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Executive Functioning and Health

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Abstract and Keywords

This review provides an overview of research on associations between the multi-component, cognitive construct executive functioning (EF) and health. Executive functioning is defined, and issues related to measurement are detailed. The categories of potential mechanisms by which EF may be associated with health and disease are described. Key research examining EF and health behaviors, stress processes, and chronic illness is reviewed with a focus on function (behavioral performance), as well as neuroanatomical research where relevant. Across these domains, there is evidence that EF is associated with health and illness in reciprocal, feed-forward fashion across the life span. Critical limitations of the current literature are noted, along with important future directions.

Keywords: Executive functioning, health behavior, smoking, alcohol use, exercise, stress, sleep, chronic illness

Executive Functioning and Health

In recent years, there has been a burgeoning interest in the association between cognitive functioning and health. Although some connections have long been understood, such as the effects of coronary artery disease and stroke on the brain, a more nuanced focus on cognitive functioning and health has emerged. Of particular interest has been executive functioning (EF). This set of neurocognitive processes has now been linked to health through a variety of mechanisms. Importantly, EF has shown substantial heritability, and it therefore is now considered a critical endophenotype-level individual-difference factor that not only is affected by disease processes, but also prospectively predicts disease onset. In this review, EF is defined, and issues related to its measurement are detailed. The potential mechanisms by which EF may be associated with health and disease are then described. The connections between EF and health behaviors, stress processes, and chronic illness are outlined, relying, where possible, on

previous reviews, meta-analyses, and well-powered empirical studies in the literature. Although the primary focus of this review is on function, where appropriate we also consider neuroanatomical underpinnings (e.g., prefrontal cortex, anterior cingulate cortex, frontal subcortical structures, white matter). Critical limitations of the current research are noted, along with important future directions. Whereas the primary focus of the review is on adults, EF and health research involving children and adolescents is also considered, as appropriate. It is beyond the scope of this article to provide an exhaustive review of all relevant research; rather, a broad overview is provided, emphasizing well-established associations as well as a theoretical framework for considering reciprocal, feed-forward associations between EF, health, and disease.

Executive Functioning: Definition and Measurement

“Executive functioning” refers to a set of higher-order cognitive processes that allow one to plan, organize, and successfully execute purposeful, goal-directed, and future-oriented actions (Cummings & Miller, 2007; Lezak, Howieson, Bigler, & Tranel, 2012; Suchy, 2015). Unfortunately, no single universally accepted definition of EF exists (Suchy, 2009). The matter is further complicated by the fact that conceptualizations of EF come from several different research traditions. In particular, definitions that originate from clinical neuropsychology (the discipline that first introduced this construct; Luria, 1980; Pribram, 1973), tend to be comprehensive, multifaceted, and clinically applicable. In this tradition, EF is typically thought to be composed of processes or abilities that are known to be deleteriously affected by neurological conditions and brain injuries. Consequently, clinical conceptualizations parallel various syndromes of executive dysfunction described in patient populations (e.g., Burgess & Shallice, 1996; Duffy, Campbell III, Salloway, & Malloy, 2001; Stuss & Alexander, 2008) (for a recent comprehensive overview and conceptual analysis of such clinically relevant processes, see Suchy, 2015).

In contrast to the clinical tradition, conceptualizations of EF that stem from cognitive psychology or cognitive neuroscience tend to be reductionist and, in some cases, even view EF as a unitary construct (e.g., Baddeley, Chincotta, & Adlam, 2001; Baddeley, Della Sala, Roberts, Robbins, & Weiskrantz, 1998). These conceptualizations are usually based on research conducted with normal or neurologically healthy populations (often college students) and tend to rely heavily on experimental tasks and factor analyses (e.g., Friedman & Miyake, 2004; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). As such, these definitions lend themselves to the study of individual differences in EF, but they are less applicable to describing deficits seen in patient populations.

Lastly, some conceptualizations stem from developmental psychology. These tend to rely on what is known about the maturation of EF in childhood and adolescence, tend to

Executive Functioning and Health

incorporate emotional processing, and often relate EF to the constructs of self-regulation or self-control (e.g., Diamond, 2013; Steinberg, 2010).

Measurement of EF can be as complex, or as simple, as the conceptual model one employs. Consequently, whereas some studies rely on multiple measures that are combined into composites presumed to measure the latent constructs (e.g., Kraybill, Thorgusen, & Suchy, 2013; Suchy et al., 2016), others virtually equate certain experimental tasks with the construct itself (e.g., Conway et al., 2005; Fan, McCandliss, Sommer, Raz, & Posner, 2002). A full accounting of approaches to EF measurement is beyond the scope of this review. Suffice it to say that, in general, more comprehensive assessments that rely on composite scores are more likely to be reliable, as well as more likely to tap into multiple components of the EF construct, which makes them more sensitive to executive dysfunction (Suchy, 2015). In contrast, some experimental tasks can reliably measure discrete executive processes. Although these tasks tend to be insensitive to certain types of EF dysfunction, they may represent important biological markers in healthy populations (Rothbart, Sheese, & Posner, 2007). One important caveat in the assessment of EF is that lower-order processes, such as comprehension of test instructions, perception of stimuli, speed of processing, or speed of motor or verbal output, must be accounted for before interpretation of results can be made (for a comprehensive review, see Suchy, Niermeyer, & Ziemnik, 2017; and Suchy, Ziemnik, & Niermeyer, 2017).

In this review, we will generally refer to EF as an overarching construct, rather than to its subdomains. This is because most tests of EF confound several EF subdomains. Additionally, although some studies refer to various components of EF specifically, these components are rarely defined, and it would not be appropriate to assume that terms such as “inhibition” and “flexibility,” for example, refer to the same constructs across studies. However, on occasion, and only when appropriate based on available evidence, we will distinguish between cognitive (working memory, mental flexibility) and behavioral (initiation, maintenance, or selection of responses) aspects of EF, and in some cases we will specify behavioral inhibition, due to its relevance for certain health behaviors.

Executive Functioning and Health: Potential Mechanisms

Executive functioning may be associated with health and disease through a variety of pathways. First, EF may be associated with health behavior. The routine engagement in both positive health behavior (e.g., exercise) and the avoidance of negative health behaviors (e.g., smoking, overeating) requires organization, persistence, and planning, as well as the ability to override emotional tendencies. Second, EF may be associated with stress processes, including stress exposure, stress reactivity, and recovery, as well as restorative processes that serve to repair stress-related damage (e.g., sleep) (Williams,

Executive Functioning and Health

Suchy, & Rau, 2009). Finally, EF may be associated with the severity and progression of chronic illness, both via direct pathophysiological mechanisms, as well as through effects on adherence to medical regimens. Across these broad health and disease categories, the associations may be bidirectional, with feed-forward and feedback reciprocal pathways between EF and health. That is, EF may be a predictor of future health behavior, stress, and disease, but it is also, in turn, altered by a variety of physiological, disease-related, and medical treatment-related processes.

