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Physical Activity and Individual Cognitive Function Parameters: Unique Exercise-Induced Mechanisms

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Abstract

Cognitive-based parameters, including memory, reasoning, concentration, and planning are key cognitions essential for optimal daily function. Emerging research demonstrates that physical activity is individually associated with each of these cognitions. Our mechanistic understanding of these relationships, however, is less understood. We comprehensively discuss the unique exercise-induced mechanisms for various individual cognitive functions. This narrative review highlights the emerging research evaluating the effects of physical activity on memory, reasoning, concentration, and planning. Herein, we discuss the unique and shared mechanistic pathways to shed light on these interrelationships. Exercise uniquely influences individual cognitive functions.

Keywords: cognition; executive function; exercise; physical activity

öz

Bellek, akıl yürütme, konsantrasyon ve planlama dahil, bilişsel temelli parametreler, optimal günlük işlev için gerekli olan temel bilişlerdir. Gelişmekte olan araştırmalar, fiziksel aktivitenin bu bilişlerin her biri ile ayrı ayrı ilişkilendirildiğini göstermektedir. Bununla birlikte, bu ilişkilerin mekanizmasına dair bilgimiz oldukça azdır. Bu çalışmada çeşitli bilişsel işlevler için özgül egzersiz kaynaklı mekanizmalar kapsamlı olarak tartışılmaktadır. Bu derlemede, fiziksel aktivitenin bellek, akıl yürütme, konsantrasyon ve planlama üzerindeki etkilerini değerlendiren yeni araştırmalar vurgulanmaktadır. Bu ilişkiselliğe ışık tutmak için paylaşılan ve özgül mekanizma yollarını tartışıyoruz. Egzersiz bireysel bilişsel işlevleri benzersiz şekilde etkiler.

Anahtar Kelimeler: biliş; egzersiz; fiziksel aktivite; yürütücü işlev

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INTRODUCTION

There are several unique parameters (e.g., memory, reasoning, concentration and planning) underlying the global domain of cognitive functioning. Exercise may, perhaps, play an influential role on these components of cognition, via diverse physiological and psychological mechanisms, as well as complex interactions among shared biological pathways (Diederich et al., 2017; Dougherty et al., 2017; Jochem et al., 2017; Macedonia & Repetto, 2017; Stillman et al., 2016). A comprehensive understanding of this mechanistic framework will direct future research towards an evaluation of the plausibility for a differential and bidirectional effect to exist between specific exercise intensities and modalities on cognitive performance.

Although the potential for exercise to exert positive benefits on cognitive function has been touted in the literature, recent evidence suggests baseline cognition may drive the subsequent augmentation of post-exercise cognitive performance. Lower psychological resources prior to the exercise stimulus may predispose certain individuals to respond more positively to exercise, via a previously hypothesized ceiling effect for exercise-induced cognitive enhancement (Crush & Loprinzi, 2017). Furthermore, conflicting results have suggested that high-intensity exercise may reduce acute cognitive performance on certain tasks (Kamijo et al., 2004; Kashihara, Maruyama, Murota, & Nakahara, 2009), but may be a viable strategy to utilize for improved performance on alternative tasks, and within specific populations (Baker et al., 2010; Frith, Sng, & Loprinzi, 2017). More work is needed to clarify these gradations, but in general, exercise (both acute and chronic) has been consistently associated with widespread, favorable, cognitive performances (Kashihara et al., 2009; Tomporowski, 2003; Tsukamoto et al., 2016).

Memory, reasoning, concentration, and planning are instrumental parameters known to moderate cognitive capacity via an interplay of highly specialized neural connections. Exercise has been shown to exert a differential effect on each of these variables, however, less attention has been focused on determining the extent to which exercise selection may alter the potency of these effects. Namely, exercise modality and intensity must be examined for the field of modern exercise psychology to progress towards an individually relevant, yet generalizable approach to enhancing cognitive performance. Individual responses to exercise participation, as well as individual differences in memory and cognitive functioning play a central role in the utility for a potential dose-response relationship to exist for exercise programs to appreciably benefit the psychological and physical mechanisms discussed herein. Ongoing research should continue to explore these individual distinctions across various populations, exploring the complex relationship between various physical activity dosages across multimodal programs, as well as the timecourse of exercise participation effects for populations whom differ by age, weight status, psychological pathology, etc. The specific evaluation of the nuances of exercise participation for special populations is beyond the scope of this review, as we aim to provide an informative primer regarding select exercise-induced mechanisms on multiple aspects of cognitive function. Although not exhaustive, we present a comprehensive discussion of relevant topics in this area, which will serve to facilitate our understanding of the complexities inherent in the dualistic mind-body relationship, and to motivate ideas for population-delimited empirical investigations. Thus, the purpose of this conceptual narrative review is to discuss four critical areas of cognitive function (i. e., memory, reasoning, concentration and planning), and describe the rationale for exercise to mediate cognition via a facilitation or upregulation of the biological pathways involved.

