

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/24439970>

Executive Functioning: Overview, Assessment, and Research Issues for Non-Neuropsychologists

Article in *Annals of Behavioral Medicine* · June 2009

DOI: 10.1007/s12160-009-9097-4 · Source: PubMed

CITATIONS

121

READS

267

1 author:



Yana Suchy

University of Utah

96 PUBLICATIONS **1,175** CITATIONS

SEE PROFILE

Executive Functioning: Overview, Assessment, and Research Issues for Non-Neuropsychologists

Yana Suchy, Ph.D.

Published online: 20 May 2009
© The Society of Behavioral Medicine 2009

Abstract

Background Executive functioning (EF) is a multifaceted neuropsychological construct that can be defined as (1) forming, (2) maintaining, and (3) shifting mental sets, corresponding to the abilities to (1) reason and generate goals and plans, (2) maintain focus and motivation to follow through with goals and plans, and (3) flexibly alter goals and plans in response to changing contingencies.

Purpose Research involving EF has become increasingly popular in a number of disciplines including behavioral medicine and personality research.

Methods The present article outlines some of the challenges faced by EF researchers, as well as briefly reviews historical perspective and neuroanatomical underpinnings of EF to provide broader context.

Results Clinical presentations of EF deficits, definitions and operationalization of the construct, as well as selection of appropriate assessment methods are provided.

Conclusion The article concludes with general cautions and guidelines for researchers.

Keywords Executive functioning · Frontal lobes · Neuropsychological assessment · Frontal-lobe syndrome · Cognition

Introduction

Executive functioning (EF) is a multifaceted neuropsychological construct consisting of a set of higher-order neurocognitive processes that allow higher organisms to make

choices and to engage in purposeful, goal-directed, and future-oriented behavior [1, 2]. EF confers an evolutionary advantage by freeing an organism from innate, hard-wired drives and reflexes, as well as from over-practiced, over-learned, and prepotent responses. A moth, for example, will be drawn to a light bulb time and time again, no matter if it burns its wings each time. In contrast, as humans, we possess the most highly evolved EF of all species, which allows us the latitude of considering options and selecting a specific response to any given stimulus based on situational contexts, previously acquired knowledge, and long-term goals.

Because EF is a highly effortful and, from an energy consumption standpoint, a costly process, it remains “dormant” for much of our waking hours, coming “on line” *only* when the novelty and/or complexity of a given situation *precludes an automatic, routine response*. Put another way, a response that is automatic or routine is, by definition, not a reflection of EF. This latter notion is important and is invoked later when the concept of “skills” will be discussed.

In clinical neuropsychology, it has been understood for a number of decades that EF represents one of a handful of core neurocognitive domains. Outside of neuropsychology, the EF construct has entered the research arena relatively recently, representing a new and exciting area of interdisciplinary inquiry. Researchers tackling this new direction face a number of challenges, most notably difficulties with defining, operationalizing, and assessing the EF construct. The present paper reviews these challenges and offers some general guidelines.

Brief Overview of EF Neuroanatomy

Traditionally, EF has been described as being associated with the so-called prefrontal cortex (PFC) of the brain,

Y. Suchy (✉)
Department of Psychology, University of Utah,
380 S. 1530 E., Rm. 502,
Salt Lake, UT, USA
e-mail: yana.suchy@psych.utah.edu

which includes all portions of the frontal lobes that are located anterior to motor and premotor cortices and the supplementary motor area (see Fig. 1). The prefrontal cortex can be divided into three main convexities: (a) the dorsolateral PFC, often described as the substrate of working memory [3]; (b) the superomedial PFC (which also includes the anterior cingulate gyrus), often described as the substrate for sustained attention, response selection, and motivation [4, 5]; and (c) the ventral (or inferior) PFC (which can be divided into orbitofrontal and ventromedial), often described as the substrate for inhibition, social appropriateness, and sensitivity to rewards and punishments [6–8]. In recent years, the most anterior portions of the frontal lobes, known as the frontal pole (which includes anterior portions of the dorsolateral and ventral prefrontal cortices), have also received much attention due to their role in morality [9], empathy [10], and higher order integration of EF [11].

The functions of the prefrontal cortex can also be further subdivided by the two cerebral hemispheres, such that the left PFC is associated with *initiation* of responses, as well as processing of information that is verbal, concrete, or detail-oriented, whereas the right PFC is associated with *inhibition* of responses, as well as processing of information that is visual-spatial, abstract or connotative, and gestalt-oriented [12, 13].

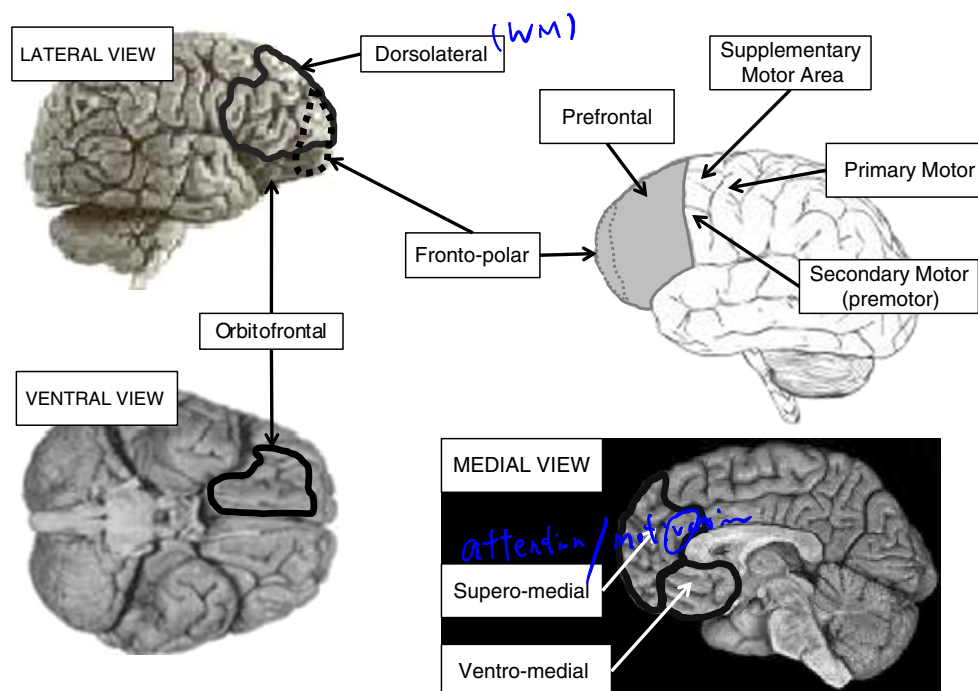
However, it is now well understood that the PFC is *not* the only brain area involved in EF. Because the frontal lobes are richly connected to a variety of other brain regions, most EF processes depend on the integrity of complex networks rather than a single frontal lobe region