Executive Functioning and Health Behavior

Smoking

Cigarette smoking is a leading cause of chronic disease and all-cause mortality (e.g., Thun et al., 2013). A recent review posits a bidirectional relationship between cigarette smoking and EF, in which better EF may reduce the potential to begin smoking and, in turn, smoking negatively affects EF. In addition, there is evidence that poor EF may impair the ability to quit smoking (Loprinzi et al., 2015). Conversely, better EF is associated with a greater likelihood of quitting smoking (Brega, Grigsby, Kookan, Hamman, & Baxter, 2008). Heavy smoking, as well as passive smoking, are associated with poorer cognition, including EF, in older adults (Anstey, von Sanden, Salim, & O’Kearney, 2007; Chen, Hu, Orton, Chen, & Wei, 2013; Lo, Pachana, Byrne, & Sachdev, 2012; Razani, Boone, Lesser, & Weiss, 2004) and increased risk for the development of Alzheimer’s disease (Lee et al., 2010; Peters et al., 2008). There is some evidence of gender differences in the strength of this association. In a study of middle-aged adults, lifetime tobacco-use disorders (as well as alcohol-related disorders) were not associated with cognitive performance among men; however, women with a diagnosis of tobacco dependence demonstrated both poorer global intellectual functioning and poorer EF (Caspers, Arndt, Yucuis, McKirgan, & Spinks, 2010).

Importantly, EF abilities at an early age predict future substance use, including cigarette smoking, in adolescence. In a study that assessed boys between the ages of 10 and 12 and followed them for two years, those at elevated risk for the development of substance use disorder (i.e., whose fathers had received a lifetime DSM-III-R diagnosis of psychoactive substance use disorder) demonstrated poorer baseline EF than children with low risk for the development of substance use disorder (i.e., whose fathers had never received a lifetime Axis I psychiatric diagnosis) (Aytaclar, Tarter, Kirisci, & Lu, 1999). Furthermore, baseline EF strongly predicted future substance use more generally, including tobacco, alcohol, and drug use in early adolescence.

With respect to mechanisms for these associations, research indicates that smoking negatively affects brain structures that subservise cognitive abilities, including EF. For example, older chronic smokers exhibited greater brain atrophy in the frontal regions over a two-year period, compared to their non-smoking counterparts (Durazzo, Insel, & Weiner, 2012). Additionally, chronic smoking is associated with electrophysiological abnormalities in the regions of the brain associated with EF, and, not surprisingly, poorer performance in a variety of cognitive domains, including EF (Durazzo, Gazdzinski, & Meyerhoff, 2007; Wang, Xu, Qian, Shen, & Zhang, 2015). Brain-related changes as a consequence of smoking are multi-determined and are likely to include decreases in both brain perfusion (Durazzo, Meyerhoff, & Murray, 2015) and general cardiovascular health. Future longitudinal research is needed to investigate the association between EF abilities prior to smoking initiation and the course of EF after initiating smoking. Lastly, the improvement of EF through healthy behaviors, for example by increasing physical activity, may aid smoking cessation (Loprinzi et al., 2015).

Alcohol Use

Moderate-to-heavy alcohol consumption is associated with cognitive dysfunction and increased risk of mortality (e.g., Rogers, Boardman, Pendergast, & Lawrence, 2015; Scheurich, 2005). In the substance abuse literature, EF deficits are virtually ubiquitous (for review, see Yücel, Lubman, Solowij, & Brewer, 2007). Regarding alcohol use in particular, recent reviews found that alcohol use is strongly associated with poorer EF specifically (Day, Kahler, Ahern, & Clark, 2015) and cognitive performance generally (Bartley & Rezvani, 2012; Stephens, Ling, Heffernan, Heather, & Jones, 2008; Field, Schoenmakers, & Wiers, 2008). Importantly, premorbid cerebral abnormalities in individuals with a family history of alcohol dependence are associated with EF difficulties that may confer risk for the development of alcohol-related problems. Specifically, a review of neuroimaging and neurophysiological studies of youth and adults with a family history of alcohol-related problems identified both gray and white matter structural abnormalities in brain regions important for EF, as well as EF limitations (Cservenka, 2016). Thus, premorbid cerebral abnormalities may contribute to EF weaknesses, which in turn tend to be associated with impulsive behavior and risk for the development of alcohol use disorder.

In addition, a recent review suggests that youth with a history of parental alcohol abuse demonstrate neurobiological alterations that are associated with compromised executive functions, yet they may not display behavioral EF deficits (Nixon, 2013). In general, alcohol use among young adults is associated with deficits in neurobehavioral processes, including EF, with history of parental alcohol use an important predictor of the onset of alcohol use (for review, see Hermens et al., 2013).

Regarding the deleterious impact of alcohol on cognition, a systematic review and meta-analysis of studies investigating alcohol-related EF impairments in social drinkers found no evidence for an association across seven studies; however, *heavy* alcohol use in participants without an alcohol-related disorder was associated with poorer EF (Montgomery, Fisk, Murphy, Ryland, & Hilton, 2012). Importantly, heavy alcohol users demonstrated EF deficits similar to those of *chronic* heavy alcohol users, consistent with the notion of bidirectional effects, such that EF deficits may confer risk for the development of alcohol-use disorder, while alcohol use contributes to neurodegenerative changes in the brain and concomitant EF deficits. In addition, a meta-analysis investigating cognitive recovery and abstinence from alcohol among alcoholics found that recovery of cognitive abilities, including EF, progresses throughout the first year of abstinence. This evidence of a persistent effect of alcoholism on cognition after initiating abstinence highlights the need to provide extra structure and scaffolding for individuals in the first year of treatment (Stavro, Pelletier, & Potvin, 2013).

The deleterious impact of alcohol on EF is a function of multiple mechanisms and includes both the direct toxic effects of alcohol on brain structures important for EF (e.g., deep white matter, cerebellum) (Elofson, Gongvatana, & Carey, 2013), as well as dietary

Executive Functioning and Health

and lifestyle factors that are often associated with heavy alcohol use and that in themselves can result in further brain damage (de la Monte & Kril, 2014). Future longitudinal research is needed to understand the association between alcohol consumption throughout the life span and EF. For example, a systematic review found that *moderate* alcohol consumption was associated with lower risk for Alzheimer's disease, but not vascular dementia or cognitive decline (Peters, Peters, Warner, Beckett, & Bulpitt, 2008). Such findings suggest that future research should examine premorbid individual differences in brain structure and function associated with EF, with longitudinal investigation of alcohol use and EF beginning in adolescence and into adulthood.

Diet

Obesity is a growing epidemic worldwide and is strongly associated with increased mortality risk, including cardiovascular, diabetes, and cancer mortality (e.g., Kelly, Yang, Chen, Reynolds, & He, 2008; Pi-Sunyer, 2009). Three recent systematic reviews have identified an association between obesity and EF, proposing that EF limitations may contribute to poor impulse control and, consequently, overeating (Fitzpatrick, Gilbert, & Serpell, 2013; Liang, Matheson, Kaye, & Boutelle, 2014; Vainik, Dagher, Dube, & Fellows, 2013). In addition to EF deficits placing individuals at risk for overeating and unhealthy weight, research also supports the notion that obesity itself has a deleterious impact on cognition. For example, there is some evidence that the association between obesity and EF increases with age (Stanek et al., 2013). A potential mechanism of this age-related increase is that obesity is a risk factor for cardiovascular disease and type 2 diabetes (see the discussion of these disorders in later sections), both of which lead to neurodegenerative changes in the brain (most notably the frontal subcortical structures and frontal white matter) that are associated with declines in EF (Sudo et al., 2012; Vincent & Hall, 2015). Importantly, weight loss is associated with improvements in cognition, including EF. A systematic review and meta-analysis investigating the association between intentional weight loss in overweight and obese adults identified a beneficial effect on EF and memory with weight loss, particularly among obese individuals (Siervo et al., 2011).