MEMORY

Description of Memory

Memory can be conceptualized as the consolidation of previously experienced stimuli. Following an acquisition of new information, the learning process by which content is translated into knowledge that can be activated by later recall, has been examined (Abdel-Majid et al., 1998). Mental representations of memory are activated by populations of neurons, known as an engram trace (Poo et al., 2016). There are many unique engram cell pathways which subserve various memory-associated parameters, suggesting the mechanisms underlying memory formation and stability are functionally connected across multiple neural structures (Loprinzi, Edwards, & Frith, 2017). Memory is an umbrella term for an array of distinct components related to recall. Areas associated with memory include, among others, classical conditioning, emotional, habitual, short-and long-term, spatial, and recognition memory (Squire, 1992). This review will provide a brief discussion of the underlying biological mechanisms and importance of selected subcomponents of memory (Eichenbaum, 2017).

Influence of Memory on Mental Performance

Aside from genetic and environmental influences, one of the most proximal underlying mechanisms of accurate learning and memory is housed in hippocampal regions (Abrous & Wojtowicz, 2015). The neurotrophin brain-derived neurotrophic factor (BDNF) is thought to regulate long-term potentiation (LTP), potentially influencing memory processes (Panja & Bramham, 2014). The induction of LTP is reliant on several cellular cascades that impact the magnitude of consolidation. The formation of new memories, as well as the subsequent consolidation and retrieval, is influenced by the neuronal strength of BDNF encoding (Rodriguez-Ortiz & Bermudez-Rattoni, 2017). Research suggests the mere activation of the hippocampus,

Table 1: Potential mechanisms through which exercise may facilitate individual cognitive parameters. Arrows indicate direct cognitive mechanisms, while dashed lines are representative of plausible moderating effects of exercise on distinct cognitive parameters.

Mental Construct		**Physiological Description**		**Proposed Link between Exercise and Cognition**
1. Memory		***Cognitive Mechanisms***		***Potential Exercise-Associated Mechanisms***
Classical Conditioning	→	Activated NMDA receptors in the amygdala, facilitate LTP	----	Stress induced modifications of neuronal circuitry within hippocampus, promote BDNF action, neural plasticity, augment neurogenesis, and contribute to structural morphologies conducive to memory
Spatial Memory	→	Activated NMDA receptors in the Hippocampus and subiculum facilitate LTP	----	Calcium influx due to NMDA receptor activation, as well as a reduced threshold for synaptic stimulation required to induce LTP.
Recognition Memory	→	The hippocampus and cortex mediate interactions between involuntary and voluntary responses during initial encoding.	----	Increased cortical brain volume, as well as serum and entorhinal cortex BDNF.
Long-term Memory	→	Consolidation is driven by protein expression and new relationships between synapses in the hippocampus	----	Late-phase LTP phosphorylates CREB, which acts directly to maximize BDNF.
Working Memory	→	Facilitated by complex neural interactions between existing synapses in the hippocampus, prefrontal cortex, and the frontal cortex, which are funneled through the subiculum	----	Neurogenesis via increased cerebral blood flow and neural plasticity in the hippocampus accelerates BDNF and LTP proliferation and serotonin expression
2. Concentration	→	The basal ganglia and anterior cingulate cortex (ACC) may prioritize relevant informational cues in accordance with executive control processes which facilitate focus	----	Basal ganglia regulation of dopamine response, as well as cognitive control and prepotent response inhibition via reduced amplitude of error-relate negativity (ERN) event potentials catalyzed in the ACC
3. Reasoning	→	Complex integration of emotion, attention, language and working memory regions of the brain, namely the medial temporal lobe, medial prefrontal cortex, in addition to various cortical areas	----	Appropriate arousal and emotional control preserves prefrontal and frontal regions via regulation of monoamines such as serotonin, dopamine, epinephrine, and norepinephrine known to impact arousal, and concomitant hormonal modulation involving the hypothalamic-pituitary axis
4. Planning	→	Evolutionary augmentation of prefrontal cortex mass promotes activation of critical frontal regions, neural plasticity and synaptic interaction	----	LTP may play an indirect role in the exercise-planning relationship, as BDNF expression is pronounced during LTP, following the phosphorylation of CREB, moderating BDNF response

namely the left hemisphere, may induce heightened retrieval, independent of BDNF action (Hariri et al., 2003). Although, it has been well established that BDNF modulates individual variability in hippocampal memory resilience, neuronal survival, and synaptic plasticity (Hariri et al., 2003; Leal, Afonso, Salazar, & Duarte, 2015).