(see Fig. 2). Thus, for example, working memory is dependent not only on the dorsolateral PFC but also on portions of the parietal lobe; response initiation is dependent not only on the left medial and ventral prefrontal cortices but also on the basal ganglia and the thalamus; sustained attention is dependent not only on the superomedial PFC but also the integrity of many regions within the right hemisphere and the thalamus; and so on and so forth. In fact, virtually all EF components require the integrity of circuits involving portions of the PFC, the basal ganglia, the thalamus, and the cerebellum, as well as cortical areas outside of the frontal lobes [14–17]. Clearly, the complexity of these networks is beyond the scope of this paper but suffice it to say that individual aspects of EF should *not* be viewed as easily localized.

Executive Dysfunction in Clinical Populations

In clinical neuropsychology, a patient with EF deficits is typically someone who has suffered some type of a brain injury or is afflicted with a neurodevelopmental or neurodegenerative illness. Typical examples would be individuals who have suffered a stroke in the frontal lobes and/or the related brain circuitry, victims of motor vehicle accidents, or patients with vascular, frontotemporal, or Parkinson's dementia. Among such individuals, EF deficits, especially if severe, can be extremely devastating. Practicing clinical neuropsychologists could likely generate virtually infinite numbers of examples: Social inappropriateness, such as persistent inappropriate sexual advances toward strangers; lapses in judgment, such as purchasing

Fig. 1 Convexities of the prefrontal cortex



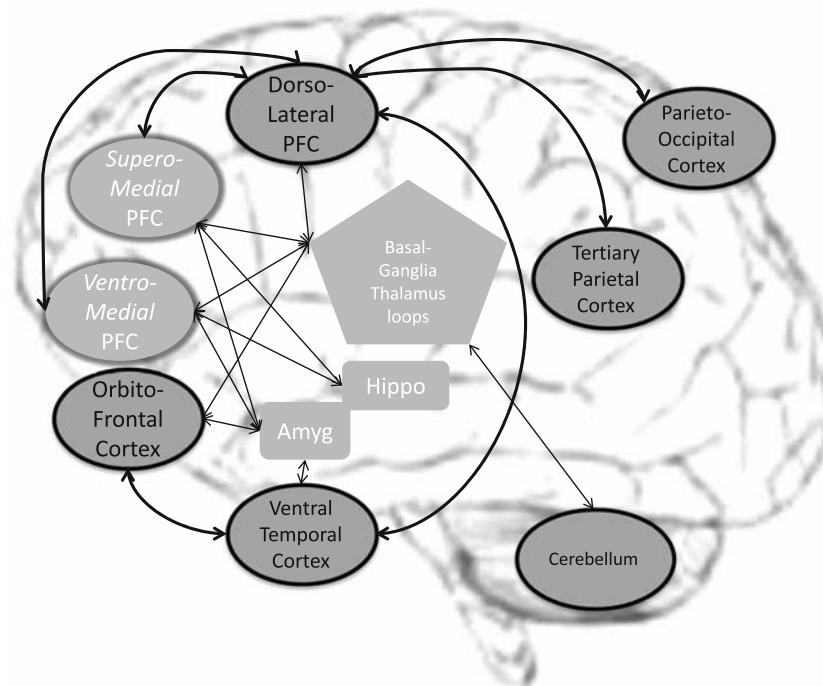


Fig. 2 Reciprocal connections between various convexities of the prefrontal cortex and limbic and subcortical structures. *Ovals outlined in black* represent lateral cortical areas and cerebellum. *Ovals outlined in gray* represent medial cortical areas. *Terms inside gray shapes*

without an outline represent subcortical or deep brain structures (amygdala, hippocampus, and basal ganglia/thalamus circuits). *Curved thick lines* represent cortico-cortical connections, whereas *thin straight lines* reflect cortico-subcortical connections

100 roles of paper towels without any understanding of how and why that happened; a complete lack of motivation, such as the inability to get out of bed to the point of laying in own excrement; or being “stimulus-bound” such that (similar to the example of the moth and the light bulb) a sight of food leads to eating, a sight of a pencil leads to drawing, a sight of a ball leads to kicking, a sight of a remote control leads to button pushing, and so on, no matter what the consequences are.

These examples illustrate the devastating functional impairments seen among individuals with severe damage to the EF substrates. They do not, however, afford appreciation of the individual differences in EF among *non*-brain-injured people. To appreciate the range of EF abilities, readers may consider simple EF lapses that all of us have experienced at one time or another. Most of us have missed a freeway exit while daydreaming; erroneously pressed the most-frequently called number on a speed dial; left our house keys in illogical places, such as the freezer or the sock drawer; or responded angrily in a situation when keeping emotions under control would have been more consistent with our intentions or goals. The frequency and seriousness of such lapses in everyday life can be viewed as a reflection of a person’s strengths or weaknesses in EF.

However, it is also important to note that *very frequent* lapses of this type signal psychopathology or neuropsychiatric illness. In fact, many Axis I disorders, such as mood

disorders, obsessive compulsive disorder, and schizophrenia [18], as well as Axis II disorders, such as antisocial [19] and borderline [20] personality disorders, have been associated with EF impairments.

