1 Introduction

Elizabeth P. Sparrow and Scott J. Hunter

Executive functioning (EF) is a cognitive capacity that is difficult to define succinctly. Most explanations of EF reflect some degree of self-regulation as applied to cognition, emotion, behavior, adaptive functioning, and even moral reasoning and choice. In line with this, multiple models have been proposed to account for and explain the range of capacities and acts that represent EF, although the predominant models are adult-centric, and as a result, less applicable to thinking about EF developmentally. It is difficult to ignore the fact that nearly every neuropsychological evaluation in the clinical setting reveals and then attempts to address some aspect of executive dysfunction (EdF). EF deficits have a significant impact in multiple domains of a person's life and the lives of others around them, including parents, teachers, siblings, and peers. The pervasive and at times quite impairing nature of EdF calls for attention and emphasis from researchers and clinicians alike.

With this volume, the reader will find that we promote a model that emphasizes the plurality of executive *functions*, as opposed to a model that views EF as a singular (albeit still dynamic) cognitive capacity. Clinical and empirical data provide examples of dissociability within EF, at both the level of neural organization and skill demonstration. We also find that EF is best represented as a spectrum, such that too much or too little of a particular EF skill can represent EdF. For example, poorly controlled emotion and behavior are EF deficits in the Disruptive Behavior Disorders (Chapter 5), and overly controlled emotion and behavior are EF deficits in the Anxiety Disorders (Chapter 10). Even when none of the EF skills assessed show a significant deficit, the cumulative effect of multiple EF deficits can result in severe impairment (i.e., the whole is greater than the sum of the parts).[1] We address this directly in Chapters 2 and 4, in particular.

With this volume, we argue that it is important to examine the differences that exist for children and adolescents in EF and EdF, specifically between the occasional lapses into executive weakness that all people experience and "true EdF" that is persistent, pervasive, and severely impairing. We emphasize that it is important to recognize the range of factors that impact and moderate EF, including stable factors such as age, gender, ethnicity, personality, and genes, and more variable factors like self-care (fatigue, pain, stress, mood, and exercise), socioeconomic status, abuse, neglect, trauma, and social rejection. Each of these factors serves to influence how EF skills develop, vary in their presentation, and remain vulnerable, both environmentally and neurobiologically. These factors are discussed at multiple points in this volume, with particular emphasis found in the chapters comprising Section II, as well as Chapters 4, 19, and 20.

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When EdF is present, it often impacts functioning widely, and hampers adjustment across academic, vocational/occupational, social and interpersonal, emotional and intrapersonal, behavioral, and adaptive functioning (see Chapters 4, 18, and 20 in particular). This also creates a recurring cycle between what EdF impacts and what impacts EdF; however, intervention has the potential to change this cycle. As a result, we place a significant emphasis across this volume on recognition and intervention for EdF.

The primary purpose of this volume is to provide a comprehensive summary of what is known about EF and EdF in childhood and adolescence, examine what these data mean in a practical sense, and consider future directions for clinical and research work. Throughout this volume, our perspective is developmental, with a focus on issues usually first seen in children and adolescents. As a result, EdF in conditions that usually show onset in adulthood, such as schizophrenia,[2,3,4,5,6] the personality disorders,[7,8] and the dementias,[9,10,11,12,13,14,15] are not covered in this volume. There are numerous review articles and texts available that address EF in the adult population, including the classic references by Stuss and Benson[16] and Lezak.[17] Books addressing EdF in pediatric populations tend to present very applied materials (i.e., worksheets for use in treatment) or very academic and theoretical perspectives, but have not integrated research and practice. This volume will fill the gap in the literature for EF in pediatric populations, as a guide for researchers and clinicians who work with children and adolescents on a daily basis.

