Neuroscience and Biobehavioral Reviews 35 (2011) 1305–1325

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

The reticular-activating hypofrontality (RAH) model of acute exercise

Arne Dietrich[a,*], Michel Audiffren[b]

[a] Department of Social and Behavioral Sciences, American University of Beirut, Beirut 1107-2020, Lebanon
[b] Centre de Recherches sur la Cognition et Apprentissage, Université de Poitiers, CNRS, France

ARTICLE INFO

Article history:
Received 11 October 2010
Received in revised form 2 February 2011
Accepted 3 February 2011

Keywords:
Arousal
Effort
Executive functions
Implicit
Reaction time
Prefrontal cortex

ABSTRACT

We present here a comprehensive, neurocognitive model to account for the psychological consequences of acute exercise. There is a substantial amount of disparate research and the proposed mechanistic explanation meaningfully integrates this body of brain and behavioral data into a single, unified model. The model's central feature is a cascading, two-step process. First, exercise engages arousal mechanisms in the reticular-activating system. This activation process, which involves a number of neurotransmitter systems, has several interrelated effects on cognition and emotion but, in general, has evolved to facilitate implicit information processing. Second, exercise disengages the higher-order functions of the prefrontal cortex. This deactivation process, which is caused in part by resource limitations, also has several interrelated effects but, in general, has evolved to keep the inefficient explicit system and unhelpful emotional processes from compromising the implicit system's functioning when optimal motor execution is needed most. In this article, we review evidence in support of this reticular-activating hypofrontality (RAH) model of acute exercise and place it into a larger evolutionary context.

Contents

* Corresponding author.
E-mail address: arne.dietrich@aub.edu.lb (A. Dietrich).

0149-7634/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neubiorev.2011.02.001

"A man cannot think deeply and exert his utmost muscular force"
– Charles Darwin, The Expression of Emotions in Animal and Man

1. Introduction

This paper outlines a comprehensive, neurocognitive model of the effects of acute exercise on cognition and emotion. It is divided into six sections. In the first, we summarize, in four brief points, the data that needs to be explained. This is followed, in the second section, by a broad overview of the basic idea behind the proposed reticular-activating hypofrontality (RAH) model. In sections three and four, we defend this model by examining more in depth the two main – and opposing – mechanisms that comprise it, the reticular-activating process and the hypofrontality process. We do this within a wider evolutionary framework that provides a rationale for why these mechanisms exist. Section five presents the model in a formal manner, including assumptions and predictions that flow from it. The sixth and final section discusses how, according to the RAH model, certain exercise parameters affect cognition and emotion.

1.1. The existing data

Any integrative, mechanistic attempt to account for the effects of acute exercise on mental function has to explain the following set of apparently contradicting results. They only appear at odds, of course, because exercise scientists have traditionally collated and analyzed the existing data without making the critical distinction between executive functions and other cognitive processes (Brisswalter et al., 2002; Etnier et al., 1997, 2006; Tomporowski, 2003). Only a model that separates them can begin to disentangle the reasons why some cognitive functions are enhanced by exercise, while others are impaired. Note also that the four points summarizing the existing data, like the RAH model in general, are only concerned with the psychological effects of acute, not chronic exercise. While acute exercise refers to the practice of a single session of exercise lasting from a few seconds to perhaps several hours, chronic exercise refers to the repetition of exercise over time during a period lasting from weeks to years (see also Table 2).

First, performance on reaction time tasks and other simple decisional tasks are enhanced during acute bouts of aerobic exercise. This is the case, details aside, across the board, that is, irrespective of the type of reaction time task and irrespective of the kind of acute aerobic exercise (Audiffren, 2009; Etnier et al., 1997; McMorris and Graydon, 2000; Tomporowski, 2003). It is one of the most robust findings of exercise psychology. This facilitation does not seem to extend into the post-exercise period (e.g., Audiffren et al., 2008; Brisswalter et al., 1997) and may even be reversed into an impairment when exercise is prolonged and/or goes to exhaustion (e.g., Cian et al., 2000, 2001).

Second, performance on cognitive tasks that substantially recruit executive processes is impaired during acute bouts of aerobic exercise. Despite their availability and widespread use in other domains of psychology, tasks requiring executive processing were not used in conjunction with exercise until a few years ago, which is the reason why this effect was documented only recently (Dietrich and Sparling, 2004). Research since then, however, (e.g., Audiffren et al., 2009; Davranche and McMorris, 2009; Del Giorno et al., 2010; Mahoney et al., 2007; Pontifex and Hillman, 2007; McMorris et al., 2009), along with the proper interpretation of even older data (Adam et al., 1997; Paas and Adam, 1991) has confirmed this phenomenon. The data does not support the notion that this decrement persists long, if at all, into the post-exercise period (e.g., Sibley et al., 2006). Indeed, studies using a chronic exercise paradigm have clearly demonstrated that the lasting effect of exercise is the opposite, an improvement of executive processes (Colcombe and Kramer, 2003; Hillman et al., 2008). There is also data suggesting that exercise induces states of effortless attention and flow (Dietrich, 2004a; Dietrich and Stoll, 2009).

Third, in making the two points above, we must acknowledge and then temporarily set aside certain complications. This gives us the opportunity to first build some theoretical scaffolding that will eventually allow us to introduce the additional complexity in a more meaningful way. To anticipate, the data are, of course, more nuanced. The facilitating effect of exercise for procedural or simple, decisional tasks shows a great deal of variability with several studies reporting no effect at all. This is hardly surprising in light of (1) the plethora of exercise modes and protocols used and (2) the sheer number and variety of tasks – and their very different cognitive demands – that have been used in exercise studies (Audiffren, 2009; Tomporowski, 2003). Moreover, we are evidently not dealing with a strong effect as it is (Etnier et al., 1997, 2006). As for tasks with manifestly explicit components, the impairment of executive functions during exercise is also likely to be a function of exercise parameters – duration and intensity, mostly – as well as the extent to which a task engages higher-order, explicit processes. The full weight of the evidence, however, suggests that we are amply justified in making the above generalizations. We will, then, ride roughshod over these complexities until we are in a position to place these two opposing effects where they belong, on the extreme ends of one continuum – actually on two, one for the explicit system and one for the implicit system, as exercise seems to affect these two information-processing systems in opposite directions (Fig. 1).

Fourth, exercise has a positive effect on emotions. In the moderate, aerobic range especially, it reduces stress, decreases anxiety, and alleviates depression (Salmon, 2001; Scully et al., 1998). Exer-

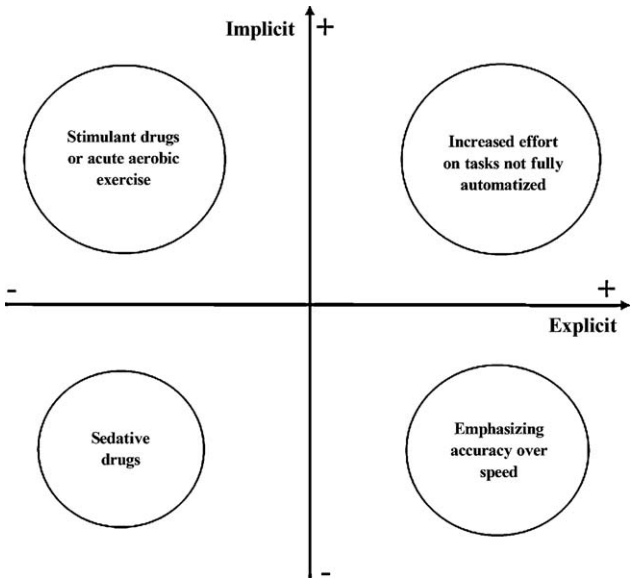

Fig. 1. Some examples of situational variables on the efficiency of explicit and implicit processes. The vertical axis represents implicit processes and the horizontal axis explicit ones. The positive sign represents a higher efficiency and the negative sign a lower one. Some stimulant drugs, such as amphetamines, or acute aerobic exercise improve the efficiency of the implicit system but have, at the same time, a negative effect on the explicit system. Motivational factors that increase mental effort to a task that is not fully automated, say, a tennis serve tend to improve the efficiency of both systems. Situations in which accuracy is paramount, in science or any other task requiring careful deliberation for instance, emphasize explicit processes and reduce the involvement of implicit processes. Finally, a sedative drug is an example in which both of the brain's information-processing systems operate below par.

cise also induces analgesia, sedation, and feelings of well being (Dietrich and McDaniel, 2004). These changes in mood states stand somewhat in contrast to those on cognitive arousal in that they are (1) generally calming and (2) typically extend well into the post-exercise period. In considering the data on emotions, we also keep a few complications temporarily clamped here, for the same reason as above.

1.2. The theoretical landscape

Theories accounting for these data, in whole or in part, have not been in short supply. They exist at all levels of psychology from neural mechanisms to energetic models, from cognitive frameworks to social theories. Theories pitched at the neurocognitive levels, and specifically built to address the effects of acute exercise, involve several neurotransmitter systems – monoamines, peptides and lipids, to name the important ones – several neuroanatomical sites, such as the prefrontal cortex, a variety of cognitive models, such as those of Kahneman (1973) or Sanders (1983), and energetic considerations, such as thermoregulation or the brain's blood and glucose distribution. Each theory, the popular but misbegotten more-blood-idea excluded (Dietrich, 2009), has some data to support it. However, each theory is also limited in that it can handle only one or two aspects, at most, of the much larger puzzle.

To give a few examples, serotonin may help explain the antidepressant effects but does not do any explanatory work when it comes to faster reaction time performance or flow states; dopamine and norepinephrine can acccount for the arousal component but run counter to any explanation for anxiolysis or sedation; the hypofrontality theory explains the impairment of executive processes but has nothing to say on analgesia or speedier motor execution; cognitive-energetic theories can model some aspects of cognitive function but fail on others, such as feelings of effortlessness; endorphins and endocannabinoids represent a possible mechanism for the sense of well being but are useless for other emotional processes.

Of course, most of these theories were not originally designed to handle all moving parts of the complex interaction between physical activity and mental function. It is possible, although not likely, that these mechanisms evolved independently, haphazardly, modulating one specific function for one specific purpose at a time. The advantage of an evolutionary perspective, however, especially when combined with current models in cognitive neuroscience, is that a synthesis emerges that subsumes all effects – and their neural mechanisms – into a single, unifying framework.

2. Basic concepts of the RAH model

A key notion underlying the present model is the well-known but apparently underappreciated distinction made between the explicit and implicit systems (see also Fig. 1). The brain operates two largely independent cognitive systems to acquire and represent information. The explicit system is rule-based, its content can be expressed verbally, and it is tied to conscious awareness. In contrast, the implicit system is skill or experience-based, its content is not verbalizable and can only be conveyed through task performance, and it is inaccessible to conscious awareness (Dienes and Perner, 1999, 2002; Schacter and Buckner, 1998; Reber, 1989; Squire, 1992; Willingham, 1998).

Advances in cognitive neuroscience have begun to identify the brain circuits underlying the explicit system. Evidence that the working memory buffer of the dorsolateral prefrontal cortex (DLPFC) holds the current content of consciousness, coupled with evidence that the executive attentional network of the DLPFC is the mechanism to select the content (Baddeley, 1996; Cowan, 1995), suggests that the explicit system is critically dependent on prefrontal regions (Ashby and Casale, 2002; Dehaene and Naccache, 2001). Abundant evidence also suggests that medial temporal lobe structures are involved (Poldrack and Packard, 2003). The neural substrates of the implicit system are less clear. The basal ganglia, cerebellum, and supplementary motor area (SMA) have been implicated most often, and they are critical for a type of implicit memory known as procedural memory (motor and cognitive skills), but contribute to other types as well, such as priming, conditioning, and habituation (Mishkin et al., 1984; Poldrack and Packard, 2003). Research on animals, brain-damaged patients, and neuroimaging studies of healthy subjects have thus demonstrated that these systems are differentiated by anatomy and function (Squire, 1992).

The existence of two distinct systems for knowledge representation indicates that each must be specialized in some way. The explicit system is understood to be a sophisticated system that is capable, thanks mostly to the computational infrastructure in the prefrontal cortex, to represent knowledge in a higher-order or conscious format; that is, it represents additional information about the information, such as, for instance, the fact that it contains the information it contains (e.g., Keele et al., 2003). This permits the information to be broadcast to a global workspace, making it usable for other parts of the system (e.g., Dehaene and Changeux, 2004). In contrast, the implicit system is taken to be a more primitive and evolutionary ancient system that does not form higher-order representations (Reber, 1989). As a consequence, the explicit system, or any other functional system in the brain, does not know about knowledge imprinted in the implicit system, making it unavailable for representation in working memory, and thus consciousness (for more details, see Cleeremans and Jiménez, 2002; Haider and Frensch, 2005; Karmiloff-Smith, 1992; Kihlstrom, 1996; MacDonald, 2008).

This makes clear why procedural knowledge is limited in its usability. The implicit system cannot represent the knowledge as a hypothetical possibility, which makes it inflexible and idiosyncratic (Dienes and Perner, 1999). This also explains why procedural knowledge, such as motor skills, is more efficient. The implicit system is not burdened by higher-order representations, which exponentially increase computational complexity (e.g., Sun, 2006). Given the already high complexity of even the simplest of motor skills, making motor knowledge explicit would become a serious resource issue. Indeed, full explicit representation of a motor skill is impossible because information in the explicit system is subject to the limits of working memory capacity (Cowan, 2001). But a motor task such as a tennis serve involves more than tossing the ball straight in the air, swinging the racket in an arc, hitting the ball as it descends, and following the motion through. Consider what it would take to write a computer program that specifies each muscle twitch in the correct order and intensity to produce a world-class tennis serve. The amount of information that must be held concurrently in the focus of attention far surpasses the computational limit of working memory. In contrast, procedural knowledge is contained in the application of the procedure and need not be extracted from general rules that are represented at a higher-order level and then applied to a specific example.

2.1. Evolutionary benefits of implicit efficiency

For action, this explicit/implicit distinction has the following consequences (see Bruya, 2009; Dietrich, 2004a; Dietrich and Stoll, 2009; Dienes and Perner, 2002; MacDonald, 2008; McGuire and Botvinick, 2009; Posner et al., 2009; Schmeichel and Baumeister, 2009; Wulf and Lewthwaite, 2009). The inherent efficiency of procedural, implicit knowledge is paramount to motor skills because purposeful motion must occur in real time. The explicit system, on the other hand, has evolved to increase flexibility but is limited,

exactly because of its ability to deal with higher-order representations, to tasks that are best solved offline and that can be broken up into chunks of complexity that do not exceed the capacity limit of working memory (Keele et al., 2003; Reber, 1989). Since this is not the case for movement, the implicit system must handle them. In other words, the explicit system owes its flexibility to its capacity for abstraction, but it is also exactly this very ability that limits its use for purposes, such as skilled motion, where time is of the essence (Dienes and Perner, 2002; Reber, 1989).

We must rigorously apply this flexibility/efficiency tradeoff that exists between the implicit and explicit systems to the computational problem of motor performance (Dietrich, 2004a; Dienes and Perner, 2002). Indeed, this position needs to go as far as it can go; it has considerably more explanatory power than has been generally recognized. As an example, consider the lightening-fast escape maneuvers of a squirrel. Lacking an overall strategy or plan, the squirrel gets to safety entirely by relying on moment to moment adjustments. Such smooth feedback-driven input–output integration can produce extremely complex movement patterns that can serve a higher goal (safety), yet requires no more than the reaction to immediately preceding input. Since these are fluid situations occurring in real time, they require, first and foremost, efficiency.

The point is perhaps most vividly exposed when we think of the deleterious effects of transferring a complex movement from implicit to explicit control (Dienes and Perner, 1999, 2002; Willingham, 1998; Wulf and Prinz, 2001). Due to the explicit system's inefficiency and capacity limit, it should be obvious that any amount of transfer of the skill from implicit to explicit execution gravely affects its quality (Beilock and Carr, 2005; Willingham et al., 2000). One only has to throw a ball with the nondominant arm to see the dramatic loss of efficiency. This is not a minor concern, if we have to, as our ancestors did, hunt for our food or are being hunted, presumably two of the most common situations in which they had to do what we today would call exercise. We can sum up the matter by stating that a well-learned motor skill is performed with maximal efficiency, if it is controlled by the implicit system (Dietrich, 2004b).

The activation of motor programs requires the engagement of the brainstem's arousal systems. It is not difficult to grasp the evolutionary significance of why, during exercise, these systems are activated. The general purpose of this machinery is (1) to activate the various nervous and endocrine mechanisms that drive and sustain the actual motion and (2) to boost performance, if ever so slightly, on the sort of mental tasks – reaction time, visual discrimination, simple choice, and so on – that are needed for the kinds of behaviors that, incidentally, also necessitated the exercise – fight-flight situations, most prominently among them.

2.2. Evolutionary benefits of hypofrontality

The evolutionary purpose of the hypofrontality is not so obvious. It comes into clear view only if we consider the flexibility/efficiency tradeoff and the complex interaction between the implicit and explicit system. Stated in one sentence, we propose that the downregulation of the explicit system evolved in humans to offset the influence of metacognitive processes in the one situation these processes are most definitely not adaptive, when survival requires fast and efficient responding. As said, tasks that require real time sensorimotor integration are best handled by the implicit system. Explicit interference in the execution of these kinds of tasks tends to decrease their effectiveness. Not only will thinking *about* movement be of little use, it will make the movement less efficient, especially if the behavior is a well-learned skill (see DeCaro and Beilock, 2009; Schmeichel and Baumeister, 2009; Ravizza, 1977; Wulf and Lewthwaite, 2009; Wulf and Prinz, 2001). A mechanism that can help minimize explicit processes from compromising the speed and accuracy of the implicit system would have a distinct survival value. This, we submit, is the adaptive purpose of the hypofrontality process.