Historical Perspective

Just as one is unaware of breathing until deprived of oxygen, one is unaware of the richness and complexity of one’s neurocognitive processes until deprived of them. Thus, historically, many cognitive functions came to the attention of scientists only after their *absence* has been observed among brain-injured individuals. The first description of the absence of EF dates back to 1835 [21], although it was another 13 years before the most well-known (and often mis-cited as the first) case of EF dysfunction came to be described. On September 13, 1848, a competent, well-liked, even-tempered railroad foreman named Phineas Gage sustained an injury in which a tamping iron penetrated his skull and severely damaged a considerable portion of his frontal lobes. Although Phineas Gage survived the accident, the injury resulted in a now well-recognized syndrome characterized by social inappropriateness, emotional dysregulation, and behavioral dyscontrol [22].

Although many other cases with similar types of deficits were later described in the literature, it was Alexander Luria, a Russian neuropsychologist, who in the 1960s and

1970s systematically studied the relationship between frontal-lobe injuries and social, emotional, and behavioral dyscontrol, popularizing the term “frontal-lobe syndrome” [23–25].

Although the term “frontal-lobe syndrome” is still occasionally used today, it has fallen out of favor. Over time, it has become increasingly clear that the syndrome (a) does not always occur following a frontal lobe injury and (b) sometimes *does* occur following injuries elsewhere in the brain. In other words, it became increasingly clear that equating the frontal lobes with cognitive and behavioral control was inadequate. This led to the introduction of the term “executive functions” [26]. Although the term is now widely adopted, our understanding of what it actually stands for continues to be a source of controversy and debate.

Approaches to Defining EF

The exact nature of the processes that fall under the umbrella of EF is still not well understood, and consequently, there is no universally accepted definition. In fact, even the approaches to defining EF vary widely among researchers. EF can be defined in a number of ways: (1) its overarching evolutionary *purpose* (e.g., ability to make choices and to engage in purposeful, goal-directed, future-oriented behavior), (2) the *clinical syndromes* observed in individuals who, due to brain damage, lose the abilities to engage in purposeful, goal directed behavior, (3) the list of the *complex skills* that contribute to purposeful, goal-directed behavior (e.g., reasoning and problem solving, planning, organization), (4) the more *elemental neurocognitive processes* that form the basis for specific EF skills (e.g., working memory, sequencing, inhibition, initiation, and response selection), (5) *atheoretical approaches*, driven by available variables or populations, (6) the *neuroanatomic substrates* that subserve complex skills and elemental neurocognitive processes (e.g., dorsolateral, superomedial, and ventral prefrontal cortices), and (7) *constructivist theories* that offer a framework for organizing the building blocks of EF. The advantages and disadvantages of each approach are considered below. An overview of the associations among the different levels of analysis of the building blocks of EF is shown in Table 1.

- (1) *Overarching evolutionary purpose.* As alluded to earlier, the overarching purpose of EF is to allow an organism to make choices and to engage in purposeful, goal-directed, and future-oriented behavior. Given the complicated, multifaceted nature of EF, defining it simply by its overarching purpose may well be the best approach, as it avoids the pitfalls of incomplete or poorly organized laundry lists of terms that themselves often need further defining. When explaining EF to

someone who is not familiar with the construct, this approach may well be the most comprehensive and conceptually meaningful. Similarly, this approach may provide a good framework for explaining the significance of EF research or its relevance for a person's success or quality of life. However, this approach is less useful when it comes to actually operationalizing EF for research or clinical observations, hence, the utility of the following approaches.

- (2) *Clinical syndromes.* In clinical neuropsychology, one classification system of EF subcomponents relies on the *syndromes* associated with different types of brain lesions [27]. In particular, lesions in the dorsolateral PFC and the related circuitry tend to lead to problems in the areas of reasoning, organization, planning, and problem solving, all of which rely on, among others, working memory. This constellation of symptoms has been referred to as the “dysexecutive” syndrome. Lesions in the orbitofrontal and ventromedial prefrontal cortices and the related circuitry tend to lead to problems in the area of social appropriateness and making adaptive choices, relying on inhibition and response selection. This constellation of symptoms has been referred to as the “disinhibited” (or sometimes “pseudo-psychopathic”) syndrome. Lesions in the superomedial PFC tend to lead to problems in the area of motivation, initiation, and sustained attention, referred to as the “apathetic” syndrome (see Table 1).

While defining EF based on clinical syndromes is useful in the clinical evaluations of neurologic populations, it is less helpful in non-neurologic settings. This is because neurologic lesions are often defined by structural, *not functional*, boundaries. For example, lesions due to strokes follow the architecture of blood vessels, *not brain functions*. Additionally, with respect to brain organization, contiguity does not always equal connectivity, as functional circuitry sometimes involves brain regions that are located far apart, even in separate lobes. And, unlike the contiguous brain areas that are affected in acquired lesions, it is these functional circuits that are often the target of various neurodevelopmental anomalies, that is, errors that occur during pre- or perinatal development. Consequently, the constellation of symptoms seen in neuropsychiatric populations does not always resemble the neurologic syndromes described above. Thus, although understanding of these syndromes may be useful in formulation of hypotheses, development of research questions, and conceptualization of results, description of individual *symptoms* (that is, deficits in either individual *complex skills* or individual *elemental neurocognitive processes*, described below) are often a more fruitful target for research of non-neurologic populations.

Table 1 Definitions of the components of executive functions (EF)

Overarching purpose	Cognitive skill	Neurocognitive processes	Corresponding neurobehavioral syndrome
Generating short-term and long-term goals OR “Set Formation”	Planning and reasoning	Focusing attention Generativity & memory retrieval Working memory Sequencing	Dysexecutive/Disorganized (primarily lesions in the dorsolateral prefrontal cortex)
Executing short-term and long-term goals OR “Set Maintenance”	Organization Follow-through Social appropriateness, judgment	Sequencing Working memory Initiation Response selection/conflict resolution Selective attention Self monitoring Attentional vigilance Inhibition, Discrepancy detection	Apathetic (primarily lesions in the superomedial prefrontal cortex) Disinhibited or Psychopathic (primarily lesions in the ventromedial and orbitofrontal cortex)
Altering short-term and long-term goals in response to changes in situations and contexts OR “Set Shifting”	Problem-solving	Discrepancy detection Cognitive flexibility Attentional shifting Generativity & memory retrieval Working memory	Dysexecutive/ Perseverative (primarily lesions in the dorsolateral prefrontal cortex)

(3) *Complex skills*. Describing EF in terms of complex skills is particularly useful in settings in which concrete observable everyday behaviors need to be identified. Planning, reasoning, problem solving, judgment, etc., are terms that can be easily understood, and behaviors that can be observed and reported on, by research participants, patients, or caregivers. Similarly, using descriptions of concrete skills, researchers can make conceptually meaningful claims that can readily be understood by other non-neuropsychology researchers. For example, one may say that patients with depression often find themselves unable to generate solutions because they suffer from impairment in reasoning, which falls under the umbrella of EF. It is likely this ease in communication that has rendered complex skills arguably the most popular approach to EF definitions.

