

PSYCHOBIOLOGY *of* PHYSICAL ACTIVITY



Edmund O. Acevedo
Panteleimon Ekkekakis

editors

Affective Responses to Acute Exercise

Toward a Psychobiological Dose-Response Model

Panteleimon Ekkekakis, PhD, FACSM

Edmund O. Acevedo, PhD, FACSM

The study of the affective responses that accompany single bouts of exercise has been a prominent research direction within exercise psychology for approximately 35 years. Based on hundreds of studies conducted during this period, the main conclusion is that participation in a bout of exercise produces a so-called “feel-better” effect, which in most cases is defined operationally as a decrease in state anxiety, an increase in perceived vigor, or a general improvement in various mood states. As described in a recent literature review, “Both survey and experimental research . . . provide support for the well publicized statement that *‘exercise makes you feel good’*” (Fox, 1999, p. 413, italics in the original). This is the same sentiment found in exercise psychology textbooks and touted in most media reports. It is now time to focus on other, finer points of the complex relationship between exercise and affect. In the last 10 or so years, the focal issue has been the shape of the dose-response relationship between the intensity of exercise and the nature of the affective response.

The Intensity-Affect Relationship

The relationship between the intensity of exercise and affective responses is important for two main reasons. One is its theoretical interest. Although the body and its activation are recognized as having an important role in most major theories of emotion, the nature and the extent of this role are and have been a hotly debated issue. Since exercise provides a way to precisely regulate the degree of bodily activation, it could potentially serve as a useful investigative platform in delineating the role of such activation in the generation of affective responses. The second reason is the practical significance of the dose-response relationship for the maintenance of exercise adherence and the prevention of dropout, two major public health concerns. Approximately 40% of the adults in the United States aged 18 years and older report no regular physical activity (United States Department of Health and Human Services

[USDHHS], 2000); two-thirds do not meet the current physical activity recommendations of 30 min of daily moderate activity (Jones et al., 1998); and only 15% participate in activities of sufficient intensity, duration, and frequency to improve or maintain cardiorespiratory fitness (USDHHS, 1996). Moreover, of those who make the decision to start an exercise program, approximately 50% drop out within the first few months (Dishman & Buckworth, 1996). The intensity of exercise might be one of the reasons contributing to this problem, as several studies have shown an inverse relation between intensity and adherence (Lee et al., 1996; Perri et al., 2002). Likewise, a meta-analysis of interventions for increasing exercise participation showed that interventions are more effective when the intensity of physical activity is lower (i.e., 50% of maximal capacity or less) rather than higher (Dishman & Buckworth, 1996). It seems reasonable to suggest that the negative relationship between the intensity of exercise and adherence is partly mediated by affect. Higher intensity might entail less pleasant or more unpleasant affect; and this may, in turn, lead to reduced adherence and increased risk of dropout, as people, in the long run, generally choose what makes them feel good and avoid what makes them feel bad.

In this chapter, we first present and critique a conceptual formulation, named the inverted U, that has served as a popular model of the dose-response relationship for many years. Second, we identify a number of critical conceptual and methodological problems in the previous dose-response literature that might have contributed to inconsistent findings. Third, we review some recently conducted dose-response studies that were based on a new research platform and have produced the first evidence of a reliable dose-response pattern. Fourth, we sketch a new theoretical framework for the dose-response relationship, named the dual-mode model. Fifth, based on neuroanatomical and neurophysiological data, we propose a possible neural basis for the dual-mode model. Sixth, we present some practical implications of the new theoretical formulation and suggest directions for future investigations.

The Inverted U As a Dose-Response Model and Its Limitations

The closest thing to a conceptual model of the dose-response relationship between the intensity

of exercise and affective responses is the time-honored inverted-U model. According to this idea, moderately vigorous exercise provides the optimal stimulus for positive affective change, whereas low-intensity exercise is insufficient to produce significant changes in affect and high-intensity exercise is either ineffective or experienced as aversive (Kirkcaldy & Shephard, 1990; Ojanen, 1994). However, it is becoming increasingly obvious that the inverted U has some serious limitations impeding further progress and thus cannot serve as the basis for future research.