The same rationale also applies to complex emotions. Their phenomenological subtraction, brought about by the downregulation of the very brain regions that compute them – prefrontal regions, mostly – has the same basic evolutionary purpose: maximize reaction time performance in simple choice situations when speed and accuracy matter most.

It follows that a movement can be executed by the explicit system and/or the implicit system but an explicit-predominant movement proceeds from a mental representation that is different in kind from one that is implicit-predominant. Since a highly practiced, implicit-predominant skill is still performed by a conscious person, it is possible for the explicit system to partake in its moment-to-moment execution. To stay with tennis, this occurs when a player buffers any part that is extraneous to the actual motion – thinking about stroke technique or reflecting on the game's importance – in a higher-order representation and allows such analysis to guide the movements. As said, however, this sort of interference is detrimental to the speed and accuracy of the action. A professional tennis player's serve, for instance, is performed implicitly during a match. Few, if any, of the serve's elements are, presumably, explicit in consciousness at that time. To find out how much the explicit system actually knows about how to do a tennis serve, Federer would simply have to perform the motion with the other arm. Now the explicit system must take over because most of the task requirements of his regular serve are irretrievably embedded in the reflexive loops of the mental representations involving the dominant arm. The problem is, however, that a tennis serve is too fast and too complicated to be executed by a mental representation that is general in nature and needs to apply its abstract knowledge, in real time no less, to a specific example. The degree of implicitness of motor competence, then, is positively related to the quality of the performance (Dietrich, 2004b). This problem of interference, we can presuppose, only arises in humans because other animals simply do not have such a highly developed explicit system in the first place that possibly could wreck the smoothness and ease of implicit motor performance.

2.3. Opponent processes

With this first, minimal bit of theoretical scaffolding erected, we can begin to see why one system is boosted and one is weakened. This is an important first step because many people find the notion of hypofrontality during exercise deeply counterintuitive. This mental block rests in part on the false belief that the improvements in mental health from exercise must emerge from some process that activates, or at least reactivates, a neural region that was hitherto not functioning at full capacity. This, too, will be fleshed out in more detail below. In any case, the 'why' does not explain the 'how'. While an evolutionary perspective helps with conceiving of the basic idea, it is the details of the neurocognitive mechanisms determining this opponent process of activation and deactivation that is the central topic of this article. We termed this yin-yang process the reticular-activating hypofrontality model to denote the idea that two specific brain regions do most of the initial heavy lifting.

On the basis of the existing data, then, the following two-step process is proposed. First, the initiation and continuation of exercise activates the various arousal systems in the reticular formation. The reticular-activating system is not one homogenous system but consists of several interrelated arousal systems that are differentiated by anatomy, neurotransmitter, and function (for detailed reviews, see Robbins and Everitt, 1995, 2007). Together, these systems mobilize the energy that sustains the physical motion; they

stimulate the motor units and activate, in a cascade of events, the autonomic nervous and endocrine systems. In addition, these arousal systems activate cortical regions that modulate sensory, attentional, and motor processes by, for instance, increasing the signal-to-noise ratio or shortening motor time for basic reflexes. Again, the adaptive value of this activation, perceptual processes included, is rather obvious when we consider the kinds of situations that necessitate large-scale, prolonged bodily motions in the animal kingdom.

Second, the brain, in order to drive the bodily motion, is forced to make profound changes to the way it allocates its metabolic resources. This follows from three fundamental principles in neuroscience: (1) the brain has a finite energy supply; (2) bodily motion is an extremely demanding task in *computational* terms – that is, to be clear, for the brain, not the body – and (3) neural processing occurs on a competitive basis. This is to say, the brain cannot maintain activation in all its networks at once and activity in one structure must come at the expense of others (Gusnard and Raichle, 2001; Nybo and Secher, 2004; Woolsey et al., 1996). For exercise, the enormous demands on motor, sensory, and autonomic brain regions result in less resource available for computations in those brain structures not directly involved in controlling the movements. So, as the brain sustains, during exercise, the massive and widespread neural activation that runs motor units, assimilates sensory inputs, and coordinates autonomic regulation, it must take metabolic resources, given their limited availability, away from neural structures whose functions are not critically needed at the time, which are, according to the transient hypofrontality theory (THT), areas of the prefrontal cortex and, perhaps, the limbic system (Dietrich, 2003, 2006).

3. The reticular-activating process

In this third section of the paper, we consider in more detail the reticular-activating process. Changes in neural activity in the ascending reticular-activating system have been, until quite recently, the sole brain mechanism enlisted by scientists to account for the effects of acute exercise on mental processes. The reason for this was that increases in catecholamine and indolamine transmission were, at the time, the only well documented neural events accompanying exercise. Faced by the lack of alternatives, exercise scientists had little choice but to make monoamines do all the explanatory work. While norepinephrine and dopamine were held responsible for the positive effects on cognition, serotonin was recruited to help out with those on mental health. This seemed to work reasonably well, at least from a perspective of a few steps back, mainly because (1) the neuroprotective effects of (chronic) exercise had not been described yet, (2) the detrimental effects of (acute) exercise on higher cognitive processes had not been described yet, and (3) the few emotional changes that did not fit in anywhere – analgesia, a sense of well being, or alteration to mental status, for instance – were either ignored or set aside in the hope that some other kind of mechanism – the uploads perhaps, after all – would emerge to take care of them. It is not surprising, then, that cognitive-energetic models, such as those of Kahneman (1973) and Sanders (1983), almost exclusively tied exercise-induced changes to cognition on these brainstem arousal mechanisms.

Mechanistic explanations expanded when the possibility of molecular mechanisms leading to synaptogenesis or even neurogenesis was discovered in the late 1990s (e.g., van Praag et al., 1999). This was fortuitous indeed because the benefits of chronic, habitual exercise – the sparing of higher cognitive function in aging (Hillman et al., 2008; Kramer et al., 2006) – were being discovered at around the same time and this phenomenon would have presented clear problems for a mechanism solely based on neurochemistry. For acute exercise, on the other hand, the various arousal systems in the reticular formation remained the only option. Although they never really worked for emotions, they continued to be used for cognition because two decisive factors kept on being confounded. First, no clear distinction was made between 'during' and 'shortly after' exercise, a difference that is, given the data, monumental for the brain (e.g., Ide and Secher, 2000). So, even when executive functions were more clearly separated from other cognitive processes and tested for in exercise studies, the perception that there probably is not a great difference between the 'during' and 'after' conditions in terms of mental effects prompted researchers to investigate this link post-exercise only, mostly because it makes test administration easier and because many researchers are only interested in post-exercise effects anyway – in school settings, for instance. Since executive functions are no longer impaired at that point – on the contrary, they seem to be improved – such protocols could not tease out the problems with the monoamines as the sole, all-in-one neural explanation for the interaction between exercise and cognition. Second, no clear distinction was made between explicit and implicit processes, although this was a quite well established division in cognitive psychology and neuroscience. But it is only in the combination of explicit processes being tested for during exercise that we can see that an arousal mechanism, even in its various manifestations, falls short of accounting for all the effects of exercise on cognition. A physiological explanation, the transient hypofrontality theory, has since been added to the list of possible brain mechanisms as a result.

It follows that the reticular-activating process, as we call this group of arousal mechanisms in our RAH model, is mostly restricted to the facilitation of bottom–up, implicit processes during, and possibly shortly after, exercise. To repeat, it is not a powerful mechanism to explain the exercise-induced changes to explicit cognition, emotional processes, mental status, or mental ability in the elderly. When seen from an evolutionary angle, this makes sense because the phylogenetically ancient arousal systems evolved to aid decision-making in the phylogenetically older implicit system in situations where only the quick make it to the next round. It did not evolve to affect the explicit system, which our ape ancestors did not have or to protect mental functions in old age, which our ape ancestors did not reach. This is not, of course, a sound evolutionary argument, but it shows, at a minimum, that the extension of the reticular-activating process to explain exercise-induced changes in explicit processing requires additional theoretical grounds.

As a neural mechanism for implicit, stimulus-driven task performance during exercise, however, catecholaminergic arousal is well established and relatively uncontroversial. We therefore allow ourselves to keep this section concise, as the only new proposal it contains is that the reticular-activating process should not be applied beyond implicit processing with respect to explaining the facilitating effects of acute exercise on cognition. The same goes for the two cognitive-energetic models exercise scientists most commonly draw on to explain the facts; we keep their discussion brief, for the same reasons. That is, they remain relatively uncontroversial when applied to implicit processing. For explicit processing, however, they can no longer be used without accommodation. The reason for this is rooted in the same cause, the failure to come to terms with the consequences of the explicit/implicit distinction, especially for bodily motion.

3.1. Arousal and activation during exercise

The reticular-activating system consists of several distinct but interrelated arousal systems that are differentiated by anatomy (specific nuclei and their projections), neurotransmitter (norepinephrine, dopamine, and serotonin, mostly) and function (Robbins and Everitt, 1995). Briefly, the noradrenergic system orig-

Table 1
Two energetical mechanisms activated by an acute bout of aerobic exercise.

Mechanism	NT system	Origin	Main function	ERP index
Arousal	Noradrenaline	Locus coeruleus	Filtering inputs	P300
Activation	Dopamine	Substantia nigra	Energizing outputs	CNV

Note: ERP: event-related potential; P300: positive potential; CNV: contingent negative variation.

inates from the locus coeruleus and projects profusely throughout the forebrain (Table 1). It mediates alertness and appears to be involved in detecting sensory signals and maintaining discrimination processes under high levels of arousal and stress (Berridge and Waterhouse, 2003; Pribram and McGuinness, 1975; Ramos and Arnsten, 2007; Robbins and Everitt, 1995). Stimuli – in any sensory modality – increase the activity of neurons in the locus coeruleus (Aston-Jones and Bloom, 1981; Grant et al., 1988; Rasmussen et al., 1986). Such fluctuations in noradrenergic activity can be detected in cortical electroencephalogram (EEG) patterns and event-related potentials (ERP) (Niewenhuis et al., 2005; Pineda et al., 1989). Increases in noradrenergic transmission are thought to improve the signal-to-noise ratio by enhancing the evoked response, suppressing the background activity, or both (Hurley et al., 2004; Moxon et al., 2007; Waterhouse and Woodward, 1980). The dopaminergic system originates from cell bodies located in the substantia nigra pars compacta and from the ventral tegmentum. It projects to (1) the dorsal and ventral striatum, which, in turn, modulate activity in a large network involving the motor thalamus, SMA, premotor area, and primary motor cortex; (2) several structures in the limbic system, such as the nucleus accumbens; and (3) cortical regions, particularly the prefrontal cortex. Together, these pathways activate or energize behavior and account for the vigor and frequency of behavioral outputs (Robbins and Everitt, 2007) (Table 1). The serotoninergic system originates from cell bodies located in the raphe nucleus and projects from there widely throughout the brain and spinal cord. This system appears to moderate the stimulating effects of catecholamine activity and thus promotes behavioral inhibition and cortical deactivation (Meeusen et al., 2006; Robbins and Everitt, 2007).

A large body of evidence shows that acute exercise activates all three of these monoamine systems (e.g., Meeusen and De Meirleir, 1995). It is not surprising, then, that they have figured prominently in mechanistic explanations of the effects of exercise on cognitive and emotional processes. The link between exercise, arousal, central catecholamines and improvements in cognitive performance is based on the idea that exercise is an arousing stressor and supported by the following set of findings from animal and human research: (1) synthesis of noradrenaline increases in the rat brain during strenuous and prolonged exercise, especially if forced; (2) concentration of plasma catecholamines increases during exercise; (3) brain noradrenergic activity increases during cortical activation; (4) the level of cortical arousal is related to the level of activity in the locus coeruleus; and (5) exercise can increase the activation of the reticular formation via somatosensory feedback provided from limb movements (see Cooper, 1973; McMorris et al., 2008). Today, the arousing effects of exercise, on peripheral and central systems, are well documented. Acute exercise also activates both the sympathetic nervous system and the hypothalamo–pituitary–adrenal axis, which results in the release of catecholamines and indolamines, both centrally and peripherally (e.g., Meeusen and De Meirleir, 1995; Wittert, 2000).

An in-depth review of exercise-induced changes in brain monoamine systems in animals is beyond the scope of the article; but reviews are available elsewhere (e.g., Chaouloff, 1997; Dishman, 1997; Meeusen and De Meirleir, 1995). In humans, the various techniques to determine concentrations of central catecholamines are, for obvious ethical reasons, not possible. Here we must rely on estimates based on plasma or urine levels. For noradrenaline (NE), the metabolite 3-methoxy 4-hydroxyphenylglycol (MHPG) is used, which readily crosses the blood–brain barrier and is excreted in urine. Urinary MHPG, however, may also be derived from peripheral NE. Two forms of MHPG can be differentiated and the sulphate form is considered a good index of brain NE, while the β-glucuronide form is considered a more sensitive indicator of peripheral NE (e.g., Peyrin, 1990; Yao et al., 1997). Recent data suggests, however, that the sulphate metabolites are also derived mainly from NE release in the periphery (Goldstein et al., 2003). For dopamine (DA), the plasma concentration of the DA metabolite homovanillic acid (HVA) is used. Exercise studies measuring these metabolites in individuals are encouraging. One study shows that MHPG and HVA levels increase up to 40% of power output and then level off (McMorris et al., 2008), an effect that appears to mirror improvements in reaction time tasks (Audiffren et al., 2008). However, the source of MHPG is not clear because the above study failed to differentiate the sulphate from the glucuronide form. Another study, which did make this distinction, also showed a positive correlation between a simple discrimination task and levels of MHPG sulphate (Peyrin et al., 1987). When taken together with the animal literature, a link emerges indicating that brain catecholamines are involved in the improvements of performance on procedural and simple decisional tasks.

This is further supported by exercise studies using ERPs. Two ERP components are tpyically used: the P300 wave and the contingent negative variation (CNV). The P300 is a positive wave recorded around 300 ms after a response signal. It has two subcomponents, a P3a reflecting a stimulus-driven frontal attention mechanism during reaction processes and a P3b reflecting the allocation of attentional resources for memory updating in temporal and parietal cortices (Polich and Criado, 2006; Polich, 2007). Brain catecholamines would, presumably, contribute more to the P3a subcomponent. The CNV is a negative slow wave that takes place in anticipation of a response, that is, between the warning and the response signal. It also has two subcomponents, an early CNV, which indicates an orienting response (e.g., Loveless and Sanford, 1974) and a late CNV, which indicates motor preparation (e.g., Vidal et al., 1995).

If acute exercise increases arousal and activation, one would expect more resources to be available for stimulus-driven attention and motor preparation. This should result in larger P300a and late CNV amplitudes as well as a shorter P300 latency during exercise (Table 1). These predictions, however, have only partly been confirmed. Moreover, previous studies did not always differentiate the two subcomponents of each wave. While P300 amplitudes are indeed larger during exercise, P300 latency is, surprisingly, longer (Grego et al., 2004; Pontifex and Hillman, 2007). Also, and importantly, the P300 amplitude rise is only present during the first 2 h of a 3-h bout of cycling exercise (Grego et al., 2004). The matter looks different again post-exercise. Here the P300 amplitude is also higher, albeit only for light and medium intensities but not for high-intensity exercise (Kamijo et al., 2004b), while the P300 latency is shorter (Hillman et al., 2003; Kamijo et al., 2004b, 2007; Magnie et al., 2000). Again, these results underscore the need, as we do below, to categorically distinguish measurements, psychological or physiological, taken during from those taken after exercise, as large-scale bodily motion alters brain activity profoundly (Dietrich, 2008b). Finally, for CNV, exercisers show lower CNV amplitudes following high-intensity but not moderate-intensity exercise (Kamijo et al., 2004a). Clearly, more work is needed here before we can draw some firm conclusion.

3.2. Cognitive-energetic models

Cognitive psychology alone cannot explain the variability of mental performance in all environmental conditions, physical exercise being a prominent case in point (Hockey et al., 1986). To handle exercise adequately, energetic considerations must supplement cognitive models. Energetic psychology is more concerned with the intensive or energizing aspects of behavior as opposed to its directional or semantic aspects. Arousal and activation are concepts, of course, that were associated early on with energy mobilization or release within the organism (Duffy, 1962). Their relation to performance goes back even further, to the earliest days of experimental psychology and neurophysiology (Yerkes and Dodson, 1908). The famous U-shaped conceptualization linking performance to the degree of arousal has maintained a place in mainstream psychology ever since.

Like the RAH model, the inverted-U model can account for some of the opposing effects of acute exercise on cognition. According to it, performance is a curvilinear function of arousal, that is, under and overaroused individuals do more poorly while optimal performance is associated with a moderate state of arousal. Also, the peak of that function can shift in both directions depending on the type of task with simple tasks being less sensitive to arousal variation than complex ones. Humphreys and Revelle (1984) included the inverted-U idea into a wider model that separated different sustained information transfer and short-term memory processes. More recently, the inverted-U model has also been observed in dopaminergic modulation of prefrontal function. While low doses of D1 agonists improve performance on tasks tapping into prefrontal-dependent cognition, higher doses worsen it (see, Arnsten and Li, 2005).

