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Etiology and Pathophysiology

The neurocognitive connection between physical activity and eating behaviour

R. J. Joseph[1], M. Alonso-Alonso[2], D. S. Bond[3], A. Pascual-Leone[2,4] and G. L. Blackburn[1]

[1]Center for the Study of Nutrition Medicine, Beth Israel Deaconess Medical Center, Boston, MA, USA; [2]Berenson Allen Center for Noninvasive Brain Stimulation, Beth Israel Deaconess Medical Center, Boston, MA, USA; [3]Weight Control and Diabetes Research Center, The Miriam Hospital, Providence, RI, USA; [4]Institut Universitari de Neurorehabilitació Guttmann, Universidad Autónoma de Barcelona, Badalona, Spain

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Address for correspondence: Dr GL Blackburn, Beth Israel Deaconess Medical Center, Department of Surgery, Center for the Study of Nutrition Medicine, Feldberg 880, East Campus, 330 Brookline Avenue, Boston, MA 02215, USA. E-mail: gblackbu@bidmc.harvard.edu

Summary

As obesity rates increase worldwide, healthcare providers require methods to instill the lifestyle behaviours necessary for sustainable weight loss. Designing effective weight-loss interventions requires an understanding of how these behaviours are elicited, how they relate to each other and whether they are supported by common neurocognitive mechanisms. This may provide valuable insights to optimize existing interventions and develop novel approaches to weight control. Researchers have begun to investigate the neurocognitive underpinnings of eating behaviour and the impact of physical activity on cognition and the brain. This review attempts to bring these somewhat disparate, yet interrelated lines of literature together in order to examine a hypothesis that eating behaviour and physical activity share a common neurocognitive link. The link pertains to executive functions, which rely on brain circuits located in the prefrontal cortex. These advanced cognitive processes are of limited capacity and undergo relentless strain in the current obesogenic environment. The increased demand on these neurocognitive resources as well as their overuse and/or impairment may facilitate impulses to over-eat, contributing to weight gain and obesity. This impulsive eating drive may be counteracted by physical activity due to its enhancement of neurocognitive resources for executive functions and goal-oriented behaviour. By enhancing the resources that facilitate 'top-down' inhibitory control, increased physical activity may help compensate and suppress the hedonic drive to over-eat. Understanding how physical activity and eating behaviours interact on a neurocognitive level may help to maintain a healthy lifestyle in an obesogenic environment.

Keywords: Eating behaviour, neurocognition, obesity, physical activity.

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Introduction

Despite increased awareness, public health campaigns and novel therapies, obesity rates remain at alarming levels in the USA (1) and are increasing worldwide (2). Lifestyle-based weight-loss interventions consistently produce a 7–10% decline in initial body weight (3), but the condition is refractory, and poor retention rates (4,5) and relapse of weight gain (6) are common. The limited long-term success of such interventions may be attributable in part to a failure to fully understand and exploit the connection between the two therapeutic cornerstones, physical activity and eating behaviours. Indeed, while much emphasis is placed on modifying both behaviours to promote an immediate energy deficit, there has been less emphasis on understanding how a change in one of these behaviours may support the longer-term maintenance of change in the other. A clearer understanding of the interaction between physical activity and eating behaviours

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based on underlying neurocognitive processes and adaptations may help to optimize the implementation, adoption and combined effect of each for healthy, sustainable weight loss (7,8).

The analysis of behaviour change via neurocognitive modelling holds promise as one of this decade's most beneficial developments in the treatment of obesity. Behaviour change implies a critical switch whereby an externally imposed intervention transits into self-regulated actions that facilitate a desired outcome. Adapted cognitive processes and resultant behaviour patterns underlie these so-called 'goal-oriented' actions, such as resisting high-calorie foods and increasing physical activity to align with a weight-loss goal. On a neurocognitive level, these behaviour changes involve the alteration of the brain's neurobiological substrates and plastic processing pathways that are cemented over time (9). Identifying these pathways and determining how to modify the neurocognitive processes involved in adopting healthy lifestyle behaviours is essential to achieving sustained weight loss. By linking two emerging lines of neuroscience literature, we posit that increased physical activity and consumption of a healthy diet share a common neurocognitive underpinning. This may help to explain the observational association and behavioural interplay between these lifestyle factors and may be useful in developing more effective weight-loss interventions.