This volume is organized into three sections. The first section provides an overview of the foundations for understanding EF and EdF. Chapter 1 reviews the historical context of EF and summarizes various efforts to define and model this complex concept. The development of EF, from infancy through young adulthood, is examined in Chapter 2. It is critical to have an understanding of this progression as individual differences[18] are superimposed on the "typical" leaps and plateaus in EF development. Chapter 3 reviews what is known about the neurobiology of EF, specifically its developmental neuroanatomical, and neurochemical correlates. Current research in this area argues against simplistic assignment of EF to the frontal lobes,[16] instead revealing the beautiful complexity of the executive networks that involve cortical, subcortical, and cerebellar areas, among others. Ironically, we are once again reaching what early scientists postulated in the 1800s, that " ... the frontal lobes are the seat of coordination and fusion of the incoming and outgoing products of the several sensory and motor areas of the cortex."[19] Chapter 4 concludes the first section with a discussion of key assessment principles and a consideration of areas to address when examining EF and EdF.

The chapters in Section II represent current advances in pediatric neuropsychology and EF by presenting examples of a variety of pediatric clinical conditions that are associated with EdF. Section II is composed of 12 chapters describing EF and EdF in a number of conditions that begin in childhood or adolescence, either acquired neurodevelopmentally or through insult to the developing brain. Each of these chapters has a similar structure to help the reader find material, including discussion of the clinical manifestations, neuroimaging data, neuropsychological findings, and future directions for research pertinent to each disorder or condition. This section includes a range of neurodevelopmental and acquired disorders involving EdF, not only the "usual suspects" such as Attention-Deficit/Hyperactivity Disorder (ADHD) and the autism spectrum disorders (ASDs), but also less frequently discussed examples, like the pediatric movement disorders, neurodevelopmental conditions secondary to trauma and insult, the seizure disorders, human immunodeficiency virus (HIV), and the sequelae of the childhood cancers. It was our goal with this section to

address the range of potential conditions seen in clinical practice, as well as to provide guidance for those less common disorders that may occasionally be referred.

The chapters in Section III represent applications of this information. Chapter 17 reviews empirically supported interventions for EdF. Chapter 18 extends this with a consideration of educational implications, including research on school-based programs to improve EdF. Cautions and caveats regarding the consideration of EF and EdF in the forensic neuropsychology and child psychiatry setting are offered in Chapter 19. This volume concludes with Chapter 20, sharing reflections on key messages and goals for research and practice.

The reader is encouraged to mark the Appendices for easy reference while reading. Appendix I lists abbreviations used throughout the volume. Appendix II provides information about tests mentioned in the volume, including names, abbreviations, and quick references to learn more about each test. Unless specified otherwise, "child" is used in this volume to refer to children and adolescents.

The contributors to this volume were carefully considered and selected to represent a range of neurodevelopment professionals who work with EdF on a daily basis. The authors represent a mix of researchers, assessors, and interventionists, across both clinical and academic settings. With their contributions, these authors are not simply summarizing what others have written, but also extending the available research literature with clinical observations, new interpretations, and guidance for the future. We hope that this volume will provide practitioners and clinical consultants with a better understanding of both EF and EdF, and provide researchers and developers of intervention programs with an impetus for continued progress in the area. By examining the foundations, manifestations, and implications of EF and EdF, we hope to convincingly demonstrate the importance of addressing these issues for the many children and adolescents who present clinically for assessment and intervention, in order to support their effective transition to productive adulthood. We also hope to challenge our colleagues and professional peers towards a broader, more dynamic view of both EF and EdF, and how they unfold and alter across development, in anticipation of more effectively coaching and guiding the families who seek our consultation.

We anticipate that this volume will be of interest to psychologists (including neuropsychologists, school psychologists, developmental psychologists, and child clinical psychologists), pediatricians, pediatric neurologists, psychiatrists, rehabilitation physicians and pediatric physiatrists, and advanced trainees in these disciplines. Additionally, it may serve as a valuable resource for educators and even parents, to support their understanding of the children with whom they live and work on a daily basis.