Given the idea that physical exercise is an arousing stressor (Cooper, 1973; Davey, 1973), one would expect an inverted-U function between exercise and cognition (Kahneman, 1973; Näätänen, 1973), and this possibility is still being pursued in recent articles on the topic (e.g., Draper et al., 2010; Kashihara et al., 2009).

However, the empirical data has not lent support to this hypothesis (McMorris and Graydon, 2000; Tomporowski, 2003). Moreover, the inverted-U model is descriptive in nature; it does not contain, like the RAH model, an explanatory mechanism at either the neural or cognitive levels. However, as will be made clear in the next section, the most important shortcoming is the fact that it does not differentiate between different types of cognitive systems (i.e. explicit/implicit). In addition to arousal, it only considers task complexity and difficulty level.

Kahneman (1973) provided perhaps the first full cognitive-energetic model. This model regards the total amount of resources, which exist in a single, undifferentiated pool, as limited. The availability of resources depends on the level of arousal, which, in turn, is determined by two sources, task demand and several other sources, such as stimulus intensity, psychoactive drugs, anxiety, or, for that matter, exercise. The model postulates a so-called allocation policy mechanism that directs and supervises the allotment of resources, which, in turn, is determined by such factors as enduring dispositions, momentary intentions, or feedback from on-going activities. The level of arousal, then, corresponds to the amount of available resources and decrements in performance occur when task demands exceed the resource availability.

The Kahneman model assumes that there is a general, nonspecific pool of energetic resources that supports all cognitive functions. But such unitary concepts of arousal have outlived their usefulness (Robbins and Everitt, 1995). They cannot explain the data, such as, for instance, the low correlations among different measures of arousal (Eysenck, 1982; Thayer, 1989) or the perfect time-sharing of two resource-demanding tasks (Wickens, 1984). In addition, the neural substrate underlying arousal, the reticular formation, is not, as already stated, a homogenous system, but consists of several highly differentiated systems.

In response to such challenges, subsequent cognitive-energetic models of human performance shifted from a unidimensional conception of resources – one unique reservoir – to a multidimensional view that permits the operation of several different supply systems. In the model of Sanders (1983, 1998), probably the most commonly used by exercise scientists, there are three energetic mechanisms: arousal, activation, and effort. Each of these resource pools influences, at the cognitive level, a specific stage of information processing. On the basis of a large number of reaction time experiments and with the aid of the additive factors method (Sternberg, 1998), Sanders linked arousal to sensory and perceptual processes – the feature extraction stage, specifically – activation to the motor adjustment stage, and effort to response selection (Table 1). An acute bout of exercise can increase the arousal and/or activation mechanism, which can modulate input (sensory processes) and/or output (motor processes), respectively. Arousal and activation are two terms that are often used interchangeably but they can be, and should be, uncoupled. If this were not so an organism would be constantly aroused by its own movements and moved (activated) by the resulting arousing input. Activation differs from arousal, then, in that it affects motor processes by maintaining a tonic readiness to respond. One function of the effort mechanism is to unyoke arousal from activation so that not all arousing stimuli provoke motor activation. In the case of reflexes, they are, for good reason, forcibly linked.

3.3. Shortcomings of cognitive-energetic models

Both cognitive-energetic models are all built on the assumption that there is only one, single cognitive system. This makes it impossible, as we will see in more detail below, to explain the full range of psychological effects because exercise seems to affect the implicit and the explicit systems in opposing ways. This failure is not a problem with the models themselves, of course, as they all predate this advance in cognitive psychology, but with the fact that none of these models were developed further, or replaced by new ones, in order to reflect this knowledge. This is curious insofar as this putative distinction between explicit and implicit processing is well-known in the field. What has not percolated though, apparently, is the implication of this distinction in computational terms, despite the obvious relevance this has to an organism's ability to perform smooth, sensorimotor integration of the kind that characterizes sports and exercise.

The trouble starts, for both models, when we consider their effort mechanism, as it is here that we enter the explicit system's domain. The first case in point is a fairly new concept in cognitive science known as effortless attention and action (Bruya, 2009; Csikszentmihalyi and Nakamura, 2009; McGuire and Botvinick, 2009; Posner et al., 2009; Schmeichel and Baumeister, 2009; Wulf and Lewthwaite, 2009). A specific example of this emerging paradigm is the idea of flow (Csikszentmihalyi, 1996), a situation in which superior performance is associated with a decrease in mental effort and attention (Bruya, 2009; Dietrich, 2004a; Dietrich and Stoll, 2009). This phenomenon is well supported by the literature (Csikszentmihalyi, 1975; Csikszentmihalyi and Csikszentmihalyi, 1988; Dobrynin, 1966; Jackson and Csikszentmihalyi, 1999). Flow, like other phenomena of effortless perfomance, is a paradox, and remains impossible to explain for traditional theories of attention and mental effort for the simple reason that they assume that better performance, *even on a well-learned task*, is associated with increased conscious effort allocated to that task. The effort mechanism, in both the Kahneman and Sanders models, also assumes that higher task demands require more effort, both objectively, in terms of caloric consumption by the brain, and subjectively, in terms of

perceived, felt effort. In flow, however, the opposite is true. Here the mental effort decreases, sometimes to the point of total effortlessness, yet such seemingly automatic action is associated with improved performance (e.g., Bruya, 2009; Csikszentmihalyi, 1996; Posner et al., 2009). Put another way, an increase in task demands is met not by an increase in felt effort, but by a decrease. In fact, people describe their action in such a flow state as if it happens by itself, without any effort at all! This is a particularly common occurrence in sports (Jackson and Csikszentmihalyi, 1999). The flow phenomenon is not a paradox at all if we uncouple explicit from implicit processes (Dietrich, 2004a). In fact, improvements on *well-learned*, stimulus-driven tasks can only occur, according to our RAH model, if the explicit system, and thus effort, is decreased. Note that this is the exact opposite prediction than the one made by both of the above cognitive-energetic models.

Another case in point is the impairment on executive tasks during exercise. Neither the Kahneman nor the Sanders model predicts such a selective effect on specific types of cognitive processes, which is likely a prominent reason why this phenomenon was not well documented until a few years ago. To clarify, both models do predict performance impairments. However, in the Kahneman model, performance is said to decline when exercise and the cognitive task compete for resources. In the Sanders model, decrements occur when that competition is over effort. Either concept, however, fails to explain why the negative effects of exercise are selective for explicit processing. To save these predictions, one would have to make an additional assumption. That is, explicit processing *always* requires more effort and/or resources and it is exactly because of their higher difficulty level that they show impairments during exercise. This is probably true in most cases, which makes the above models able to account for a good deal of the data on explicit processing as well. The trouble is, however, that there are a few data points that just do not fit and it is typically such awkward facts that force a theoretical re-examination of the issue. Consider, for instance, the Dietrich and Sparling (2004) study, in which participants took two standard intelligence tests during exercise. These tests cost considerable mental effort and no participant reported that they were easier, or demanded less effort, than the heavily executive tasks they also completed. Evidently, both types of tasks required effort and concentration, to about the same degree; yet, there was a selective impairment for one type of task but not the other. Data like these cannot easily be explained by the Kahneman and Sanders models, which focus on resources and effort, respectively. While the theoretical framework of our RAH model leads us to slightly different predictions for some cases, note that the RAH model also makes predictions – for the flow experience, for instance – which are diametrically opposed to those of Kahneman and Sanders.

The reason for the shortcomings in these cognitive-energetic models is simply the fact that neither makes a distinction between the explicit and implicit system. This renders them unable to make specific predictions, aside from difficulty level, about how exercise affects cognition. To give another example, patients with prefrontal damage have great difficulty planning the course of a day. We can assume, therefore, that this sort of fluid, prospective planning requires substantial amount of executive processes (Shallice and Burgess, 1991). Yet, healthy individuals do this rather effortlessly? Our RAH model predicts that this task would be impaired during exercise, a prediction other cognitive-energetic theories do not make, given that this task does not appear to consume much felt effort. The models of Kahneman and Sanders, as well as other such theories (e.g., Humphreys and Revelle, 1984; Hockey, 1997), make several more predictions that are also off the mark as soon as we factor into the equation the state of knowledge in cognitive neuroscience. The RAH model, on the other hand, is not only informed by current understanding of (1) neurophysiology, the existence of several different arousal systems, (2) cognitive psychology, the existence of two different cognitive systems, but also (3) functional neuroanatomy, a field from which we can draw several predictions about how exercise, given the neural activation pattern it produces, affects mental function. This last advantage of the RAH model becomes vividly clear when we consider emotions in a later section, which none of the above cognitive-energetic models can adequately explain.

3.4. Localizing the facilitation effect

For implicit processing, the previous cognitive-energetic models, especially that of Sanders, can help us localize the facilitating effects of exercise on reaction time and simple decisional tasks in the cognitive architecture of the implicit system. Information processing here is separated into sensory, perceptual, decisional, and motor stages (Sanders, 1983, 1998). Setting aside the decisional stage, for the above mentioned reasons, empirical data suggest that steady-state aerobic exercise does not affect all of these stages (Audiffren, 2009).

There are several ways of doing this. The additive factors method, for instance, is based on the discrete serial information-processing model (Sternberg, 1998). It considers reaction time to be the sum of the duration of all processing stages that take place between the occurrence of the stimulus and the initiation of a response. For each stage, at least one computational factor exists that directly and selectively affects reaction time duration without modifying processing quality. It follows from this that when two factors do not influence any stage in common, one can expect their effects to be additive. When two factors influence at least one stage in common, one can expect their effects to interact in an overadditive manner. In the Sanders (1983, 1998) model, four stages are distinguished from the robust pattern of additivity among four computational factors: (1) a preprocessing stage influenced selectively by signal intensity; (2) a feature extraction stage influenced by signal quality; (3) a response selection stage influenced by stimulus-response compatibility; and, finally, (4) a motor adjustment stage influenced by the foreperiod duration. The additive factors method allows us to localize the effects of acute exercise on implicit tasks within this sequence of information-processing stages. Empirical data collected using this framework suggest that the facilitating effects of acute exercise on mean reaction time occur at the level of motor processes (Arcelin et al., 1998).

We obtain a similar result by fractionating the reaction time. This can be done by dividing the electromyographic (EMG) activity of the agonist muscle into two components: the time interval between the response signal and EMG activity onset, termed premotor time (PMT) and the time interval between EMG activity onset and motor response onset, termed motor time (MT) (Botvinick and Thompson, 1966). MT reflects the duration of the electromechanical transduction within muscular fibers, while PMT reflects the duration of all earlier stages. By separating PMT from MT, it is possible to determine whether the facilitating effects of acute bouts of exercise on reaction time occur before or after the onset of EMG activity and, therefore, whether it influences early cortical integration processes or late motor processes (Hasbroucq et al., 2001). Fractionating reaction time this way, several studies have confirmed that acute exercise influences motor processes (e.g., Audiffren et al., 2008; Davranche et al., 2005, 2006).

Finally, a variety of methods are available that permit us to determine whether performance improvements in a sustained information transfer task (Humphreys and Revelle, 1984) also come about through the modulation of sensory processes. In the flicker fusion task, for instance, exercise is known to enhance flickering or fusion detection (Davranche and Audiffren, 2004; Davranche et al., 2005; Davranche and Pichon, 2005; Lambourne et al., 2010)

and contrast sensitivity (Woods and Thomson, 1995). In a signal detection paradigm, exercise enhanced sensory sensitivity by increasing the value of d' (Audiffren et al., 2007). The sum of the evidence shows that the facilitating effects of acute exercise on such data-driven, bottom–up processing is modulated by arousal and activation, that is, the reticular-activating process speeds up reaction times by enhancing early sensory as well as motor processes. This mechanism, however, cannot explain all the data on tasks requiring more executive processes.

4. The hypofrontality process

The fourth section of the paper delineates the hypofrontality process, which has mental effects opposite to those induced by the reticular-activating process. The flexibility/efficiency tradeoff between the explicit and implicit system is the very bedrock on which any type of mechanistic explanation of human motor performance must be anchored. What is critical here is not only the view that both systems have evolved for fundamentally different purposes and thus carry out fundamentally different functions, but the interaction between them, that is, the understanding that flexibility precludes efficiency and vice versa (see also Fig. 1). This vividly exposes the error in thinking that exercise can enhance both processes at the same time. From an evolutionary point of view, a hypofrontality process of some kind must occur in a pressure situation involving physical motion; it simply is not adaptive to engage higher-order executive processes, at least not to the extent humans could, in a do-or-die situation. Without factoring in this tradeoff, the idea of hypofrontality does indeed seem to violate common sense. With this in place, we now turn our attention to the actual neural mechanism inducing the hypofrontality process.

Although evidence for a transient downward shift in activity in prefrontal regions during exercise has been accumulating for decades, in animals and humans, and from several directions, the general idea that exercise could deactivate brain areas, let alone that such decrease in brain activity could explain some of the psychological data, was so completely against the accepted wisdom of the time that even when the dots were connected in the form of the transient hypofrontality theory (Dietrich, 2003, 2006) many people tended to dismissed the possibility. This was due, for the most part, to the fact that the hypofrontality concept ran counter to a number of widely held but mistaken beliefs about the effects of exercise on brain health and, by extension, mental function. Perhaps the most harmful of these were – actually, still are: (a) exercise increases blood supply to the brain and, therefore, oxygen and glucose uptake; (b) bodily motion is taxing for muscles, no doubt, but it is not something that forces the brain into its computational reserves; and (c) that improvements to mood and cognition must surely come from a mechanism that boosts neural activity, a belief so pervasive that it has been called the activation-is-good fallacy (Dietrich, 2009). It simply did not make much sense that all those creative ideas and positive feelings one experiences while exercising can come from a process that tends to deactivate the pinnacle of human evolution, the prefrontal cortex.

The central idea behind the hypofrontality process is that the brain is forced, during exercise, to make profound changes to the way it allocates its metabolic resources (Dietrich, 2003, 2006). A cornerstone of cognitive science holds that we have a limited information-processing capacity at the bottleneck of consciousness. But there also exists a total cap on the total amount of information processing – unconscious and parallel, that is – that can occur at any one time. In the case of exercise, the enormous demands on motor, sensory, and autonomic structures, powered by the reticular-activation process, result in fewer resources available for processes, cognitive or emotional, that are not involved directly in maintaining the motion (Dietrich, 2003, 2006; Nybo and Secher, 2004). The reality of such local, need-based shifts in cerebral metabolism is powerfully demonstrated in every neuroimaging study; indeed, they serve as the very rationale for functional neuroimaging in general.

The problem arises when we fail to understand motion as a biocomputation of the highest order. If this failure is compounded by the mistaken notion that the brain is the recipient of additional resources – blood, oxygen, glucose, or otherwise – during exercise (see Astrand and Rodahl, 1986; Ide and Secher, 2000), it is impossible to grasp the pivotal resource limitation the brain must solve during exercise. Motor control in general just is simply not a minor part of the brain's daily chores and when it comes in a sustained manner involving large muscle groups, as it does in sports and exercise, large-scale changes occur as a result. For this reason, we first offer a few crutches for the imagination, to help with the more counterintuitive claims of the hypofrontality process before we then proceed with a brief summary of the evidence showing that the simple act of, say, running activates vast areas of the brain and thus requires the redistribution of much of its resources.

4.1. Three intuition pumps

In artificial intelligence, motion has long been recognized as a gigantic *computational* problem. Human artificers have built machines that make the number one world ranking in chess readily; yet, they are not even close to make a robot that walks nicely on two feet, let alone one that makes a decent tennis serve. It certainly is not because they cannot make the moveable equipment – arms, legs, joints, etc (the main problem seems to be balance – Kuo et al., 2005). The reason is that sensorimotor integration, in real time, requires an astronomical amount of number-crunching. Even for the simple act of walking, the brain must control an uncountable number of muscle fibers to precise specification, with every twitch affecting the strength of the contraction of the next. This is computationally, and thus metabolically, very costly, even when the movement is controlled mostly by lower brain centers. Programming this into a robot has yet to be done successfully (Kuo et al., 2005). We still must await the time when the first two-legged robot is the number one tennis player in the world.

Next, consider, the brain's motor system. By simply listing the number of structures devoted to movement you can get an appreciation of the complexity of moving the body around: primary motor cortex, secondary motor cortices (i.e. premotor and SMA), basal ganglia, the motor thalamus, cerebellum, red nucleus, substantia nigra, the massive pathway systems, and the motor neurons all along the spinal cord, among rather many others. This represents not just an enormous amount of brain volume but also a very high number, in percentage terms, of neurons. Why, for instance, does the cerebellum have more neurons than any other structure in the brain, including the entire cerebral cortex! The cerebellum does the brunt work of fine motor coordination, the very thing for which brute computational power is so critically needed. Movement also occurs through space so any motor activity must integrate sensory processes and soon we are at yet another, nearly equally long list of brain structures that must be activated in order to process the relevant perceptual information during exercise. Add to this all the nuclei mediating autonomic regulation such as, for instance, in the hypothalamus, the reticular formation, and many nuclei in the medulla, and we arrive at a long list of brain regions that must be activated for nothing other than the simple act of running!

To end, let us try a third, more sensitive intuition pump. The male human brain is about 150 g heavier than the female one. It is universally understood that this is due to the male's higher body mass. But consider what this really means. The male brain has, on average, 8–10% more brain mass only so that he can throw around

what amounts to no more than a few pounds of body mass. It is hard to believe that moving around a few more pounds of muscle and bone requires so much more brain mass, especially in percentage terms, given that we are animals who are already copiously equipped with neuronal mass. But it does. Also keep in mind that the human motor system is more highly evolved than that of other animals. Animals with much smaller brains can produce very complex movements, movements we find extraordinary, but what they cannot do is learn motor acts for which they are counterprepared, let alone to such a state of perfection the way humans can. Just think of our ability to swim butterfly, pole vault, or play the violin, all actions we are not designed to perform. Again, these arguments are not sound evidence as far as neuroscience goes; we simply offer them here so that we can start thinking of movement in terms of its neural costs.