The associative link between physical activity and diet: observations and theoretical foundations

Whereas the effect of physical activity and dietary habits on disease has been established (10,11), the ability of these two behaviours to indirectly modify each other has been sparsely documented. The few studies that examined the interaction between habitual physical activity and healthy diet suggest an interactive and reinforcing relationship (12,13). A study by Brodney *et al*. found that higher cardiorespiratory fitness levels were associated with lower percentage of energy from fat, higher dietary fibre intake, reduced cholesterol intake and closer adherence to dietary recommendations (14). Findings from a study of 2004 New England households suggested that moderately to very active individuals tend to consume more fibre, less total fat and less saturated fat than sedentary participants (15). In one of the few prospective studies in this area, men and women who reported increases in their activity levels from age 33 to 42 also reported improvement in the overall quality of their diet (16). Taken together, these studies suggest a link between physical activity and diet, opening the possibility that engagement in higher levels of physical activity may influence the amount and quality of dietary intake or vice versa. However, crude and imprecise measures of physical activity, small sample sizes, reliance on dietary self-report and mixed samples of normal and overweight/obese individuals may undermine the reliability of the data. Such epidemiologic studies can, at best, identify potential associations and help formulate hypothesis that warrant follow-up testing. The extent and direction of causality and underlying mechanisms of this behavioural covariance remains largely unexplored given the observational nature of these studies.

Studies also indicate that physical activity and dietary behaviours comprise a constellation of similar lifestyle behaviours that cluster together (17,18). A review article by Blair *et al*. indicates that physical activity may indirectly influence health behaviours like smoking, substance abuse, risk taking, over-eating and stress management (19). Conversely, sedentary behaviour and unhealthy eating habits fit into the cluster of behaviours that are adverse to health. One study, which documented the prevalence and cluster patterns of lifestyle factors among a random sample of adolescents, adults and seniors from a large Midwestern health plan, found that overall, 14.5% of the health plan members met recommended guidelines for four healthy lifestyle factors – physical activity, non-smoking, high-quality diet and healthy weight (20). An investigation into the prevalence and clustering of lifestyle risk factors such as sedentary behaviour, smoking, low fruit and vegetable consumption and excessive alcohol consumption among Dutch men and women found significant clustering, with about 20% of the participants having three risk factors simultaneously (21). Analysis of data from the Cardiovascular Risk in Young Finns Study reported similar grouping (22). Specifically, physical inactivity has been associated with smoking (23) and increased dietary fat consumption (24), and those who smoke and drink regularly tend to consume less fruits and vegetables (25). In one study that specifically examined the patterns of adolescent physical activity and dietary behaviours, the most prevalent cluster of related health behaviours was formed by not meeting the physical activity and fruit and vegetable recommendations (26). These studies imply that physical activity and dietary behaviours do not occur in isolation. The synchronized tracking of lifestyle factors suggests that these are complex, multidimensional behaviours that coexist, occur collectively in distinct patterns and perhaps reinforce each other.

For decades, health psychologists, who examine how biological, behavioural and psychological factors influence health and illness, have laid the groundwork to explain the connection between lifestyle behaviours through observation, theories and tested psychological constructs. A variety of social cognitive models (27), each of which provides a slightly different view or perspective, have been developed and adapted to describe the cognitive determinants of health-related behaviour change. Intra-personal models that have built a reputable evidence base to explain and guide lifestyle changes include the Health Belief

Model (28), Social Cognitive Theory (29), the Theory of Reasoned Action/Theory of Planned Behavior (30), Self-Determination Theory (31) and the Transtheoretical Model of Change (32). Each model includes a set of health cognitions, thoughts and feelings that the individual associates with health-related behaviours. In combining physical activity, eating behaviour, smoking, alcohol consumption, etc. into an indistinguishable group of health behaviours, these models inherently support observational evidence of clustering.

Models from social cognitive theory have been adapted to behavioural weight-loss interventions, many of which report a positive interaction between reduced caloric intake and increased physical activity. In an examination of the comparative and cumulative effects of diet and physical activity behaviour changes during a 2-year weight-loss programme, investigators noted synergistic improvements in both factors (33). A study by Jakicic *et al*. showed that after 18 months of participation in a behavioural weight-loss programme, subjects reported increased leisure-time physical activity and adherence to eating behaviour associated with weight loss (34). In turn, new behaviour modification strategies are being developed (35) and integrated into the lifestyle interventions of large-scale clinical trials such as Look AHEAD (36). Although multiple lines of research imply a symbiotic interaction, it remains to be determined whether or not the concurrent adoption of elevated physical activity and dietary choices are independent processes or if these lifestyle behaviours somehow track together and might even be mediated by shared neurobiological substrates, which if identified, might lead to more effective interventions.