First and foremost, the model does not appear to be consistent with emerging data. For example, it has been found that low-intensity and short-duration exercise can produce transient but significant positive changes (Ekkekakis et al., 2000; Saklofske, Blomme, & Kelly, 1992; Thayer, 1987); that high-intensity exercise, such as an incremental exercise protocol to volitional exhaustion, in addition to negative changes such as increases in fatigue, may also lead to some positive changes such as improvements in self-esteem (Pronk, Crouse, & Rohack, 1995); and that, during moderate-intensity exercise, some individuals may respond positively whereas others may respond negatively (Van Landuyt et al., 2000). Second, the inverted-U model is descriptive, not mechanistic. As such, it cannot provide any insights into the psychobiological processes underlying the observed responses. And, third, the model is nomothetic, making no provisions for patterns of interindividual variability in affective responses to the same intensity of exercise. It is clear that not all individuals respond in the same way to the same exercise stimulus (Gauvin & Brawley, 1993; Van Landuyt et al., 2000).

Previous Dose-Response Findings and Weaknesses in the Literature

In total, the relationship between the intensity of exercise and affective responses has been examined in approximately 55 studies. Of these, 31, conducted until 1998, were reviewed by Ekkekakis and Petruzzello (1999). These studies could be divided into two groups. One group contained 26 studies that examined affective changes from before the exercise to various time points after. Of these, the majority (14 of the 26 or 54%) did not show reliable evidence of a dose-response effect. The other group contained studies that examined affective

responses during the exercise bouts. In these studies, there was a consistent dose-response pattern, with higher intensities being associated with less positive or more negative affective responses (e.g., Acevedo, Rinehardt, & Kraemer, 1994; Hardy & Rejeski, 1989; Parfitt & Eston, 1995; Parfitt, Eston, & Connolly, 1996; Parfitt, Markland, & Holmes, 1994). Similar findings, distinguishing between assessments from before to after exercise and assessments during exercise, have also emerged from the studies conducted since 1998.

The Ekkekakis and Petruzzello (1999) review also described some of the limitations in previous studies that might have obscured the identification of dose-response effects. The first limitation is apparent from the aforementioned findings of the review and pertains to the timing of affect assessments. In the majority of the studies, affect was assessed from before to after exercise, presumably based on the assumption that any changes that take place in the interim would be linear. However, it is clear that this is not the case. The studies that have examined responses during the exercise bout have shown that, depending on the intensity, the trajectory of change could be nonlinear. Specifically, when the intensity is high enough to induce a negative change, the decline follows a quadratic trend during exercise (even when the workload is continuously adjusted to keep the rate of oxygen consumption stable) and is followed by a rapid rebound once the exercise bout is terminated (e.g., Acevedo et al., 1996). Given this pattern, the failure to find consistent dose-response effects in studies that used before-to-after assessments is not surprising, since dose-response differences that might have appeared during exercise had likely dissipated by the time the postexercise assessment took place (in many cases, after a cool-down or a period of recovery).

The second problem pertains to the assessment of affective responses. In most dose-response studies, researchers used multi-item self-report measures of state anxiety or mood states (see Ekkekakis & Petruzzello, 2000, for a review). This, however, was not based on evidence that these are the only affective variables relevant to exercise across various intensities. Instead, it was based on the fact that standardized self-report measures of state anxiety and mood states, appropriate for use with nonclinical samples, were commonly available and becoming popular at the time that this line of research got under way (i.e., in the early 1970s). Thus, in essence, the outcome variables were dictated by the available measures rather than the other way around. In actuality, the affective

responses that are likely to occur in different individuals under different conditions across the range of exercise intensities are impossible to predict at this stage. Therefore, in the context of dose-response studies, it would be advantageous to assess affective responses from a broad perspective, one that could capture all major variants of affective experience. Of course, because it would be impractical to ask participants whether they do or do not feel every affective state represented in the English vocabulary, this approach would have to involve a compromise; some degree of specificity must be sacrificed in exchange for breadth of scope. This can be accomplished by dimensional models of affect. According to the conceptual basis of dimensional models, affective states are systematically interrelated, such that their similarities and differences can be modeled parsimoniously along as few as two basic dimensions. The dimensional model with the longest history in affective psychology and the most extensive research base is the circumplex (Russell, 1980). In the circumplex model, the global affective space is defined by the orthogonal and bipolar dimensions of affective valence (ranging from displeasure to pleasure) and perceived activation (ranging from low to high). Given its breadth of scope, parsimony, balance, and domain-general nature, the circumplex model can be used as a template of affective space upon which the responses to various intensities of exercise can be mapped and compared (Ekkekakis & Petruzzello, 2002a).