4.2. Review of the evidence

Several techniques such as ^{133}Xe wash out, radioactive microsphere, and autoradiography as well as EEG, SPECT, and PET, have been used to measure brain activity during exercise. Converging evidence from these studies indicates that exercise is associated with profound regional changes in motor, sensory, and autonomic regions of the brain. Marked increases in activation occur in neural structures responsible for generating the motor patterns that sustain the physical activity. In particular, the primary motor cortex, secondary motor cortices, basal ganglia, cerebellum, various midbrain and brainstem nuclei, motor pathways, as well as several thalamic nuclei are involved. In addition, exercise activates structures involved in sensory, autonomic, and memory function, particularly primary and secondary sensory cortices, sensory pathways, brainstem nuclei, hypothalamus, and the sensory thalamus. Cerebral blood flow (CBF) and local cerebral glucose utilization (LCGU), both indexes of the functional activity of neurons, have confirmed this pattern of neural activity in exercising animals (Gross et al., 1980; Holschneider et al., 2003; Sokoloff, 1991; Vissing et al., 1996). Taken together, these neural regions represent a substantial percentage of the total brain mass confirming that physical exercise requires massive neural activation in a large number of neural structures across the entire brain. It follows that prolonged, aerobic exercise would require the sustained activation of this large amount of neural tissue.

Physiological data on human brain activity during exercise, though remarkably sparse, consolidate, not surprisingly, the data in the animal literature. The various methods used to map neural activation patterns in animals cannot be used in humans for obvious ethical reasons and most neuroimaging techniques are also not viable options because they preclude head movement (Dietrich, 2008a). In addition, it is also not possible to scan subjects – with fMRI, for instance – immediately following exercise. Neuroimaging studies show that the pattern of neural activation associated with a particular task rapidly returns to baseline levels after the cessation of that task. Indeed, this is the very theoretical basis of interpreting neuroimaging studies; the temporal association of task performance with brain activation. This suggests that a delay of even a few seconds would be sufficient to start normalizing any exercise-induced regional changes in neural activity. In other words, dualism aside, if brain activation did not change once a person stops moving, the person would still be moving (Dietrich, 2008b).

There are ways around this, through, and in one PET study, increased brain activation was recorded in the "primary sensory cortex, primary motor cortex, supplementary motor cortex as well as the anterior part of the cerebellum" in response to cycling (Christensen et al., 2000, p. 66), while a single photon emission computed tomography (SPECT) study found increases in regional CBF in the supplementary motor area, medial primary sensorimotor area, the striatum, visual cortex, and the cerebellar vermis during walking (Fukuyama et al., 1997). These and other studies are still limited, as evidenced by a comparison with the brain activation seen in rCBF studies in animals, because they are based on keeping the head as still as possible. However, this requires the whole body to move as little as possible. As we will see later, this is a fatal confound. The resource limitation issue of motion largely depends on the amount of muscle mass moved as well as the intensity and duration it is moved, with whole-bodily motion at a strenuous intensity being an entirely different matter than cycling, in supine position, for a few minutes at an intensity so low that subjects manage to keep the head still enough to produce artifact-free images in the brain scanner. For the same reason, brain activation measured by neuroimaging studies using mental imagery as a substitute for real motion bears little resemblance to the brain activation present in individuals that are actually in full motion (Dietrich, 2008b).

Another way around this entire problem is the 18 fluorodeoxyglucose (FDG) PET technique. 18 FDG allows for functional mapping of brain areas during motion because the glucose uptake occurs during the movement. Because this compound is not readily metabolized by neurons, it stays fixed long enough until the scanner can detect the regional changes of glucose uptake later. In other words, the main disadvantage of PET, compared to fMRI – its poor temporal resolution – can be used to our advantage here. This allows one to produce a functional index of the moving human brain. So far, two studies have done this (Kemppainen et al., 2005; Tashiro et al., 2001). Both confirmed the massive brain activation that occurs as a function of large-scale bodily movement. Importantly, they found, as predicted by the THT, general cortical deactivation and, specifically, highlighted the deactivation in prefrontal regions (Tashiro et al., 2001) and the link of this prefrontal hypometabolism to fatigue (Kemppainen et al., 2005). In both cases, subjects ran for a mere 5 and 10 min, respectively, and the THT predicts that this deactivation would become ever more severe with longer exercise duration. Such a study has yet to be done, though.

Additional evidence in support of the THT comes from EEG studies, some of which are already, as mentioned, decades old. EEG data have consistently demonstrated that exercise is associated with alpha and theta enhancement, particularly in the frontal cortex (Boutcher and Landers, 1988; Kamp and Troost, 1978; Kubitz and Pothakos, 1997; Nybo and Nielsen, 2001; Petruzzello and Landers, 1994; Pineda and Adkisson, 1961; Yagi et al., 1999; Youngstedt et al., 1993). An increase in alpha activity is a putative indicator of decreased brain activation (Kubitz and Pothakos, 1997; Petruzzello and Landers, 1994). For instance, Kubitz and Pothakos (1997, p. 299) concluded that "exercise reliably increases EEG alpha activity", while Petruzzello and Landers (1994, p. 1033) stated that "there was a significant decrease in right frontal activation during the post-exercise period." In a more recent study correlating EEG with the rating of perceived exertion (RPE), Nybo and Nielsen (2001) recorded from three placements (frontal, central, and occipital cortex) during submaximal exercise and found that "altered EEG activity was observed in all electrode positions, and stepwise forward-regression analysis identified core temperature and a frequency index of the EEG over the frontal cortex as best indicators of RPE" (p. 2017). This finding suggests that exercise is not only associated with decreases in frontal activity but also that the degree of physical effort might be correlated with the severity of frontal deactivation.

When reading these, in part 30 years old, EEG articles, one is struck by the extent to which the authors struggled to explain their electrophysiological results. Not a single one concluded, presumably because of what they falsely thought this meant for mental health, that exercise deactivates neocortical regions. It just did not make sense, though the evidence was right there.

Unlike EEG, ERP studies of executive functions have rarely been used in conjunction with exercise. The anterior cingulate cortex (ACC) is of interest in this context because it has been associated with cognitive control and internal conflict monitoring (van Veen and Carter, 2002a). ERPs and fMRI studies suggest that the activity of the ACC increases in choice reaction time tasks during error trials immediately following the wrong response (Carter et al., 1998; Kiehl et al., 2000), but also in inhibition tasks (Go/No Go) and conflict tasks (Eriksen flanker task) prior to a response on correct incongruent trials (van Veen and Carter, 2002b). Two event-related potentials are supposed to reflect response conflict detection by the ACC: (1) the error-related negativity (ERN), observed 50–150 ms following the occurrence of a fast, impulsive error and (2) the frontocentral negativity (N2) observed around 200 ms after a stimulus that elicits a response conflict. The amplitude of these two waves is considered an index of efficiency for cognitive control and conflict monitoring. Given this, we would predict that hypofrontality induced by exercise leads to a decrease in N2 and ERN amplitudes.

On the basis of Lacey's principle of directional fractionation (Lacey, 1967), acute exercise should not be expected to uniformly increase physiological parameters, such as cortical activity (e.g., amplitude of ERPs), sympathetic activity (e.g., heart rate) or somatic activity (e.g., amplitude of EMG burst in muscles). The RAH model predicts that, while exercising, some systems respond with an increase (e.g., in P300 and CNV amplitudes) due to noradrenergic and dopaminergic activation, while others respond with a decrease (e.g., in N2 amplitudes) due to the deactivation of prefrontal areas.

In addition, near-infrared spectroscopy (NIRS) has been used to limn the neurophysiological changes that accompany exercise (Perrey, 2008). Studies using this tool have also largely supported the predictions of the THT; that is, strong decreases in HbO_2 (oxy-hemoglobin), the main neurovascular index for cortical oxygenation, have been reported in most NIRS studies, especially for exercise of longer duration and/or subjects close to exhaustion (Bhambhani et al., 2007; Ide et al., 1999; Racinais et al., 2007; Rupp and Perrey, 2008; Timinkul et al., 2008). For the initial stages of exercise, however, at least two studies have reported an initial increase in prefrontal HbO_2 (Rupp and Perrey, 2008; Timinkul et al., 2008). In other words, it appears that prefrontal oxygenation "increased in the first few minutes of exercise but decreases markedly from the workload corresponding to the second ventilatory threshold up to exhaustion" (Rupp and Perrey, 2008, p. 153). The initial increase of prefrontal oxygenation for the first few minutes reported by these two NIRS studies stands in contrast to the PET, SPECT and CBF/LCGU studies showing decreased prefrontal activity, even for the initial phases of exercise. It is most likely, however, that the NIRS data for the first few minutes of exercise are simply due to a novelty effect. Unlike 18FDG PET for instance, wearing a NIRS apparatus feels a bit odd at first, an effect that increases with the onset of movement. In addition, although NIRS is somewhat tolerant of motion, it is still necessary for the subject to keep the head as still as possible to reduce the occurrence of motion artifacts. Running like this, much like running with brand new shoes, is likely to activate attentional mechanisms in the beginning, an effect that then subsides as the runner gets used to the new situation. This confound can easily be controlled for by habituating subjects to the novel exercise environment prior to taking measurements.

Single cell recording in exercising cats has also provided support for decreased activation in prefrontal regions. Recording from 63 neurons in the prefrontal cortex, units associated with the control of the movement showed increased activity during locomotion, while other prefrontal units decreased their discharge (Criado et al., 1997).

And, finally, there is the evidence we mentioned above showing that tasks involving executive processes tend to be impaired during exercise (Adam et al., 1997; Audiffren et al., 2009; Davranche and McMorris, 2009; Del Giorno et al., 2010; Dietrich and Sparling, 2004; Mahoney et al., 2007; Paas and Adam, 1991; Pontifex and Hillman, 2007). To these data we must add the largely anecdotal evidence that long, physical exercise – ultramarathons, for instance – can profoundly alter mental status, including hallucinations, the loss of ego boundaries, and a sense of timelessness (Dietrich, 2003; Dietrich and McDaniel, 2004). In sum, the weight of the animal and human literature permits the conclusion that the pattern of neural activity during aerobic exercise should be regarded as a state of generalized brain activation with the specific exclusion of the executive system in prefrontal and other cortical regions.

4.3. Implications for emotions

According to the THT, an exercising individual enters, sooner or later, a mental state marked by a transient decrease in prefrontal function. Less neural activity in the prefrontal cortex does not mean, however, that a runner operates at the cognitive level of a prefrontal patient. The effects are much more subtle, for most exercise modes at least, and are perhaps best seen in the consequences of acute exercise on emotions. It is here that the explanatory power of the hypofrontality process comes into full view because the reticular-activating process, or the cognitive-energetic models of Kahneman (1973) and Sanders (1983, 1998) cannot accommodate these data. Nor can the effect on mood states be accounted for, as is commonly believed, by the activation of the opioid or serotonin systems (Dietrich and McDaniel, 2004).

Several anxiety and depressive disorders show evidence of hyperactivity in some brain regions. In obsessive compulsive disorder (OCD) for instance, the ventromedial prefrontal cortex (VMPFC), which has been implicated in complex emotions, exhibits widespread hypermetabolism (Baxter, 1990; Baxter et al., 1987), while individuals with post traumatic stress disorder or phobia show hyperactivity in the amygdala (LeDoux, 1996). Given the analytical, emotional and attentional capacities of the prefrontal cortex, the excessive activity is thought to generate a state of hyper-vigilance and hyper-awareness leading to anxiety. PET studies reveal a similar picture for depression, which is also marked by hyperactivity in the VMPFC and the amygdala (Mayberg, 1997; Mayberg et al., 1995). Conversely, the dorsolateral prefrontal cortex (DLPFC), which is associated with higher cognitive functions, shows less than normal activity in depression, depriving the individual of the higher cognitive abilities that might help mitigate the negative mood. Treatment with serotonin reuptake inhibitors results in a normalization of the malfunctioning of this complex prefrontal circuitry (Mayberg et al., 1995), pointing to an abnormal interaction between the VMPFC and the DLPFC rather than global prefrontal dysfunction (Starkstein and Robinson, 1999). Interestingly, healthy subjects asked to think sad thoughts show a similar pattern of activity (Damasio et al., 2000). Considering the similarities in brain activation, it is not surprising that OCD patients frequently develop comorbid major depression, and that the treatment of choice for both disorders is serotonin reuptake inhibitors (Starkstein and Robinson, 1999).

Before the advent of neuroimaging, prior to the mid-1990s perhaps, it was not known that affective states, such as depression and several of the anxiety disorders, are accompanied by *excessive* activity in prefrontal and limbic regions. Exercise scientists, following mainstream neuroscience, tried to account for the effects of exercise on emotions almost exclusively in neurochemical terms, in no small measure, no doubt, due to the success of neuropharmacology in treating these disorders (Schatzenberg and Nemeroff, 2009). The functional neuroanatomical components of the pathology of affective disorders were never integrated into the interaction between exercise and mental health.

With the concept of exercise-induced hypofrontality, however, a novel neural mechanism by which exercise might be beneficial to mood becomes immediately apparent. To spell it out, the massive neural activity caused by the large-scale bodily motion, coupled with the brain's finite metabolic resources, makes it impossible for the brain to sustain excessive neural activity in structures, such as the prefrontal cortex and the amygdala, that are not needed at the time. As the brain must run on safe mode the very structures that appear to compute the information that engender stress, anxiety, and negative thinking in the first place, we experience relief from life's worries. In other words, without enough metabolic resources to activate the very brain regions that can possibly figure out what, say, today's stock market crash does to your retirement fund, such anxieties are simply less likely to reach consciousness. It is, so to speak, a budget crunch that causes specific phenomenological subtractions (for details, see Dietrich, 2003, 2006).

4.4. Opioids and lipids

Despite our ambition to advance an integrative and comprehensive framework, we acknowledge that there are a few mental phenomena accompanying exercise that fall outside the explanatory purview of either the reticular-activating or the hypofrontality process. Most prominently among them are, perhaps, analgesia and sedation as well as the much rarer occurrence of euphoria (Dietrich and McDaniel, 2004). In light of the fact that whole body motion engages so many different bodily systems, it should come as no surprise that exercise also elicits many different kinds of compensatory mechanisms. The suppression of pain sensations, a sense of calm and well being, as well as the occasional experience of a euphoric state, especially among endurance athletes, has long been ascribed to endorphin release, but convincing evidence for this possibility has only been supplied very recently (Boecker et al., 2008). In addition, there is also evidence that the endocannabinoid system is involved (Sparling et al., 2003). To what extent these mental phenomena are mediated by opioid and lipid neurotransmitters is not clear, but there is, at present, no alternative explanation for them.

5. The formal model

The RAH model is based on three fundamental energetic principles in neuroscience:

1. The brain receives a constant and limited supply of metabolic resources. Although local changes in energy utilization occur, global cerebral metabolism is stable.
2. Bodily motion is a biocomputation of the highest order. Movement requires, therefore, a substantial allocation of metabolic resources to those brain regions – motor, sensory, and autonomic – that control the movement. The exact amount of the increase depends primarily on two factors: (a) the quantity of muscle tissue involved in the motion and (b) the intensity of the motion.
3. Neural processing occurs on a competitive basis. Given the global cap on resources, local increases in neural activity in some brain areas must be offset by concomitant decreases in others. In the case of movement, the brain must shift metabolic resources, given their limited availability, to neural structures that sustain the movement, which leaves fewer resources for brain regions computing functions that are not critically needed at the time.

From these three basic principles, we can derive the following set of corollaries. The computational cost of controlling motion is inherently high. This already high cost increases even further as a function of three parameters. First, the more muscle mass is involved the higher is the cost. Physical exercise requiring large muscle groups, then, necessitates a greater shift of metabolic resources than exercise that does not. According to the rate law – intensity is coded by the rate of neuronal firing – a similar positive relationship holds for the second parameter, intensity. Exercise of higher intensity, then, creates a greater need for reshuffling than exercise of lower intensity. It would follow from this that exercise of maximum intensity involving the whole body generates the highest possible metabolic demands in brain regions associated with the exercise and thus forces the greatest possible downregulation in unrelated brain regions. Intensity, however, invokes a limiting factor, the cardiovascular system. Exercise at intensities above the anaerobic threshold cannot be maintained at the muscular level. Accordingly, anaerobic exercise, despite the massive neural changes it brings about, is unlikely to have profound consequences for overall brain function for the simple reason that it is unsustainable, which makes any ensuing psychological effects, albeit intense, limited in time. This introduces the third parameter, duration. The longer the duration of the exercise the longer is the brain required to maintain the lopsided allocation policy that draws resources away from neural areas not pertinent to the exercise. In other words, the longer the exercise the more profound is the associated downregulation in these neural areas.

Cerebral perturbation is greatest, therefore, when the exercise involves the entire body and is performed for an extended period at the highest intensity possible for that time frame. A marathon run at, or just below, the anaerobic threshold is a good example. It is this physical workload that exacts the heaviest, overall toll on the brain's energy supply, causing a reallocation that is heavy in magnitude and long in duration. Naturally, it is also this physical workload that has the most profound consequences for emotional and cognitive processes.