Classical Conditioning and Its Underlying Mechanisms

Research in classical conditioning aims to habituate a subject to a repeated stimulus over time, such that, ultimately, a conditioned physiological or psychological response will manifest to an unconditioned stimulus (McSweeney & Bierley, 1984). For example, if a child is repeatedly bullied on the school bus, they may learn to associate school buses with a negative, fear-inducing experience. When the child's family moves to a new school district, the child may continue to feel fear and aversion when riding the bus, as the bus has become a conditioned stimulus that is associated with aversive memories. Previous work examines fear conditioning and the profound influence stressors may exert on memory capacity. Learned fear is a constituent of classical conditioning, and has been significantly associated with memory of pleasant and unpleasant experiences (Hamann, Ely, Grafton, & Kilts, 1999). Classical conditioning is mediated by neuronal activation of the amygdala, which serves as the critical juncture between unconditioned and conditioned information (Clugnet, LeDoux, & Morrison, 1990). The link between conditioned and unconditioned stimuli is thought to arise from the lateral nucleus of the amygdala. Neural activation in this region is critically augmented by the nature of the conditioned stimulus preceding the unconditioned behavioral outcome (Repa et al., 2001). Impaired activity of the amygdala has been shown to markedly diminish fear conditioned responses (Cousens & Otto, 1998; Goosens, 2001). Although the amygdala may not play a vital role in the storage of long-term knowledge, this region may govern consolidation properties in other brain regions, especially during inhibitory avoidance (McGaugh, 2002; McGaugh, Cahill, & Roozendaal, 1996; Wilensky, Schafe, & LeDoux, 2000). Long-term potentiation (LTP) is a key mechanism by which fear memory may be mediated. Amygdular LTP is similar to hippocampal LTP in that access of previously experienced fear memories is often catalyzed by NMDA receptor activation and subsequent protein activity. Specifically, consolidation of many types of memory involves the cellular response to cAMP response element binding protein (CREB). Action potentials released by the amygdala, coupled with excitatory postsynaptic potentials released following exposure to the conditioned stimulus, induces a cellular influx of calcium through NMDA receptors, which initially promotes short-term fear memory, with continued calcium entry facilitating long-term storage (Blair, 2001).

Role Through Which Exercise May Influence Classical Conditioning

The mild stress associated with physical activity may elevate classically conditioned memory. Acute and long-term stressors have been shown to exert an inhibitory effect on hippocampal memory capacity, while concomitantly facilitating amygdular-dependent memory via LTP cascades (Vyas, Mitra, Shankaranarayana Rao, & Chattarji, 2002). This understanding provides plausibility for a multidirectional component to align with classical memory development. Although one pathway may be downregulated, exercise may uniquely assist the upregulation of another (i. e., stress-associated amygdular learning) (Sapolsky, 2003). Importantly, if the amygdala is temporally stimulated, transient potentiation may progress to protein-dependent LTP. Although, exercise elicits physiological responses similar to acute stressors (Zschucke, Renneberg, Dimeo, Wustenberg, & Strohle, 2015), voluntary, or self-directed exercise participation may be a *eustressor* capable of inducing favorable psychological anxiolytic effects, which are thought to exert a downstream influence on the enhancement of motivation and vigilance, positive mood and cognitive functioning. Specifically, the hypothalamus-pituitary axis (HPA) is known to play a critical role in the synthesis and release of cortisol, which is a primary stress hormone. Exercise has been linked with an attenuation in pathological HPA axis reactivity to acute and chronic stress-related threats to homeostasis via an adaptive regulation of negative feedback mechanisms governing excess cortisol release (Martikainen et al., 2013; Rimmele et al., 2009). Another possible explanation is the protective effect of physical activity on hippocampal structures, which may override stress-associated inhibition (Greenwood, Strong, Foley, & Fleshner, 2009). Exercise may alter the neuronal circuitry of the hippocampus, promote BDNF action, neural plasticity, augment neurogenesis, and contribute to structural morphologies that increase learning and memory (Cotman & Engesser-Cesar, 2002; Eadie, Redila, & Christie, 2005; Ekstrand, Hellsten, & Tingstrom, 2008; Neeper, Gomez-Pinilla, Choi, & Cotman, 1996). Pavlovian fear conditioning involves hippocampal and extra-hippocampal structures. Specifically, physical activity increases contextual learning, even under sub-optimal acquisition conditions, whereas the amygdala is linked to the consolidation of newly acquired information from fear-conditioning scenarios (Rattiner, Davis, & Ressler, 2005).