Physical activity and homeostatic versus hedonic eating

Different lines of research suggest a positive influence of physical activity on the self-regulation of eating behaviour by enhancing the sensitivity of the physiological satiety signalling system, adjusting macronutrient food choices or requirements and/or changing the hedonic response to food stimuli. One area of research, which investigates the effect of physical activity on the homeostatic mechanisms that control appetite, indicates that active people have sharper hunger–satiety mechanisms, and hence, improved control of appetite (37). A review by Blundell *et al*. (38) showed that contrary to the popular belief that physical activity increases appetite and calorie intake, men and women can tolerate exercise-induced acute energy deficits and do not compensate by eating more. Data indicate that this holds true for both lean (39) and obese (40) cohorts. Although compensation may increase in the long term (16+ d), this compensation is partial or incomplete. Conversely, it has been shown that reducing physical activity from 1.8 to 1.4 × RMR (resting metabolic rate) does not induce a compensatory reduction in energy intake, leading instead to a positive energy balance (41). The above findings suggest that increased physical activity may have significant effects on metabolic processes, and hence, energy balance in the short term. However, these studies are limited by short duration (days or weeks) and use of predominantly normal-weight subjects. Although some prospective studies that relate physical activity levels to longer-term changes in energy intake (42) and macronutrient preference (43) do exist, assessment via dietary recall has shortcomings in accurately reflecting nutrient intake and eating patterns. Measures that capture changes in the behavioural components of physical activity and food intake will help to distinguish between physiologic and behavioural changes over yearlong periods.

This discussion introduces the important distinction between the impact of physical activity on short-term metabolic adaptations versus long-term behavioural adaptations, both of which influence energy intake, but through separate mechanisms. The metabolically regulated, more automatic appetite is controlled by homeostatic mechanisms. The gut influences feeding behaviour by generating hunger and satiety signals, which are communicated to the brain via peptides from the gastrointestinal tract (e.g. ghrelin) and adipose tissue (e.g. leptin) (44). The homeostatic system is comprised of the hypothalamus and the brainstem, which drive food intake based on caloric needs and energy balance (45,46). The characterization of the biochemical signals and neurobiological processes involved in hunger, meal initiation and satiety has been the subject of extensive research since the early 20th century (47). Numerous theories – lipostatic (48), aminostatic (49), thermostatic (50), glucostatic (51) and others (52,53) – have explored the metabolic basis of food intake. For example, the glucostatic theory incorporates neurophysiologic considerations and builds upon the shared premise that glucose uptake and utilization is central to the control of hunger, satiety and control of energy balance. Experimental evidence from prospective studies links hypoglycaemia to weight gain (54), which may be exacerbated by modern realities including sleep-deprivation or increased use of computers for knowledge-based work, both of which have been shown to induce spontaneous energy intake related to changes in glycaemic control (55,56). Although this review limits its focus to the neurocognitive dimension of hedonic eating behaviour, it is important to recognize that such hypotheses have generated complementary, rather than alternative evidence of the obesogenic consequences of modern lifestyle.

Regardless of whether physical activity transiently impacts energy intake (57) or whether perturbations in glycaemia increase caloric consumption in the short term (54), it is evident that sustainable weight loss requires enduring changes in the motivation to eat (58). In contrast to metabolic mechanisms that regulate satiety, this dimen-

sion of food intake is cognitive-behavioural in nature. Within the last decade, it has been recognized that an increasing proportion of human food consumption is driven by the pleasure, known as 'hedonic hunger' (59), rather than the homeostatic principles of energy balance. In turn, increased attention has been focused on the influence of reward sensitivity (60), the brain reward system (61) and the mechanisms underlying liking and wanting (62). This reward-related appetite is centred in the brain's mesolimbic dopamine system, including the ventral tegmental area of the midbrain and extending to the nucleus accumbens in the striatum (63). In an obesity-promoting environment, the hedonic response to food stimuli can disrupt and override the homeostatic mechanisms that regulate satiety (64).

Presently, the distinction between the homeostatic and hedonic appetite is blurred by evidence that these influences may change over time (65). In future studies, it will be important to delineate the separation between these forces. For the purposes of this review, which focuses on long-term weight control for 2+ years, the discussion concentrates on the behavioural self-regulation of the hedonic response to food. Changes in behaviour patterns from those that promote hedonic over-eating to those that support healthy weight maintenance result from changes in neurobiological substrates and the formation of new connections between neural circuits. In order to better identify, effectively target and ultimately adapt the impulsive, reward-seeking component of eating behaviour, a deeper understanding of the underlying neurocognitive mechanisms is essential.