The RAH model is based on just three more fundamental principles at the neurocognitive level, which, together, explain the psychological implications of the model:

4. Brain catecholamines and indolamines mediate arousal in humans and modulate information processing. Exercise is an arousing stressor that activates these brain monoamine systems.
5. The brain operates two largely independent cognitive systems: the implicit and explicit systems. The key insight, with respect to the RAH model, is the flexibility/efficiency tradeoff, which flows readily from an evolutionary analysis that considers the different formats each system uses for knowledge representation.
6. When the brain is taxed by exercise, or overtaxed by prolonged, strenuous, full-body motion, the resulting downregulation in brain areas irrelevant to the motor task progresses from areas supporting the highest cognitive functions, down the functional hierarchy, one phenomenological subtraction at a time, to areas supporting basic ones. The prefrontal cortex, being the most zenithal higher-order structure, is the first region affected by the heavy metabolic burden of exercise, making its computations less likely to be supported sufficiently to figure in phenomenology or any subsequent decision-making process.

On the basis of this limited set of six fundamental principles, we can derive a number of predictions at different operational levels about the neural and psychological effects of acute exercise. At the neurophysiological level, we predict, for instance, that:

1. The inherently high computational requirement of motion is exacerbated by three exercise parameters: muscle mass, intensity, and duration.

Corollary 1.1. The larger the muscle mass involved, (a) the higher is the metabolic need in exercise related brain areas, (b) the greater

is the overall shift in resources, and (c) the more profound is the concomitant downregulation in unrelated brain areas.

Corollary 1.2. The same three-step, relationship also holds for exercise intensity.

Corollary 1.3. The same three-step, relationship also holds for exercise duration.

Corollary 1.4. When combining considerations of intensity and duration, an additional variable must be factored in. Intensity sets a limit to duration, making it necessary to modify the simple relationship that we predict holds for each parameter in isolation. We predict that the greatest perturbation of the cerebral metabolism, in terms of magnitude and time, occurs for prolonged exercise at the anaerobic threshold.

Corollary 1.5. The compensatory downregulation of brain areas not involved in the exercise task advances from the topmost layers of the functional hierarchy, in a kind of onion-peeling process, towards more basic ones. It follows that areas of the prefrontal cortex would show the strongest hypoactivity effect during exercise. The size of this effect depends on the interaction of the three exercise parameters as outlined above.

2. Physical exercise leads to the release of brain monoamines in humans.

   At the psychological level, we can make the following, additional predictions:
3. Implicit, procedural processes tend to be facilitated during exercise due to the increased activity of catecholamines.
4. Explicit or executive processes tend to be impaired during exercise due to the brain's compensatory response with respect to the allocation resources. For this effect, we can also predict the shape of the function. We expect that the three exercise parameters, as outlined above, aggravate the impairment of prefrontal-dependent processes.
5. Emotional dysfunctions that are caused by excessive neural activity in the prefrontal cortex and/or limbic structures – some types of depression and anxiety disorders, for instance – are alleviated by exercise. We expect that this effect is also a function of the three above exercise parameters, for the same reason and in the same manner.

   Still more predictions that flow from the above set of fundamental principles can be made at the psychophysiological level. For instance:
6. The amplitude of event-related potentials that are identified as indices of arousal or motor readiness (e.g., P300a or late CNV) increases for exercise compared to rest.
7. The indices of motor neuron efficiency (e.g., motor time or the amplitude of the EMG burst) increase for exercise compared to rest.
8. The amplitude of event-related potentials that are identified as indices of executive processes (e.g., N2 or ERN) decreases as a function of the interaction of the three exercise parameters.

   Finally, at the behavioral level, our RAH model yields the following predictions:
9. Performance on stimulus-driven, procedural, automatized, and/or choice reaction time tasks tends to be improved by acute exercise compared to rest. A rough approximation as to the kinds of tasks that qualify here, we predict improvements on neuropsychological tasks that are not sensitive to detecting prefrontal damage.
10. Performance on tasks with demands on executive functions tends to be impaired during exercise. This impairment varies as a function of two factors. First, the more significant a task's executive components, as, again, roughly indexed by prefrontal patients, the stronger, and earlier, it is impaired during exercise. Second, the impairment is made worse by the interaction of the three exercise parameters.
11. Scores on state anxiety inventories tend to be lower during, compared to post, exercise because the skewed distribution of resources is also more acute. Again, the effect size depends on the three exercise parameters.
12. In a task that allows for a separation of implicit from explicit processes – Jacoby's process dissociation procedure or tests of implicit and explicit memory – we predict that indices of implicit processes are improved by acute exercise while indices of explicit processes are impaired. Again, the three exercise parameters modulate this effect.
13. The extent to which a task is automated alters the exercise effect because the neural control of the task shifts from a more explicit representation to a more implicit one. Accordingly, the more a task is practiced the more likely it is facilitated by exercise. Indeed, we predict that it is possible that the same task, when newly learned, can be impaired by acute exercise and subsequently facilitated after it has been automated.
14. A derivative of the flexibility/efficiency tradeoff is that the participation of explicit processes in the execution of well-learned, procedural tasks tends to decrease their effectiveness. Put another way, increased mental effort directed towards such a task can have a detrimental effect on performance. We predict, therefore, that acute exercise facilitates such experiences of effortless attention and action because the strength of explicit processes is reduced, making them less likely to influence implicit execution.

6. Exercise and task parameters

In the sixth, and last, section, we consider how the RAH model accounts for a number of exercise parameters. The data on the interaction between exercise and psychological function appear to be prohibitively varied. Participants' tests scores go up, or down, or stay the same depending on whether they run, or cycle, or do some other thing; whether they do it in the heat, with coffee in their veins, or in the early morning; whether they are tested before, or during, or after exercise; whether they exercise hard, or long, or both; whether they are fit, or young, or have an anxiety disorder; and, of course, whether the task they are asked to complete involves reacting, or ignoring, or memorizing some sort of stimuli. The accumulated evidence, seen in its entirety, does not seem to yield consistent relationships, especially not of the straightforward kind – positive linear (Etnier et al., 2006) or inverted U (McMorris and Graydon, 2000; Tomporowski, 2003). Almost always, data sets in science are not that bewilderingly complex; such a state of affairs only prevails when a discipline has yet to identify the proper axes that would align the data just right. That no clean relationship appears to describe the effects of acute exercise on emotion and cognition is, we claim, the result of throwing the whole of the data into one pot and analyzing it *en masse*. Since there are, according to the RAH model, two opponent processes at play, some of the data may simply cancel themselves out.

We propose the RAH model because it explains and predicts a large part of the variation in the data. By organizing the data set along two opponent processes, the reticular-activating process and the hypofrontality process, we can disentangle the effects of several confounding factors that smooth the data into the amorphous hodgepodge it presently is. To start, we must differentiate, in a categorical manner, between (1) explicit and implicit processing rather than, as is traditionally the case, place cognitive tasks along a unidimensional continuum of task difficulty or mental effort and (2) a person who is in motion and one who is not, as for the brain this is a difference that could not be any bigger. It is these two factors

Table 2
Acute versus chronic exercise.

Type of effect	Mode of exercise	Type of change	Type of brain mechanism
Acute effect	Single bout of exercise	Transient	Physiological: modulation in the activity of neural networks
Chronic effect	Regular exercise	Durable	Anatomical: morphological changes in the brain structure

that allow us to start unwrapping the complexities we have been keeping clamped so far.

Engaging the issues in reverse order, we can first separate out all studies in which cognitive performance was measured after exercise, however long the pause. For reasons that we hope need no further scrutiny at this point, cognitive functions during exercise are not comparable to those post-exercise, given what we know about the brain activation in these two conditions (Dietrich, 2006, 2008b). Since all recent reviews on the topic have failed to make this distinction in a categorical manner (Brisswalter et al., 2002; Etnier et al., 1997, 2006; Tomporowski, 2003), any global conclusions drawn from them should be considered confounded.

The RAH model is an explanation for the psychological changes during a single bout of exercise. Mechanisms of acute exercise are predominantly physiological ones, that is, they involve a transient modulation in the activity of neural networks (Table 2). No sooner is the action over, so are the physiological changes, give or take a bit of time. Exercise, however, also has long-term effects on mental function, such as the sparing of higher cognitive functions in aging (Colcombe and Kramer, 2003; Hillman et al., 2008; Kramer et al., 2006). Such neuroprotective effects, the fruits of a life-long habit of exercising, must have different mechanisms underlying them. The durability of chronic effects cannot come from changes in neuronal activity patterns but require relatively permanent structural changes to the nervous system, such as angiogenesis (e.g., Swain et al., 2003), synaptogenesis (e.g., Chu and Jones, 2000), or neurogenesis (e.g., Cotman and Engesser-Cesar, 2002; van Praag et al., 1999). These molecular and cellular mechanisms are entirely outside the domain of our present model.

6.1. Nature of the task

Before we can firmly distinguish the mental effects of exercise into the further categories 'during' and 'immediately after' exercise, we must first clarify matters related to the nature of the task. A task is, of course, not inherently explicit or implicit. Which neural network controls the task's execution changes as a function of learning. It is not so simple, then, to tell how much a task, at any one time and for any one person, requires the engagement of prefrontal, executive processes, as this depends on the degree of automation. Even the most complex of tasks – language, for instance – can be, with countless hours of practice, imprinted in the implicit system. This does not merely alter how exercise affects performance on that task but fully reverses, according to our model, the direction of the effect – from negative to positive. The same task that would be impaired in novices, who must use explicit processes, would be enhanced in experts, who can use implicit processes. This is a unique prediction of our model and some data to this effect has already been reported (Delignières et al., 1994). The neural mechanism that provides the rationale for this prediction is as follows.

The effortful acquisition of skilled human movement – tapping a finger sequence, for instance – recruits prefrontal, premotor, primary motor, and parietal cortices as well as the cerebellum (Jenkins et al., 1994). Due to the full engagement of the frontal attentional network at the time of acquisition, we are typically unable to attend to anything else (Broadbent, 1958; Cowan, 1995). It is thought that during this acquisition process the basal ganglia acts as a passive observer. However, studies have also shown that shifts in neural control occur as a function of practice so that the details of a motor task become gradually controlled by the basal ganglia (Mishkin et al., 1984) in a circuit that also includes the supplementary motor cortex, the motor thalamus, and the hippocampus (Jenkins et al., 1994). Put another way, with practice the brain builds a second, implicit representation of the task requirements, which is the equivalent of what is known conversationally by the unfortunate misnomer 'muscle memory'. A thus automated motor pattern can be controlled entirely by this basal ganglia/supplementary motor cortex circuit and little prefrontal activity is required during its routine execution. This is the brain's conquer and divide principle: as the basal ganglia/supplementary motor cortex execute a complex motor skill, aided by direct perceptual input from the parietal cortex, attentional resources in the prefrontal cortex are no longer tied up with the details of the movement. At that point, the execution of the motor skill bypasses consciousness. In other words, by automizing a motor task, it becomes less dependent on the prefrontal supported explicit system, and it is this, in the end, that makes it more efficient. Driving a car on a familiar route is the canonical example for this phenomenon (Cleeremans and Jiménez, 2002).

Any cognitive task in which sensory input and motor output must be integrated in real time does not – cannot – require substantial prefrontal engagement; to be *effective*, it must be run implicitly. It is certainly possible to follow a previously established strategic plan, say, avoiding an opponent's strong backhand or making minor tactical decisions during the game, but moment-to-moment execution must always rely on reflexive loops that, as a result of thousands of hours of highly dedicated practice, have the application embedded in the procedure. With this in mind, we can examine one issue that has led many exercise scientists astray in their theorizing about the exercise and cognition interaction. This issue involves the extent to which exercise requires explicit processes. The source of this error in thinking is easy to understand and runs like this. Exercise, say, in the form of a team sport like basketball, surely requires a whole array of prefrontal-dependent, executive processes. Players must, among many other things, do strategic planning, deliberately retrieve memories, focus attention, time events, keep in mind the score, and so forth. How can this be reconciled with the hypofrontality process, which, presumably, takes place in the player's brains at the time of the game?

Before we solve this deceptive conundrum, let us first examine to what extent implicit or explicit processes might control the execution of a given tasks, keeping in mind that these are not inherent features of the tasks themselves but can change by way of automation. Any reaction time task – by definitional fiat, almost – is, especially over time, mostly controlled by the implicit system, irrespective of how complex the stimulus or the choice options, as long as they do not exceed the load limit of working memory. The RAH model predicts that such tasks, especially if well-learned, *tend* to be enhanced during exercise through the various components of the reticular-activating process (Table 3). The extent to which this improvement in performance occurs depends, of course, on the muscle mass that is set in motion, as well as the duration and the intensity of the motion. This point we will be examined in more detail below.

In contrast, any task in which several representations must be buffered at the same time – two concurrent rules, for instance – requires executive processes. If the requirements of the task are perpetually changing, so that they can never be chunked together to make them all fit below the capacity limit of working memory, we

Table 3
Hypothetical bidirectional effect of steady-state exercise on cognitive processes.

Improvement of performance	Impairment of performance
Implicit	Explicit
Stimulus-driven	Goal-driven
Automatic	Effortful
Bottom–up	Top–down
Unconscious	Conscious

Note: Processes from the right column tend to be improved, while processes from the left column tend to be impaired by acute exercise. Each row should be viewed as a continuum. There is no dimensional overlap, that is, for instance, a top–down process may be fully unconscious, while a stimulus-driven process may require allocation of effort.

are dealing with a task that is inherently explicit. That is, it cannot be automated. The standard example is perhaps prospective planning, in real-life situations. However, as said, any task – however complex initially – in which the requirements can be chunked, perhaps because they reoccur predictably, is amenable to automation and thus can become controlled, over time, by the implicit system. To crystallize this into a more precise prediction with respect to our RAH model, any task that is sensitive to prefrontal-damaged patients, irrespective of practice, should show decrements during exercise (Table 3). Again, the strength of this effect depends on the exact exercise parameters. This prediction differs from the assumption that executive tasks are simply more difficult and thus more readily impaired when resources are scarce (Kahneman, 1973). Daily planning, for instance, is an almost effortless task and our model predicts this ability to be impaired. Some intelligence tests, on the other hand, are quite difficult, but if they do not emphasize executive functions, as some do, our model predicts that they tend not to be impaired (Dietrich and Sparling, 2004).

Cognitive tasks are also commonly performed by both information-processing systems, each doing the part best suited to their purpose. Again putting aside exercise specifications for now, the RAH model predicts that such mixed tasks, as we might call them, would tend to be unchanged by exercise for the simple reason that the two processes controlling them, explicit and implicit, are affected by exercise in opposite directions. In short, they might wash out. We can state this also in the form of a prediction. There are methods, such as Jacoby's process dissociation procedure (Destrebecqz et al., 2005; Jacoby, 1991) that allow implicit processes to be separated from explicit ones within the same task. In memory research, it is also possible to differentiate between implicit and explicit tasks (Eich and Metcalfe, 2009). Using such methods, our RAH model makes a prediction that other cognitive-energetic models do not make, that is, the index of implicit process efficiency of a given task would be selectively improved, while the index of explicit process efficiency would be selectively impaired.

If we reorganize, along those lines, the data on acute exercise summarized in a recent review (Tomporowski, 2003) and add to it the work done since that time, the effects of exercise on cognitive function – for studies assessing cognition during exercise only – exhibit a clear pattern. In Tables 4–6, we have illustrated this. Table 4 lists all studies performed to date that combine exercise with tasks containing manifestly executive components. They

Table 4
Studies using cognitive tasks during moderate exercise that require more substantial explicit processing.

Task	Direction of effect	Exercise parameters	Reference
Short-term memory	Impairment	20 min cycling at 75% MAP	Paas and Adam (1991)
Short-term memory	Speed-accuracy tradeoff	20 min cycling at 75% MAP	Adam et al. (1997)
Wisconsin card Sorting task	Impairment	45 min cycling or running at 75% HRmax	Dietrich and Sparling (2004), exp. 1
Paced auditory serial addition task	Impairment	65 min running at 75% HRmax	Dietrich and Sparling (2004), exp. 2
Vigilance task	Impairment	30 min walking with/without obstacles and a 40-kg load	Mahoney et al. (2007)
Ericksen flanker task	Impairment	6.5 min cycling at 60% HRmax	Pontifex and Hillman (2007)
RNG adjacency score	Strategy shift	35 min cycling at 90% VT	Audiffren et al. (2009)
Ericksen flanker task	Impairment	30 min cycling at 50% MAP	Davranche and McMorris (2009)

Note: MAP: maximum aerobic power; HRmax: maximum heart rate; VT: ventilatory threshold; RNG: randon number generation.

Table 5
Examples of studies using cognitive tasks during moderate exercise that require substantial implicit processing.

Task	Direction of effect	Exercise parameters	Reference
Choice reaction time	Facilitation	20 min cycling at 75% MAP	Paas and Adam (1991)
Choice reaction time	Facilitation	20 min cycling at 75% MAP	Adam et al. (1997)
Quick decision-making	No effect on accuracy; facilitation on speed	Cycling at 70% and 100% MPO	McMorris and Graydon (1997)
Choice reaction time	Facilitation	10 min cycling at 60% MAP	Arcelin et al. (1998)
Choice reaction time	Facilitation	17 min cycling at 50% MPO	Davranche and Audiffren (2004)
Choice reaction time	Facilitation	15 min cycling at 50% MAP	Davranche et al. (2005)
Choice reaction time	Facilitation	15 min cycling at 50% MAP	Davranche et al. (2006)
Choice reaction time	Facilitation	35 min cycling at 90% VT	Audiffren et al. (2008)

Note: MAP: maximum aerobic power; MPO: maximum power output; HRmax: maximum heart rate; VT: ventilatory threshold; RT: reaction time.