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Section I **Foundations of Executive Function/Dysfunction**

Chapter 1

Models of executive functioning

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Scientists have approached executive functioning (EF) from a variety of perspectives, including neuroanatomical, neurochemical, evolutionary, syndrome-based, and statistical. Many have attempted to concisely define EF and executive dysfunction (EdF) by listing functions or underlying operations,[1,2] while others have focused on its neuroanatomical or neurophysiological correlates.[3] There is some degree of overlap among these descriptions, but no consensus. Perhaps the confusion regarding exactly what constitutes EF reflects the ways in which it has been examined historically. Early studies were *adult-based*, examining behaviors produced by brains that had already developed. These studies, while informative about adults with acquired EdF, did not take into account issues of development, such as how an insult impacts EF in a still-developing brain or how neurodevelopmental disorders impact brain and function. Early work in the field primarily examined the effects of insults to the *frontal lobes*, which led to a circular argument that "damage to the frontal lobes causes EdF, therefore EF must be regulated by the frontal lobes." This was later refined and modified with attribution of EF to the prefrontal cortex (PFC), but the assumption of one-to-one correspondence between function and structure, with limited consideration of the rich network we now know is involved in EF, remained the dominant model. Yet this model failed to account for evidence of intact functioning after removal of the frontal lobe,[4] EdF experienced after damage to other brain areas,[5] or evidence of EdF in the absence of a known neurologic insult (as is the case with some of the neurodevelopmental disorders).

Another key assumption in early work regarding EF was that there is a *homogenous* executive construct. This oversimplification led to a substantial degree of inflexibility in planning, executing, and interpreting studies. As Salthouse has observed, the *diversity of variables* used in assessing EF reveals a lack of agreement regarding the construct; different researchers use different tasks as the primary EF variable, and even standard EF batteries have very little overlap in tests used.[2] The lack of a "gold standard" significantly limits evaluation of EF measures beyond face and predictive validity. Efforts to examine construct validity generally find that performance on tests hypothesized to measure EF is moderately correlated with many other constructs, not just EF (i.e., low discriminant validity).[2]

Without a unifying plan or concept, investigations of EF and EdF have gone in a number of directions. Some researchers and clinicians continue to emphasize the simplistic explanation they learned early in their training, that EF is most related to the frontal lobes, and that it is primarily seen after brain injury or in Attention-Deficit/Hyperactivity Disorder (ADHD). Lack of agreement regarding what processes are part of EF contributes

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to the confusion, as different definitions and classifications complicate comparisons of research findings. For example, opinions regarding whether working memory (WM) is part of EF impact interpretation of high correlations between EF measures and intelligence quotient (IQ) test results that are primarily moderated by the Working Memory Index (cf. data reported by Arffa).[6] This confusion regarding the EF construct limits cohesion in the field, and thus limits progress.

In this chapter, we present a historical context within which to understand current models of EF and EdF. We conclude with suggestions for reaching an improved, integrated model of EF that accounts for changes in neural networks and skills across child and adolescent development.

Evolutionary context

For many years, an assumption has been that EF is a singular, phylogenetically higher-order cognitive skill that identifies us as human and differentiates us both behaviorally and dynamically from other mammalian species. Bernstein and Waber, among others, have reminded us that this fallacious concept ignores the reality that problem solving is an inherent aspect of mammalian development.[7] Although it was initially believed that the size of the PFC was a critical difference between humans and other species, comparative analyses indicate that the frontal lobe to total brain volume ratio is consistent across primates, including humans.[8–10] It appears that white matter volume is a differentiating factor, with humans having more white matter than other primates.[11] However, it is difficult to know whether this represents basic species differences, or whether modern human brains reflect high levels of stimulation that cause greater interconnectivity.[12] Ultimately, it remains important to remember that, as highlighted by Bernstein and Waber:[7]

> "the frontal lobes are neither new in evolutionary terms nor special to humans: they have been part of the neural apparatus of the mammalian line for 176 million years. The goal-oriented behavior that these neural systems support is not only common to all mammals, but also critical to their survival and to the evolutionary success of the whole mammalian enterprise . . . [The EF-directed] control processes are – indeed, must be – inextricably embedded in the total package of biological systems that all animals need to obtain food, reproductive partners, and other critical resources" (p. 40).

Historical context

The concepts referred to as EF today have been considered for centuries. The Bible seems to mention EF in verses written in approximately 900 B.C., "A man without self-control is like a city broken into and left without walls."[13] In 1835, deNobele described impairment after damage to the frontal lobes,[14] although the more famous case is that of Phineas Gage, who was described by Harlow in 1848.[15–18] The importance of these cases was realized retrospectively, after prominent neuropsychologists began to draw attention to this concept.