Overall, future longitudinal research is needed to investigate EF performance before and after the development of obesity. Furthermore, the association between EF and dietary choices across the life span should be investigated using well-validated tests of EF (Cao et al., 2015). For example, a systematic review investigating dietary consumption and EF in children and adolescents indicated that a healthy diet is positively associated with EF (Cohen, Gorski, Gruber, Kurdziel, & Rimm, 2016). In addition, it is possible that the individuals who are more vulnerable to becoming executive depleted by sleep deprivation then engage in overeating; this interpretation would be consistent with past research suggesting that overeating and inhibitory control are associated only under

Executive Functioning and Health

states of executive depletion, such as in the context of food restriction (Bartholdy, Dalton, O'Daly, Campbell, & Schmidt, 2016; Chapman, Benedict, Brooks, & Schioth, 2012).

Exercise and Physical Activity

Insufficient physical activity is associated with chronic diseases, including obesity, cardiovascular disease, cancer, osteoporosis, and type 2 diabetes; increasing physical activity reduces the risk of developing these diseases and is beneficial for cognitive functioning (e.g., Dishman et al., 2006; Etnier et al., 1997). In general, physical activity promotes cognitive functioning, including EF (Astill, Van der Heijden, Van Ijzendoorn, & Van Someren, 2012; Bherer, Erickson, & Liu-Ambrose, 2013; Chang & Hung, 2010; Chang, Labban, Gapin, & Etnier, 2012; Guiney & Machado, 2013; Guzman-Cortes, Villalva-Sanchez, & Bernal, 2015; Jedrziwski, Lee, & Trojanowski, 2007; Loprinzi, Herod, Cardinal, & Noakes, 2013; Middleton, Barnes, Lui, & Yaffe, 2010; Miller & Taylor-Piliae, 2014). A growing body of research indicates that physical exercise is associated with better cognition and EF in children and adolescents (Best, 2010; Carson et al., 2016; Chaddock, Pontifex, Hillman, & Kramer, 2011; Esteban-Cornejo, Tejero-Gonzalez, Sallis, & Veiga, 2015; Fedewa & Ahn, 2011; Khan & Hillman, 2014; Lees & Hopkins, 2013; Mura, Vellante, Nardi, Machado, & Carta, 2015; Tomporowski, Lambourne, & Okumura, 2011; Verburgh, Konings, Sherder, & Oosterlaan, 2014). Systematic reviews and meta-analyses provide clear evidence that physical activity, including aerobic exercise, tai chi, cognitive-motor training, low-intensity exercise, walking, and overall mobility, is associated with better cognition and EF across the adult life span (Carvalho, Rea, Parimon, & Cusack, 2014; Franco-Martin, Parra-Vidales, González-Palau, Bernate-Navarro, & Solis, 2013; Kelly et al., 2014; Lehert, Villaseca, Hogervorst, Maki, & Henderson, 2015; Ludyga, Gerber, Brand, Holsboer-Trachsler, & Pühse, 2016; Scherder et al., 2014; Tse, Wong, & Lee, 2015; Tseng, Gau, & Lou, 2011; Valenzuela & Sachdev, 2009; Wayne et al., 2014; Zhao, Tranovich, & Wright, 2014; Zheng, Liu, Li, Huang, Tao, & Chen, 2015). Furthermore, a meta-analysis of prospective studies indicated that physical activity protects against cognitive decline (Sofi et al., 2011).

Randomized controlled trials also provide evidence that exercise benefits EF. A meta-analytic review investigating aerobic exercise interventions and neurocognitive performance found a positive association between aerobic exercise training and EF (Smith et al., 2010). In addition, meta-analyses of physical activity interventions to improve cognitive performance in older adults found that physical activity promotes cognitive functioning and EF (Colcombe & Kramer, 2003; Hindin & Zelinski, 2012; Karr, Areshenkoff, Rast, & Garcia-Barrera, 2014; Kelly et al., 2014). Furthermore, research indicates that resistance exercise training has positive effects on older adult's cognitive functioning (Chang, Pan, Chen, Tsai, & Huang, 2012). Lastly, the combination of both cognitive and physical activity has been found to benefit EF in older adults (Bamidis et al., 2014; Barcelos et al., 2015; Lauenroth, Ioannidis, & Teichmann, 2016; Law, Barnett, Yau, & Gray, 2014). Similar findings have emerged in children and adolescents—cognitively engaging aerobic exercise interventions result in greater improvements in EF than non-cognitively engaging aerobic exercise (Best, 2010). Importantly, there is also

Executive Functioning and Health

evidence of reciprocal associations, such that better EF before beginning an exercise regimen is associated with greater adherence, an effect mediated by self-efficacy (McAuley et al., 2011).

Research indicates that physical activity is positively associated with EF through several mechanisms. Aerobic exercise is believed to benefit EF by promoting the up-regulation of brain-derived neurotrophic factor (BDNF), a protein that is involved in neuronal growth (Huang, Larsen, Ried-Larsen, Moller, & Andersen, 2014; Pang & Lu, 2004; Yang, Lin, Chuang, Bohr, & Mattson, 2014), and by increasing oxygen and nutrition to the brain (Hotting & Roder, 2013). In a study of older adults, BDNF mediated the effect of an exercise group on EF, specifically for participants aged 71 years and older (Leckie et al., 2014). In addition, research indicates that physical exercise promotes EF by increasing the neuronal efficiency, functional connectivity, and gray matter volume of brain regions subserving EF, with some evidence that physical exercise may also increase and protect white matter (Voelcker-Rehage & Niemann, 2013). Furthermore, coordinated exercise (movements that require coordination and balance) is posited to increase functional connectivity and gray matter volume in the medial-temporal and fronto-parietal areas (Voelcker-Rehage & Niemann, 2013). In addition, physical exercise benefits EF through neuroplasticity mechanisms and by protecting against the development of diseases that compromise cognitive functioning (Hotting & Roder, 2013). Importantly, exercise may improve weight loss outcomes by increasing EF (Joseph et al., 2011), another potential mechanism for protection against cognitive decline.

Overall, exercise is a promising health behavior for the promotion of cognitive abilities, particularly EF, given the brain's malleability during developmental periods of change (childhood through adolescence and old age) (Hotting & Roder, 2013; Ludyga, Gerber, Brand, Holsboer-Trachsler, & Puhse, 2016). However, systematic reviews have highlighted the lack of definitive evidence for the positive effect of exercise and physical activity on cognition for older and young to middle-aged adults (Cox et al., 2016; Snowden et al., 2011; van Uffelen, Chin A Paw, Hopman-Rock, & van Mechelen, 2008), and there is some evidence that strenuous exercise in young children could be detrimental to brain development and, consequently, EF (Howard et al., 2016). Thus, future research examining this association across the life span is needed to further investigate the benefits of physical activity on cognition, understand mechanisms, and refine exercise interventions for EF improvement (Etnier, Nowell, Landers, & Sibley, 2006; Lubans et al., 2016).

Executive Functioning and Stress

Association with psychosocial stress is another pathway by which EF may be linked to health and disease. In this section, recent research on EF and stress is reviewed using a previously proposed theoretical framework (Williams, Suchy, & Rau, 2009). In this

Executive Functioning and Health

framework, stress is conceptualized as a set of processes, including stress exposure, stress reactivity, stress recovery, and restoration. EF is hypothesized to both predict these processes, as well as to be influenced by them, in reciprocal feedback.

Stress Exposure

“Stress exposure” refers to events that may be minor daily “hassles” (e.g., conflict with a co-worker, traffic) or major life events (e.g., death of close family member, job loss). Stress exposure may also include internal “events” that involve recall of a past stressor (i.e., rumination) or anticipation of a potential future stressor (i.e., worry).