Table 6
Examples of studies using cognitive tasks during moderate exercise that require a mixture of both, explicit and implicit processes.

Task	Direction of effect	Exercise parameters	Reference
Short-term memory	No effect	Cycling at ever higher VO_2max	Sjoberg (1980)
Mental arithmetic	U-shaped function	Cycling at ever higher VO_2max	Reilly and Smith (1986)
RNG redundancy score	No effect	50 min cycling at ever higher load	Travlos and Marisi (1995)
Global/local priming	Improvement	12 min cycling at 60% VO_2max	Pesce et al. (2003)
Brief Kaufman Intelligence test	No effect	45 min cycling or running at 75% HRmax	Dietrich and Sparling (2004), exp. 1
Peabody picture vocabulary test	No effect	65 min running at 75% HRmax	Dietrich and Sparling (2004), exp. 2

Note: HRmax: maximum heart rate; VT: ventilatory threshold; RT: reaction time; VO_2max: maximum oxygen uptake.

all show impairment. We are not aware of any published report to the contrary. Table 5 highlights studies using tasks that do not have to be controlled by explicit processes because they are simple and/or can be automated. They all show facilitation. There are many more studies fitting this category and we could have picked eight others to make the point. Although there are some studies in this class reporting no effect, we are not aware of any published work that describes impairments for such tasks. What stands out clearly is the relative weight, that is, the overwhelming majority of research shows that such procedural tasks are enhanced during exercise. Finally, sifting through the research using tasks that appear to tap into both, implicit and explicit processes, we find mostly evidence of no effect in exercise studies on cognitive function. Table 6 lists six reports that are, we think, exemplary for this group. In light of the fact that an important moving part of the puzzle is still kept collapsed – exercise duration and intensity, specifically – which, naturally, can weaken or intensify these effects, the comfortable fit of the entire data set into the above categories strikingly demonstrates the explanatory power of the opponent processes that underlies the RAH model.

With a better grasp of task characteristics, we can now return to our deceptive conundrum from above. To see why a supposedly strategic sport such as basketball is unlikely to require activation of the explicit system *during the game*, and thus substantial prefrontal involvement, consider the many examples of coordinated hunting activity in the animal kingdom. Here, too, there is a broad strategy, an overall goal, a number of intermediate goals towards the main goal, conscious retrieval of relevant memories, temporal integration, as well as sustained and directed attention. What's more, success depends on keeping in mind, at any one time, the preferences and capabilities of your teammates and opponents (or prey), their positions, the layout of the land, and so on. These cognitive processes are all, without a doubt, executive processes, are they not? Yet, the prefrontal cortex of a predatory animal appears adequate for them.

Exercise-induced hypofrontality has been dismissed on the false premise that it is a sort of switch that turns off all higher cognitive functions once a person gets moving. What must be understood, it seems, is what, exactly, the human prefrontal cortex does and whether any of its functions are truly needed for a given task. This is not easy; after all, this issue bedeviled the field of intelligence testing for decades (Hebb, 1939) and a clear understanding of this matter did not emerge until Damasio (1994) reported the case of the patient Elliot.

As we have argued at length throughout the article, any task that requires the efficient execution of sensorimotor tasks, which would include any sport involving speedy stimulus-driven responding, cannot extensively be controlled by the explicit system if the organism is to survive. What the hypofrontality process simply proposes, then, is that exercise relegates the very top layers of the cognitive hierarchy, those mental faculties unique to our species, to a lower priority, and thus temporarily downregulates their computation. As said, this does not have negative consequences. Sports like basketball do not only readily accommodate a state of prefrontal hypofunction, they must do so, according to our RAH model, if the performance enhancement caused by the reticular-activating process is not to be nullified by pointless interference from the explicit system.

We can draw this out from yet another angle. Exercise downregulates, based on the brain's resources, neural structures performing functions that an exercising individual can afford to disengage from on-going activities. This must be conceded as part of the notion of competitive neural processing. This also means, of course, that if the individual cannot afford disengaging these functions, the brain regions sub-serving them are obviously also not downregulated, at least not until the resource issue becomes really critical. The above analysis, however, makes it clear that higher-order cognitive computations do not figure prominently in sports requiring speedy and efficient motor execution at any rate; it just is not their evolutionary purpose.

6.2. Duration and intensity

We have been setting aside all this time one crucial variable that significantly modulates the effect sizes – in both directions, presumably – of the psychological consequences of exercise: duration and intensity. Unfortunately, and surprisingly, there is very little dose–response data on this topic, especially for the one factor the RAH model would predict matters more: duration. Prior empirical research has rather heavily relied on cross-sectional or pre-post comparisons (Etnier et al., 2006). This leaves us with little choice but to approach this issue in a somewhat unconventional manner and embellish the available data with (1) some anecdotal evidence and by (2) filling in the gaps with predictions that are informed, as would be expected, by the present RAH model. Common sense alone tells us that a few minutes of light jogging would not change a person's mental status significantly. But a few hours of hard running, total physical exhaustion for all but the Haile Gebrselassies in the human population, can drastically change our belief system. Even a Friedrich Nietzsche (1878, p. 212), who was not exactly noted for his sporting prowess, realized this when he wrote: "A few hours of mountain climbing turn a villain and a saint into two rather equal creatures. Exhaustion is the shortest way to equality and fraternity." The question, then, is how we get from mindset A to mindset B as we keep on walking. What is the shape of the curve that describes the progressive decline in cognitive function that must take place? Likewise, when – and why – is the initial facilitation of implicit processes reversed into a disability, which empirical tests confirm it is (Cian et al., 2000, 2001)? And, where is the peak of that function?

To find some answers, we will escort an imaginary runner on his way to exhaustion. Suppose, then, that our runner, after a few minutes of slowly warming up, reaches a steady-state plateau just below the anaerobic threshold – a typical marathon pace. Suppose further, to keep things simple, that neither hydration nor glucose availability nor heat enters into the equation and that our runner has the muscular and cardiovascular fitness to run long enough to make matters interesting – for the brain, that is.

Our imaginary runner activates the various arousal systems in the brainstem. This activation supplies the energy needed for the action by allocating much of the available pool of metabolic resources to the relevant motor, sensory, and autonomic brain structures. Within a few minutes, given the data (e.g., Arcelin et al., 1998; Audiffren et al., 2008; Pesce et al., 2003), this reticular-activating process also stimulates cortical and subcortical regions that modulate sensory, attentional, and motor processes. The net effect of this activation is the improvement of various information-processing stages involved in the execution of implicit, sensorimotor tasks. Since we lack detailed information of dose–response relationships, we do not know, for any given task, when this facilitation starts exactly, how strong it is, or how long it lasts.

The limited data we do have suggest the following generalizations. The facilitation effect seems to starts soon after movement commences, it is not dramatic enough to be noticed by the naked eye; that is, precise measurement is needed to detect it, and it can last for a considerable amount of time, certainly more than 40 min into the run (e.g., Adam et al., 1997; Audiffren et al., 2008; Collardeau et al., 2001). Intensity appears to play a minor role here. Extreme cases aside – a slow walk or a full sprint – the facilitation of reaction time in binary choice situations is reliably present between, approximately, 40–80% of maximum

oxygen uptake, without further increases within this range (e.g., Allard et al., 1989; Delignières et al., 1994; Isaacs and Pohlman, 1991; McMorris and Keen, 1994). This correlates moderately with increased catecholamine release in the brain, as measured by plasma concentrations of the NE metabolite MHPG and the DA metabolite (HVA), which increase at minimal intensity but then quickly reach an asymptote (McMorris et al., 2000, 2008). Some of this brainstem activation – in serotenergic pathways, specifically – is likely inhibitory and serves the purpose of moderating the excitatory effects of norepinephrine and dopamine (Meeusen et al., 2006). Although a direct link has not been established, serotonin may also contribute to the fact that our runner feels more positive about life in general at this stage of the run.

The diversion of such a large part of the energetic resources has consequences for all brain structures that do not contribute to the on-going action. Within 5–10 min, apparently, given the data (Kemppainen et al., 2005; Tashiro et al., 2001), some prefrontal, other cortical and limbic regions show decreased metabolic activity. As this hypofrontality process sets in, information processing in the explicit system is held back by the shortage of resources. Since this sort of explicit processing is not needed – or desirable – in most cases, this effect, too, goes unnoticed by the naked eye; that is, a specific neuropsychological test is required to detect this phenomenon. Again, due to the lack of dose–response data, we do not know the strength of this effect. We can assume, though, that with continued running, and thus continued metabolic taxation, it can only get worse. This is a prediction that deserves empirical scrutiny, although anyone who has ever done LSD (long slow distance) running can readily attest to this phenomenon. There is also a second effect and this one is noticeable. The hypofunction of prefrontal, and perhaps limbic, areas eliminates from phenomenology negative emotional processes, such as ruminating about the past or worrying about the future, that are caused by these very regions being hyperactive. As this excessive activity is checked by the on-going motion, our runner becomes calm and relaxed.

If we take a snapshot of our runner's mental and/or brain state at 40 min, we cannot say that there is much drama. Aside from a bit of catecholaminergic arousal to quicken response latency and a bit of hypofrontality to take the edge off, effects which are both so weak that they escape most attempts of introspective detection, all that has noticeably changed up to this point is that our runner feels somewhat more peaceful and calm. If lucky, he might experience a state of mild flow and meditative contemplation.

Should our runner stop here, matters will never reach a boiling point and all bodily systems return to normal. How quickly this occurs for the brain is not well-known. From a theoretical point of view, we can surmise that motor regions of the brain must instantly return to baseline activity, as this is, of course, the cause of the stopping in the first place (Dietrich, 2008b). This, in turn, should immediately free resources that can then be allocated elsewhere. What is far from clear, however, is how long it takes for this change to affect the brain globally. Lactate, glucose and oxygen data (Ide et al., 2004) as well as EEG recordings (Ángyán and Czopf, 1998) indicate that this normalization occurs gradually but rapidly, a matter of seconds to minutes, apparently. This explains perhaps why, immediately after a marathon, the effects of both opposing mechanisms on cognition – implicit enhancement and explicit impairment – can still be detected (Eich and Metcalfe, 2009).

We should emphasize openly at this junction that the RAH model is designed specifically to account for the psychological effects during exercise. Given the lack of detailed data about the time it takes for the brain to resume pre-exercise status, and how the two opponent processes underlying the RAH model contribute to it, we can be much less confident in using the theoretical framework of the model to predict changes in emotion and cognition post-exercise. We do not want to fall into the trap of trying to explain too much too fast. Surely though, any attempt to explain the psychological effects after exercise must start with an informed view of what happens in the brain during exercise. But the possibility that there are additional, perhaps compensatory, neural mechanisms that might come into play once exercise is terminated is ample reason to be cautious about extending the RAH model beyond its domain.

One could reasonably expect, however, that the instant change in neural activation patterns that effectively ends the exercise period has an equally instant effect on implicit and explicit processes performance, as the very basis that affects both, the reticular-activating process and the hypofrontality process, is no longer operating. The psychological data confirm this expectation. The trouble is that not all exercise-induced effects so dutifully fall back to baseline. The weight of the evidence post-exercise does show that the facilitation effect for implicit-predominant tasks is much harder to detect and in most cases disappears (e.g., Audiffren et al., 2008; Collardeau et al., 2001; Hogervorst et al., 1996). We acknowledge that there is much more variability in this data set (see Tomporowski, 2003), which is likely a product of the time it takes to start and administer a task.

Matters take a different – and highly interesting – turn for tasks with manifestly explicit components. Here the weight of the evidence indicates that the negative effect not only fades but swings, after a few minutes, into its opposite. All in all, there are more studies showing an enhancement on those kinds of cognitive tasks than studies reporting no effect in the post-exercise period (e.g., Hillman et al., 2003; Sibley et al., 2006). These data are difficult to explain, and should not be explained, with the RAH model, especially if testing occurred several minutes after exercise. Evidently, there are other neural mechanisms that must assert their influence here. The same seems to hold for the changes to mood states. Research indicates that the antidepressant and anxiolytic effects during exercise linger well into the post-exercise period (Scully et al., 1998; Salmon, 2001). This is surely better accounted for by neurochemical theories, with perhaps serotonergic (Chaouloff, 1997) or endocannabinergic (Dietrich and McDaniel, 2004; Sparling et al., 2003) modulation, rather than in neurophysiological terms of the RAH model.

Suppose, though, that our imaginary runner is sufficiently trained to push past 40 min. From here on forward, accompanying our runner effectively turns, due to the utter lack of hard data, into a mini thought experiment. The only thing for which we have empirical support beyond this point is that somewhere, somehow, the initial facilitation of implicit processing also turns into a deficit (Cian et al., 2000, 2001), which is also indicated by the disappearance of the P300 amplitude rise, which indicates cortical arousal, in the final hour of a 3-h cycling stint (Grego et al., 2004). This is easily corroborated by common sense; just imagine your own cognitive performance, simple decisional tasks included, after doing an all-day hike to exhaustion. For the explicit system, we can presuppose that the unrelenting shortage of metabolic resources only further compromises its ability to process information. But what neural mechanism might lie behind the reversal of fortunes for the implicit system? There are several proposals for the so-called central fatigue hypothesis, neurobiological (Nybo and Secher, 2004) and psychological (Noakes et al., 2004), and the RAH model might inform this body of work with an additional, admittedly speculative, proposal.

The basic idea is that whole body motion, at a strenuous pace and sustained duration, could, conceivably, be so computationally costly in fact that it may inherently outstrip the brain's limited energy supply. This scenario is conceivable if we consider that there are few tasks, if any, that require the kind of massive neural activation large-scale bodily movements do. In the same way, then, that we can live large on credit cards for some time with-

out repercussions, so might the brain be able to afford to lower itself into a temporary metabolic hole and compensate for it later. For people so physically fit that they do not succumb to peripheral fatigue first – muscular or cardiovascular, that this – the brain's position, in terms of energetic resources, could become increasingly precarious indeed. Brains would not have had the time to evolve a mitigating mechanism for this insult for the simple reason that we humans have had only recently the motivation to acquire this ability. Inevitably, when somebody lives too long beyond his means, there will be, at some point, a rude awakening. Should this indeed be so, the brain would be forced, like a sinking ship, to throw overboard more and more neural networks until it reaches even those that drive the very motion itself. This process would, presumably, start at the highest-order brain structure, the prefrontal cortex, and continue down the functional hierarchy, one phenomenological subtraction at a time (Dietrich, 2007). Conditions such as hyperthermia, hypoxia, hypoglycemia, and dehydration only serve to speed up this decline. In the end, the failure to drive the relevant motor units might even contribute to central fatigue.

Apart from our RAH model, we know of no other theory, neuroscientific or psychological, that can account for the dramatic effects of exercise-induced altered states of consciousness. The cognitive effects, which include such experiences as ephemeral attention, timelessness, and silent introspection simply cannot be explained, as is commonly believed, by neurotransmitter theories. Nor can they be explained by current cognitive-energetic theories. These phenomena, however, readily conform, especially in terms of phenomenology, to a state of profound prefrontal hypofunction (Dietrich, 2003).

References

Adam, J.J., Teeken, J.C., Ypelaar, P.J.C., Verstappen, F.T.J., Paas, F.G.W., 1997. Exercise-induced arousal and information processing. International Journal of Sport Psychology 28, 217–226.

Allard, F., Brawley, L., Deakin, J., Elliot, F., 1989. The effect of exercise on visual attention performance. Human Performance 2, 131–145.

Ángyán, L., Czopf, J., 1998. Exercise-induced slow waves in the EEG of cats. Physiology and Behavior 64, 268–272.

Arcelin, R., Delignières, D., Brisswalter, J., 1998. Selective effects of physical exercise on choice reaction processes. Perceptual and Motor Skills 87, 175–185.

Arnsten, A.F.T., Li, B.M., 2005. Neurobiology of executive functions: catecholamine influences on prefrontal cortical functions. Biological Psychiatry 57, 1377–1384.

Ashby, G.F., Casale, M.B., 2002. The cognitive neuroscience of implicit category learning. In: Jiménez, L. (Ed.), Attention and Implicit Learning. John Benjamins Publishing Company, Amsterdam & Philadelphia, pp. 109–141.

Aston-Jones, G., Bloom, F.E., 1981. Norepinephrine-containing locus coeruleus neurons in behaving rats exhibit pronounced responses to non-noxious environmental stimuli. Journal of Neuroscience 1, 887–900.

Astrand, P., Rodahl, K., 1986. Textbook of Work Physiology, 3rd ed. McGraw Hill, New York.

Audiffren, M., 2009. Acute exercise and psychological function: a cognitive-energetic approach. In: McMorris, T., Tomporowski, P.D., Audiffren, M. (Eds.), Exercise and Cognitive Function. Wiley, New York, pp. 3–39.

Audiffren, M., Abou-Dest, A., Possamai, C.-A., 2007. Steady-state exercise aerobic increases sensory sensitivity. In: Lemoine, L. (Ed.), Sensory Control. Paper Presented at 12th ACAPS Metting. Leuven, Belgium.

Audiffren, M., Tomporowski, P., Zagrodnik, J., 2008. Acute aerobic exercise and information processing: energizing motor processes doing a choice reaction time task. Acta Psychologica 129, 410–419.

Audiffren, M., Tomporowski, P., Zagrodnik, J., 2009. Acute aerobic exercise and information processing: modulation of executive control in a random number generation task. Acta Psychologica 132, 85–95.