Despite its popularity, this approach is not without problems. The major weakness of this approach is, ironically, inherent in its very reliance on *skills*—a learned ability to do something competently. In other words, *skills*, by definition, refer to something one can acquire, learn, practice, and improve, and as such they come on line in routine, or previously practiced, contexts. Recall that EF, in contrast,

does *not* come on line in situations in which behavior can rely on learned, routine, or automatic responses.

But the problem is *not* with the word “skills” but rather with the actual abilities that fall under the EF skill category. Take reasoning, for example. Reasoning skills are systematically taught and developed across all levels of formal schooling, with individuals who achieve higher levels of education typically having better developed reasoning skills. Consequently, an identical situation may then require a relatively minimal involvement of EF from a person with well-developed reasoning, in contrast to a relatively heavy reliance on EF from a person with poorly developed skills. Or, conversely, depending on the context, the same person may *appear* to have adequate EF in a situation that has, for them, become routine, but poor EF in another, more novel situation. Consider, for example, an elderly person who lives alone and independently takes care of her daily needs: She pays bills, shops, and cooks; that is, she engages in purposeful and goal-directed behaviors, the presumed output of intact EF. However, when faced with a life-changing situation, such as a move to a new home, this individual suddenly “develops” a severe EF deficit: Cooking, shopping, and bill paying are suddenly beyond her EF capacity.

Having a good conceptual grasp on the construct of EF allows one to understand that such individuals do *not*

suddenly develop dementia. Rather, when removed from a routine situation that requires minimal EF involvement and placed into a novel situation in which EF needs to be on line much of the time, evidence of functional impairment may become apparent. In other words, the latter situation is simply a more valid “test” of actual EF abilities. And, thus, in the final analysis, the use of specific *skills* for defining the construct of EF may lead to paradoxical conclusions: *The better practiced the skills, the less reflective of EF they actually are.*

Given these issues, researchers need to use caution when using a list of complex skills to define EF. As stated above, such definitions may facilitate communication or may be necessary when ratings of everyday behaviors are used as the dependent variables. But in laboratory settings, discrete neurocognitive processes likely represent a better and more valid approach to defining and operationalizing the EF construct.

- (4) *Elemental neurocognitive processes.* Unlike complex skills, discrete *elemental neurocognitive processes* are less susceptible to prior learning, familiarity with a situation, or educational achievement. They are the stuff that cognitive psychology and cognitive neuroscience are made of. Under the umbrella of EF, one can find such processes as initiation, inhibition, response selection, and working memory (see Table 1). Such processes have been identified by breaking down performances on various cognitive tasks into their most basic components. Historically, this has been accomplished by often ingenious manipulations of stimulus onset asynchronies, stimulus presentation latencies, the order of stimulus presentation, or the location of stimuli in one’s visual field. In recent years, such cognitive methods have been supplemented with functional neuroimaging, allowing further validation of discrete neurocognitive processes by demonstrating their reliance on predictable neuroanatomic networks [28–30].

Despite its considerable advantages and ever-increasing popularity, there are some pitfalls in this approach, which call for caution. In particular, although various discrete aspects of EF identified via these methods do correlate with neurocognitive, neuroanatomic, physiologic, genetic, and psychological variables, none of these components *alone* defines EF. Consequently, to be conceptually meaningful and complete, a definition of EF needs to rely on a *list* of such elemental processes. Unfortunately, these processes themselves are somewhat imprecisely defined, and the potential interpretations of individual terms are nearly as numerous as the researchers who use them. For that reason, a careful and consistent use of terminology, as well as thoughtful defining of terms, should always be a goal for researchers tackling this line of study.

- (5) *Atheoretical approaches.* As if the above approaches to defining EF were not challenging enough, things are further complicated by EF definitions that are opportunistic and/or atheoretical; that is, driven by which variables, tasks, or populations are available for any given study. Such approaches generally miss the boat with respect to the conceptual understanding of EF. On the one hand, readers may encounter definitions that oversimplify the construct of EF, using a single term or a phrase as a synonym for the entire construct and failing to consider EF’s multifaceted nature. For example, EF has been referred to by single terms such as “cognitive control,” “response selection,” “problem solving,” or “selective attention.” On the other hand, readers may encounter definitions of EF that are essentially long laundry lists of terms that do not fall neatly into any of the above-listed categories but rather mix and match some subset of the purposes, skills, processes, substrates, and syndromes.

Additionally, because purposeful, goal-directed behavior is central to human existence, the notion of EF permeates psychology even when the construct itself is not invoked. In fact, self-regulation, self-control, emotion regulation, delay of gratification, attentional control, self-monitoring, and response modulation, to name a few, all rely on some aspects of EF [31–36]. From among the terms listed above, attentional control deserves further mention, as there is considerable overlap between attention and executive processes. Specifically, *automatic* attention occurs spontaneously in response to stimuli and is present even among primitive species or among individuals who are considerably cognitively impaired, whereas *directed* attention is under EF control [37] and is sometimes included in the list of EF skills.