Prior reviews suggest that stress exposure prospectively predicts diminished EF, though much of the research has focused on underlying neuroanatomical changes. Animal research has demonstrated that stress exposure has detrimental effect on the structure and function of the prefrontal cortex (Arnsten, 2009; Shansky & Lipps, 2013), known to play an important role in some aspects of EF. The prior literature has shown that exposure to stress in childhood (e.g., maltreatment, poverty) affects the development of neural networks underlying EF (Blair, 2010), and associations between child maltreatment and EF has been extended into middle adulthood (Nikulina & Widom, 2013). Experimental manipulation of chronic stress in adults has also been shown to alter aspects of EF, as well as fMRI-assessed functional connectivity within the frontoparietal network; however, these effects appear to be reversible (Liston, McEwen, & Casey, 2009). There is also limited evidence that exposure to moderate, controllable stress may benefit performance on EF tasks (Henderson, Snyder, Gupta, & Banich, 2012). In a recent meta-analysis of experimental acute-stress-induction studies in healthy adults, there was evidence that acute stress diminished performance on tasks assessing the more cognitive aspects of EF that are important for reasoning, planning, and problem solving (e.g., working memory and cognitive flexibility), but it enhanced performance on EF tasks measuring behavioral inhibition (Shields, Sazma, & Yonelinas, 2016; see also Hermans, Henckens, Joëls, & Fernandez, 2014). Also relevant to associations with stress exposure, post-traumatic stress disorder (PTSD) is associated with neurocognitive deficits, including EF; importantly, however, a recent meta-analysis could not determine whether the observed cognitive functioning deficits resulted from trauma exposure, reflected preexisting vulnerability, or stemmed from an interaction between the two (Scott et al., 2015).

Stress exposure can also occur in the form of worry (i.e., anticipating a possible future stressor) and rumination (i.e., reliving a past stressor)—sometimes collectively termed “intrusive thoughts” or “perseverative cognition.” Indeed, a recent meta-analysis of 60 studies concluded that there was consistent evidence that perseverative cognition is associated with both cardiovascular and endocrine psychophysiology, suggesting pathways to adverse stress-related health outcomes (Ottaviani et al., 2016). With respect to EF, there is evidence that individuals with disorders characterized by worry and rumination, such as depression, have poorer EF (e.g., Gohier et al., 2009) and that the

Executive Functioning and Health

propensity to engage in negatively valenced repetitive thought is associated with poorer EF (Segerstrom, Roach, Evans, Schipper, & Darville, 2010). Moreover, EF training mitigates intrusive thinking (Bomyea & Amir, 2011). There is also evidence of reciprocal effects—rumination has been found to prospectively predict decreases in EF among adolescents (Connolly et al., 2014), and experimental worry induction is associated with diminished cognitive performance (Salters-Pedneault, Suvak, & Roemer, 2008), as is experimental rumination induction in dysphoric individuals (Philippot & Brutoux, 2008).

There is surprisingly little research on the prospective association between individual differences in EF and stress exposure. However, alterations in EF via traumatic brain injury are prospectively related to stress coping difficulty (e.g., Marschark, Richtsmeier, Richardson, Crovitz, & Henry, 2000), which may be associated with greater stress exposure over time. It is also the case that disorders characterized by poor EF, such as attention deficit hyperactivity disorder (ADHD), are associated with more daily stress (Hirvikoski et al., 2009).

With respect to mechanisms for the association between stress exposure and EF, the enhancement of behavioral inhibition is theoretically consistent with the long-held notion that high stress is associated with increased anxiety, which in turn is associated with increased error monitoring and behavioral inhibition (Ballard, 2001). The mechanisms for the deleterious impact of experimentally induced acute stress on EF are not fully understood—some effects may be mediated by stress hormones (e.g., cortisol), which are sometimes associated with decreases in dopamine in the prefrontal cortex (PFC), although this effect is typically seen in response to more chronic stress (Robbins & Arnsten, 2009). In the case of more severe stress exposure (e.g., trauma), a meta-analysis of neuroimaging studies generally supported neurocircuitry models of PTSD that posit disrupted fronto-limbic circuitry (Patel, Spreng, Shin, & Girard, 2012).

In summary, prior research on the association between EF and stress exposure has primarily examined the effects of acute stress on cognitive functioning. The strongest evidence is for the deleterious effects of stress on EF during childhood, when the PFC is still developing. Sound longitudinal research is lacking in adulthood, but experimental research suggests that negative effects of stress on EF are reversible, at least in healthy, cognitively intact adults. Moreover, at least in acute stress, there is some evidence for enhancement of some aspects of EF, such as response inhibition.

Stress Reactivity

The term “stress reactivity” typically refers to the immediate physiological response to a stressor (e.g., blood pressure, heart rate, heart rate variability, cortisol). Broadly defined, stress reactivity also encompasses cognitive (e.g., appraisal) and emotional responses to stressful events. Considerable evidence suggests that exaggerated cardiovascular

Executive Functioning and Health

reactivity to stress confers risk for the development of atherosclerosis, hypertension, and future myocardial infarction (Schwartz et al., 2003; Treiber et al., 2003).

Because EF relies, in part, on brain regions associated with modulation of the autonomic nervous system (Critchley, 2005), there should be reliable associations with stress reactivity. In particular, the parasympathetic nervous system (PNS) influences cardiac function via the vagus nerve, which has rich connections to the PFC. Importantly, PNS activation, indexed by high-frequency heart rate variability (HF-HRV) or respiratory sinus arrhythmia (RSA), is associated with prefrontal activity (Lane, Reiman, Ahern, & Thayer, 2001; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). That is, EF and aspects of PNS functioning are co-localized in these brain regions, suggesting that individual differences in EF may be reflected in stress-related autonomic physiology (e.g., blood pressure, HF-HRV) and affect. Indeed, there is substantial evidence for the involvement of the PFC in the regulation of both autonomic and hypothalamic-pituitary-adrenal (HPA) axis stress responses (McKlveen, Myers, & Herman, 2015).

Although recent models of self-regulation such as the Neurovisceral Integration Model (Thayer & Lane, 2009) and the Polyvagal Theory (Porges, 2011) highlight the functional association between the autonomic nervous system and the PFC, there has been surprisingly little focus on EF and stress reactivity. Hendrawan and colleagues (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012) found that performance on one measure of EF—Letter Fluency—was associated with less cortisol and skin-conductance reactivity (as well as less anxiety) to a laboratory psychosocial stressor in a small sample ($n = 32$). However, this finding may have been confounded by the fact that performance on Letter Fluency itself is highly susceptible to the effects of anxiety—thus, not surprisingly, anxiety-prone individuals will exhibit poorer performance.

Importantly, both exaggerated cardiovascular reactivity and dampened reactivity have been associated with poor health outcomes. In a longitudinal study, dampened blood pressure reactivity to stress in young adulthood was associated with poorer performance on an EF task (Stroop) in midlife in the Coronary Artery Risk Development in Young Adults (CARDIA) study (Yano et al., 2016). Studies that administer cortisol have found immediate deficits on cognitive aspects of EF, such as working memory, and enhanced performance on inhibition tasks, with delayed (one hour post-administration) enhancement in working memory (Shields, Bonner, & Moons, 2015). However, it is important to differentiate experimental studies that examine cortisol administration from studies of individual differences in reactivity. In general, cortisol administration studies find that the effects are reversed over time.

With respect to affective reactivity to stress, Compton and colleagues found that poorer performance on one EF-related task (emotional Stroop) predicted greater negative-affect responses to daily life stressors in an experience-sampling paradigm (Compton et al., 2011). Although preliminary, these findings suggest that individual differences in EF may confer vulnerability to greater emotional responses to stress in daily life.

