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Executive function in the context of chronic disease prevention: Theory, research and practice

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ABSTRACT

Objective. To provide an overview of the nature, organization and measurement of executive function, and describe its significance for preventive medicine theory, research and practice.

Method. A conceptual and narrative review linking the operation of executive control systems to health behavior performance and health outcomes, within the context of chronic illness prevention.

Results. Stronger executive function is linked with more consistent performance of a variety of health protective behaviors, less performance of health risk behaviors, and greater longevity in the existing observational research literature. These effects are not fully explained by demographic factors such as education, income and socioeconomic status, but may in some cases interact with them, or mediate their effects on other outcomes. Experimental manipulations of executive control suggest that the effect of executive function is causal, particularly in relation to the modulation of appetitive craving responses that may compete with healthy behaviors (or facilitate unhealthy behaviors).

Conclusion. Executive function is a potentially important variable in explanatory frameworks for health behavior and health outcomes. The size of effect and its endurance remain uncertain, though the causal status of its influence on some behaviors is becoming increasingly clear. Additional understanding of the relation between executive control and demand imposed by ecological context is an important frontier for research on changing behavior to prevent disease, and may be an explanatory factor in social patterning of these same conditions.

Why do human beings behave in ways that are ultimately in conflict with their own health interests? What is responsible for the seeming default status of such tendencies? The answers to these questions are complex and our understanding of the underlying causal forces remains incomplete. However, important advances from the fields of social and cognitive neurosciences are beginning to push forward the frontier of our understanding. In this paper we review the contributions of two processes that have potentially important, interdependent roles in producing health behavior trajectories that generate predispositions to chronic illness development. The first is executive function (EF), which enables controlled processing of information and top-down (i.e., non-stimulus driven) regulation of behavior, as directed by conscious thought. The second, related process is that of automaticity, typified by the withdrawal of controlled processes and yielding to bottom-up behavioral control by the environment, situational cues, and visceral drives. As we will discuss, the interplay of these two modes of operation (driven by the convergence of multiple systems of influence) may explain some of the all-too-human tendency to engage in behaviors that encourage the development of chronic diseases over the lifespan.

What is executive function?

Executive function (EF) is a set of cognitive abilities that collectively serve to assist with top-down (i.e., non-stimulus driven) control of behavior, emotion and thought (Baddeley, 1996; Miyake and Friedman, 2012; Miyake et al., 2000; Shallice and Burgess, 1993). EF has an overarching unitary quality, but also correlated (but dissociable) subfunctions including inhibition (i.e., the ability to suspend prepotent/default responses), mental flexibility (i.e., the ability to switch back and forth between rules or response sets), and working memory (i.e., the ability to hold in mind and work with finite packets of task-relevant information; Miyake and Friedman, 2012). Statistically speaking, the most "pure" facet of EF is inhibition, being perfectly correlated with unitary EF, while working memory and mental flexibility are thought to include a mixture of both pure EF and unique components (Miyake and Friedman, 2012). The coordination of all three sub-facets of EF allows us to engage in more complex self-regulatory actions such as effortful goal pursuit, resisting temptations, and maintaining

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focus on a long-term goal in the presence of distracters or other less important, competing goals (Hofmann et al., 2012). Although the definition and scope of EF as a theoretical concept is debated, most models minimally include the three components mentioned above (inhibition, working memory, and mental flexibility), while others expand to include some of the functions enabled by EF such as planning, prospective memory, and control of attention.

Individual differences in EF are related to, but conceptually distinct from, general cognitive dispositions such as IQ and broad personality traits such as conscientiousness. While IQ is the most general of the cognitive functions (in fact, the aggregation of all cognitive functions into a single undifferentiated metric), EF is much more specific, pertaining mostly to the self-control facet of cognition. Personality factors such as conscientiousness are conceptually similar to EF, but reflect only the self-perceived regularities in behavior that are acknowledged by the individual and reported as such. The patterns of association between IQ/conscientiousness and health outcomes mirror those of EF (Bogg and Roberts, 2013; Deary, 2012), but in fact EF partially explains both of these associations in a statistical sense (i.e., IQ and conscientiousness predict health outcomes because of their overlap with EF processes; Hall et al., 2009, 2013a).

Demographic influences on EF

EF has important associations with demographic characteristics including education and socioeconomic status. Poverty in childhood has been a particular focus of study. The impact of poverty and the moderation of the effects by genetic differences are exemplified in a recent prospective study of children in low income families in North Carolina (Raver et al., 2013). These children were followed from birth and EF measured at 48 months using measures of working memory, mental flexibility and inhibitory control. Executive function was reduced by 0.10 standard deviations for each year of the first four years of life these children lived below the poverty threshold. Importantly these effects were not evident in all children. Those who had temperaments characterized by high levels of reactivity were those most negatively affected.