- (6) *Neuroanatomic substrates.* In the wake of what appears to be an unending frustration, the definition of EF has recently come the full circle, invoking, once again, the all but forgotten “frontal-lobe syndrome.” In particular, Donald Stuss, a prominent EF researcher, has explicitly elected to focus on the functional neuroanatomy of the human frontal lobes first and the EF construct second. He writes, “We have limited our research to patients with focal lesions of the frontal lobes....In our opinion this is the first step in limiting the terms of reference in the study of executive and frontal functions” (p 291) [38]. From a neuropsychological standpoint, this may, in the end, be the most reasonable approach: After all, one cannot argue with what functions should be included in this construct, as the physical boundaries are undeniably clear. However, the utility of this approach for personality and psychopathology research is questionable.

- (7) *Constructivistic definitions.* Given the complex nature of EF and the difficulties with defining it, a number of researchers have generated theoretical models that introduce new constructs, or latent variables, that could explain the EF structure and function. Such models sometimes propose a single unifying entity that is hoped to capture the EF construct in its entirety, such as the “central executive” [39, 40]. This and other similar models provide a very useful framework for the study of the construct of EF. However, they do not always lend themselves to research in which EF strengths and weaknesses among different populations are studied, as the ability of such models to truly capture all clinically and theoretically relevant aspects of EF has not yet been demonstrated. In other words, whether a particular model is useful needs to be determined based on the theoretical questions of any given study and on the aspects of EF that, in theory, uniquely characterize a given population.

For the purpose of organizing information into Tables 1, 2, and 3, a tripartite model of EF is used, which consists of three entities: the abilities to (1) form, (2) maintain, and (3) shift mental set [41–43]. These components correspond with the abilities to (1) reason and problem solve, (2) maintain motivation to follow through with a response in the absence of external structure, and (3) alter goals and plans as needed in response to changing contingencies.

The advantage of this model is that it affords descriptions of different profiles of strengths and weaknesses among the three major EF subcomponents (i.e., set formation, set maintenance, and set shifting), which can lead to certain predictable behavioral responses. In particular, individuals with a strength in the area of forming mental set generate new ideas easily and readily come up with plans or solutions to problems. However, such a set of strengths does not necessarily translate into successful *execution* of goals or plans. For that, adequate set maintenance needs to also be present, as well as the ability

Table 2 Review of clinical measures of executive functioning and subcomponent processes

Clinical tasks	Nonexecutive components	Set formation		Set shifting	Set maintenance			
		Working memory	Generativity	Cognitive flexibility	Response selection	Inhibition	Initiation	Attent'l vigilance
WCST-CA	Visual-spatial		*	*				
WCST-PR	Visual-spatial			*		*		
WCST-FMS		*						*
Figural fluency-unique designs	Visual-spatial, graphomotor, processing speed		*		*		*	
Figural fluency errors	Visual-spatial	*		*	*	*		
Verbal fluency words	Language, processing speed		*		*		*	
Verbal fluency errors	Language, processing speed	*		*	*	*		
Stroop color and word test	Visual spatial, language, processing speed				*	*		*
Trail making test-part B	Visual spatial, language, processing speed	*		*				*
Porteus mazes errors	Visual spatial	*				*		
Rey Figure-Copy	Visual spatial	*						
Towers	Visual spatial	*		*				
CPT-impulsivity						*		
CPT-vigilance								*
Go/No-go commissions						*		
Go/No-go omissions								*

“Towers” examples include Tower of Hanoi and Tower of London; “Verbal fluency” examples include Controlled Oral Word Association (COWA) and FAS; “Figural fluency” examples include Ruff Figural Fluency Test (RFFT)

WCST-CA Wisconsin Card Sorting Test-categories achieved, *WCST-PR* WCST perseverative responses, *WCST-FMS* WCST failure to maintain set, *CPT* continuous performance test

Table 3 Review of experimental measures of executive functioning and subcomponent processes

Experimental tasks	Nonexecutive components	Set formation		Set shifting Cognitive flexibility	Set maintenance			
		Working memory	Generativity		Response selection	Inhibition	Initiation	Attent'l vigilance
ANT-Exec Control	Visual spatial abilities				*			*
Reversal Learning	Visual spatial abilities	*	*	*	*			*
Cued Switching Tasks- Switching Costs	Verbal or visual spatial abilities, depending on the task			*	*		*	
Uncued Switching Tasks- Costs	Verbal or visual spatial abilities, depending on the task	*		*	*		*	*
Cued Switching Tasks- Switching errors	Verbal or visual spatial abilities, depending on the task					*		
Uncued Switching Tasks- Errors	Verbal or visual spatial abilities, depending on the task	*				*		*
N-Back	Processing speed	*						
Iowa Gambling Task		*			*	*		

ANT attention network test

to shift to a new plan if new situational constraints present themselves. Conversely, some individuals may have a weakness with respect to conceptualization and problem solving, but when presented with a reasonable plan (generated by someone else), they can successfully follow through with the plan's execution [44] (see Table 1).

Assessment Approaches

Assessment of EF has traditionally been fraught with difficulties. The first and the most obvious issue is the lack of a definition or a theory that would provide an adequate framework for assessment and test development. Another central problem with EF assessment is the rich connectivity (via rich neuroanatomic connections) of the EF system with other cognitive systems. Consequently, it is virtually impossible to assess EF without incidentally assessing other, nonexecutive processes.

The third central challenge is the fact that EF weaknesses often become apparent only in situations that *lack structure*. For example, some people with EF weaknesses find it difficult to *spontaneously* switch from one mental set to another, finding themselves stuck when problems in everyday life arise. However, when cued by someone that switching needs to take place, they are capable of generating ideas and solving problems. Unfortunately, most clinical and experimental testing situations unavoidably provide structure, which is often enough to help individuals with mild EF deficits to overcome their weaknesses. As an example, tests that assess

switching abilities often provide explicit instructions to switch [45]. It is against the backdrop of these challenges that the remainder of this section needs to be considered.

Clinical Tests

From a historical standpoint, given the initial conceptualization of EF impairments as manifestations of the “frontal-lobe syndrome,” the earliest assessment instruments were developed with the goal of identifying individuals with frontal-lobe lesions. Well-known examples include the Wisconsin Card Sorting Test (WCST) [46], the Trail Making Test-Part B (TMT-B) [47], and measures of verbal and figural fluency [48] (see Table 2). These tests have been extensively validated via demonstration of their sensitivity to frontal-lobe lesions [49–51] and continue to be popular in clinical and research settings to this day. In fact, some of them represent among the most sensitive, if *not specific*, EF instruments.