Executive Functioning and Health

In summary, current models of self-regulation (and, thus, stress and emotion regulation) emphasize associations between the PFC, the anterior cingulate cortex, and the autonomic nervous system, suggesting that there should be robust associations between EF and stress reactivity. Although preliminary evidence supports this association, this is an area ripe for investigation. Individual differences in EF should confer risk for poor cardiovascular and emotional regulation in response to stress. Moreover, much of the research reviewed in the preceding “Stress Exposure” section implicates stress reactivity as the likely mechanism for prospective associations with poorer EF over time.

Stress Recovery

“Stress recovery” typically refers to how quickly individuals return to baseline levels of physiological arousal following a stressor in the context of laboratory stress paradigms. There is evidence that cardiovascular recovery from stressors is an important predictor of the development of cardiovascular disease (Brosschot, Gerin, & Thayer, 2006; Schwartz et al., 2003). To some extent, recovery has also been examined in ambulatory experience-sampling protocols. Expanding the original concept of this stress process, end-of-day arousal might also be considered “recovery”—the extent to which an individual is able to “wind down” after a stressful day. The construct of pre-sleep arousal captures this latter notion of stress recovery and predicts the development of chronic sleep disturbance (Fernandez-Mendoza et al., 2010).

Given the associations between EF and PNS functioning described heretofore, it is hypothesized that EF should predict physiological recovery from stressful events. Although evidence for this association is limited, preliminary research suggests that poorer EF is associated with longer heart-rate-recovery times from stress among older adults (Roiland, Lin, Phelan, & Chapman, 2015).

Restoration

During and after the experience of stress, restorative processes operate to repair cellular damage and to return an individual to baseline levels of physiological activity (Cacioppo & Berntson, 2007; Robles & Carroll, 2011). Sleep, wound healing, and DNA repair are examples of restorative processes that occur from organism-level to genome. In this review, the associations between sleep and EF are considered. Sleep problems in the United States are widespread, with approximately one-third of adults reporting at least one nocturnal insomnia symptom (Hossain & Shapiro, 2002; Morin & Jarrin, 2013), and 30% reporting average sleep duration of six hours or less (Luckhaupt, Tak, & Calvert, 2010). In general, insufficient duration and quality of sleep are associated with poorer cognitive functioning, including EF (Waters & Bucks, 2011). Research investigating the association between EF and sleep includes studies that assess naturally occurring sleep disruption (predominantly insomnia) and experimental sleep restriction and deprivation. A meta-analysis investigating cognitive performance of individuals with insomnia and

Executive Functioning and Health

normal sleepers found small to moderate cognitive impairments for individuals with insomnia (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012). In addition, meta-analyses have identified an association between sleep deprivation and diminished cognitive performance, including EF (Lim & Dinges, 2010; Philibert, 2005; Pilcher & Huffcutt, 1996; Wickens, Hutchins, Laux, & Sebok, 2015).

Poor cognition and EF due to sleep disruption have been demonstrated across the life span, including in children (de Freitas Araujo & de Almondes, 2014; Turnbull, Reid, & Morton, 2013). Importantly, a meta-analysis found sleep duration is positively associated with EF and negatively associated with behavioral problems among healthy school-aged children (Astill, Van der Heijden, Van Ijzendoorn, & Van Someren, 2012). Furthermore, a systematic review found a clear deficit in psychomotor vigilance among adolescents in the context of sleep deprivation, but no effects on cognition from partial sleep restriction (de Bruin, van Run, Staaks, & Meijer, 2016). Among older adults, systematic reviews and meta-analyses indicate that insomnia is a risk factor for dementia (de Almondes, Costa, Malloy-Diniz, & Diniz, 2016) and have provided evidence that self-reported short and long sleep durations (i.e., fewer than six and more than nine hours/night) are associated with poorer cognitive performance, including EF (Devore, Grodstein, & Schernhammer, 2016; Lo et al., 2016). Further research is needed to better understand the association between sleep and cognition among older adults, including the investigation of moderators such as comorbid medical and mental health conditions (Brewster, Varrasse, & Rowe, 2015).

Executive functions rely heavily on the PFC, which is characterized by a high metabolic rate during wakefulness (Maquet et al., 1990). Accordingly, the restorative benefits of sleep have been posited as essential for maintaining EF (Harrison & Horne, 2000; Harrison, Horne, & Rothwell, 2000; Horne, 1993; Horne, 2012). A meta-analysis found that acute sleep deprivation is associated with decreased activation of brain regions known to be involved in EF, including the PFC, and increased activation of other areas, indicating a complex activation and deactivation interaction effect from sleep loss (Ma, Dinges, Basner, & Rao, 2015). Importantly, better EF may confer resilience to the adverse effects of sleep disruption. In one study, individuals who demonstrated better baseline EF also demonstrated better psychomotor vigilance after 41 hours of sleep deprivation than individuals who performed poorly on baseline EF tasks (Killgore, Grugle, Reichardt, Killgore, & Balkin, 2009). Overall, research indicates that EF is impaired in the context of sleep disruption; however, individual differences in EF may buffer the deleterious effects of sleep disruption on cognitive functioning. Most prior research has focused on either sleep disorders (e.g., insomnia) or experimental sleep deprivation; thus, additional research is needed to examine EF and naturally occurring sleep disruption.

Executive Functioning and Chronic Illness

Executive Functioning and Health

In the next sections, evidence for associations between EF and prominent chronic illnesses are reviewed. In most instances, the research has focused on the effects of disease or medical treatment on cognitive functioning, including EF. However, these associations are thought to be reciprocal. EF may predict disease onset and severity, disease processes may degrade EF over time, and EF in turn may influence the progression of disease by affecting disease management, such as adherence to medical regimens.

Cardiovascular Disease

Heart disease is the leading cause of death in the United States and is associated with cognitive impairment, including EF dysfunction (e.g., Eggermont et al., 2012). In fact, longitudinal research with cognitively normal older adults shows that history of coronary heart disease predicts declines in global cognition, including EF, *prospectively* (Zheng et al., 2012).

Importantly, heart disease is often associated with other conditions that show association with EF deficits. Most notably, heart disease often occurs in the context of the metabolic syndrome, which is characterized by dyslipidemia, high blood pressure, dysregulated glucose homeostasis, abdominal obesity, and insulin resistance (Kassi, Pervanidou, Kaltsas, & Chrousos, 2011). Across a number of recent studies collectively examining thousands of patients, metabolic syndrome has been consistently associated with EF dysfunction (Dearborn et al., 2014; Exalto et al., 2015; Falkowski, Atchison, Debutte-Smith, Weiner, & O'Bryant, 2014; Rouch et al., 2014). Furthermore, metabolic syndrome *prospectively* predicts poorer EF performance (Reijmer et al., 2011; see also Siervo, Harrison, Jagger, Robinson, & Stephan, 2014, for a review of metabolic syndrome and general cognitive decline).

Regarding which comorbidities of heart disease represent the most likely mechanisms behind its association with EF dysfunction, hypertension is the most prominent culprit, as hypertension is known to contribute to damage in subcortical regions of the brain that make up the frontal-subcortical circuitry crucial for EF. In fact, it is well recognized that hypertension is associated with subcortical ischemic vascular dementia (SIVD), which in turn is characterized by EF deficits (Sudo et al., 2012).