Transitioning from behaviour to neurocognition

Health psychology and social cognitive theory built the framework upon which cognitive neuroscience is now investigating neural systems and neurophysiologic mechanisms that underlie behaviours. Theoretical concepts from behavioural psychology such as reasoning, self-control and decision making are now being identified through quantitative tests that target cognitive processes and identify underlying brain regions and circuits (66). With regard to physical activity and eating behaviours, in order to build upon the behavioural association and to propose a neurocognitive explanation, a brief overview of the neurocognitive basis of human behaviour is instructive.

Behaviours often involve decision making, irrespective of whether these choices are evaluated rationally (67) or influenced by emotions (68). This decision-making process is mediated by our neural systems (69). Individuals constantly monitor behaviour through a process called self-regulation, which involves re-evaluating beliefs, setting goals and keeping thoughts and behaviours in line with such goals (70). Self-regulation falls under the loosely defined umbrella of executive functions, a group of advanced cognitive processes that break the normal flow from impulse to action. By halting automatic actions, executive processes facilitate the execution of goal-directed behaviour, intentioned steps to complete a task. Goal-oriented processes include the ability to initiate and stop actions, to monitor and change behaviour as needed and to plan future behaviour when presented with novel situations and environments. Experimentally, executive functions manifest in various cognitive components such as working memory, mental set shifting and inhibitory control (71), as well as more complex functions such as decision making, delayed gratification or self-regulation. Neuroimaging studies have mapped these executive functions (also called cognitive control) to the brain's prefrontal cortex, a region that coordinates thought and action in accordance with internal goals (72). A growing body of literature indicates that in addition to the homeostasis and reward centres of the brain, the prefrontal cortex is integrally involved in the regulation of the lifestyle behaviours that are contributing to the obesity epidemic (73).

Until recently, neuroscientific obesity-related research has focused primarily on the connection between the hypothalamus and the gastrointestinal system in the regulation of appetite (74). However, as evidenced by the prevalence of obesity, the metabolic circuits that regulate energy homeostasis can be impaired and desensitized, leading to over-eating; the human perception of and behaviour towards food can override neuroendocrine satiety signals. For our ancestors, food intake was a means of subsistence and vigorous physical activity was a daily requirement. Modern realities have now shifted these necessities into the category of 'lifestyle behaviours' (75). The 'toxic environment' (76) in which we live promotes sedentary behaviour and overconsumption of calories. From ubiquitous junk food advertisements to the diminished imperative for physical activity, our neural systems are repeatedly bombarded with health-deterring stimuli. In essence, healthy behaviours are no longer supported externally; we must cultivate and fortify them internally. Aside from considerations of socioeconomic status, cultural norms and religious beliefs, each individual now bears the onus of implementing a set of health behaviours that collectively comprise lifestyle.

The prefrontal cortex, 'top-down' inhibitory control and eating behaviour

Our cognitive processes underlie our behaviour, which in turn, determines aspects of lifestyle, such as dietary pattern, and hence, nutritional intake. For the purposes of this discussion, our eating behaviour is best conceptualized as an extension of Satpute and Leiberman's neurocognitive approaches (77) of the reflexive and reflective dual process models (73,78) in the field of social cognition. The reflexive system is the 'default responder', meaning that it is respon-

sible for our automatic, unconscious reactions. We share the reflexive system with other primates, suggesting that this system is evolutionarily old. The reflexive system favours reward and has been traced to dopamine-responsive neural circuits in the limbic and paralimbic structures. Hence, this system drives hedonic over-eating. In contrast, the reflective system is the more recent human adaptation that enables us to make rational, typically conscious decisions. This system demands greater cognitive resources in order to override the reflexive system and execute goal-oriented actions. The reflective system controls our executive processes and has been traced to the prefrontal cortex and associated cortices, anatomical brain regions that have underwent a remarkable expansion in the human species. Importantly, the reflective system is also more plastic (79), meaning that it is adaptable and capable of integrating new habits (80). Thus, despite predetermined genetic differences in anatomy and connections, such initial substrates might be modified and ultimately lead to new and improved lifestyle behaviours.

With regard to nutrient intake, executive functions are necessary to regulate and inhibit the impulsive, hedonic response to food (81). Impulsivity can be generally defined as the tendency to think, control and plan insufficiently, which is therefore inaccurate or maladaptive (82). This behavioural trait suggests a shortfall or repression of our reflective system, the system that includes our executive functions. When defined as an insufficient response inhibition, impulsivity can be seen as the inability to stop a predominant action, e.g. a movement that has been overlearned. Inhibitory control exerts the stop signal necessary to override these automatic responses, thereby enabling goal-oriented actions. The prefrontal cortex guides this 'top-down' processing in order to align behaviour with internal states or intentions (83). For example, if a cookie lover is presented with a warm chocolate chip cookie, the automatic response would be to eat the cookie; however, if this impulse to eat conflicts with that person's dietary goals, executive functions must suppress the impulse to maintain dietary restriction.