The issue of *specificity* is an important one and deserves further discussion, as typical clinical measures of EF virtually always assess several different components of EF as well as other *nonexecutive* functions. This is not an insurmountable problem in clinical settings, where entire batteries of tests are used and the locus of a deficit can be deduced through process of elimination. In other words, if a patient performs well on measures of processing speed and working memory, but poorly on TMT-B, then it can be deduced that cognitive flexibility is the culprit (see Table 2). However, in many research situations, such painstaking deduction process is not

practical, and in the final analysis, most research findings that are based on these instruments offer minimal discrimination with respect to discrete neurocognitive processes.

An additional problem with the use of clinical measures in research is that some of these tests yield relatively narrow ranges of scores with low ceilings. This, again, is not necessarily a problem in clinical settings, where only impaired individuals need to be identified and where normal distribution among healthy persons is not necessarily relevant. However, in research, normally distributed scores with adequate range of scores are crucial. The WCST, arguably the most misused neuropsychological measure in non-neuropsychological research, is a good example of this problem. The scores on this test are almost never normally distributed, and most healthy young individuals, unless neurologically impaired, produce virtually perfect scores. Consequently, studies that use this test for identification of subtle EF weaknesses among individuals with depression, anxiety disorders, or personality disorders frequently fail to reject the null hypothesis.

And finally, some clinical measures are based on the “skills” model of EF, relying heavily on *practiced* abilities such as reasoning, problem solving, and planning. While the WCST is somewhat problematic in this regard, even more problematic are the various Tower tests, and, even more so, the 20 Questions test that is part of the Delis–Kaplan Executive Function System battery [48]. Consider the following situation: Subject A grew up in a family in which playing the 20 Questions game was a common and popular pastime. Subject B, on the other hand, has never played the game in his life. When performing the 20 Questions test, Subject A can readily rely on over-learned sets of strategies, rambling off a series of questions about the mystery object’s semantic category or the size of the bread box. In other words, Subject A performs the test with minimal, if any, EF involvement. In contrast, Subject B needs to generate *novel* strategies, relying heavily on EF. Clearly, for each subject, the test is differentially sensitive to EF or may even measure an entirely different construct. While in clinical settings, the appropriateness of the measure for any given patient can be determined via clinical interview; this is again not practical in research situations.

Despite their weaknesses, there are some advantages to using clinical measures in research. The most notable among these is the fact that clinical measures virtually always provide published norms. Thus, if a studied group differs from a control group, one can use the norms to determine the clinical significance of such a difference, i.e., whether the studied group is characterized by *deficits* (that is, whether performances fall into a bona-fide impaired range), or just a relative *weakness*. Such determinations typically cannot be made with the use of experimental instruments, which are discussed next.

Experimental Tasks

Despite their lack of norms, experimental measures have a number of advantages over clinical instruments: They (a) tend to be designed to maximize sensitivity and specificity to *discrete aspects* of EF, (b) generate normally distributed scores, and (c) are relatively resistant to previously learned skills. Most such measures do not lend themselves to clinical evaluations for reasons that are beyond the scope of this paper but can, if used and interpreted properly, answer a variety of research questions. The tasks of this type can generally be divided into three main categories: (1) cognitive control, (2) working memory, and (3) emotional decision making. For a list of tasks, see Table 3.

Switching tasks [45], Go/No-Go and reversal learning tasks [52, 53], computer-administered Stroop tasks [54], and, recently, the Attention Network Test [55] are the most typical examples of *cognitive control* measures. All these tasks share in common the need to select one of several possible responses to a given stimulus (i.e., conflict resolution). This often involves, among others, inhibiting a response that is prepotent. The prepotent status of a given response is either due to the response being previously over-learned (e.g., on a Stroop task, word reading is prepotent to naming the color of print) or due to being primed as part of the experimental procedure (e.g., on a switching task, a participant generates a particular response on one trial, but that same response is incorrect on the subsequent trial). It is important to note that although all these types of tasks are often referred to as assessing cognitive control, each assesses somewhat different sets of control processes. For example, only inhibition of a response is needed on Go/No-Go tasks, which require that participants respond to some stimuli, while avoid response of any type to some other stimuli. In contrast, response selection is needed on Switching Tasks, which require that participants respond to a set of stimuli in a particular way (e.g., the *colors* of presented designs), until cued to respond in another way (e.g., the *shapes* of presented designs). See Table 2.

In contrast to cognitive control tasks, *working memory* tasks only require that participants hold information in working memory, without any conflict resolution requirement. Such tasks often utilize the so-called N-back paradigm, in which participants need to continually hold in mind a stimulus that was presented an N number of trials (most typically two) back.

Finally, the *emotional decision making* tasks, such as the Iowa Gambling Task (IGT), require that participants weigh, toward a specific goal, the relative contributions of rewards and punishments [56]. In the IGT, participants are instructed to select cards that are presented face down in four decks, with the goal of winning as much money as possible. Selecting cards from two of the decks (the “bad”

decks) results in large winnings but even larger losses, while selecting cards from the other two the decks (the “good” decks) results in small winnings but even smaller losses. While the IGT is the most well-known example of these types of tasks, a number of other tasks have been utilized in a similar manner, such as the passive avoidance task utilizing varied reward and punishment schedules in psychopathy research [57, 58].

Some caution in interpreting experimental tasks is in order. In particular, because such tasks measure very discrete neurocognitive processes, they may in some situations be less sensitive to a generalized EF weakness than clinical measures would be. This is because some EF deficits only become apparent as a result of the cumulative effect of a number of slight weaknesses across a number of discrete EF subcomponents. In contrast, in other situations, assessment of discrete processes may artificially inflate the impression of a deficit. This happens when a given population is characterized by a considerable anomaly in a single discrete EF process, an anomaly that only becomes apparent under certain very specific situations. The result, if robust and replicated, may have some very useful theoretical implications but may also have very limited implications for how one’s actual EF manifests itself in everyday life.