A number of variables may explain the link observed including genetic endowment and education, each of which may be associated with poverty and thereby influence EF. A recent systematic review attempted to disentangle the causal links between poverty and a range of outcomes for children including health, social, behavioral and cognitive outcomes (Cooper and Stewart, 2013). The authors concluded that the evidence relating to poverty to cognitive development and school achievement was the clearest in indicating a causal role of poverty upon cognitive development.

Physiological substrates of EF

As a theoretical entity, EF is in fact an emergent quality of cognitive function that arises from the operation of several interconnected centers within the human cortex, most prominently featuring the prefrontal cortex (PFC; Garavan et al., 2002; Miller, 2000; Miller and Cohen, 2001), but also parietal areas (Bellebaum and Daum, 2007a; Van der Werf et al., 2003). Within the PFC, there are several substructures that have been implicated in the neurobiology of EF, including the dorsolateral, ventromedial, and inferior regions (Miller, 2000; Miller and Cohen, 2001). Aside from such internal substructures, arguably the most important feature of the executive control network is its interconnections with other brain centers and systems, enabling its potential for modulation of these same structures. Specifically, the PFC is highly interconnected with evolutionarily older structures implicated in the generation of emotion (limbic system), motor responses (motor cortex) and reward responsivity (the striatum; Alexander et al., 1986; Crowe et al., 2013; Cummings, 1995; Groenewegen et al., 1997; Miller, 2000; Miller and Cohen, 2001; Tekin and Cummings, 2002).

Developmental aspects of EF

The lifespan trajectory of EF development is more complex than some other cognitive functions. From birth to adulthood, the development of EF is driven primarily by the rate of maturation of the PFC and other structures that support it. In contrast with other regions of the brain that are in place by adolescence (12–18), the PFC continues to develop well beyond adolescence and into young adulthood (late teens, early 20s; (Diamond and Lee, 2011)). The aging process beyond middle adulthood (40 to 50 years) further influences the integrity of the executive control network; the PFC and its connecting fibers (to other regions mentioned above) are among the most sensitive to the effects of age-related cognitive decline in late life (Dodge et al., 2011; MacPherson et al., 2002; O'Sullivan et al., 2001). As such, over the lifespan from birth to death, the brain regions supporting EFs are subject to considerable influence from the environment, the aging process, and possibly even behavior itself. Nonetheless, rank ordering of individual differences—within each of these spheres of influence—is subject to substantial genetic influence (Friedman et al., 2008). These individual differences, regardless of origin, have many implications for outcomes in financial, social and health domains (Moffitt et al., 2011). We explore the latter in this review.

Measurement of EF

Typically EF has been assessed by neuropsychological testing or cognitive paradigms, both of which can be administered either in person or by computer. Examples of common EF measures include the Stroop task, Trails B, Stop Signal task, Go/No-Go task and the Flanker task, among others. Many of these tasks require participants to override a habitual or reflexive response in order to substitute another, more novel, response in its place. Combinations of errors (of commission or omission) and/or reaction times (faster is generally better) are used as metrics to quantify EF strength. Tests examining the working memory facet include N-back task, digit span and reading span tasks, while mental flexibility is quantified by tasks such as the Navon figure, and the Wisconsin Card Sort. Complex EF tasks—i.e., tasks that tap several different facets of EF at the same time—include the Tower of Hanoi, Tower of London and the Iowa Gambling task. It is also possible to combine these "behavioral" measures with functional neuroimaging methods (e.g., fMRI, fNIRS and EEG) to gauge the amount of blood flow, oxygenation, neuroelectric activity in the areas of the brain that support EF processes. Although behavioral tests of EF are intended for adolescents and throughout the adult lifespan, versions for children have also been developed (eg., Zalazo, 2006). For a review of these and other measures see Jurado and Rosselli (2007), as well as Miyake et al. (2000); Banich (2009) provides an accessible description linking performance on some of these to underlying neurophysiological substrates.

Relationship among EFs, health behavior and health outcomes

At least two prospective observational studies have linked individual differences in EF with all-cause mortality; in both studies, those initially healthy older adults with stronger baseline EF had significantly longer 10-year survival than their low EF counterparts (Duff et al., 2009; Hall et al., 2009), and this effect was demonstrated to be independent of age, sex and education (Hall et al., 2009). Moreover the excess mortality in the low EF group in one of these two studies (Hall et al., 2009) appeared to be attributable to increased new occurrence of chronic illnesses—those illnesses with high behavioral imperatives for prevention—in the low EF group. These findings, of course, beg the question: Why is stronger EF associated with longer survival and lower incidence of chronic illness? Several possibilities exist (see Fig. 1), for instance, EF may be lowered by the early (and undetected) progression of insidious disease process, which in turn eventually