Spatial Memory and Its Underlying Mechanisms

Spatial memory involves constructing a cognitive reference point for environmental stimuli, which may be contextually associated with concurrently presented cues (Bannerman et al., 2014). Hippocampus, subiculum, and cortical regions are inextricably involved in areas related to explicit skills, such as spatial memory (Squire, 1992). Hippocampal pyramidal cells are believed to drive encoding of specific object locations (O'Keefe, 1979). The hippocampus is responsible for connecting spatial information introduced in temporally-mediated sequences (Teng & Squire, 1999). The retrosplenial cortex is located between hippocampal, thalamic, and cortical limbic structures implicated in episodic navigation and contextual representation features critical for optimal spatial memory (Miller, Vedder, Law, & Smith, 2014). Another structural component of hippocampal circuitry, the subiculum primarily delivers information to cortical structures, and has been linked to LTP facilitation. Lesions in the subiculum may produce decrements in spatial memory integration (Morris, Schenk, Tweedie, & Jarrard, 1990). Recent evidence indicates that short-term administration of insulin-like growth factor-1 (IGF-1), a peptide which regulates brain activity across the lifespan (Fernandez & Torres-Aleman, 2012; Pardo et al., 2016), counteracts the deleterious influence of aging on hippocampal integrity in rat models (Pardo et al., 2016). This neuroprotective effect is proposed to target proliferation of new neurons in the dentate gyrus, as well as concomitant astrocyte branching, with longer-term exposure to IGF-1 speculated to exert a more robust effect (Pardo et al., 2016). Further, NMDA antagonists, such as AP5, are also expected to impede spatial memory via reduced LTP induction (Collingridge, Kehl, & McLennan, 1983; Morris, Anderson, Lynch, & Baudry, 1986; Morris et al., 1990). The importance of NMDA action will be detailed later in this review, but it is important to note that deficits in spatial skills resulting from NMDA blockage may be attenuated by task familiarity. Interestingly, spatial learning may not require potentiation for consolidation. Effective learning may be achieved when the context can be associated with previous experiences (Otnaess, Brun, Moser, & Moser, 1999).

Role Through Which Exercise May Influence Spatial Memory

Exercise is believed to aid plasticity of neural structures, specifically contributing to hippocampal neurogenesis, LTP, and BDNF, which have been shown to protect and advance spatial memory (Bjornebekk, Mathe, & Brene, 2005; O'Callaghan, Ohle, & Kelly, 2007; Vaynman, Ying, & Gomez-Pinilla, 2003, 2004). A growing body of literature has touted the beneficial effects of environmental enrichment on spatial memory. Exercise may provide a viable form of enrichment, as well as reduce the threshold for synaptic stimulation required to induce LTP, which is reliant on several cellular cascades that impact the magnitude of consolidation (Farmer et al., 2004). LTP is supported by synaptic transmissions, primarily within the hippocampus, regulated by NMDA receptors, which open ligand and voltage channels enacting an influx of postsynaptic calcium (Ascher & Nowak, 1986; Bliss & Collingridge, 1993; Collingridge et al., 1983). NMDA receptors governing synaptic plasticity are thought to undergo age-dependent changes from early life through adulthood, which may exert intensified effects on the efficiency of sensory memory (Heynen & Bear, 2001). This further reinforces the temporal component of potentiation, as well as supplies rationale for previously experienced events to not only impact plasticity, but also partially drive LTP activity. Distinct synapses may additionally activate an LTP response via sufficient voltage-regulated calcium loading alone (Bliss & Collingridge, 1993; Grover & Teyler, 1990). Synaptic thresholds vary among neural structures, but after reaching the critical threshold of activation, the resultant response is an accelerated, all-or-none potentiation (Petersen, Malenka, Nicoll, & Hopfield, 1998). The subsequent insertion of AMPA membrane receptors is one proposed interpretation of fast-acting LTP response. CaMKII is a major protein constituent of neuronal mass, and is found in both pre-and postsynaptic vesicles, although activity may be centralized in postsynaptic sites, where calcium concentration is expected to exert cellular effects linked to neural plasticity. CaMKII has been referred to as a "memory molecule" (Shen, Slack, & Tosh, 2000) with an important caveat being that the intensity of kinase expression is associated with potentiation effects leading to enhancement of spatial memory and fear conditioning (Bejar, Yasuda, Krugers, Hood, & Mayford, 2002; Shen et al., 2000). Exercise has been shown to facilitate neuroplasticity via increased AMPA receptor subunits, GluR1 and GluR2/3. Additional receptor subunit proliferation implies that neuronal modulation may be expressly modifiable with regular physical activity (Real, Ferreira, Hernandes, Britto, & Pires, 2010).

Recognition Memory and Its Underlying Mechanisms

Recognition memory has been operationally defined as an automatic response that occurs during the initial encoding phase of information processing (Tulving & Thomson, 1973). Although widely disputed, and believed to emerge subconsciously, recognition memory may be associated with intelligence. In fact, recognition memory may be displayed along a continuum between involuntary and effortful reactions (Fagan, 1984). The visual paired-comparison task assesses recognition of new information, compared with previously experienced stimuli. Amnesic individuals with hippocampal lesions have been shown to incur no detriment to performance, provided the new object or picture is presented immediately following the familiar cue. This finding reinforces the argument that hippocampal memory consolidation may be time-sensitive (McKee & Squire, 1993).

Role Through Which Exercise May Influence Recognition Memory

Whiteman et al. examined the potential moderating effect of exercise on recognition memory utilizing brain-imaging measurements to directly observe morphological changes in brain structure and serum BDNF (Whiteman, Young, Budson, Stern, & Schon, 2016). Their findings support the rationale for increased cortical thickness in the entorhinal cortex to contribute to memory performance (Diamond, Ingham, Johnson, Bennett, & Rosenzweig, 1976), and neuronal survival. BDNF levels are known to be highly concentrated in the entorhinal cortex, which could be a potential explanation for augmented recognition memory. Another conclusion may be the effect of exercise-driven angiogenesis on increased brain volume (Palmer, Willhoite, & Gage, 2000).