The third problem is the standardization of exercise intensity. In the first wave of dose-response studies, participants exercised at the same level of heart rate or against the same level of resistance. The failure of this approach to take into account individual fitness levels soon became apparent, so the norm in most subsequent studies was to use percentages of measured or estimated maximal exercise capacity (maximal heart rate or oxygen uptake). Even this approach, however, is problematic for at least two reasons. First, the levels of intensity were essentially selected on an arbitrary basis (i.e., there was no conceptual reason for selecting, for example, 40%, 60%, and 80% as opposed to 30%, 50%, and 70%). Second, percentages of maximal capacity fail to take into account differences in the underlying metabolic processes, namely the contribution of aerobic and anaerobic metabolism. This is a problem identified in exercise physiology in the 1950s (Wells, Balke, & Van Fossan, 1957) and one that has resurfaced numerous times since then (e.g., Katch et al., 1978). At 75% of maximal oxygen uptake, for

example, it is possible that one individual uses almost entirely aerobic sources whereas another requires substantial anaerobic supplementation. Given the rather profound ventilatory, cardiovascular, and endocrine changes that occur as the organism transitions to anaerobic supplementation, it cannot be argued that a percentage of maximal capacity, such as 70%, can provide an effective method of standardizing the intensity of exercise across individuals. The solution is to take the underlying metabolic processes into account by selecting intensities in relation to a transition marker, such as the lactate or the gas exchange ventilatory threshold (Wells, Balke, & Van Fossan, 1957).

The fourth problem stems from the phenomenon of interindividual variability in affective responses. Statistical analyses of change in dose-response studies were conducted at the level of group aggregates, and thus individual differences were treated as error. However, individual differences could have considerable psychological significance. For example, they could be accounted for by relevant traits or situational appraisals. Furthermore, it is important to keep in mind that, perhaps unlike most other affect-inducing stimuli, exercise can have a bidirectional impact on affect, producing pleasure or displeasure. Therefore, in the context of exercise, individual differences may manifest themselves not only in the form of quantitative differences in the extent to which an individual feels better or worse, but also in the form of qualitatively different responses to the same stimulus (such as changes toward pleasure or displeasure). In fact, in a study involving a 30 min bout of moderate-intensity (60% of estimated maximal aerobic capacity) stationary cycling, 44.4% of the participants reported gradual improvements in affective valence during the bout, whereas 41.3% reported gradual declines (Van Landuyt et al., 2000). As a result of these divergent patterns, the average rating across the entire sample appeared unchanged. Clearly, however, this conclusion would have misrepresented the actual responses. Therefore, the examination of patterns of change at the individual level, in addition to the traditional aggregate-level analyses, is necessary.

The "Next Generation" of Dose-Response Studies

A series of studies addressing most of the limitations of previous research has appeared in

recent years and has provided the first reliable evidence of a dose-response relationship between the intensity of exercise and affective responses. The main finding, which has now been replicated by at least three independent laboratories, is that the point of transition from aerobic metabolism to anaerobic supplementation, operationally defined as either the gas exchange threshold or the lactate threshold, seems to be the turning point toward a decline in affective valence (reduced pleasure and, ultimately, increased displeasure) during exercise.

Bixby, Spalding, and Hatfield (2001) compared the effects of two exercise intensities, one corresponding to the gas exchange threshold and one corresponding to 75% of the gas exchange threshold, among 27 university students. Affect was assessed not only before and after exercise, but also during exercise. Specifically, the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was administered immediately before exercise, at the 20th minute of a 30 min bout on the cycle ergometer, and at the 20th minute of a 30 min recovery period. Furthermore, a visual analog scale of affective valence (pleasure-displeasure) was administered three times at baseline, three times during exercise (min 10, 20, 30), and three times during recovery (min 10, 20, 30). The results showed that the lower-intensity exercise led to an improvement, whereas the higher intensity led to a decline in affective valence during exercise. However, there were no significant differences between the two conditions after exercise, as there was a rapid "rebound" in affective valence in the higher-intensity condition as soon as the exercise bout ended.

Hall, Ekkekakis, and Petruzzello (2002) reported the results of a study examining ratings of affective valence, using the Feeling Scale (FS; Hardy & Rejeski, 1989), obtained at 1 min intervals during an incremental treadmill test to volitional exhaustion. The FS is an 11-point, single-item, bipolar measure of pleasure-displeasure, ranging from -5 to +5, and anchors at zero ("Neutral") and all odd integers, from "Very Good" (+5) to "Very Bad" (-5). The participants were 30 university students. To standardize the intensity of the activity despite variable test durations until the point of exhaustion, the first 2 min, the 4 min surrounding the gas exchange threshold (the minute before, the minute of, and the 2 min following it), and the last 2 min were retained for further analysis. The results showed that it was only once the participants exceeded the gas exchange threshold that signifi-

cant and nearly homogeneous declines in affective valence occurred. Consistent with the results of Bixby, Spalding, and Hatfield (2001), there was a significant improvement in the affective state within just the first minute of a cool-down walk.