A useful cognitive test to assess inhibitory control is the stop-signal task (84), during which participants are directed to respond to a visual cue by pressing a button, but to stop pushing when they hear a stop tone. Nederkoorn and colleagues have employed the stop-signal task to show that in general, obese women (85) and children (86) tend to be more impulsive. From this follows a series of studies showing that a less efficient response inhibition (i.e. greater impulsivity) is related to increased food intake and over-eating (87), increased weight and obesity (88) and less weight loss during weight reduction treatment (89). This group has also demonstrated a causal connection between over-eating and impulsivity by inducing impulsivity in high- and low-restrained eaters (90). Data indicate that over-eating follows from an interaction between restraint and impulsivity. High-restraint, high-impulsive subjects tend to over-eat after being primed with a tasty preload or the aroma of food; in contrast, those who exhibit high restraint but are lower on the impulsivity scale do not over-eat to the same extent (91). Thus, behavioural disinhibition, a deficit in or repression of this inhibitory check, may play a prominent role in over-eating, and hence facilitate the development of obesity over time.

Insight into the neurocognitive link between disinhibition and over-eating comes from conditions explained by defective inhibitory control, most notably attention-deficit/hyperactivity disorder (ADHD). In his *Unifying Theory of ADHD*, Russell A. Barkley argues that inhibitory control sets the occasion for the performance of self-directed actions by providing the delay necessary for them to occur (92). The prefrontal cortex provides the brake necessary for the execution of goal-oriented behaviour, and this brake is deficient in ADHD. Over-eating may be partially explained by this model as studies confirm strong associations between overweight/obesity and symptoms of ADHD in children, adolescents and adults (93,94). Research has also shown that higher than expected rates of binge eating, which is characterized by bouts of unrestrained eating, occur in individuals with ADHD (95).

A second way to gauge impulsivity is to think of it as sensitivity to reward, which can be assessed by the ability to delay gratification, or to wait in order to attain something one wants (96). Walter Mischel *et al.*'s famous 'marshmallow experiments' (97), which examined the cognitive mechanisms that enable a young child to forego immediate satisfaction and to wait for a larger but delayed reward, understood the inherent link between delay of gratification and eating behaviour. Continuing research with the original participants suggests that the ability to delay gratification during the pre-school years predicts future outcomes, including body mass index and drug use (98). Recently, a prospective study showed that students who failed the delayed gratification task at 4 years of age were more likely to be overweight at 11 years old (99). Similarly, increased adiposity has been linked to poorer performance on the Iowa Gambling Task (100), a psychological test that assesses reward sensitivity, among other cognitive components, by choosing between immediate versus eventual monetary gains. Delay of gratification and delay discounting in the Iowa Gambling Task are two constructs that have been useful for examining inhibition of impulsive over-eating (101,102). Of course, the intuitive link between over-eating and impulsivity is that successful weight loss, a delayed reward, requires avoiding triggers and sacrificing the immediate gratification of over-eating.

Neuroimaging studies in both the right (103) and the left (104) prefrontal cortex bolster Mischel *et al.*'s theory by suggesting this region's involvement in delaying gratifica-

tion. Furthermore, neuroimaging studies confirm a role for the prefrontal cortex in hedonic feeding inhibition by linking activation patterns with motivation to consume palatable foods. A study published in 2001 (105) examined the activation patterns in response to continuous ingestion of chocolate by self-identified chocolate fanatics. Decreased motivation to consume more chocolate over time was associated with increased activation of the lateral prefrontal cortex, suggesting an inhibitory signal. In contrast, exposure to appetizing food cues results in preferential activation of hedonic regions of the brain, with obese individuals showing greater activation in these areas (106,107). Transcranial magnetic stimulation, a technique that non-invasively modulates cortical activity, is now being tested as a therapeutic intervention for such impulsive eating habits (supported by the Klarman Family Foundation). Preliminary trials indicate that enhancing activity in both the left (108) and the right (109) dorsolateral prefrontal cortices with non-invasive brain stimulation can suppress food cravings (110). These studies suggest that the amount of activity in the prefrontal cortex may determine the degree of inhibition over downstream reward circuits that encourage what was once an evolutionarily advantageous impulse to over-eat.