Long-term Memory and Its Underlying Mechanisms

Long-term memory is suggested to rely on the coherence of multiple neural structures in facilitation of a stable memory trace. Neuronal and molecular modulation subserving long-term memory storage is accomplished by neurogenesis and enhancement of synaptic plasticity over time, a process requiring increased metabolic energy and efficient network communication (Suzuki et al., 2011). The neocortex is responsible for accessing remote information (Squire, 1992). While the hippocampus is equipped to store information immediately, synaptic alterations within the neocortex occur over an extended time frame. Support for this idea is evidenced by previous research demonstrating that lesions of the hippocampus may not impede recollection of distant memories; however, the degree of impairment is dependent upon the severity of damage. Further, more recent evidence shows hippocampal involvement in long-term memory retrieval, specifically via increased activation of neural firing during protocols using neural imaging measurements (Nadel, Samsonovich, Ryan, & Moscovitch, 2000). Long-term memory is also influenced by protein expression, event-related genetic activation, as well as new relationships between synapses.

Role Through Which Exercise May Influence Long-Term Memory

Research has shown that both aerobic and anaerobic exercise may improve executive functioning linked to long term memory performance (Potter et al., 2005; Winter et al., 2007). Long-term memory is essentially a reservoir containing an indeterminable amount of successfully consolidated information, which may be readily accessed and is resistant to dissolution, despite lengthy delays. Muscular and neural physiological adaptations to skeletal muscle after exercise training are thought to modulate long-term brain functioning and stable memory adaptations (Lista & Sorrentino, 2010; Sofi et al., 2011), perhaps synergistically. A primary mechanism by which exercise may positively influence long-term memory is associated with preservation of neocortex volume across the lifespan. Higher fitness levels are suggested to attenuate gray matter loss, and prevent age-related atrophy of prefrontal and hippocampal regions essential to long-term memory performance (Erickson, Leckie, & Weinstein, 2014). Additionally, late-phase LTP (L-LTP) stimulation catalyzes an increase in post-synaptic calcium concentration (Zagaar, Dao, Levine, Alhaider, & Alkadhi, 2013). Short-term memory and long-term memory biology may be explained, in part, by analogues in LTP durability. LTP is uniquely characterized by distinct phases of activity, much like the complex distribution of human memory. The first, transient phase persists for only a couple of hours, and is independent of protein activity characteristic of long-term memory. However, the second phase of LTP lasts several hours to several weeks, contingent upon de novo protein synthesis. Further, CaMK IV, a distinct kinase associated with L-LTP, phosphorylates CREB (Barco, Alarcon, & Kandel, 2002; Bramham & Messaoudi, 2005; Kandel, 2001), which acts directly to maximize BDNF expression (Barco et al., 2002; Bramham & Messaoudi, 2005; Kandel, 2001). Previous work has confirmed the residual cognition effects of BDNF, which may remain increased weeks following physical activity (Berchtold, Castello, & Cotman, 2010; Berchtold, Chinn, Chou, Kesslak, & Cotman, 2005). In addition, habitual exercise may also increase the size of the hippocampus via augmented cell proliferation, and/or attenuate age-associated loss of hippocampal volume, which correlates with serum BDNF production (Erickson et al., 2011; Redila & Christie, 2006). Preservation, or increased size, of dendritic complexity within hippocampus may elicit preventative effects on long-term memory capacity, particularly during later adulthood (Erickson et al., 2011; Redila & Christie, 2006).

Working Memory and Its Underlying Mechanisms

Working memory is considered to have a limited capacity; only retaining memory over the short term, ensuring this information is available until it is needed. While working memory is stored, it is sustained by the continued firing of delay neurons in, for example, the prefrontal cortex (Baeg et al., 2003; Khan & Muly, 2011). Neurotransmitter (e.g., glutamate) release and neural oscillations in the brain have been used to evaluate working memory (Khan & Muly, 2011). The amplitude of gamma and theta oscillations (Howard et al., 2003; Mainy et al., 2007; Meltzer et al., 2008; van Vugt, Schulze-Bonhage, Litt, Brandt, & Kahana, 2010) are associated with working memory (Raghavachari et al., 2001). Acute exercise is associated with an increase in neurotransmitter release and neuronal firing (Khan & Muly, 2011).