Ekkekakis, Hall, and Petruzzello (2004) replicated this finding using two different treadmill protocols, with 30 participants in each, to eliminate the possibility that the decline in affective valence that was initiated with the gas exchange threshold was protocol specific. They used a data reduction method similar to that used by Hall, Ekkekakis, and Petruzzello (2002). Consistent with the previous results, trend analyses demonstrated that during both protocols, the only 3-point segments for which the quadratic declining trends in self-ratings of affective valence were significant were those initiated with the minute at which the gas exchange threshold occurred.

Ekkekakis, Hall, and Petruzzello (2001) extended this line of research by examining affective responses to constant-speed treadmill exercise, as opposed to the incremental protocols used in the two previous studies. Thirty volunteers ran on a treadmill for 15 min at three intensities (on separate days, in random order): 20% of maximal aerobic capacity below, at, and 10% above their gas exchange threshold. The changes in self-ratings of affective valence during exercise were not statistically significant for the conditions below and at the level of the gas exchange threshold. Only the responses during the run above the gas exchange threshold showed a significant quadratic declining trend. Eighty percent of the participants (24 of the 30) in this condition reported declines in affective valence by an average of 3.17 units on the 11-point FS. It is important to point out that the differences in treadmill speed between the conditions at and 10% above the gas exchange threshold were very small (typically less than one-half mile per hour) and so were the differences in heart rate (10 bpm difference from condition to condition at the end of the runs). Yet the significant differences in the pattern of affective responses underscore the importance of exceeding the gas exchange threshold. Also consistent with previous studies (Bixby, Spalding, & Hatfield, 2001; Hall, Ekkekakis, & Petruzzello, 2002), there were no differences between the three conditions after a cool-down, since affective valence showed a very rapid rebound as soon as the runs stopped.

Acevedo and colleagues (2003) examined the FS responses to three running intensities, one 10% of maximal aerobic capacity below, one at, and one

10% of maximal aerobic capacity above the onset of blood lactate accumulation (by convention, a concentration of 4 mM lactate per liter of blood) among 11 distance runners. Each run lasted for 5 min. The FS declined significantly and became negative only when the intensity exceeded the onset of blood lactate accumulation.

Collectively, these studies provide considerable support to the idea that the transition to anaerobic supplementation represents an important event from an adaptational standpoint and, as such, is accompanied by a strong signal to conscious awareness in the form of a decline in affective valence. Ekkekakis, Hall, and Petruzzello (2004) and Acevedo and colleagues (2003) expressed the view that perhaps this affective change, since it is apparently perceptible and salient, could be used by exercisers as a guide in regulating their exercise intensity. Importantly, a considerable literature suggests that an intensity at or just below the aerobic-anaerobic transition is effective in improving fitness and maintaining health (see Ekkekakis, Hall, & Petruzzello, 2004, for a review).

On the basis of this idea, Lind, Joens-Matre, and Ekkekakis (2005) examined the intensity that a sample of 23 middle-aged, healthy, but sedentary women self-selected when they were asked to exercise on a treadmill for 20 min. On average, at the 15th and the 20th minute of the exercise session, their treadmill speed, oxygen uptake, rating of perceived exertion, and rating of affective valence were not different from the values corresponding to their gas exchange threshold as identified from a previous graded treadmill test. This suggests that, even intuitively, without being taught to do so, exercisers might use the aerobic-anaerobic transition as a guide in regulating their intensity. Stated differently, it is possible that exercisers tend to choose an intensity that is not high enough to induce unpleasant affective responses. In fact, ratings of affective valence remained positive and stable throughout the session at self-selected intensity. Lind, Joens-Matre, and Ekkekakis (2005), however, found that, beyond the average trend, individuals within the group selected intensities that were from as low as 60% to as high as 160% of the oxygen uptake corresponding to the gas exchange threshold. It could be argued, then, that this is where the challenge for future research lies; namely, in developing an understanding for the sources of this variability and appropriate intervention methods for teaching both the overestimators and the underestimators to select intensities that are effective, safe, and

likely to elicit positive (or, at least, nonnegative) affective responses.

The Dual-Mode Theory

From the data reviewed so far, it seems clear that the aerobic-anaerobic transition acts as a turning point, beyond which affective valence begins to decline. It is interesting to ponder why this would be the case. The answer perhaps lies in the adaptational significance of this event.