Conversely, the ability to adopt and maintain healthy eating habits has been linked to greater inhibitory control. Studies show that increases in cognitive restraint and self-efficacy (111,112) and decreases in emotional, disinhibited eating (113) are predictive of successful weight maintenance. As shown in analysis of members of the National Weight Control Registry (114), an ongoing study of more than 7000 weight-loss maintainers, one of the critical predictors of successful weight-loss maintenance is a lower level of dietary disinhibition (115,116). This is supported by neuroimaging studies of successful weight-loss participants, which report greater activation in the prefrontal regions in response to food cues (117). After ingestion of a meal, successful dieters show increased neural activity in the dorsolateral prefrontal cortex, which is involved in behavioural control and self-restraint (118). In contrast, obese men (119) and women (120) demonstrate less activation of the dorsolateral prefrontal cortex in response to a meal than do lean or formerly obese men and women.

This evidence lends strong support to the notion that eating behaviour is not regulated solely by homeostatic mechanisms centred in the hypothalamus; executive functions, particularly inhibitory control, and the prefrontal lobes also appear to play an important role. While this concept of impulsivity may connote a lack of self-control or willpower, we maintain that impulsivity is becoming a more compulsory response in our obesogenic environment. The reflective brain network, which encompasses the prefrontal circuits that guide executive functions and goal-oriented behaviours, is of a limited capacity (77). Controlled processes, such as abiding by dietary goals and resisting temptations to indulge, tax and eventually exhaust the cognitive resources that regulate more emotional, automatic responses. This strain is exacerbated by stimuli in the obesogenic environment, which suggests that maintaining a healthy weight in our modern lifestyle demands an elevated and durable cognitive reserve. Regardless of whether or not the individual has a predisposed deficit in executive function or a dysfunction of prefrontal circuits, the absence of external support to enable healthy eating habits places the burden of behaviour change on the individual. The aforementioned association between cognitive disinhibition and excessive caloric intake begs the question of whether or not the cognitive resources necessary to counteract obesogenic stimuli and fortify inhibitory control can somehow be 'exercised' and enhanced internally. If so, it logically follows that goal-oriented behaviour, as epitomized by healthy dietary choices, can be improved, refined and perhaps maintained. In determining how to boost executive functions, it is now instructive to consider the burgeoning literature on the cognitive benefits associated with the companion health behaviour: physical activity.

Physical activity can improve executive functions, self-efficacy and mood

A surge of research over the past decade provides supportive evidence that physical activity is positively correlated with neurogenerative, neuroadaptive and neuroprotective processes. Data from cross-sectional studies and randomized clinical trials suggest positive influences of fitness training on human brain structure and cognitive function (121). After assessing 18 studies published from 1966 to 2001 in a meta-analysis, it was reported that exercise broadly improves cognitive function across a number of domains, particularly executive functions, which appear to be more sensitive to enhancement as compared to other aspects of cognition (122). Kramer *et al*. showed that over a 6-month period, a group that engaged in moderate walking not only became more aerobically fit, but also showed enhanced executive processing, as indexed by better performance in task switching, stopping and selective attention tasks, compared with the stretching and toning control group (123). In agreement with these findings, functional magnetic resonance imaging data show that modulation of physical activity disproportionately influences tasks that necessitate greater amounts of executive control (124). This coincides with evidence that brain regions related to executive control, including prefrontal circuits, are more plastic (125), structurally sound (126) and even prone to growth (127) with elevated levels of aerobic fitness.

Given the evidence that increasing physical activity improves executive function, it seems that decreased physical activity would contribute to or at least be associated

with deficits in similar cognitive processes. Indeed, when researchers at Berlin's Center for Space Medicine put 24 healthy men into the bed rest model of prolonged weightlessness, scores on the Iowa Gambling Task, which has been associated with regulation of eating behaviour, were significantly lower during bed rest than before or after the intervention (128). This finding is supported by studies which report that a combination of high disinhibition, a disposition for opportunistic eating, and low self-restraint is associated with higher sedentary behaviour (129). Exercise has been shown to exert a positive influence on those with a high disinhibition score by decreasing motivation to eat (130) and increasing preference for low-fat foods (131). These cognitive measures are supported anatomically by the intertwined development between the adjacent prefrontal and motor cortices (132). Since exercise has been shown to enhance connectivity between distinct functional networks in separate brain regions (124), increased use of the motor cortex during physical activity may enhance executive processes and vice versa. Although this remains speculative, neurocognitive underpinnings may help to explain the reported correlation between sedentary activities, such as time spent watching television, and energy intake (133).

