by the evidence presented in this review, which also identifies ways in which this insight can advance clinical neuroscience research. We propose that the field will be able to draw clearer conclusions regarding cognitive abnormalities in psychopathology and their connections to neurobiology if the efficiency of evidence accumulation is recognized as a major driver of neurocognitive differences.

Keywords: Cognitive control • Diffusion model • Executive function • Linear ballistic accumulator • Mathematical psychology • Tran's Diagnostic risk

Introduction

The consistent observation that impairments in executive functions and cognitive control are observed Trans diagnostically across multiple mental disorders, such as schizophrenia, depression, and anxiety, is the driving force behind this work [1]. Clinical neuroscience research to better comprehend the psychological and neurobiological foundations of psychopathology and deficits in higher-order cognition has mushroomed. In addition, major funding agency initiatives like the Research Domain Criteria project and the Computational Psychiatry Program place a significant emphasis on this work [2]. The objective of this review is to provide a critical perspective on the state of the science at the moment; we argue in favour of a new framework and identify a number of interrelated challenges faced by current methods. The dominant fractionation paradigm, which aims to break cognitive functions down into constituent elements with selective relationships to clinical disorders, is reviewed in the Fractionation Paradigm and Recent Challenges section. It also details recent findings that pose serious challenges to this approach [3]. The efficiency of evidence accumulation, a central individual difference dimension measured in sequential sampling

models of cognition, is the primary focus of the alternative paradigm presented in the following three sections, which are based on computational modeling. We look at the evidence to show that EEA has several advantages over fractionation-based metrics for explaining individual and clinical differences in cognitive performance across a wide range of seemingly quite distinct cognitive tasks [4]. In conclusion, we draw attention to important clinical neuroscience implications of this framework.

Materials and Method

The Concept of Fractionation and Recent Obstacles

Higher-order cognition, according to this framework, is made up of a number of component functions, each of which represents a relatively distinct individual difference dimension [5]. In clinical neuroscience research, where it is common to postulate that disorders involve selective impairments in particular functions, this last assumption is especially relevant. A main tool for fractionation is a collection of carefully designed experimental tasks with the goal of selectively engaging particular functions. For instance, in the Strop task's incongruent condition participants must respond to a word's ink color while disregarding the word's semantic meaning, which indicates a different color [6]. It is believed that this discrepancy activates a process of inhibition that suppresses the dominant tendency to provide a word response. It is assumed that the inhibition process is not engaged in an otherwise similar congruent condition in which the color of the word and its meaning are identical. As a result, it is assumed that individual inhibition is precisely measured by performance differences between the two conditions. Factor analysis is frequently used in conjunction with tasks like the Strop to investigate patterns of covariance across task batteries. Miyake et al.'s foundational work uncovered evidence for response inhibition, task switching, and updating working memory, and this framework continues to be the most influential fractionation taxonomy [7].

Ssms Computational Psychiatry and Mathematical Psychology

A crucial paradox has been highlighted in a number of recent psychiatry, clinical psychology, and other behavioural sciences commentaries: Even though formal mathematical process models have significantly improved precision, theory development, and cumulative knowledge for other sciences, these fields have largely avoided using them [8]. The subfield of mathematical psychology, which has a long history of using formalisms to specify and rigorously test theories about the mechanisms that underpin cognitive processing, is one notable exception. In addition to the general advantages of mathematical modeling in science, this method has recently shown remarkable promise for identifying connections between human cognition and neural function. These connections include greater explanatory clarity and stronger empirical tests of theoretical predictions. In addition, the models of mathematical psychology are beginning to play a significant role in the burgeoning field of computational psychiatry [9]. In this field, the models are used to identify potential biobehavioral dimensions associated with psychopathology that may have clearer relationships with neurobiological mechanisms than the cognitive constructs that are currently in use.

Reduced EEA as a Trans diagnostic Risk Factor for Psychopathology in Neurocognition

SSMs have been used the most extensively to study ADHD because RT variability has long been of interest. Meta-analytic effect size estimates for comparisons with healthy participants are in the moderate to large range, as reviewed by others and supported by subsequent work [10]. Individuals with ADHD consistently exhibit reduced EEA in SSM analyses. The variety of domains in which EEA reductions are observed is arguably the most striking feature of these effects. These domains include simple perceptual decision making, sustained attention inhibition, pattern learning, and interval timing. Additionally, it has been demonstrated that stimulant medication treatments for ADHD improve EEA in both healthy adults and children, indicating that EEA may mediate the effects of treatment. Stimulants both increased EEA in an incongruent task condition and a congruent task condition in the latter study. This finding suggests that both ADHD-related deficits and treatment-related improvements in EEA are domain-general, spanning diverse tasks and conditions with varying levels of complexity and executive demands. This is consistent with the pattern of cross-task effects observed in ADHD. Reduced EEA has been found in schizophrenia, depression, and people at risk for frequent substance use, in addition to ADHD. Our most recent work has demonstrated that EEA is a Tran's diagnostic risk factor for psychopathology, expanding on

these findings. We found a negative correlation between the overall severity of individuals' cross-disorder psychopathology symptoms and a latent EEA factor derived from multiple tasks that was significantly lower in patients with ADHD, schizophrenia, and bipolar disorder compared to healthy participants in a large sample drawn from the UCLA Consortium for Neuropsychiatric Phonemics. Due to the simplifying assumption made in this study that the standard DDM can provide adequate measures of EEA on inhibition tasks, it is necessary to replicate these findings using more complex modeling procedures.

Conclusions

The new literature on the use of mathematical process models to study individual and clinical differences in Neurocognition was evaluated in this review. Trait EEA, a fundamental individual difference dimension formalized in computational models, is likely a primary driver of observed deficits on tests of neurocognitive abilities across clinical disorders, according to our argument. Clinical neuroscience may move away from biologically amorphous constructs and measures with poor psychometric properties if an EEA-focused research approach is adopted. EEA, on the other hand, is a precisely defined construct that exhibits strong psychometric properties, clear links to psychopathology, and is well-positioned to yield richer connections with neurobiology. It also has strong psychometric properties.

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