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An exploration of exercise-induced cognitive enhancement and transfer effects to dietary self-control

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ABSTRACT

The primary objective of this study was to examine the effects of aerobic exercise on executive function, specifically inhibitory control, and the transfer to self-control in the dietary domain. It was hypothesized that exercise would enhance inhibitory control, and that this enhancement would facilitate self-control in a laboratory taste test paradigm. Using a crossover design, 51 participants completed counterbalanced sessions of both moderate exercise (experimental condition) and minimal effort walking (control condition) using a treadmill; the intersession interval was 7 days. Prior to each exercise bout participants completed a Stroop task. Following each bout participants completed a second Stoop task, as well as a bogus taste test involving three appetitive calorie dense snack foods and two control foods; the amount of each food type consumed during the taste test was covertly measured. Results revealed that moderate exercise significantly improved performance on the Stroop task, and also reduced food consumption during the taste test for appetitive calorie dense snack foods; there was no exercise effect on control food consumption. Exercise-induced gains in Stroop performance mediated the effects of moderate exercise on appetitive snack food consumption. Together these findings provide evidence that a bout of a moderate aerobic exercise can enhance inhibitory control, and support for cross-domain transfer effects to dietary self-control. © 2016 Elsevier Inc. All rights reserved.

1. Introduction

A substantial proportion of the global chronic disease burden may be attributed to individual level risk behaviors (e.g., overconsumption of calorie dense foods, physical inactivity, smoking). These behaviors are recognized as being detrimental to one's health, yet they are relatively common in the general population. The maintenance of healthy dietary habits in particular may be essential to prevent the onset of numerous chronic diseases, as the overconsumption of calorie dense foods is associated with an increased risk for excessive adiposity and subsequently obesity, type 2 diabetes, coronary artery disease, and cerebrovascular disease (Bailey, Sullivan, Kirk, & Donnelly, 2010; Du et al., 2009; Misra, Singhal, & Khurana, 2010; Rosenheck, 2008). However, calorie dense foods are somewhat hard to resist, partially due to an evolved preference for foods that are high in fat and sugar, which makes the consumption of such foods the natural default

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to resist the allure of appetitive food stimuli, and otherwise exert dietary self-restraint to limit the consumption of such foods. Such self-regulatory abilities may partially depend on neurobiologicallybased cognitive abilities such as executive functions (Hall, 2016; Vainik, Dagher, Dubé, & Fellows, 2013). Executive function (EF) is an overarching term that encapsulates a variety of higher level cognitive processes implicated in the reflective top-down (i.e., non-stimulus driven) control over behavior, thought, and emotion (Baddeley, 1996; Miyake & Friedman, 2012; Miyake et al., 2000). Conceptually, the unitary EF construct can be decomposed into three interconnected, but dissociable hasic subcomponent: working memory mental flowibil

Drewnowski, Kurth, Holden-Wiltse, & Saari, 1992). As such, indi-

vidual health and wellbeing is in part dependent on our capacity

(Drewnowski, 1997; Drewnowski & Greenwood,

sociable, basic subcomponents: working memory, mental flexibility, and inhibition. Of these subcomponents, inhibition is (from a statistical standpoint) a pure manifestation of executive abilities, as it perfectly correlates (1.0) with the unitary EF factor; working memory and task switching consist of a mixture of this fundamental EF construct and unique, but unrelated processes. A definitive aspect of behavioral inhibition is the capacity to temporarily override reflexive (i.e., prepotent) responses to stimuli, and behave in a





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goal-directed manner. In the context of dietary behaviors, the capacity to override visceral (autonomic) responses to appetitive food stimuli is crucial to effectively regulate the consumption of calorie dense foods; therefore, inhibition may be more pertinent to dietary self-regulation than the other EF facets.

Indeed, prior observational studies have confirmed that stronger (i.e., better) executive control abilities are associated with the reduced consumption of calorie dense snack foods, and that this effect is evident when food consumption is assessed via selfreport or objectively, and is independent of important demographic confounders (Allan, Johnston, & Campbell, 2010; Allan, Johnston, & Campbell, 2011; Allom & Mullan, 2014; Hall, 2012; Hall, Elias, & Crossley, 2006; Hall & Fong, 2013; Kakoschke, Kemps, & Tiggemann, 2015a; Kakoschke, Kemps, & Tiggemann, 2015b: Riggs, Chou, Spruiit-Metz, & Pentz, 2010: Riggs, Spruiit-Metz, Sakuma, Chou, & Pentz, 2010: Vainik et al., 2013). Likewise, carefully controlled behavioral experiments have identified specific situational contexts in which such effects emerge; specifically, when eating environments include cues that suggest consumption is normative or expected, EF has a more pronounced effect on eating behavior (Hall, Lowe, & Vincent, 2014; Hall et al., 2014). Given the significance of EFs in explaining successful dietary selfrestraint, particularly in facilitating contexts, it is important to understand how such abilities can be optimized.