Both cognitive testing and neuroimaging studies show that regular physical activity enhances executive functions, and these same cognitive processes are implicated in the regulation of impulsive eating behaviour. This cognitive convergence is further supported by research on depression and mood, which are integrally linked to cognition and for which both physical activity and eating behaviour appear to be key mediators (134,135). Depression and obesity are positively related in a bidirectional relationship (136). Emotional eating, which refers to the tendency to eat in response to negative emotions, has been associated with elevated levels of depressive symptoms (137), the consumption of high-calorie foods (138) and higher body mass index (139). In line with this, a lack of physical activity self-efficacy, a person's confidence in his or her ability to be physically active on a regular basis, has also been linked to higher depressive symptoms (140). A recent study by Kontinnen *et al*. (141) used mediation analysis to confirm both of these associations within the same cohort. Although this study examined emotional eating and physical activity self-efficacy as independent pathways between depressive symptoms and higher adiposity, the negative correlation between these factors suggests an interaction.

In contrast, successful adoption of a physically activity lifestyle may positively influence eating behaviour through its effect on variables such as motivation, commitment and self-efficacy (31,142). Evidence shows that increases in exercise motivation and self-efficacy predict positive changes in the self-regulation of eating behaviour, and the resulting confidence could 'spill-over' into other behaviours important for weight management (143). Both structured exercise (e.g. running, cycling, fitness classes) and lifestyle activities (e.g. walking) have been positively associated with weight control (144) and eating self-regulation. Andrade *et al*. recently showed that both vigorous exercise and lifestyle activities improve several eating behaviour variables that describe dietary self-regulation (145). These variables mediated the relationship between physical activity and 1-year change in body weight. This indicates that adoption of physical activity may promote a maintained decrease in body weight not only through an immediate increase in energy expenditure, but also by changing psychological markers of eating behaviour, and may also explain why physical activity has been shown to play the paramount role in long-term weight management (7).

Depression provides insight into possible mechanisms by which increased physical activity can improve weight control via strengthening the cognitive resources needed for hedonic restraint. Negative emotional states promote over-eating by depleting the cognitive resources necessary to resist the temptation to over-eat (146). Physical activity, perhaps by improving mood (147) and/or reducing anxiety and stress (148), counters the reflexive impulse to over-eat. In the largest randomized control trial to date, with a cohort of 200 clinically depressed adults, Blumenthal *et al*. showed that 4 months of aerobic exercise achieved reductions in depression comparable to standard antidepressant medication and greater than placebo (149). Neurocognitive research shows that depression is characterized by hypoactivity of the prefrontal cortex (150). In the presented framework of prefrontal inhibitory control, this would be predicted to be associated with a dampened or suppressed reflective system. Hence, given physical activity's therapeutic effect on depression and depression's link to unrestrained eating, it seems plausible that physical activity could enhance those same cognitive processes that regulate hedonic eating behaviour.

Physical activity modifies eating behaviour through a neurocognitive connection

Connecting the pieces between physical activity and executive function with eating behaviour and executive function suggests that rather than a coincidental association, physical activity and eating behaviour may share a common neurocognitive platform. As shown diagrammatically in Fig. 1, physical activity builds the cognitive resources, namely inhibitory control, necessary to block impulsive actions that may have adverse health consequences, specifically over-eating. In this sense, physical activity is a 'gateway behaviour' that may induce people to improve their diets over time (151,152). Since the mid 1980s, health psychologists have posited that physical activity can be an inherently rewarding activity that contributes to both health and subjective vitality. By filling a psychological

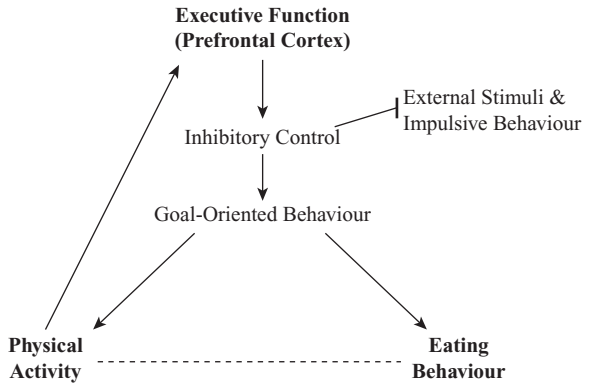

Figure 1 A diagrammatic representation of the hypothesis linking physical activity to eating behaviour via executive function. In the proposed model, physical activity can enhance executive function by generating, strengthening and refining neural circuits in the prefrontal cortex. These executive functions are requisite for inhibitory control, the ability to suppress impulsive behaviour and responses to external stimuli. Due to the abundance of negative food stimuli in the environment that prompt unhealthy dietary habits, practising healthy dietary choices demands a high degree of inhibitory control. Inhibitory control acts as the brake, thereby enabling the execution of goal-setting behaviour and self-control. Thus, physical activity indirectly modifies eating behaviour by strengthening executive function. Over time, physical activity and healthy dietary habits become behavioural adaptations connected by their goal-oriented nature.

need of competence and enjoyment, adherence to physical activity may affirm other healthy behaviours (153). In today's world, in an environment that fosters sedentary behaviour and over-eating, practicing regular physical activity and adhering to healthy diet are necessarily self-regulatory, goal-oriented behaviours. From a neurocognitive perspective, top-down inhibitory control enables us to repress health-aversive stimuli and execute health-promoting behaviours.