Goal-directed actions require the capacity to utilize mental constructs of previous experiences, temporally ordering spatial and non-spatial input to formulate appropriate behaviors. Conceptually, working memory facilitates goal-oriented action via the dynamic interplay between manipulations of semantic, visual, spatial, and higher-order central executive processes (D'Esposito & Postle, 2015). Working memory predominantly involves neural interaction between the hippocampus, prefrontal cortex, and the frontal cortex, which are funneled through the subiculum, an area believed to facilitate the processing of information sent to various brain regions. Strengthening existing connections between synapses may be vital to the storage of memory (Cajal, 1909). A multitude of active neurons may be available for information processing, however only the synapse appropriate to the received information will be stimulated to strengthen the efficiency of neural firing for relevant memory processes (Hebb, 1949). Stronger neural networks may be the key to exceptional memories. Beyond fronto-hippocampal regions, the connections within the anterior cingulate cortex (ACC) are suggested to coordinate with various structures linking emotion, motor function, and sensory processing of information (Bush, Luu, & Posner, 2000). The ACC is also proposed to direct working memory efficiency and augment cognitive control, while reducing conflicting neural activations that distract attention from online task-demands (Colcombe et al., 2004; Hillman, Erickson, & Kramer, 2008). There are a litany of underlying biological mechanisms that offer plausibility for enhanced synaptic integrity, although a critical mechanism of interest is LTP (Bliss & Lomo, 1973; Lomo, 1966). Outside of the hippocampus, which is considered the locus of LTP activity, cortical structures involved in working memory were among the first brain regions linked to LTP and memory (Doyere, Burette, Negro, & Laroche, 1993).

Role Through Which Exercise May Influence Working Memory

A potential mechanism by which physical activity may foster working memory is the proposed beneficial effect of exercise on tasks requiring higher degrees of executive control (Hogervorst, Riedel, Jeukendrup, & Jolles, 1996; Lichtman & Poser, 1983; Tomporowski PD, 2005). Executive control is comprised of memory processes including choice, planning, and coordination (Meyer & Kieras, 1997), which govern appropriate behavioral responses via inhibitory control over irrelevant or incorrect stimuli (Hogervorst et al., 1996; Lichtman & Poser, 1983; Tomporowski PD, 2005). Executive control may enhance the efficiency of working memory in areas related to active storage, maintenance, and management of temporally dependent information (Kane & Engle, 2002; Postle, 2006). Research on the association between working memory and exercise has demonstrated improvements in task performance in both abbreviated reaction time latency and higher response accuracy following acute bouts, and chronic physical activity engagement (Hogervorst et al., 1996; Lichtman & Poser, 1983; Tomporowski, 2003; Tomporowski PD, 2005). Exercise increases cerebral blood flow (Querido & Sheel, 2007) and neural plasticity in the hippocampus (Friedman & Goldman-Rakic, 1988), acting to accelerate proliferation of BDNF, LTP, and serotonin expression, molecules known to promote neurogenesis (Hillman et al., 2008; van Praag, Kempermann, & Gage, 1999; Vaynman & Gomez-Pinilla, 2005).

CONCENTRATION

Concentration is a broad construct encompassing interest and attentional regulation. Concentration is largely modifiable, as a function of pursued behavioral interest, or the purposeful maintenance of goal-directed focus (Posner & Petersen, 1990). Underlying success in sustainable concentration are physiological arousal regulation, psychological alertness, and delegation of attentional processes (Posner & Petersen, 1990). The brain structures involved in concentration include cortical regions, the thalamus, and the basal ganglia (Fan, McCandliss, Sommer, Raz, & Posner, 2002; Posner & Rothbart, 2007). The anterior cingulate cortex (ACC) may play an especially important role in prioritizing relevant informational cues in accordance with executive control process which facilitate focus (Fan et al., 2002; Posner & Rothbart, 2007; Seeley et al., 2007). Counterintuitive to attentional preservation, mind-wandering may be mitigated by alternative cortical structures, including the posterior cingulate cortex, and the para-hippocampal gyrus (Buckner, Andrews-Hanna, & Schacter, 2008; Mason et al., 2007). Stimuli requiring longer attentional control often recruit additional mental processes, such as monitoring and updating, and inhibiting prepotent responses to achieve target goals.

Role through which Exercise May Influence Concentration

Certain modalities and/or intensities of exercise require targeted focus on physical behaviors and the execution of coordinated motor movements. As heart rate and respiration increase in line with physical exertion, so must physical awareness of fatigue, effort, and movement (Mothes, 2014). Exercisers must constantly self-regulate their level of focus throughout the exercise bout. Effortful concentration must be applied for physical activity to continue safely and efficaciously. Further, rumination or anxiety regarding disconnected contexts, may be less attended to while participating in physical activity (de Bruin, van der Zwan, & Bogels, 2016). Researchers have linked exercise in this capacity to dispositional mindfulness, as attentional control and cognitive executive functioning may be ubiquitously employed during engagement in these scenarios. Similarly, mental focus is necessary when undertaking mindfulness-based techniques (de Bruin et al., 2016).

Inhibition of prepotent responses may require an individual to override their natural instincts and situational affective state. Children with ADHD have exhibited a reduced ability to maintain cognitive control when presented with competing stimuli (Nigg, 2001), which may be partly explained by the activity of dopamine transporters and receptors expressed in the basal ganglia and prefrontal cortex. Aerobic capacity has also been linked to the function of dopaminergic basal ganglia in the human brain (Aron et al., 2007). Basal ganglia-regulated dopamine response is thought to influence cognitive control performance and response inhibition (Aron et al., 2007). Physical activity may also reduce the amplitude of error-relate negativity (ERN), which is an event potential catalyzed in the ACC (Hillman et al., 2008). Decreased ERNs are associated with increased self-regulatory behaviors. Thus, cognitive performance may be aided via ACC-mediated decrements in mental conflict-processing of diverse environmental cues concurrently competing for attention (Colcombe et al., 2004). Therefore, physical activity may improve regulation and efficiency of context-specific action potentials linked to concentration.