Summary

The present paper provides an overview of challenges associated with EF research, especially when conducted with non-neurologic populations. A conceptual overview of different approaches to defining and assessing EF, as well as strengths and weaknesses of such approaches, is provided, with the goal of affording researchers options for selection of an approach that best fits their purposes and contexts. Readers are encouraged to adhere to the following guidelines when using the EF construct in their research:

1. Develop a solid conceptual understanding of the construct of EF to facilitate appropriate study design and interpretation of findings.
2. Carefully define the EF construct in their writing, considering the audience and the purpose of their reference to EF; in other words, different definition approaches may be appropriate for conceptual discussions of EF versus empirical investigations of EF.
3. Carefully define all EF terms and variables, as no uniform definitions exist and the literature on the whole is fraught with confusion and misinterpretation.
4. Carefully select assessment instruments, considering the studied population, the expected nature and severity of EF weaknesses, and the degree to which clinical versus theoretical questions need to be answered.

Acknowledgment The author wishes to thank Drs. Paula Williams, Timothy Smith, and Gordon J. Chelune for valuable feedback and suggestions.

References

1. Cummings JL, Miller BL. *Conceptual and clinical aspects of the frontal lobes. The human frontal lobes: Functions and disorders*. 2nd ed. New York, NY, US: Guilford Press; 2007:12–21.
2. Gazzaley A, D’Esposito M, Miller BL, Cummings JL. *Unifying prefrontal cortex function: Executive control, neural networks, and top-down modulation. The human frontal lobes: Functions and disorders*. 2nd ed. New York, NY, US: Guilford Press; 2007:187–206.
3. Fuster JNM. The prefrontal cortex of the primate: A synopsis. *Psychobiology*. 2000; 28: 125–131.
4. Bush G, Frazier JA, Rauch SL, et al. Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the Counting Stroop. *Biol Psychiatry*. 1999; 45: 1542–1552.
5. Swards TV, Swards MA. Representations of motivational drives in mesial cortex, medial thalamus, hypothalamus and midbrain. *Brain Res Bull*. 2003; 61: 25–49.
6. Angrilli A, Palomba D, Cantagallo A, Maietti A, Stegagno L. Emotional impairment after right orbitofrontal lesion in a patient without cognitive deficits. *Neuroreport*. 1999; 10: 1741–1746.
7. Mathiesen BB, Farster PLV, Svendsen HA. Affect Regulation and Loss of Initiative in a Case of Orbitofrontal Injury. *Neuro-Psychoanal*. 2004; 6: 47–62.
8. Tremblay L, Schultz W. Reward-related neuronal activity during go-nogo task performance in primate orbitofrontal cortex. *J Neurophysiol*. 2000; 83: 1864–1876.
9. Moll J, Eslinger PJ, de Oliveira-Souza R. Frontopolar and anterior temporal cortex activation in a moral judgment task. *Arq Neuropsiquiatr*. 2001; 59: 657–664.
10. Ruby P, Decety J. How would you feel versus how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *J Cogn Neurosci*. 2004; 16: 988–999.
11. Koechlin E, Basso G, Pietrini P, Panzer S, Grafman J. The role of the anterior prefrontal cortex in human cognition. *Nature*. 1999; 399: 148–151.
12. Lezak MD, Howieson DB, Loring DW. *Neuropsychological Assessment*: Oxford University Press; 2004.
13. Stuss DT, Alexander MP, Floden D, et al. *Fractionation and localization of distinct frontal lobe processes: Evidence from focal lesions in humans, Principles of frontal lobe function*. London: Oxford University Press; 2002:392–407.
14. Aron AR. Progress in executive-function research: From tasks to functions to regions to networks. *Curr Dir Psychol Sci*. 2008; 17: 124–129.
15. Braver TS, Ruge H, Cabeza R, Kingstone A. *Functional neuroimaging of executive functions. Handbook of functional neuroimaging of cognition*. 2nd ed. Cambridge, MA US: MIT Press; 2006:307–348.
16. Ottowitz WE, Dougherty DD, Savage CR. The neural network basis for abnormalities of attention and executive function in major depressive disorder: Implications for application of the medical disease model to psychiatric disorders. *Harv Rev Psychiatry*. 2002; 10: 86–99.
17. Tekin S, Cummings JL. Frontal-subcortical neuronal circuits and clinical neuropsychiatry: An update. *J Psychosom Res*. 2002; 53: 647–654.
18. Moritz S, Birkner C, Kloss M, et al. Executive functioning in obsessive-compulsive disorder, unipolar depression, and schizophrenia. *Arch Clin Neuropsychol*. 2002; 17: 477–483.