In 1964, Teuber published a paper entitled, "The Riddle of the Frontal Lobes".[19] Much of his commentary could be reiterated today, nearly 50 years later. He wrote,

> "Man's frontal lobes have always presented problems that seemed to exceed those encountered in studying other regions of his brain . . . these assorted aspects of frontal lobe dysfunction, in infrahuman species, are dissociable, since any one of these symptoms can be shown to occur

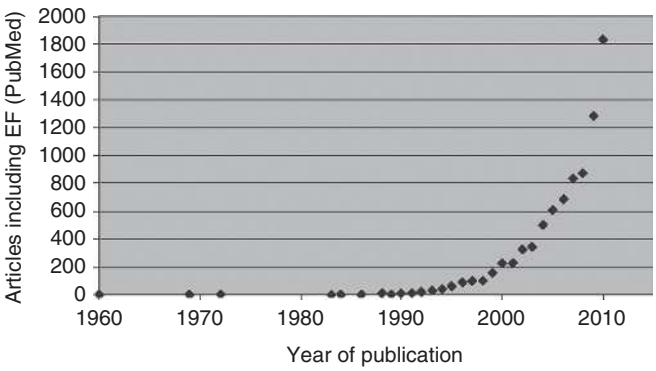

Fig. 1.1. Number of articles indexed in PubMed that included EF or EdF (search terms ="executive function "OR" executive dysfunction" OR "executive functions" OR "executive dysfunction" OR "dysexecutive "OR" executive deficit "OR" executive deficits")

> without the others . . . Despite the conspicuous efforts and ingenuity that went into behavioral analysis of these frontal lobe symptoms, their interpretation, in terms of some basic alterations in carnivore or simian behavior, remains elusive . . . During the first third of this century, there developed a substantial majority opinion which ascribed to the frontal lobes all the highest conceivable functions – from "abstract behavior," foresight, and intellectual synthesis, to capacity for ethical behavior, control of affect, awareness of self, and recent memory. In the early forties and through the fifties of the century, the pendulum swung in the other direction – toward a denial of any especial importance of human prefrontal structures" (p. 25).

A next landmark in the consideration of EF is "the frontal lobe syndrome" described by Luria in 1969.[20,21] This term was coined at a time when EF deficits were believed to occur in conjunction with insults to the frontal lobes. Although Luria[22] did not explicitly refer to EF in writing, he did attribute deficits in the ability to solve complex problems to the frontal lobes as the locus for the "complex process of formation and execution of a programme" (p. 219).

The first intentional use of "executive" in the published literature was noted in Baddeley and Hitch's model of the "central executive," which is discussed later in this chapter.[23] This concept gradually gained popularity, including Schaie's description of the "executive stage of adult cognitive development" that he postulated emerged in the 30s and 40s.[24] Shallice[25] used the phrase "executive functions and their disorders" in a 1982 publication applying the Norman and Shallice supervisory attentional system concept to explain performance of patients with brain lesions (p. 199). Although the first edition of Lezak's well-referenced volume[26] did not include a section about EF, she addressed many of the components we now consider as such. In the second edition,[27] she included a chapter entitled "Executive Functions and Motor Performance," describing the problems inherent in assessing EF.

Although the earliest publications about EdF in clinical populations focused on brain injury, the late 1980s showed an emerging interest in the EF concept as it was exhibited in other clinical conditions including schizophrenia,[28] ADHD,[29] and phenylketonuria (PKU).[30] This slow trickle of research quickly became a torrent, with the 1990s showing an explosion of articles regarding EF in various conditions. The number of articles per year focusing on and addressing this concept has only increased (see Fig. 1.1).

In the following sections, a handful of influential models will be examined, including evolution of language, the central executive, the Supervisory Attentional System, Theory of

Mind, self-regulation, "hot" and "cold" EF, the Cognitive Complexity and Control theory, and structural equation modeling with latent variables. These models are presented roughly in the order of publication, although it can be difficult even for theorists to know whose idea came first. Each of these models has influenced other models and impacted our current views of EF and EdF.