Baddeley, A., 1996. Exploring the central executive. Quarterly Journal of Experimental Psychology 49A, 5–28.

Baxter, L.R., 1990. Brain imaging as a tool in establishing a theory of brain pathology in obsessive-compulsive disorder. Journal of Clinical Psychiatry 51 (Suppl.), 22–25.

Baxter, L.R., Phelps, M.E., Mazziota, J.C., Guze, B.H., Schwartz, J.M., Selin, C.E., 1987. Local cerebral glucose metabolic rates in obsessive-compulsive disorder: a comparison with rates in unipolar depression and normal controls. Archives of General Psychiatry 44, 211–218.

Beilock, S.L., Carr, T.H., 2005. When high-powered people fail Working memory and "choking under pressure" in math. Psychological Science 16, 101–105.

Berridge, C.W., Waterhouse, B.D., 2003. The locus coeruleus–noradrenergic system: modulation of behavioral state and state-dependent cognitive processes. Brain Research Reviews 42, 33–84.

Bhambhani, Y., Malik, R., Moorkerjee, S., 2007. Cerebral oxygenation declines at exercise intensities above the respiratory compensation threshold. Respiratory Physiology & Neurobiology 156, 196–202.

Boecker, H., Sprenger, T., Spilker, M.E., Henriksen, G., Koppenhoefer, M., Wagner, K.J., Valet, M., Berthele, A., Tolle, T.R., 2008. The runner's high: opioidergic mechanisms in the human brain. Cerebral Cortex 18, 2523–2531.

Botvinick, J., Thompson, L.W., 1966. Premotor and motor components of reaction time. Journal of Experimental Psychology 71, 9–15.

Boutcher, S.J., Landers, D.M., 1988. The effects of vigorous exercise on anxiety, heart rate, and alpha enhancement of runners and nonrunners. Psychophysiology 25, 696–702.

Brisswalter, J., Arcelin, R., Audiffren, M., Delignieres, D., 1997. Influence of physical exercise on simple reaction time: effects of physical fitness. Perceptual and Motor Skills 85, 1019–1027.

Brisswalter, J., Collardeau, M., Arcelin, R., 2002. Effects of acute physical exercise characteristics on cognitive performance. Sport Medicine 32, 555–566.

Broadbent, D.A., 1958. Perception and Communication. Pergamon, New York.

Bruya, J., 2009. Effortlessness attention. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 1–28.

Carter, C.S., Braver, T.S., Barch, D.M., Botvinick, M.M., Noll, D.C., Cohen, J.D., 1998. Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280, 747–749.

Chaouloff, F., 1997. The serotonin hypothesis. In: Morgan, W.P. (Ed.), Physical Activity & Mental Health. Taylor & Francis, Washington, pp. 179–198.

Christensen, L.O., Johannsen, P., Sinkjaer, N., Peterson, N., Pyndt, H.S., Nielsen, J.B., 2000. Cerebral activation during bicycle movements in man. Experimental Brain Research 135, 66–72.

Chu, C.J., Jones, T.A., 2000. Experience-dependent structural plasticity in cortex heterotopic to focal sensorimotor cortical damage. Experimental Neurology 166, 403–414.

Cian, C., Barraud, P.A., Melin, B., Raphel, C., 2001. Effects of fluid ingestion on cognitive function after heat stress or exercise-induced dehydration. International Journal of Psychophysiology 42, 243–251.

Cian, C., Koulmann, N., Barraud, P.A., Raphel, C., Jimenez, C., Melin, B., 2000. Influences of variations in body hydration on cognitive function: effects of hyperhydration, heat stress, and exercise-induced dehydration. Journal of Psychophysiology 14, 29–36.

Cleeremans, A, Jiménez, L., 2002. Implicit learning and consciousness: a graded dynamic perspective. In: French, R.M., Cleeremans, A. (Eds.), Implicit Learning and Consciousness. Psychology Press, pp. 1–40.

Colcombe, S., Kramer, A.F., 2003. Fitness effects on the cognitive function of older adults: a meta-analytic study. Psychological Science 14, 125–130.

Collardeau, M., Brisswalter, J., Audiffren, M., 2001. Effects of a prolonged run on simple reaction time in well trained runners. Perceptual and Motor Skills 93, 679–689.

Cooper, C.J., 1973. Anatomical and physiological mechanisms of arousal with specific reference to the effects of exercise. Ergonomics 16, 601–609.

Cotman, C.W., Engesser-Cesar, C., 2002. Exercise enhances and protects brain function. Exercise and Sport Sciences Reviews 30, 75–79.

Cowan, N., 1995. Attention and memory: an integrated framework. Oxford Psychology Series, No 26, Oxford University Press, New York.

Cowan, N., 2001. The magical number 4 in short-term memory: a reconsideration of mental storage capacity. Behavioral and Brain Sciences 24, 87–185.

Criado, J.M., de la Fuente, A., Heredia, M., Riolobos, A.S., Yajeya, J., 1997. Electrophysiological study of prefrontal neurons of cats during a motor task. European Journal of Physiology 434, 91–96.

Csikszentmihalyi, M., 1975. Beyond Boredom and Anxiety. Jossey–Bass Publishers, San Francisco.

Csikszentmihalyi, M., 1996. Creativity. Harper Perennial, New York.

Csikszentmihalyi, M., Csikszentmihalyi, I.S., 1988. Optimal Experience: Psychological Studies of Flow in Consciousness. Cambridge University Press, Cambridge, New York.

Csikszentmihalyi, M., Nakamura, J., 2009. Effortless attention in everyday life: a systematic phenomenology. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 179–190.

Damasio, A.R., 1994. Descartes' Error: Emotion, Reason and the Human Brain. G.P. Putnam, New York.

Damasio, A.R., Grabowski, T.J., Bechera, A., Damasio, H., Ponto, L.L.B., Parvizi, J., Hichwa, R.D., 2000. Subcortical and cortical brain activity during the feeling of self-generated emotions. Nature Neuroscience 3, 1049–1056.

Davey, C.P., 1973. Physical exertion and mental performance. Ergonomics 16, 595–599.

Davranche, K., Audiffren, M., 2004. Facilitating effect of exercise on information processing. Journal of Sports Sciences 22, 419–428.

Davranche, K., Burle, B., Audiffren, M., Hasbroucq, T., 2005. Information processing during physical exercise: a chronometric and electromyographic study. Experimental Brain Research 165, 532–540.

Davranche, K., Burle, B., Audiffren, M., Hasbroucq, T., 2006. Physical exercise facilitates motor processes in simple reaction time performance: an electromyographic analysis. Neuroscience Letters 396, 54–56.

Davranche, K., McMorris, T., 2009. Specific effects of acute moderate exercise on cognitive control. Brain & Cognition 69, 565–570.
Davranche, K., Pichon, A., 2005. Critical flicker frequency threshold increment after an exhausting exercise. Journal of Sport & Exercise Psychology 27, 515–520.
Dehaene, S., Changeux, J.P., 2004. Neural mechansims for access to consciousness. In: Gazzaniga, M.S. (Ed.), The Cognitive Neurosciences. , 3rd ed. MIT Press, Cambridge, MA, pp. 1145–1158.
Dehaene, S., Naccache, L., 2001. Towards a cognitive science of consciousness: basic evidence and a workspace framework. Cognition 79, 1–37.
Delignières, D., Brisswalter, J., Legros, P., 1994. Influence of physical exercise on choice reaction time in sports experts: the mediating role of resource allocation. Journal of Human Movement Studies 27, 173–188.
DeCaro, M.S., Beilock, S.L., 2009. The benefits and perils of attentional control. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 51–74.
Del Giorno, J.M., Hall, E.E., O'Leary, K.C., Bixby, W.R., Miller, P.C., 2010. Cognitive function during acute exercise: a test of the transient hypofrontality theory. Journal of Sport & Exercise Psychology 32, 312–323.
Destrebecqz, A., Peigneux, P., Laureys, S., Degueldre, C., Del Fiore, G., Aerts, J., Luxen, A., Van Der Linden, M., Cleeremans, A., Maquet, P., 2005. The neural correlates of implicit and explicit sequence learning: interacting networks revealed by the process dissociation procedure. Learning and Memory 12, 480–490.
Dienes, Z., Perner, J., 1999. A theory of implicit and explicit knowledge. Behavioural and Brain Sciences 5, 735–808.
Dienes, Z., Perner, J., 2002. A theory of the implicit nature of implicit learning. In: French, R.M., Cleeremans, A. (Eds.), Implicit Learning and Consciousness. Psychology Press, pp. 68–92.
Dietrich, A., 2003. Functional neuroanatomy of altered states of consciousness: the transient hypofrontality hypothesis. Consciousness and Cognition 12, 231–256.
Dietrich, A., 2004a. Neurocognitive mechanisms underlying the experience of flow. Consciousness and Cognition 13, 746–761.
Dietrich, A., 2004b. The cognitive neuroscience of creativity. Psychonomic Bulletin & Review 11, 1011–1026.
Dietrich, A., 2006. Transient hypofrontality as a mechanism for the psychological effects of exercise. Psychiatry Research 145, 79–83.
Dietrich, A., 2007. Introduction to Consciousness. Palgrave Macmillan, London.
Dietrich, A., 2008a. Mind on the run. Methods 45, 253–254.
Dietrich, A., 2008b. Imaging the imagination: the trouble with motor imagery. Methods 45, 319–324.
Dietrich, A., 2009. The cognitive neuroscience of exercise: the transient hypofrontality theory and its implications for cognition and emotion. In: McMorris, T., Tomporowski, P.D., Audiffren, M. (Eds.), Exercise and Cognitive Function. Wiley, New York, pp. 69–90.
Dietrich, A., McDaniel, W.F., 2004. Cannabinoids and exercise. British Journal of Sports Medicine 38, 536–541.
Dietrich, A., Sparling, P.B., 2004. Endurance exercise selectively impairs prefrontal dependent cognition. Brain and Cognition 55, 516–524.
Dietrich, A., Stoll, O., 2009. Effortlessness attention hypofrontality and perfectionism. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 159–178.
Dishman, R.K., 1997. The norepinephrine hypothesis. In: Morgan, W.P. (Ed.), Physical Activity & Mental Health. Taylor & Francis, Washington, pp. 199–212.
Dobrynin, N., 1966. Basic problems of the psychology of attention. In: Psychological Science in the USSR 274–291. US Dept of Commerce, Clearinghouse for Federal Scientific and Technical Information, Washington, DC.
Draper, S., McMorris, T., Parker, J.K., 2010. Effect of acute exercise of differing intensities on simple and choice reaction and movement times. Psychology of Sport and Exercise 11, 536–541.
Duffy, E., 1962. Activation and Behavior. Wiley, New York.
Eich, T.S., Metcalfe, J., 2009. Effects of the stress of marathon running on implicit and explicit memory. Psychonomic Bulletin & Review 16, 475–479.
Etnier, J.L., Nowell, P.M., Landers, D.M., Sibley, B.A., 2006. A meta-regression to examine the relationship between aerobic fitness and cognitive performance. Brain Research Reviews 52, 119–130.
Etnier, J.L., Salazar, W., Landers, D.M., Petruzzello, S.J., Han, M., Nowell, P., 1997. The influence of physical fitness and exercise upon cognitive functioning: a meta-analysis. Journal of Sports & Exercise Psychology 19, 249–277.
Eysenck, M.W., 1982. Attention and Arousal. Springer–Verlag, Berlin.
Fukuyama, H., Ouchi, Y., Matsuzaki, S., Nagahama, Y., Yamauchi, H., Ogawa, M., Kimura, J., Shibasaki, H., 1997. Brain functional activity during gait in normal subjects: a SPECT study. Neuroscience Letters 228, 183–186.
Goldstein, D.S., Eisenhofer, G., Kopin, I.J., 2003. Source and significance of plasma levels of catechols and their metabolites in humans. The Journal of Pharmacology and Experimental Therapeutics 305, 800–811.
Grant, S.J., Aston-Jones, G., Redmond, D.E.J., 1988. Responses of primate locus coeruleus neurons to simple and complex sensory stimuli. Brain Research Bulletin 21, 401–410.
Grego, F., Vallier, J.-M., Collardeau, M., Bermon, S., Ferrari, P., Candito, M., Bayer, P., Magnié, M.-N., Brisswalter, J., 2004. Effects of long duration exercise on cognitive function, blood glucose, and counterregulatory hormones in male cyclists. Neuroscience Letters 364, 76–80.
Gross, P.M., Marcus, M.L., Heistad, D.D., 1980. Regional distribution of cerebral blood flow during exercise in dogs. Journal of Applied Physiology 48, 213–217.
Gusnard, D.A., Raichle, M.E., 2001. Searching for a baseline: functional imaging and the resting human brain. Nature Review Neuroscience 2, 685–694.
Haider, H., Frensch, P.A., 2005. The generation of conscious awareness in an incidental learning situation. Psychological Research 69, 399–411.
Hasbroucq, T., Burle, B., Bonnet, M., Possamai, C.-A., Vidal, F., 2001. Dynamique du traitement de l'information sensori-motrice: Apport de l'électrophysiologie [The dynamics of information processing: Electrophysiological arguments]. Canadian Journal of Experimental Psychology 56, 75–97.
Hebb, D.O., 1939. Intelligence in man after large removal of cerebral tissue: report of four left frontal lobe cases. Journal of General Psychology 21, 73–87.
Hillman, C.H., Erickson, K.I., Kramer, A.F., 2008. Be smart, exercise your heart: exercise effects on brain and cognition. Nature Reviews Neuroscience 9, 58–65.
Hillman, C.H., Snook, E.M., Jerome, G.J., 2003. Acute cardiovascular exercise and executive control function. International Journal of Psychophysiology 48, 307–314.
Hockey, G.R.J., 1997. Compensatory control in the regulation of human performance under stress and high workload: a cognitive-energetical framework. Biological Psychology 45, 73–93.
Hockey, G.R.J., Coles, M.G.H., Gaillard, A.W.K., 1986. Energetical issues in research on human information processing. In: Hockey, G.R.J., Gaillard, A.W.K., Coles, H.M.G. (Eds.), Energetics and Human Information Processing. Martinus Nijhoff Publishers, Dordrecht, pp. 3–21.
Hogervorst, E., Riedel, W., Jeukendrup, A., Jolles, J., 1996. Cognitive performance after strenuous physical exercise. Perceptual and Motor Skill 83, 479–488.
Holschneider, D.P., Maarek, J.-M.I., Yang, J., Harimoto, J., Scremin, O.U., 2003. Functional brain mapping in freely moving rats during treadmill walking. Journal of Cerebral Blood Flow and Metabolism 23, 925–932.
Humphreys, M.S., Revelle, W., 1984. Personality, motivation, and performance a theory of the relationship between individual differences and information processing. Psychological Review 91, 153–184.
Hurley, L.M., Devilbiss, D.M., Waterhouse, B.D., 2004. A matter of focus: monoaminergic modulation of stimulus coding in mammalian sensory networks. Current Opinion in Neurobiology 14, 488–495.
Ide, K., Horn, A., Secher, N.H., 1999. Cerebral metabolic response to submaximal exercise. Journal of Applied Physiology 87, 1604–1608.
Ide, K., Secher, N.H., 2000. Cerebral blood flow and metabolism during exercise. Progress in Neurobiology 61, 397–414.
Ide, K., Schmalbruch, I.K., Quistorff, B., Horn, A., Secher, N.H., 2004. Lactate, glucose and O_2 uptake in human brain during recovery from maximal exercise. Journal of Physiology 522, 159–164.
Isaacs, L.D, Pohlman, E.L., 1991. Effects of exercise intensity on an accompanying timing task. Journal of Human Movement Studies 20, 123–131.
Jackson, S.A., Csikszentmihalyi, M., 1999. Flow in Sports. Human Kinetics, Champaign, IL.
Jacoby, L.L., 1991. A process dissociation framework: separating automatic from intentional uses of memory. Journal of Memory and Language 30, 513–541.
Jenkins, I.H., Brooks, D.J., Nixon, P.D., Frackowiak, R.S.J., Passingham, R.E., 1994. Motor sequence learning: a study with positron emission tomography. Journal of Neuroscience 14, 3775–3790.
Kahneman, D., 1973. Attention and Effort. Prentice–Hall, Englewood Cliffs, NJ.
Kamijo, K., Nishihira, Y., Hatta, A., Kaneda, T., Kida, T., Higashiura, T., Kuroiwa, K., 2004a. Changes in arousal level by differential exercise intensity. Clinical Neurophysiology 115, 2693–2698.
Kamijo, K., Nishihira, Y., Hatta, A., Kaneda, T., Wasaka, T., Kida, T., Kuroiwa, K., 2004b. Differential influences of exercise intensity on information processing in the central nervous system. European Journal of Applied Physiology 92, 305–311.
Kamijo, K., Nishihira, Y., Higashiura, T., Kuroiwa, K., 2007. The interactive effect of exercise intensity and task difficulty on human cognitive processing. International Journal of Psychophysiology 65, 114–121.
Kamp, A., Troost, J., 1978. EEG signs of cerebrovascular disorder, using physical exercise as a provocative method. Electroencephalography and Clinical Neurophysiology 45, 295–298.
Karmiloff-Smith, A., 1992. Beyond Modularity: A Developmental Perspective on Cognitive Science. MIT Press, Cambridge, MA.
Kashihara, K., Maruyama, T., Murota, M., Nakahara, Y., 2009. Positive effects of acute and moderate physical exercise on cognitive function. Physiological Anthropology 28, 155–164.
Keele, S.W., Ivry, R., Mayr, U., Hazeltine, E., Heuer, H., 2003. The cognitive and neural architecture of sequence representation. Psychological Review 110, 316–339.
Kemppainen, J., Aalto, S., Fujimoto, T., Kalliokoski, K.K., Långsjö, J., Oikonen, V., Rinne, J., Nuutila, P., Knuuti, J., 2005. High intensity exercise decreases global brain glucose uptake in humans. Journal of Physiology 568, 323–332.
Kiehl, K.A., Liddle, P.F., Hopfinger, J.B., 2000. Error processing and the rostral anterior cingulate: an event-related fMRI study. Psychophysiology 33, 282–294.
Kihlstrom, J.F., 1996. Perception without awareness of what is perceived, learning without awareness of what is learned. In: Velmans, M. (Ed.), The Science of Consciousness: Psychological, Neuropsychological and Clinical Reviews. Routledge, London, pp. 23–46.
Kramer, A.F., Erickson, K.I., Colcombe, S.J., 2006. Exercise, cognition and the aging brain. Journal of Applied Phsiology 101, 1237–1242.
Kubitz, K.A, Pothakos, K., 1997. Does aerobic exercise decrease brain activation? Journal of Sport & Exercise Psychology 19, 291–301.
Kuo, A.D., Donelan, J.M., Ruina, A., 2005. Energetic consequences of walking like an inverted pendulum: step-to-step transitions. Exercise & Sport Sciences Reviews 33, 88–97.
Lambourne, K., Audiffren, M., Tomporowski, P., 2010. Effects of acute exercise on sensory and executive processing tasks. Medicine & Science in Sports & Exercise 47, 1396–1402.