The dorsolateral prefrontal cortex (DLPFC) is considered to be the central neural substrate underlying inhibitory control, and EFs more broadly (Floden, Vallesi, & Stuss, 2011; Miller & Cohen, 2001; Smolker, Depue, Reineberg, Orr, & Banich, 2014; Stuss, 2011). In addition, an accumulating body of evidence has demonstrated that the differential operation of the DLPFC is implicated in the successful, self-initiated control of dietary behaviors, such as the cognitive regulation of hedonic food cravings (Kober et al., 2010), effective dietary self-control (Hare, Camerer, & Rangel, 2009), and suppressing the motivation to eat (Yoshikawa, Tanaka, Ishii, Fujimoto, & Watanabe, 2014). These findings suggest that cortical activity in the DLPFC in response to appetitive food stimuli may predict individual differences in dietary self-control. such that attenuated activity may predispose individuals to selfregulatory failures. Therefore, optimizing activity in the DLPFC could theoretically improve dietary self-restraint, potentially via enhanced inhibitory control abilities. Consistent with this notion, at least six laboratory studies involving cortical stimulation methods have demonstrated that increasing activity in the DLPFC reduces both subjective snack food cravings following cue exposure, as well as objectively measured consumption (Fregni et al., 2008; Goldman et al., 2011; Jauch-chara et al., 2014; Lapenta, Sierve, de Macedo, Fregni, & Boggio, 2014; Uher et al., 2005). Further, Lowe, Hall, and Staines (2014) reported that temporary disruptions in the executive control network-via inhibitory transcranial magnetic stimulation to the left DLPFC-resulted in enhanced cravings for, and consumption of, appetitive calorie dense snack foods; this effect was mediated by changes in inhibitory control abilities. Together, these findings support the contention that the integrity of the executive control network can causally influence dietary self-restraint.

Given the causal significance of inhibitory control in relation to dietary behaviors, there is continuing interest in EF enhancement strategies. One promising avenue is physical exercise, which has been shown to enhance cognitive function both broadly, and specifically in relation to EFs (Chang, Labban, Gapin, & Etnier, 2012; Lambourne & Tomporowski, 2010; McMorris & Hale, 2012; McMorris, Sproule, Turner, & Hale, 2011; Smith et al., 2010). At least 20 min of acute aerobic exercise is necessary to observe a positive effect on cognitive functions, with the largest effects being observed 11–20 min following the exercise bout (Chang et al., 2012); these effects are present for up to 52 min after exercise cessation (Joyce, Graydon, McMorris, & Davranche, 2009). In addition, there is some evidence that the beneficial effects of physical exercise may vary depending on initial EF strength, such that individuals with poor EFs are the most amendable to exercise induced improvements in EF (Drollette et al., 2014).

Although the mechanisms underlying exercise-induced improvements in EF are not entirely understood, prior research has suggested several non-exclusive possibilities (Erickson, Hillman, & Kramer, 2015), including (1) increased cerebral blood flow to the prefrontal cortex (Endo et al., 2013; Giles et al., 2014; Rooks, Thom, McCully, & Dishman, 2010; Yanagisawa et al., 2010), (2) increased expression of brain derived neurotropic factor (BDNF; Ferris, Williams, & Shen, 2007; Tsai et al., 2014), or (3) exercise-induced brain glycogen supercompensation (Matsui et al., 2012). While it is evident that further research is needed to develop a convincing model outlining the mechanisms underlying the exercise-induced effects on EF, it remains clear that aerobic exercise, undertaken both acutely and over an extended period of time, can be used to enhance EFs by optimizing the brain structures and associated neurochemistry supporting them.

It is possible that acute exercise may also improve dietary selfcontrol via this same route. Yet, the direct application of exerciseinduced EF enhancement to the problem of dietary self-restraint remained largely unexplored. Only one prior study has examined EF as a mediator of exercise effects on eating behavior, but the results were ambiguous due to methodological limitations (Lowe, Hall, Vincent, & Luu, 2014). Several prior studies have documented improvements in dietary self-restraint following acute exercise (i.e., reduced food intake; Balaguera-Cortes, Wallman, Fairchild, & Guelfi, 2011; Guelfi, Donges, & Duffield, 2013; Hagobian et al., 2013; Martins, Morgan, & Truby, 2008; Schubert, Desbrow, Sabapathy, & Leveritt, 2013; Sim, Wallman, Fairchild, & Guelfi, 2014; Thivel et al., 2012), but without considering (or measuring) EF as a mediator. At present, the mechanisms underlying effect of exercise on dietary behavior have remained obscure, and exercise-induced improvements in EF have not been previously considered as a potential mediating pathway.

The present study used within-subjects crossover design to test the effects of a bout of acute aerobic exercise on dietary selfcontrol via exercise-induced improvements in inhibitory control (i.e., direct cross-domain "transfer" effect). The primary hypotheses are: (1) that acute aerobic exercise will enhance inhibitory control, and (2) acute exercise will facilitate dietary self-control in a subsequent laboratory taste test, and (3) that the former effect will mediate the latter. Secondary hypotheses are that the exerciseinduced enhancements in inhibitory control will be strongest for those with relatively weak baseline EF, and that the transfer effects to dietary self-control will be specific to calorie-dense snack food, and will not generalize to less calorie dense control foods.

2. Methods

2.1. Participants

A sample of 51 female undergraduate students, aged 17–28 (M = 19.08; SD = 1.736), participated in this study. Participants were predominately Caucasian (64%) with a normal body mass index (BMI; M = 21.81; SD = 3.071). All participants were recruited from undergraduate psychology courses using an online participant recruitment system. In exchange for their participation, participants received two course credits. Similar to the procedure reported in Lowe, Hall, and Staines (2014) and Lowe, Hall, Vincent, and Luu (2014), participants were preselected based on self-reported food cravings for milk chocolate and potato chips. Participants were excluded if they had been clinically diagnosed

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