In addition to this neurocognitive basis, the choice of physical activity as an effective first target for weight loss also garners critical support from the physiologic/homeostatic angle. Regular physical activity improves insulin sensitivity (154), increases fat oxidation and enhances the body's response to leptin (155). Starting weight loss with physical activity may represent an investment in physiological functionality to counteract the hypoglycaemia and weight regain that often follows weight loss (156). This point reinforces the idea that the more immediate, homeostatic regulation of appetite and energy intake complements and perhaps interacts with and/or stimulates some of the neurocognitive adaptations and subsequent behaviour changes over time. Physical activity has been shown to be one of, if not the most important, factor in long-term weight maintenance (8), but whether this is due to metabolic changes, neurocognitive adaptations or more likely a combination of both remains to be determined. Collaborative projects that integrate multiple aspects of eating behaviour and physical activity are a priority for future research.

This hypothesis is grounded in the link between executive function and eating behaviour, which suggests that suboptimal prefrontal cortex function, or more aptly cognitive resources under constant strain in an obesogenic environment, may contribute to the aetiology of obesity. The mounting evidence that positively correlates increased physical activity with enhanced executive function may represent a viable therapeutic approach to controlling and modifying eating behaviour. The first step in building an evidence base for this hypothesis would be to establish the causal relationship between physical activity and enhanced cognition. Then, it would be important to investigate whether or not exercise causes an enhancement in inhibitory control, or other key components of executive functions. It would also be instructive to concurrently encourage and monitor new studies investigating the behavioural impact of physical activity interventions and pharmacotherapies on ADHD patients as a positive impact would presumably act through a similar neurocognitive mechanism. Whereas this review covers physical activity in the broad sense, including both exercise and lifestyle activities, it will be equally critical to determine intensity thresholds, durations or specific activities that best strengthen and refine executive functions. Can different modes of exercise, such as aerobic and resistance training, produce the same outcome on these cognitive tests? We would need to develop an evidence-based programme to adopt physical activity in order to induce and sustain the desired neurobiological effects and consequential eating behaviour modifications. Connecting these spheres of research opens expansive investigative opportunities with important translational implications for the treatment of obesity and adoption of a healthy lifestyle.

Conclusion

To the best of our knowledge, this is the first review to combine the robust literature on physical activity and eating behaviour in order to hypothesize a neurocognitive link. This proposal is meant to spark interaction among neuroscientists, psychologists, endocrinologists, exercise physiologists, dieticians and clinicians to promote integrative, translational research. Our hypothetical model has widespread implications for both clinical care and public policy. When designing the most effective and efficient approach to weight loss and subsequent weight-loss maintenance, it is critical to understand which to target first – physical activity or diet – based not only on its effect on energy balance, but on its ability to positively impact the other behaviour. Perhaps, elevated physical activity can facilitate changes in related healthy lifestyle behaviours by

'training' the cognitive resources necessary for their execution. Considering the fallibility of fad diets and the underperformance of clinical treatment strategies, it seems plausible to approach from the physical activity angle. In this way, we may facilitate healthy and steady weight loss by dimming the often overbearing spotlight on weight loss. With regard to public policy, we take particular issue with the fact that financially strained school systems are cutting physical education classes and unstructured recess time. By reducing physical activity in schools, we may be propagating the childhood obesity crisis not only by reducing energy expenditure, but also by worsening eating habits. Ironically, too much work with no play may be weakening those cognitive abilities that we are so eager to test (157).

As embodied by our developmentally advanced prefrontal cortex, humans display complex yet interrelated behaviours, the underlying neurocognitive components of which must be elucidated to help treat lifestyle-related diseases. Investigating physical activity and other possible modes of enhancing executive function may increase our understanding of how to control appetite and enhance inhibitory mechanisms that regulate eating behaviour. This could improve adherence to diet and exercise therapy, ultimately facilitating adoption of these behaviours.

Conflict of Interest Statement

No conflict of interest was declared.

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