REASONING

Reasoning and its Underlying Mechanisms

Reasoning is a complex navigational framework for making inferential decisions relative to the environment (Zeithamova, Schlichting, & Preston, 2012). Affective states, or moods, may be a primary determinant of incidental reasoning, whereas affective traits, or personality constructs are more likely to respond to cues based on a fundamentally stable frame of mind (Blanchette & Richards, 2010). Rational thinking may be viewed as an abstraction of cognition and is distinctly individual. In situations requiring correct reasoning responses, individual differences designate the degree of cognitive separation of objective and subjective appraisal to form rational thoughts. The seminal work of Lefford in 1946 pioneered the classic research assumption that emotion could deleteriously alter the logicality and psychological efficiency of proper reasoning (Lefford, 1946). However, recent work has found emotion to exert a positive effect when the emotional construct is intrinsically valued (Blanchette, 2006). Nevertheless, an unequivocal consensus has yet to be posited for the positive or negative affect of emotion on reasoning capabilities. One potential explanation for oppositional results may be that incidental states divert attentional allocation from the task demands, while integral traits draw focus inward, allowing individuals to formulate personally meaningful conclusions (Blanchette & Richards, 2010; Laney, Campbell, Heuer, & Reisberg, 2004). Reasoning is suggested to integrate attention, language and working memory regions of the brain, namely the medial temporal lobe, medial prefrontal cortex, in addition to various cortical areas (Goel, Gold, Kapur, & Houle, 1998). However, a frontal language comprehension center, Broca's area, has also been implicated in reasoning research, and is thought to induce encoding via personally valuable internal dialogue that must be created prior to resultant behavior (Goel et al., 1998).

Role through which Exercise May Influence Reasoning

Exercise, when intrinsically valued, has the ability to positively influence acute affect, as well as augment self-efficacy over the long-term (Rhodes & Kates, 2015). Interestingly, modifications in exercise-specific affect may occur before, during, or after the exercise bout. Further, intrinsic regulation facilitates mood and trait-associated expectations for future physical activity engagement (Rhodes & Kates, 2015). As affect has been linked to reasoning capacity, the rationale for exercise to act in parallel is justified. Depressed individuals are often more likely to experience deficits in executive functioning and reasoning (Steffens et al., 2006). A mechanism by which exercise may attenuate the detrimental impact of depressive symptomology is proliferation of monamines such as serotonin, dopamine, epinephrine, and norepinephrine, known to influence affect and arousal (Craft & Perna, 2004; Loprinzi, Herod, Cardinal, & Noakes, 2013). Physical activity may also enhance coping efficacy in highly arousing situations through hormonal modulation (Hotting, Schickert, Kaiser, Roder, & Schmidt-Kassow, 2016), especially in reference to the hypothalamic-pituitary axis (McEwen, 2002). Evidence suggests exercise may be profoundly beneficial in attenuating adverse psychological stress responses and could enable the proposed enhancement effects of moderate arousal on memory processes to take precedence (Gagnon & Wagner, 2016; Sandi & Pinelo-Nava, 2007). Exercise may also function as an appropriately arousing stimuli, affecting goal-directed reasoning (Nguyen-Michel et al., 2006). Fitness plays an instrumental role in preserving functionality of prefrontal and frontal neural regions associated with reasoning (Colcombe & Kramer, 2003), as well as may moderate verbal memory function (Etnier, 1997). Lastly, as previously mentioned, components involved in working memory consolidation may be inherent to reasoning function. Acute bouts of exercise have been indicated to influence working memory performance via serum BDNF increases (Lee et al., 2014).