Evolution of language

Bronowski, a physicist, mathematician, linguist, philosopher, and educator, presented his theory in 1967 to explain why human language was different from communication in other species.[31] Elements of his model included (1) a delay between a stimulus and a response, (2) separation of affect/emotional charge, or processing content rather than simply responding to affective charge, or self-regulation of emotion, (3) prolongation of reference, or the capacity to recall the past to inform future decisions while keeping in mind the present situation (much like what we now call WM), (4) internalization of language, referring to private self-reflection and self-exploration allowing consideration of options before responding, and (5) reconstitution, analyzing parts of a situation and synthesizing them to construct a new response. He summarized these steps as allowing humans to disengage from their current context and influence their environment by understanding it, and connected the ability to delay response to development of the frontal lobes.[32] Although Bronowski's model focused on language, it showed consideration of what would come to be called EF in a few years. This model was a primary influence for Barkley's[33] model of executive functions in ADHD (see below).

Working memory (central executive)

In 1974, Baddeley and Hitch published a chapter describing their three-part model of WM[23] that was developed in response to concerns about the existing model that a short-term store held information until it could be entered into long-term memory (LTM). Their tripartite model was controlled by a system of limited attentional capacity, the "central executive," which was subserved by two storage or "slave" systems, the phonological loop and visuospatial sketchpad. Baddeley and Hitch were not the first to use the term "working memory," but they were the first to include specific mention of an executive component.[34] This model of a unitary attentional executive within a WM system was challenged by findings of intact immediate memory in patients with severely impaired LTM, findings that surpassed the capacity of finite slave systems. The original central executive was not believed to have any integrational capacity, which meant the model could not explain facilitation of short-term memory (STM) by LTM (as occurs in chunking) nor did it allow for crosstalk between the visuospatial and auditory slave systems. After the proposal of a dual-process control by Norman and Shallice[35] (see below), Baddeley added the "episodic buffer" to his model, a finite system controlled by the central executive that holds information long enough to be integrated.[36] Baddeley ties the central executive in this model to a distributed neural network, writing "[t]he central executive is likely to engage multiple brain regions in a functionally coherent network, including dorsolateral prefrontal cortex" (p. 836).[34] The central executive model has been criticized for creating a homunculus within the brain.[37]

Attentional control (supervisory attentional system)

The information processing model of attentional control was presented by Norman and Shallice in 1980.[35] Rather than a single executive, Norman and Shallice proposed two control processes: (1) routine habits, or pre-existing "schemas," that control a specific overlearned action or skill, and (2) an intentional "supervisory attentional system" (SAS) that comes online when a situation is non-routine. This grew out of the information processing movement related to artificial intelligence solutions to problem solving. Even in routine situations, "contention scheduling" selected a certain number of schemas to activate, inhibiting the others until environmental demands changed. The SAS serves to bias contention scheduling such that different schemas are activated.

In a subsequent paper, Shallice referenced Luria's work with frontal lobe lesions, stating that the study of executive functions and their disorders was a good area for interaction of cognitive psychology and neuropsychology.[25] He then evaluated what his model could predict about "high-level cognitive impairment" (p. 202). Shallice hypothesized that an inoperative SAS would leave a person reliant on the contention scheduling system; this would produce predictable behaviors that were slow to change (i.e., perseveration). In the presence of strong environmental triggers that activated schemas, the person would have difficulty inhibiting (i.e., distractibility), and in the absence of strong environmental triggers he would likely do nothing at all (i.e., apathy). He drew strong parallels between these predictions and observations of animals and humans with frontal lobe lesions. Like the central executive model, the SAS has been criticized for creating a homunculus within the brain.[37]

Theory of mind

The concept "theory of mind" was first described by Hebb in a 1958 lecture.[38] The term appeared again in 1978 when Premack and Woodruff[39] discussed its relevance to understanding chimpanzees, defining theory of mind as the ability to attribute mental states not only to self, but also to others (e.g., "I *think* John likes you," or, "I *guess* she will be happy with this gift."). They postulated that this ability allowed chimpanzees to make inferences about what others believe to be the case and thus make predictions about their probable actions. They concluded with a discussion of preliminary data from human children showing that they did not show evidence of inferred mental states until about 4 years old.