Consistent with the notion that hypertension explains, at least in part, the association between heart disease and EF dysfunction, a recent study of individuals with metabolic syndrome found that hypertension was the strongest predictor of EF deficits (Levin et al., 2014). Whereas this is in contrast to a meta-analysis (of nine studies) that found no significant association between blood pressure and EF (Gifford et al., 2013), evidence for the deleterious impact of hypertension on cognition is considerable and should not be disregarded. Indeed, the use of hypertensive medications has been shown to stop the

Executive Functioning and Health

progression of cognitive decline in SIVD (Shah et al., 2009a,b; see also Gorelick et al., 2012).

Recent research indicates that maternal pregnancy-related hypertension is associated with offspring EF deficits (Wade & Jenkins, 2016). Research also suggests that hypertension is associated with EF impairment in children and adolescents (Cha, Patel, Hains, & Mahan, 2012; Kupferman, Lande, Adams, & Pavlakis, 2013). In summary, hypertension is associated with cognitive deficits, including EF, across the life span, with evidence that the deleterious effects of hypertension on EF can be transmitted in utero to offspring.

Although heart disease and its comorbidities undoubtedly contribute to cognitive decline, evidence also suggests that premorbid EF weaknesses place one at risk for the development of heart disease. Specifically, in a longitudinal study of older adults at risk for coronary heart disease, individuals who scored in the lowest third of EF tests in comparison to those who scored in the highest third had greater risk of coronary heart disease (1.85-fold) and stroke (1.51-fold) over time (Rostamian et al., 2015).

Cerebrovascular Disease

Cerebrovascular disease can be broadly divided into two categories: large vessel disease and small vessel disease. Large vessel disease, also known as cerebrovascular accident or stroke, involves either a blockage or a rupture of a major cerebral vessel. Although stroke is among the leading causes of death worldwide (Donnan, Fisher, Macleod, & Davis, 2008), the majority of stroke victims survive. The extent to which stroke survivors exhibit EF dysfunction depends in part on which blood vessels have been affected. Somewhat predictably, strokes that involve blood vessels that supply the frontal lobes (particularly the PFC and the anterior cingulate cortex), posterior parietal lobes, or the cerebellum are most likely to lead to EF deficits, given the role of these regions in EF (Suchy, 2015). However, outcomes can also depend on an array of additional individual differences, including the efficiency and distribution of vasculature, localization of function, and premorbid patterns of strengths and weaknesses in cognition (Chuang et al., 2011; De Silva, Silva, Amaratunga, Gunasekera, & Jayesekera, 2011; Wright et al., 2013). Thus, approximately 50–75% of stroke patients exhibit at least some EF limitations, regardless of lesion location (Chung, Pollock, Campbell, Durward, & Hagen, 2013; Pulsipher, Stricker, Sadek, & Haaland, 2013). The specific type of stroke-related EF dysfunction can vary greatly, depending on lesion location, and may include apathy, disinhibition, inappropriateness, or impaired reasoning and problem solving. Because strokes involving the frontal lobes can also be associated with hemiparesis or hemiplegia in the arm, the leg, or both, patients face considerable challenges in adjusting to their limitations in mobility. The extent to which these patients utilize rehabilitation services and successfully employ compensatory strategies (Hayes, Donnellan, & Stokes, 2015), as well as the

Executive Functioning and Health

extent to which they cope effectively with their stroke-related disability (Kegel, Dux, & Macko, 2014), depend in part on whether EF has been preserved.

In contrast to large vessel disease, small vessel disease usually does not present with a sudden onset of symptoms, but rather with a gradual decline in EF, along with declines in processing speed, visual-spatial abilities, and motor functions (Cosentino et al., 2004; Sudo et al., 2012). These gradual declines eventually culminate in SIVD (Haaland & Swanda, 2008). Unlike large vessel disease, which affects primarily the cerebral cortex, small vessel disease affects deep white matter and subcortical gray matter, although it can also be comorbid with Alzheimer's disease, and is thought to be a risk factor for the development of Alzheimer's dementia (Smith & Bondi, 2008).

Importantly, as documented by the Centers for Disease Control and Prevention (CDC; http://www.cdc.gov/stroke/risk_factors.htm), both small and large vessel disease can be comorbid with cardiovascular disease and diabetes, as well as other risk factors such as smoking, high cholesterol and high blood pressure, obesity, and a lack of exercise. As such, both diseases can be affected by lifestyle choices. As outlined before, lifestyle choices depend in no small part on individual differences in EF. Not surprisingly, then, preliminary findings show that low EF predicts future incidence of stroke (Oveisgharan & Hachinski, 2015).

Cancer

Cancer is a group of related diseases that share the features of abnormal cell growth and proliferation. The most common forms of cancer are breast, lung, prostate, colon, and bladder (American Cancer Society, 2016). The association between cancer and EF has been well documented. There are known negative effects of cancer treatment on cognitive functioning, particularly EF (e.g., Ferguson & Ahles, 2003; Stewart, Bielajew, Collins, Parkinson, & Tomiak, 2006)—a phenomenon now commonly termed “chemobrain.” However, poorer cognitive functioning has been identified in cancer patients even prior to beginning chemotherapy (e.g., Bender et al., 2015; Wefel, Lenzi, Theriault, Davis, & Meyers, 2004), in addition to changes during treatment, suggesting that at least some forms of cancer may have negative pathophysiological effects on brain functioning. In fact, neuroimaging research confirms the presence of both structural and functional brain abnormalities prior to treatment (Simó, Rifà-Ros, Rodriguez-Fornells, & Bruna, 2013). Moreover, cognitive changes can occur both during and after treatment, not limited to chemotherapy. Although long-term associations between EF and cancer or cancer treatment have been less studied, there is evidence of EF deficits in breast cancer survivors 10 or more years following treatment (Yamada, Denburg, Beglinger, & Schultz, 2010). In a meta-analysis of 14 fMRI studies, de Ruyter & Schagen (2013) report evidence of hypoactivation in prefrontal and parietal brain regions during EF tasks among breast cancer survivors 5–10 years following treatment, suggesting reduced neural functioning as a result of chemotherapy. On the other hand, long-term cognitive changes (Ahles & Saykin, 2007), as well as frontal cortex hypoactivation (Simó, Rifà-Ros, Rodriguez-

Executive Functioning and Health

Fornells, & Bruna, 2013), appear to occur only in a subset of chemotherapy-treated patients. The improved survival rates among cancer patients have made the understanding of long-term cognitive changes of paramount interest, particularly in survivors of childhood cancer, where there have been clearly documented EF deficits (e.g., Kahalley et al., 2010; Robinson et al., 2014).

With respect to mechanisms, in addition to the evidence of reduced prefrontal neural functioning described here, decreased frontal gray matter density has been found following chemotherapy (McDonald, Conroy, Smith, West, & Saykin, 2013), as well as decreased functional connectivity in the default mode network (Dumas et al., 2013) and decreased regional connectivity and global network organization and integration (Hadi Hosseini, Koovakkattu, & Kesler, 2012). Indeed, in a review of neuroimaging studies, Simo and colleagues (2013) reported evidence of functional and structural changes prior to treatment, as well as during chemotherapy. Beyond the direct effects of chemotherapy, lifestyle factors such as obesity, physical activity, and sleep disruption are also related to cognitive functioning in cancer survivors (Hartman, Marinac, Natarajan, & Patterson, 2015). Other potential mechanisms include inflammation due to the disease itself, drugs (beyond chemotherapy) used in treatment, hormone changes, nutritional deficiencies, and stress.