19. Morgan AB, Lilienfeld SO. A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clin Psychol Rev.* 2000; 20: 113–156.
20. LeGris J, van Reekum R. The Neuropsychological Correlates of Borderline Personality Disorder and Suicidal Behaviour. *Can J Psychiatry.* 2006; 51: 131–142.
21. Lyketsos CG, Rosenblatt A, Rabins P. Forgotten frontal lobe syndrome or 'Executive Dysfunction Syndrome'. *Psychosomatics.* 2004; 45: 247–255.
22. Macmillan M. *An odd kind of fame: Stories of Phineas Gage.* Cambridge, MA US: The MIT Press; 2000.
23. Luria AR. *The frontal lobes and the regulation of behavior. Psychophysiology of the frontal lobes.* Oxford England: Academic Press; 1973.
24. Luria AR, Homskey ED, Blinkov SM. Impaired selectivity of mental processes in association with a lesion of the frontal lobe. *Neuropsychologia.* 1967; 5: 105–117.
25. Luria AR, Majovski LV. A frontal lobe syndrome due to a cerebrovascular accident affecting the corpus callosum. *Clin Neuropsychol.* 1979; 1: 17–19.
26. Lezak MD. The problem of assessing executive functions. *Int J Psychol.* 1982; 17: 281–297.
27. Duffy JD, Campbell JJ III, Salloway SP, Malloy PF. *Regional prefrontal syndromes: A theoretical and clinical overview. The frontal lobes and neuropsychiatric illness.* Arlington, VA, US: American Psychiatric Publishing, Inc.; 2001:113–123.
28. Fink GR, Halligan PW, Marshall JC, et al. Neural mechanisms involved in the processing of global and local aspects of hierarchically organized visual stimuli. *Brain* (Vol. 120). Oxford: Oxford Univ Press, <http://www.oup.com>; 1997: 1779–1791.
29. Nielson KA, Langenecker SA, Garavan H. Differences in the functional neuroanatomy of inhibitory control across the adult life span. *Psychology & Aging* (Vol. 17). Washington, DC: American Psychological Assn, <http://www.apa.org>; 2002, 56–71.
30. Sylvester C-Y, Wagner TD, Lacey SC et al. Switching attention and resolving interference: fMRI measures of executive functions. *Neuropsychologia* (Vol. 41). Amsterdam: Elsevier Science, <http://www.elsevier.com>; 2003, 357–370.
31. Carlson SM, Wang TS. Inhibitory control and emotion regulation in preschool children. *Cogn Dev.* 2007; 22: 489–510.
32. Knolle-Veentjer S, Huth V, Ferstl R, Aldenhoff JB, Hinze-Selch D. Delay of gratification and executive performance in individuals with schizophrenia: Putative role for eating behavior and body weight regulation. *J Psychiatr Res.* 2008; 42: 98–105.
33. Magar ECE, Phillips LH, Hosie JA. Self-regulation and risk-taking. *Personality Individ Differ.* 2008; 45: 153–159.
34. Osaka N. The frontal lobe correlates of metacognition: Neural interpretation based on the cognitive neuroscience of working memory. *Jpn Psychol Rev.* 2007; 50: 216–226.
35. Suchy Y, Kosson DS. State-dependent executive deficits among psychopathic offenders. *J Int Neuropsychol Soc.* 2005; 11: 311–321.
36. Vohs KD, Baumeister RF, Schmeichel BJ, et al. Making choices impairs subsequent self-control: A limited-resource account of decision making, self-regulation, and active initiative. *J Pers Soc Psychol.* 2008; 94: 883–898.
37. Posner MI, Dehaene S. Attentional networks. *Trends Neurosci.* 1994; 17: 75–79.
38. Stuss DT, Alexander MP. Executive functions and the frontal lobes: A conceptual view. *Psychological Research.* 2000; 63: 289–298.
39. Baddeley A, Della Sala S, Roberts AC, Robbins TW, Weiskrantz L. *Working memory and executive control. The prefrontal cortex: Executive and cognitive functions.* New York, NY US: Oxford University Press; 1998:9–21.
40. Baddeley A. The central executive: A concept and some misconceptions. *J Int Neuropsychol Soc.* 1998; 4: 523–526.
41. Osmon DC. *Complexities in the evaluation of executive functions., Forensic neuropsychology: Fundamentals and practice.* Lisse, Netherlands: Swets & Zeitlinger Publishers; 1999:185–226.
42. Osmon DC, Suchy Y. Fractionating frontal lobe functions: Factors of the Milwaukee Card Sorting Test. *Arch Clin Neuropsychol.* 1996; 11: 541–552.
43. Suchy Y, Kosson DS. Forming, switching, and maintaining mental sets among psychopathic offenders during verbal and nonverbal tasks: Another look at the left-hemisphere activation hypothesis. *J Int Neuropsychol Soc.* 2006; 12: 538–548.
44. Suchy Y, Blint A, Osmon DS. Behavioral Dyscontrol Scale: Criterion and predictive validity in an inpatient rehabilitation unit population. *Clin Neuropsychol.* 1997; 11: 258–265.
45. Jersild AT. Mental set and shift. *Archives of Psychology.* 1927, *Whole number 89.*
46. Heaton RK, Chelune GJ, Talley JL, Kay GG, Curtiss G. *Wisconsin Card Sorting Test Manual: Revised and Expanded.* Lutz: Psychological Assessment Resources, Inc.; 1993.
47. Reitan RM. *Trail Making Test: Manual for administration, scoring, and interpretation.* Indianapolis: Indiana University Medical Center; 1958.
48. PsyCor. *Delis-Kaplan Executive Function System.* San Antonio: The Psychological Corporation; 2001.
49. Demakis GJ. A meta-analytic review of the sensitivity of the Wisconsin Card Sorting Test to frontal and lateralized frontal brain damage. *Neuropsychology* (Vol. 17). Washington, DC: American Psychological Assn, <http://www.apa.org>, 2003, 255–264.
50. Demakis GJ, Harrison DW. Relationships between verbal and nonverbal fluency measures: implications for assessment of executive functioning. *Psychol Rep.* 1997; 81: 443–448.
51. Reitan RM, Wolfson D. Category Test and Trail Making Test as measures of frontal lobe functions. *Clin Neuropsychol.* 1995; 9: 50–56.
52. Buss AH. Some determinants of rigidity in discrimination-reversal learning. *J Exp Psychol.* 1952; 44: 222–227.
53. Drewe EA. Go-no go learning after frontal lobe lesions in humans. *Cortex.* 1975; 11: 8–16.
54. Quinn SOB, Quinn EP. The Stroop Interference Effect in Young Children: A Developmentally Appropriate Approach. *Ind Diff Res.* 2005; 3: 183–187.
55. Fan J, McCandliss BD, Sommer T, Raz A, Posner MI. Testing the efficiency and independence of attentional networks. *J Cogn Neurosci.* 2002; 14: 340–347.
56. Bechara A, Damasio H, Tranel D, Damasio AR. The Iowa Gambling Task and the somatic marker hypothesis: Some questions and answers. *Trends Cogn Sci.* 2005; 9: 159–162.
57. Newman JP, Patterson CM, Howland EW, Nichols SL. Passive avoidance in psychopaths: The effects of reward. *Pers Individ Differ.* 1990; 11: 1101–1114.
58. Newman JP, Schmitt WA. Passive avoidance in psychopathic offenders: A replication and extension. *J Abnorm Psychol.* 1998; 107: 527–532.