Lacey, J.I., 1967. Somatic response patterning and stress: some revisions of activation theory. In: Appley, M.H., Trumbull, R. (Eds.), Psychological Stress. Century Crofts, New York, pp. 14–42.
LeDoux, J., 1996. The Emotional Brain. Touchstone, New York.
Loveless, N.E., Sanford, A.J., 1974. Slow potential correlates of preparatory set. Biological Psychology 1, 303–314.
MacDonald, K.B., 2008. Effortful control, explicit processing, and the regulation of human evolved predispositions. Psychological Review 115, 1012–1031.
Magnie, M.N., Bermon, S., Martin, F., Madany-Lounis, M., Suisse, G., Muhammad, W., Dolisi, C., 2000. P300 N400, aerobic fitness, and maximal aerobic exercise. Psychophysiology 37, 369–377.
Mahoney, C.R., Hirsch, E., Hasselquist, L., Lesher, L.L., Lieberman, H.R., 2007. The effects of movement and physical exertion on soldier vigilance. Aviation, Space, and Environmental Medicine 78, B51–B57.
Mayberg, H.S., 1997. Limbic-cortical dysregulation: a proposed model of depression. Journal of Neuropsychiatry and Clinical Neuroscience 9, 471–481.
Mayberg, H.S., Mahurin, R.K., Brannon, K.S., 1995. Parkinson's depression: discrimination of mood-sensitive and mood insensitive cognitive deficits using fluoxetine and FDG PET. Neurology 45, A166.
McGuire, J.T., Botvinick, M.M., 2009. The impact of anticipated cognitive demand on attention and behavioral choice. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 103–120.
McMorris, T., Collard, K., Corbett, J., Dicks, M., Swain, J.P., 2008. A test of the catecholamines hypothesis for an acute exercise–cognition interaction. Pharmacology Biochemistry and Behavior 89, 106–115.
McMorris, T., Davranche, K., Jones, G., Hall, B., Corbett, J., Minter, C., 2009. Acute incremental exercise, performance of a central executive task, and sympathoadrenal system and hypothalamic-pituitary-adrenal axis activity. International Journal of Psychophysiology 73, 334–340.
McMorris, T., Graydon, J., 1997. Effects of exercise on the decision making of soccer players. In: Reilly, T., Bangsbo, J., Hughes, M. (Eds.), Science and Football III. E and FN Spon, New York, pp. 279–284.
McMorris, T., Graydon, J., 2000. The effect of incremental exercise on cognitive performance. International Journal of Sport Psychology 31, 66–81.
McMorris, T., Keen, P., 1994. Effect of exercise on simple reaction times of recreational athletes. Perceptual and Motor Skills 78, 123–130.
McMorris, T., Sproule, J., Draper, S., Child, R., Sexsmith, J.R., Forster, C.D., Pattison, J., 2000. The measurement of plasma catecholamine and lactate theresholds: a comparison of methods. European Journal of Applied Physiology 82, 262–267.
Meeusen, R., De Meirleir, K., 1995. Exercise and brain neurotransmission. Sports Medicine 20, 160–188.
Meeusen, R., Watson, P., Hasegawa, H., Roelands, B., Piacentini, M., 2006. Central fatigue: the serotonin hypothesis and beyond. Sports Medicine 36, 881–909.
Mishkin, M., Malamut, B., Bachevalier, J., 1984. Memory and habit: two neural systems. In: Lynch, G., McGaugh, J.J., Weinberger, N.M. (Eds.), Neurobiology of Learning and Memory. Guilford Press, New York, pp. 66–77.
Moxon, K.A., Devilbiss, D.M., Chapin, J.K., Waterhouse, B.D., 2007. Influence of norepinephrine on somatosensory neuronal responses in the rat thalamus: a combined modeling and in vivo multi-channel, multi-neuron recording study. Brain Research 1147, 105–123.
Nietzsche, F., 1878–2008. Human, All Too Human. Prometheus Books, Amherst, NY.
Näätänen, R., 1973. The inverted-U relationship between activation and performance: a critica review. In: Kornblum, S. (Ed.), Attention and Performance IV. Academic Press, New York, pp. 155–174.
Niewenhuis, S., Aston-Jones, G., Cohen, J.D., 2005. Decision making, the P3, and the locus coeruleus-norepinephrine system. Psychological Bulletin 131, 510–532.
Noakes, T.D., St Clair Gibson, A., Lambert, E.V., 2004. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans. British Journal of Sports Medicine 38, 511–514.
Nybo, L., Nielsen, B., 2001. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. Journal of Applied Physiology 91, 2017–2023.
Nybo, L., Secher, N.H., 2004. Cerebral perturbations provoked by prolonged exercise. Progress in Neurobiology 72, 223–261.
Paas, F.G.W., Adam, J.J., 1991. Human information processing during physical exercise. Ergonomics 34, 1385–1397.
Perrey, S., 2008. Non-invasive NIR spectroscopy of human brain function during exercise. Methods 45, 289–299.
Pesce, C., Capranica, L., Tessitore, A., Figura, F., 2003. Focusing of visual attention under submaximal physical load. International Journal of Sport and Exercise Psychology 1 (3), 275–292.
Petruzzello, A., Landers, D.M., 1994. State anxiety reduction and exercise: does hemispheric activation reflect such changes. Medicine and Science in Sports and Exercise 26, 1028–1035.
Peyrin, L., 1990. Urinary MHPG sulphate as a marker of central norepinephrine metabolism: a commentary. Journal of Neural Transmission General Section 80, 51–65.
Peyrin, L., Pequignot, J.M., Lacour, J.R., Fourcade, J., 1987. Relationships between catecholamine or 3-methoxy 4-hydroxy phenylglycol changes and the mental performance under submaximal exercise in man. Psychopharmacology 93, 188–192.
Pineda, A., Adkisson, M.A., 1961. Electroencephalographic studies in physical fatigue. Texas Reports on Biology and Medicine 19, 332–342.
Pineda, J.A., Foote, S.L., Neville, H.J., 1989. Effects of locus coeruleus lesions on auditory, longlatency, event-related potentials in monkey. Journal of Neuroscience 9, 81–93.
Poldrack, R.A, Packard, M.G., 2003. Competition among multiple memory systems: converging evidence from animal and human brain studies. Neuropsychologia 41, 245–251.
Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. Clinical Neurophysiology 118, 2128–2148.
Polich, J., Criado, J.R., 2006. Neuropsychology and neuropharmacology of P3a and P3b. International Journal of Psychophysiology 60, 172–185.
Pontifex, M.B., Hillman, C.H., 2007. Neuroelectric and behavioural indices of interference control during acute cycling. Clinical Neurophysiology 118, 570–580.
Posner, M.I., Rothbart, M.K., Rueda, M.R., Tang, Y., 2009. Training effortless attention. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 409–424.
Pribram, K.H., McGuinness, D., 1975. Arousal, activation and effort in the control of attention. Psychological Review 82, 116–149.
Racinais, S., Bishop, D., Denis, R., Lattier, G., Mendez-Villaneuva, A., Perrey, S., 2007. Muscle deoxygenation and neural drive to the muscle during repeated sprint cycling. Medicine & Science in Sports & Exercise 39, 268–274.
Ramos, B.P., Arnsten, A.F.T., 2007. Adrenergic pharmacology and cognition: focus on the prefrontal cortex. Pharmacology & Therapeutics 113, 523–536.
Rasmussen, K., Morilak, D.A., Jacobs, B.L., 1986. Single unit activity of locus coeruleus neurons in the freely moving cat I. During naturalistic behaviors and in response to simple and complex stimuli. Brain Research 371, 324–334.
Ravizza, K., 1977. Peak performances in sports. Journal of Humanistic Psychology 4, 35–40.
Reber, A.S., 1989. Implicit learning and tacit knowledge. Journal of Experimental Psychology General 118, 219–235.
Reilly, T., Smith, D., 1986. Effect of work intensity on performance in a psychomotor task during exercise. Ergonomics 29, 601–606.
Robbins, T.W., Everitt, B.J., 1995. Arousal systems and attention. In: Gazzaniga, M.S. (Ed.), The Cognitive Neurosciences. The MIT Press, Cambridge, MA, pp. 703–720.
Robbins, T.W., Everitt, B.J., 2007. A role for mesencephalic dopamine in activation: commentary on Berridge (2006). Psychopharmacology 191, 433–437.
Rupp, T., Perrey, S., 2008. Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. European Journal of Applied Physiology 102, 153–163.
Salmon, P., 2001. Effects of physical exercise on anxiety, depression, and sensitivity to stress: a unifying theory. Clinical Psychological Review 21, 33–61.
Sanders, A.F., 1983. Towards a model of stress and human performance. Acta Psychologica 53, 61–97.
Sanders, A.F., 1998. Elements of Human Performance: Reaction Processes and Attention in Human Skill. Lawrence Erlbaum Associates, Mahwah, NJ.
Schacter, D.L., Buckner, R.L., 1998. On the relationship among priming, conscious recollection, and intentional retrieval: evidence from neuroimaging research. Neurobiology of Learning and Memory 70, 284–303.
Schatzenberg, A.F., Nemeroff, C.B., 2009. Essentials of Clinical Psychopharmacology (4th). The American Psychiatric Publishing, Inc., Arlington, VA.
Schmeichel, B.J., Baumeister, R.F., 2009. Effortful attention control. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 29–50.
Scully, D., Kremer, J., Meade, M.M., Graham, R., Dudgeon, K., 1998. Physical exercise and psychological well being: a critical review. British Journal of Sports Medicine 32, 111–120.
Shallice, T., Burgess, W., 1991. Deficits in strategy application following frontal lobe damage in man. Brain 114, 727–741.
Sibley, B.A., Etnier, J.L., Le Masurier, G.C., 2006. Effects of an acute bout of exercise on cognitive aspects of Stroop performance. Journal of Sport & Exercise Psychology 28, 285–299.
Sjoberg, H., 1980. Physical fitness and mental performance during and after work. Ergonomics 23, 977–985.
Sokoloff, L., 1991. Measurements of local cerebral glucose utilization and its relation to functional activity in the brain. Advances in Experimental and Medical Biology 291, 21–42.
Sparling, P.B., Giuffrida, A., Piomelli, D., Rosskopf, L., Dietrich, A., 2003. Exercise activates the endocannabinoid system. Neuroreport 14, 2209–2211.
Squire, L.R., 1992. Memory and the hippocampus: a synthesis from findings with rats, monkeys and humans. Psychological Review 99, 195–231.
Starkstein, S.E., Robinson, R.G., 1999. Depression and frontal lobe disorders. In: Miller, B.L., Cummings, J.L. (Eds.), The Human Frontal Lobes: Functions and Disorders. The Guilford Press, New York, pp. 247–260.
Sternberg, S., 1998. Discovering mental processing stages: the method of additive factors. In: Scarborough, D., Sternberg, S. (Eds.), An Invitation to Cognitive Science, Methods, Models, and Conceptual Issues. MIT Press, Cambridge, MA, pp. 703–863.
Sun, R., 2006. Cognition and Mutli-agent Interaction: From Cognitive Modeling to Socia Stimulation. Cambridge University Press, Cambridge.
Swain, R.A., Harris, A.B., Wiener, E.C., Dutka, M.V., Morris, H.D., Theien, B.E., Konda, S., Engberg, K., Lauterbur, P.C., Greenough, W.T., 2003. Prolonged exercise induces angiogenesis and increases cerebral blood volume in primary motor cortex of the rat. Neuroscience 117, 1037–1046.
Tashiro, M., Itoh, M., Fujimoto, T., Fujiwara, T., Ota, H., Kubota, K., Higuchi, M., Okamura, N., Ishi, K., Berecski, D., Sasaki, H., 2001. 18F-FDG PET mapping of regional

brain activity in runners. Journal of Sports Medicine and Physical Fitness 41, 11–17.
Thayer, R.E., 1989. The Biopsychology of Mood and Arousal. Oxford University Press, New York.
Timinkul, A., Kato, M., Omori, T., Deocaris, C.C., Ito, A., Kizuka, T., et al., 2008. Enhancing effect of cerebral blood volume by mild exercise in healthy young men: a nearinfrared spectroscopy study. Neuroscience Research 61, 242–248.
Tomporowski, P.D., 2003. Effects of acute bouts of exercise on cognition. Acta Psychologica 112, 297–324.
Travlos, A.K., Marisi, D.Q., 1995. Information processing and concentration as a function of fitness level and exercise induced activation to exhaustion. Perceptual and Motor Skills 80, 15–26.
Vidal, F., Bonnet, M., Macar, F., 1995. Programming the duration of a motor sequence – role of the primary and supplementary motor areas in man. Experimental Brain Research 106, 339–350.
van Praag, H., Kempermann, G., Gage, F.H., 1999. Running increases cell proliferation and neurogenesis in the adult mouse dentate gyrus. Nature Neuroscience 2, 266–270.
van Veen, V., Carter, C.S., 2002a. The anterior cingulate as a conflict monitor: fMRI and ERP studies. Physiology & Behavior 77, 477–482.
van Veen, V., Carter, C.S., 2002b. The timing of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience 14, 593–602.
Vissing, J., Anderson, M., Diemer, N.H., 1996. Exercise-induced changes in local cerebral glucose utilization in the rat. Journal of Cerebral Blood Flow and Metabolism 16, 729–736.
Waterhouse, B.D., Woodward, D.J., 1980. Interaction of norepinephrine with cerebrocortical activity evoked by stimulation of somatosensory afferent pathways in the rat. Experimental Neurology 67, 11–34.
Wickens, C.D., 1984. Processing resources in attention. In: Parasuraman, R., Davies, D.R. (Eds.), Varieties of Attention. Academic Press, Orlando, FL, pp. 63–102.
Willingham, D.G., 1998. A neuropsychological theory of motor skill learing. Psychological Review 105, 558–584.
Willingham, D.B., Wells, L.A., Farrell, J.M., Stemwedel, M.E., 2000. Implicit motor sequence learning is represented in response locations. Memory & Cognition 28, 366–375.
Wittert, G., 2000. The effect of exercise on the hypothalamo-pituitary-adrenal axis. In: Warren, M.P., Constantini, N.W. (Eds.), Sports Endocrinology. Humana Press, Totowa, pp. 43–55.
Woods, R.L., Thomson, W.D., 1995. Effects of exercise on aspects of visual function. Ophthalmic and Physiological Optics 15, 5–12.
Woolsey, T.A., Rovainen, C.M., Cox, S.B., Henegar, M.H., Liang, G.E., Liu, D., et al., 1996. Neuronal units linked to microvascular modules in cerebral cortex: response elements for imaging the brain. Cerebral Cortex 6, 647–660.
Wulf, G., Lewthwaite, R., 2009. Effortless motor learning? An external focus of attention enhances movement effectiveness and efficiency. In: Bruya, B.J. (Ed.), Effortless Attention: A New Perspective in the Cognitive Science of Attention and Action. MIT Press, Cambridge, MA, pp. 74–102.
Wulf, G., Prinz, W., 2001. Directing attention to movement effects enhances learning: a review. Psychonomic Bulletin & Review 8, 648–660.
Yagi, Y., Coburn, K.L., Estes, K.M., Arruda, J.E., 1999. Effects of aerobic exercise and gender on visual and auditory P300, reaction time, and accuracy. European Journal of Physiology 80, 402–408.
Yao, J.K., Zhu, P., Wilds, D.J., van Kammen, D.P., 1997. A simplified routine assay for urinary 3-methoxy-4-hydroxyphenylglycol. Journal of Neural Transmission 104, 967–975.
Yerkes, R.M., Dodson, J.D., 1908. The relation of strength of stimulus to the rapidity of habit formation. Journal of Comparative and Neurology and Psychology 18, 459–482.
Youngstedt, S., Dishman, R.K., Cureton, K., Peacock, L., 1993. Does body temperature mediate anxiolytic effects of acute exercise. Journal of Applied Physiology 74, 825–831.