Importantly, many cancer survivors perceive deficits in EF, even as their performance on neuropsychological tests may be within the normal range. For many survivors, this may be a perception of a *relative* drop in cognitive functioning (i.e., from high to average functioning). Thus, the perception of change in functioning is accurate, even if within normal limits. This type of perception may result in self-regulatory changes that can affect quality of life and mood (e.g., withdrawing from daily activities to compensate for perceived cognitive problems). In summary, the association between cancer and cognitive functioning, EF included, is well documented but not yet fully understood.

Diabetes

Diabetes mellitus (DM) is an endocrine disorder characterized by the inability of the body to properly process blood glucose. This could result either from the inability of the pancreas to produce insulin (i.e., type 1 diabetes), or insulin resistance, in which cells in the body fail to appropriately respond to insulin (i.e., type 2 diabetes). Type 1 diabetes typically has a childhood onset. Onset of type 2 diabetes tends to occur in adulthood, though its prevalence in pediatric populations is on the rise in conjunction with increases in childhood obesity. Type 2 diabetes is a relatively common condition, afflicting about 8% of the world population; type 1 diabetes is considerably less common, representing only about 10% of all DM cases (CDC, 2014).

DM is relevant in the discussion of EF for three reasons. First, DM complications, including both hyperglycemia and iatrogenic hypoglycemia, are known to have a deleterious impact on the brain and on cognition. Specifically, hyperglycemia has been

Executive Functioning and Health

associated with micro- and macro-vascular changes (Ambler, Fairchild, Craig, & Cameron, 2006) that increase one's risk for stroke and for the development of SIVD (see the cerebrovascular disease section herein for more details, as well as discussion of the metabolic syndrome in the section on cardiovascular disease). Iatrogenic hypoglycemia, caused by excessive exogenous insulin intake, can lead to coma or death (Desrocher & Rovet, 2004), but it is also associated with poorer cognitive outcomes (Hannonen, Tupola, Ahonen, & Riikonen, 2003) and neuronal loss (Ferguson et al., 2003). In children with early onset type 1 diabetes, prolonged and repeated hypoglycemia can lead to the development of seizure disorder and intellectual disability (Menni et al., 2001). Given these effects, it is not surprising that EF is deleteriously affected by DM (for a comprehensive review, see Bade-White & Obrzut, 2009).

Second, successful management of DM requires that patients follow a strict regimen, which involves checking their blood glucose multiple times each day, calculating appropriate carbohydrate intake, administering insulin, and maintaining a proper diet and exercise routine. The ability to follow such a complex regimen on a daily basis requires considerable cognitive, behavioral, and emotional control. Thus, it has been hypothesized that EF is an important contributor to the maintenance of glycemic control, with recent research providing support for this notion (Duke, Raymond, & Harris, 2014; Suchy et al., 2016). Lastly, whereas the development of type 1 diabetes is unrelated to behavioral choice, it is a different matter with type 2 diabetes, wherein poor diet, obesity, and lack of exercise play an important contributing role to the onset and course of the disease.

Human Immunodeficiency Virus/Autoimmune Deficiency Syndrome

Although there has been progress in reducing the spread, and in effective treatment of, human immunodeficiency virus (HIV)/autoimmune deficiency syndrome (AIDS) infection, it remains a major health threat and cause of death, particularly among certain demographic groups. As of 2013, there were over 900,000 people living with HIV infection in the United States. Rates of infection are continuing to increase for those aged 25–29, and it is the eighth leading cause of death among those aged 25–34 (CDC, 2014). Whereas improvements in treatment have increased survival rates, the effects of HIV/AIDS on cognitive functioning, especially EF, have now been well documented. Categories of impairment include asymptomatic neurocognitive impairment (ANI), mild neurocognitive disorder (MND), and extensive cognitive decline referred to as HIV-associated dementia (HAD). A meta-analysis of 41 studies and over 8,000 participants indicated that EF, motor functioning, and information-processing speed show the greatest decline across disease progression from asymptomatic HIV status to AIDS (Reger, Welsh, Razani, Martin, & Boone, 2002). In a review of 56 studies comparing HIV+ and HIV- individuals, Dunbar and Brew (1997) found that a majority of studies (71%) found significantly poorer EF performance among individuals with advanced HIV infection. Both age and cognitive reserve moderate cognitive decline in HIV+ individuals: older age

Executive Functioning and Health

(Sacktor et al., 2010) and lower cognitive reserve (years of education, occupational attainment, and estimated premorbid intelligence; Stern, Silva, Chaisson, & Evans, 1996) are associated with greater decline.

Executive functioning prospectively affects HIV/AIDS progression by influencing medication adherence. Current treatment of HIV/AIDS includes a combination of drugs called highly active antiretroviral therapy (HAART), the efficacy of which depends on strict adherence to the regimen. In a review of 11 studies, Lovejoy and Suhr (2009) found that impaired neuropsychological functioning, including EF, was associated with poorer adherence to HAART. The association is reciprocal, with strong prospective associations between EF and medication adherence; higher adherence, in turn, predicts improvements in EF, as well as in processing speed and motor functioning (Ettenhofer, Foley, Castellon, & Hinkin, 2010). The latter findings are consistent with research documenting that HAART mitigates cognitive impairment (Sacktor et al., 2006).

Mechanisms for HIV-EF associations have been investigated at multiple levels. On a cellular level, it is known that HIV enters the brain early in infection, apparently relying on proteins expressed by mature monocytes (Williams, Anastos, Morgello, & Berman, 2015). Recent research suggests that HIV binds to neuronal proteins and blocks an essential autophagy process in the brain. Specifically, HIV appears to prevent efficient disposal of damaged proteins in the brain, resulting in neuronal death (Fields et al., 2016). On a more molar level, HIV is known to target primarily white matter and/or frontal-subcortical structures (Paul, Cohen, Navia, & Tashima, 2002) that are important for EF, resulting in a cognitive profile that is reminiscent of SIVD (see “Cerebrovascular Disease” section). The effects of HAART on lipid abnormalities represent additional potential mechanisms for the association with cognitive impairment (e.g., Cruse, Cysique, Markus, & Brew, 2012; Farrugia, Lucariello, & Coppola, 2009). To the extent that EF is associated with poor health behavior and stress regulation (see preceding sections), there may also be behavioral pathways to HIV progression.

Of note, different subtypes, or clades, of HIV are present around the world, and there is emerging evidence suggesting that some clades are associated with greater cognitive impairment than others (Witten, Thomas, Westgarth-Taylor, & Joska, 2015). This picture is further complicated by the fact that individuals with lower socioeconomic status are at a greater risk for cognitive dysfunction in association with HIV, probably due to other health disparities (Arentoft et al., 2015). Consequently, such individuals are at an increased risk for poorer treatment adherence and concomitant cognitive decline. With increased globalization and migration across countries and continents, international collaborative research is needed to improve our understanding of risk for cognitive impairment among different patient groups.

Chronic Pain

Executive Functioning and Health

Approximately one-third of adults in the United States experience chronic pain, most commonly attributed to low back pain, followed by osteoarthritis (Johannes et al., 2010). There is evidence for EF deficits across a range of chronic pain syndromes, suggesting that the association is not disease-specific (see Moriarty, McGuire, & Finn, 2011, for review). A recent meta-analysis of the magnitude of the association between chronic pain and cognitive test performance found consistent small-to-moderate effects for test performance on a variety of EF tests (Rathbone et al., 2016). Among older adults, those with more severe pain have poorer EF (van der Leeuw et al., 2016). There is preliminary evidence that EF prospectively predicts the occurrence of chronic pain. For example, patients with poorer EF pre-surgery are more likely to report pain a year later (Attal et al., 2014).