PLANNING

Planning and its Underlying Mechanisms

The human brain has tripled in size over the past 2.5 million years, with much of this mass increase attributable to adaptations of the prefrontal cortex, which has experienced an approximated growth rate two times larger than the rest of the brain (Deacon, 1997). An important constituent of the prefrontal cortex, Brodmann's area 10, is shown to increase allometrically, relative to the brain at large. This is notable, as Brodmann's area 10 is definitively involved in planning and coordination of future action (Carpenter MB, 1983; Schoenemann, 2006). Active working memory in the right prefrontal-parietal brain regions are believed to store intentional thought and preemptively assess problem states related to this fundamental planning paradigm. The left posterior dorsolateral prefrontal cortex has been shown in fMRI research to be instrumental for the selection of appropriate sub-goals throughout the planning process. The perceptual sub-goaling strategy has been suggested to explain the temporal sequence of goal inception, which must be determined along with the number of goals required for eventual task success. Specific examination of the time course involved across the formation of task-relevant goals may be imperative in understanding the construction and solidification of conscious plans. Frontal activation of the prefrontal cortex has been implicated in such planning behaviors. Frontal parietal and subcortical structures may be coactivated, although not all activated structures may command equal recruitment under variable planning demands. Individual variability may manifest during the development, manipulation, and processing of goals preceding planned action. The nature of the cognitive demand is thought to determine the active region utilized for efficacious planning. Superior planning regarding the sub-goaling strategy is perhaps most successful when there is a functional dissociation between recruited frontal regions and the left inferior frontal gyrus (Bachevalier, Alvarado, & Malkova, 1999). Planning complexity may also delegate neural activation in the brain to the polar frontal cortex, although the evidence is equivocal (Abraham, Dragunow, & Tate, 1991; Abraham, Masonparker, Williams, & Dragunow, 1995). The adaptive control of thought-rational cognitive modeling is one plausible theory to explain individual differences in the manipulation of goal-oriented cognition. Self-generated representations, cognitive branching, and the preservation of a hierarchy of goal-significance must also be taken into consideration when describing planning distinctions on the individual level, where modern research is lacking.

Role through which Exercise May Influence Planning

Planning is a mental construct, couched under the umbrella term of executive functioning, which comprises a host of goal-directed behaviors (Miyake et al., 2000). The prefrontal cortex, along with additional cortical structures implicated in conscious planning, have been associated with BDNF expression, which has been shown to exhibit exercise-dependent concentrations that facilitate learning and memory (Berchtold et al., 2010; Berchtold et al., 2005). BDNF is linked to increased cognition and memory performance and is also elevated with exercise participation (Berchtold et al., 2010; Berchtold et al., 2005). In addition, LTP may play an indirect role in the exercise-planning relationship, as BDNF expression is pronounced in late-phase LTP (Alhaider, Aleisa, Tran, & Alkadhi, 2011), following the phosphorylation of CREB, which moderates the BDNF response, and mediates afferent synaptic signaling cascades, neuronal differentiation, and cellular survival (Finkbeiner et al., 1997). LTP has been previously identified as a biological determinant of synaptic plasticity, and is associative, durable, and selective (Bliss & Collingridge, 1993). LTP is intimately associated with the hippocampus, and substrates shown to initiate or inhibit hippocampal function, have been shown to exert similar effects on LTP proficiency (Diamond, Dunwiddie, & Rose, 1988; Diamond et al., 1976; Greenstein, Pavlides, & Winson, 1988; Larson, Wong, & Lynch, 1986; Morris et al., 1986; Rose & Dunwiddie, 1986). The effects of acute and long term exercise on BDNF and LTP processes have been detailed previously, with one of the most prolific impacts of exercise-associated frontal activation on behaviors involved in executive functioning (Farmer et al., 2004; Kane & Engle, 2002; Tomporowski, 2003; Tomporowski PD, 2005; Vaynman & Gomez-Pinilla, 2005; Vaynman et al., 2003, 2004).

CONCLUSION

In conclusion, and as demonstrated in this review, there are unique mechanisms that influence each of the evaluated cognitions, and exercise appears to activate each of these pathways. Exercise may alter neural networks within the hippocampus, promote BDNF and plasticity, and facilitate neurogenesis to enhance learning and memory consolidation (Cotman & Engesser-Cesar, 2002; Eadie et al., 2005; Ekstrand et al., 2008; Neeper et al., 1996). The activity of dopamine transporters and receptors expressed in the basal ganglia and prefrontal cortex has been linked to the executive functioning involving concentration (Aron et al., 2007). Basal ganglia-regulated dopamine response is thought to influence cognitive control performance and response inhibition (Aron et al., 2007). LTP may also play an indirect role in the exercise-planning relationship, as BDNF expression is expressed in line with LTP (Alhaider et al., 2011). Emotion has been linked to reasoning capacity and may mitigate depressive symptomology shown to contribute to deficits in executive functioning and reasoning (Steffens et al., 2006). Specifically, the release of monamines such as serotonin, dopamine, epinephrine, and norepinephrine are evidenced to markedly influence affect (Craft & Perna, 2004; Loprinzi et al., 2013). In addition to these unique mechanistic pathways for the different cognitions, there are some shared pathways, such as LTP. As demonstrated above, LTP appears to play an important role in memory, reasoning, and planning, and encouragingly, there is experimental evidence to support the role of exercise in modulating LTP. Thus, through unique and shared mechanistic pathways, exercise may help to serve as a gateway behavior to help facilitate numerous cognitive functions.

Future research should elucidate the influence of temporal placement of the exercise bout, relative to learning and memory processes. For example, identifying if physical activity before, during, or after learning exerts a differential effect would be instrumental in the operationalization of practical recommendations for memory consolidation. Similarly, experimental studies should assess the extent to which exercise intensity may differentially influence BDNF expression, LTP proliferation, and mediation of affect and arousal in the context of unique cognitive function parameters.

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