Alan Leslie presented on theory of mind in autism at a 1983 conference,[40] later publishing a paper with Uta Frith and her doctoral student, Simon Baron-Cohen, on the subject.[41] They described a model of "metarepresentational development . . . being able to conceive of mental states: that is, knowing that other people know, want, feel, or believe things" (p. 38), saying that the ability to form second-order representations was a prerequisite for existence of this theory of mind which subserves social skills.

Frith and colleagues examined theory of mind with a variety of clever manipulations dealing with beliefs and false beliefs. In the well-known "Sally and Anne scenario" (an example of a "false-belief" task), Sally puts a marble in a basket and then leaves the room. Anne hides Sally's marble in her box. Sally returns, and the experimenter asks, "Where will Sally look for her marble?" If theory of mind is present, the child will recognize that Sally expects the marble to be where she left it, and he says she will look in the basket. In the absence of theory of mind, the child does not discriminate between his knowledge and Sally's knowledge, and he says Sally will look in the box. There were

four questions asked: Naming (names for each girl in the story), Belief (where does Sally believe the marble is), Reality (where is the marble really), and Memory (where was the marble in the beginning). All members of the clinical samples (children with autism and children with Down Syndrome) and normal control groups replied accurately to the Naming, Reality, and Memory questions. The Belief question was answered accurately by 85% of the normal control group, 86% of the Down Syndrome group, and 20% of the autism group ($p < 0.001$); the children with autism pointed to the current marble location when asked where Sally would look for the marble. In other words, the children with autism did not demonstrate a theory of mind.

Over 1500 articles about theory of mind have been published since that time, many examining the construct in autism. Theory of mind has been explored in typical development[42–44] as well as in clinical groups beyond autism, including disruptive behaviors,[45,46] Fetal Alcohol Spectrum Disorders (FASD),[47] traumatic brain injury (TBI),[48] bipolar disorder,[49] and schizophrenia.[50] Although there are differing views regarding whether theory of mind has EF components, whether EF is a prerequisite for theory of mind, whether theory of mind is a prerequisite for EF, or whether theory of mind is actually an example of an EF skill, results from a number of analyses show a close relationship between theory of mind and EF,[45,46,51–56] with a meta-analysis finding a strong effect size of 1.08 for the relationship.[57]

Self-regulation in ADHD

In 1997, Barkley proposed a formal model of executive dysfunction in ADHD.[33] His model reflected a core deficit in "behavioral inhibition" that influenced four specified EF skills (WM, internalized speech, self-regulation of affect/motivation/arousal, and reconstitution); all five of these components impacted the motor control systems.[33] Barkley[58] proposed that "behavioral inhibition sets the occasion for the executive functions to occur" (p. 193). He[58] described behavioral activation as playing a support role, writing, "I would not consider arousal or activation to be executive functions in themselves" (p. 194). He criticized past descriptions of the central executive, saying they produce another black box or homunculus within the brain. Barkley[58] proposed instead that the central executive might actually be "time, or the individual's sense of the future," (p. 202) in that review of the past and future-directed goals represent self-regulation relative to time.

The influence of Bronowski's[31] model on Barkley's[33,58] work is clear, as the new model expanded Bronowski's five elements beyond language, adding an explicit component of the motor control system. The element of time can be linked to Fuster's premise that "cross-temporal contingencies," as subserved by temporal organization, temporal integration, attention, "executive working memory," and preparatory set, are critical for the timely organization of behavior and language.[59,60] Barkley also acknowledges the influence of Douglas' early work on self-regulation as an underlying deficit in ADHD,[61] Goldman-Rakic's WM model,[62] and Damasio's somatic marker hypothesis.[63]

Barkley's model is considered by some critics to be incomplete, and in need of broader validation than just with a sample of individuals with ADHD. As well, it is likely that this model serves to capture some but not all of the processes underlying what we conceptualize as EF. However, it does facilitate and foster an empirical approach to examining how EF interacts with and is guided by broader cognitive capacities.