With respect to mechanisms linking chronic pain and EF, Moriarty and colleagues (2011) describe the overlap between the neuroanatomical and neurochemical underpinnings of pain and cognition, noting multi-level evidence of pain-related cognitive changes ranging from behavioral to molecular. Their proposed model integrates prior theoretical frameworks, including (1) competing limited resources, consistent with prior self-regulation models (e.g., Nes, Roach, & Segerstrom, 2009); (2) neuroplasticity; and (3) dysregulated neurochemistry. Such a broad-based multi-level framework captures reciprocal feed-forward associations between EF and pain over time. However, as a cautionary note, chronic pain can in some cases represent a psychiatric, rather than physical, condition, and in such cases cognitive dysfunction itself may need to be explained by other (i.e., behavioral and psychiatric) mechanisms (Suesse, Wong, Stamper, Carpenter, & Scott, 2015).

Multiple Sclerosis

Multiple sclerosis (MS) is a progressive neurodegenerative condition marked by a chronic autoimmune process that results in the degeneration of myelin in the central nervous system. The age of onset is typically between the ages of 20 and 40 years, with MS representing a second most common cause of disability among young and middle-aged adults (Adelman, Rane, & Villa, 2013;). Although motor, sensory, and autoimmune symptoms are often the most noticeable, cognitive limitations are common, affecting more than 50% of patients (Amato et al., 2010; Chelune, Scott, & Pinkston, 2008). Like stroke, MS is characterized by heterogeneity of lesion locations and lesion burden, resulting in heterogeneity of cognitive presentations. That said, approximately 80% of patients with detectable cognitive limitations have been reported to exhibit impairment in EF (García, Plasencia, Benito, Gómez, & Marcos, 2009). Interestingly, rather than lesion location, it is the lesion burden and whole brain atrophy that are the best predictors of the severity of EF deficits (Parmenter et al., 2007). This association may be explained by the fact that white matter integrity in MS patients relates more to processing speed than to EF (Genova, DeLuca, Chiaravalloti, & Wylie, 2013). In fact, it has been suggested that the presumed EF deficits in this population can fully be explained by deficiencies in speed of processing (Leavitt et al., 2014). However, the debate about the unique contributions of speed of processing, working memory, and EF to the cognitive presentation of MS patients has been ongoing for some time (Drew, Starkey, & Isler, 2009; Macniven et al., 2008), and it is unlikely that any one study will settle this debate in the foreseeable future.

MS is frequently associated with psychiatric symptoms, most notably depression, with a lifetime prevalence three times that of the general population (Nicholl, Lincoln, Francis, & Stephan, 2001; Voss et al., 2002). Importantly, depression in this population cannot be explained by physical disability alone, as rates are higher among patients with MS compared to patients suffering from other chronic illnesses (Chwastiak et al., 2002). Regarding bidirectional relationships between EF and illness, clearly MS diagnosis cannot be attributed to any EF-related lifestyle choices. However, once a person is diagnosed with EF, depressive symptoms appear to mediate the relationship between EF dysfunction and *complaints* of cognitive symptoms (Feinstein, 2006; Randolph, Arnett, & Freske, 2004). Given the well-documented association between EF and depression (Douglas & Porter, 2009; Murrough, Iacoviello, Neumeister, Charney, & Iosifescu, 2011), depression and stress (Siegrist, 2008; Von Werne Baes, de Carvalho Tofoli, Martins, & Juruena, 2012), and EF and stress as reviewed earlier in this chapter, one should not underestimate the potential contribution of EF to depressive and stress symptoms in this population. Importantly, the potentially deleterious impact of stress on inflammatory processes associated with MS further highlights the importance of continued research in this area.

Summary and Future Directions

Across the domains reviewed—health behavior, stress processes, and chronic illness—significant associations with EF have been demonstrated. Substance use and exercise have robust associations with EF; there is also growing evidence that individual differences in EF affect eating behavior and the development of obesity. Additionally, longitudinal research in healthy individuals would be particularly informative in teasing apart the direction of effects over time. Moreover, individual differences in EF should be given greater consideration in health behavior intervention research. Additional supports to compensate for poorer EF might improve outcomes in smoking cessation, weight loss, and alcohol and drug treatment.

With respect to stress processes, most research thus far has focused on the effects of acute stress on EF; although aspects of EF appear to be (temporarily) negatively affected, there is evidence that response inhibition may improve during acute stress. However, severe chronic stress, particularly in childhood, may have more permanent effects on EF. It is surprising that there has been little focus on EF as a vulnerability factor (vs. outcome) in stress regulation. Future research should consider individual differences in EF as a predictor of stress exposure—both daily hassles (e.g., time management, interpersonal conflict) and cognitive stressors (i.e., worry and rumination). Further examination of EF and physiological stress reactivity and recovery is needed, as is investigation of affective responses to stress. Reciprocal relations between EF and stress-related sleep disruption also warrant further study. This type of research would inform early intervention in sleep disorders. For example, to the extent that individual differences in EF predict pre-sleep cognitive and physiological arousal (a risk factor for the development of insomnia), precise targets for intervention could be identified.

Virtually all the chronic illnesses reviewed evidence associations with EF that include direct pathophysiological effects of disease and effects of medical treatment. Importantly, there is evidence that poorer EF is related to medical regimen adherence—a key consideration in efforts to improve behavioral intervention in chronic illness. Not surprisingly, the majority of research on EF and chronic illness has examined EF/cognitive functioning as the outcome (vs. predictor). Nevertheless, it is hypothesized that across health domains, there is a reciprocal, feed-forward association with EF. In a rare example of longitudinal associations between EF and future mortality risk, Batty, Deary, and Zaninotto (2016) examined the prospective association between cognitive functioning (a composite of EF, memory, and processing speed) and mortality risk in different disease categories in the large sample (>11,000) English Longitudinal Study of Aging. Cognitive performance predicted greater odds of death by cancer, cardiovascular disease, respiratory illness, and other causes, controlling for a host of other risk factors (including health behavior). Importantly, statistical examination of the potential for reverse causality did not change the hazard ratios. These findings suggest that individual differences in cognitive functioning, including EF, are an independent risk factor for poor health

Executive Functioning and Health

outcomes. The findings also suggest that the association is not disease-specific and is not fully explained by associations with poor health behavior (though, as described here, that is also a viable pathway).

Findings from the prospective examination of EF and mortality suggest that there is still much to be done in understanding both the direction of effects as well as the mechanisms for those effects in EF and health associations. Longitudinal studies of individuals who are healthy at baseline would be particularly informative. Furthermore, with some notable exceptions, there is still a tendency to utilize single tests of EF (vs. composites of multiple tests), leading to less reliable measurement, as well as less sensitive tests of EF relationships (since not all aspects of this multi-component construct are captured in any single test). In addition, few studies control for lower-order processes when examining EF-health associations, leading to ambiguity in the interpretation of findings.

To conclude, EF has emerged as a critical construct in the maintenance of health and the development of disease. EF predicts the onset of health-risk behavior and is, in turn, negatively affected by such behavior. It is a critical construct in our understanding of stress regulation, as well as the mechanisms by which childhood trauma predicts future adverse mental and physical health outcomes. There is increasing evidence that (1) individual differences in EF prospectively predict a broad array of chronic illnesses and resultant mortality risk, (2) many chronic diseases result in poorer EF, and (3) adherence to medical regimens is more difficult in the face of such cognitive changes. Collectively, these findings suggest that continued study of EF will inform our understanding of mechanisms underlying health and disease, as well as improve primary, secondary, and tertiary interventions.

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