Although exercise performed within the aerobic range can be continued for a long period of time, exercise that significantly exceeds the point of transition from aerobic metabolism to anaerobic supplementation precludes the maintenance of a physiological steady state, induces fatigue, and creates the need to stop the activity. Activity that requires a substantial anaerobic contribution increasingly depends on a relatively limited reservoir of metabolic resources (i.e., the adenosine triphosphate and creatine phosphate pool in the muscles and anaerobic glycolysis) compared to the resources available to aerobic metabolism (i.e., muscle and liver glycogen, free fatty acids from adipose tissue, and, to a lesser degree, body proteins). Furthermore, beyond the aerobic-anaerobic transition, a multitude of physiological adjustments takes place, drastically transforming the internal environment. Primarily, there is a rapid accumulation of lactate and hydrogen ions dissociated from lactic acid. This, in turn, has been linked to several processes that contribute to fatigue, including the accelerated breakdown of creatine phosphate (McCann, Mollé, & Caton, 1995), the gradual inhibition of glycolysis and glycogenolysis (Spriet et al., 1989), the inhibition of lipolysis (Boyd et al., 1974), and the interference with the calcium triggering of muscle contractions (Favero et al., 1995). In addition, lactic acidosis stimulates the release of catecholamines (Goldsmith et al., 1990), and thus the lactate threshold has been found to occur in close proximity to a catecholamine threshold (Urhausen et al., 1994; Weltman et al., 1994). In turn, catecholamines have widespread effects that further push the organism toward its functional limits, including a breakpoint in the relationship between the double product (the product of heart rate and systolic blood pressure) and work rate (Riley et al., 1997; Tanaka et al., 1997). Moreover, to compensate for metabolic acidosis, above the point of transition to anaerobic supplementation there is an increase in the frequency and depth of

ventilation (Wasserman, 1978). Finally, the transition to anaerobic supplementation is accompanied by the recruitment of low-efficiency fast-twitch muscle fibers (Helal, Guezennec, & Goubel, 1987; Nagata et al., 1981; Shinohara & Moritani, 1992), thus increasing the oxygen cost of work and disrupting coordination patterns.

Overall, these conditions begin to pose a challenge to the maintenance of homeostasis. Damasio (1995) has written that "certain configurations of body state" (p. 21), such as during a heart attack, induce an innate, preorganized affective response. We would argue that the aforementioned physiological adjustments that occur beyond the aerobic-anaerobic transition constitute a quintessential example of such a "configuration of body state." Given the possible implications of this condition for adaptation and survival, it would make good sense if this "configuration of body state" induced a pronounced negative affective response. In fact, studies have shown that as exercise intensity increases, the negative correlations between affective valence and various indices of metabolic strain (heart rate, ventilation, respiratory rate, oxygen consumption, blood lactate) increase in magnitude (Acevedo, Rinehardt, & Kraemer, 1994; Hardy & Rejeski, 1989).

Importantly, Acevedo and colleagues (2003) reported that, although affective valence shows no significant relationships with physiological variables below or at the onset of blood lactate accumulation, valence was significantly related in a negative direction to both heart rate and ventilation when the intensity exceeded the onset of blood lactate accumulation. Ekkekakis (2003, see p. 232) has also reviewed evidence showing that during exercise performed at an intensity exceeding the gas exchange threshold, but not below, physiological variables (respiratory exchange ratio, percentage of peak heart rate) accounted for significant portions of the variance in affective valence. Collectively, these findings suggest that, once exercise intensity exceeds the level of transition from aerobic metabolism to anaerobic supplementation, affective valence is strongly related to physiological indices of metabolic strain.

Besides physiological variables, affective responses to exercise have also been shown to have significant relationships with cognitive variables. Of these, self-efficacy has been studied most extensively. Although the strength of the relationship between self-efficacy and affective responses appears to differ as a function of exercise intensity, the pattern is still not entirely clear. On the basis

of the idea that the role of self-efficacy as a mediator of affective responses is strengthened in the face of challenging stimuli, McAuley and Courneya (1992) first proposed that the association between physical self-efficacy and affect should become stronger when exercise intensity reaches a level where bodily cues become unequivocally aversive (tentatively specified as above 70% of maximal heart rate). At such an intensity level, a highly efficacious person is expected to exhibit more positive affect compared to a less self-efficacious one. More recently, McAuley and colleagues (2000) extended the earlier hypothesis of McAuley and Courneya (1992) by proposing that "at high intensities, physiological cues . . . override cognitive processing" (p. 312). Therefore, cognitive influences on affective responses are theorized to become stronger when the intensity of exercise presents a substantial but not overwhelming challenge. Conversely, the influence of cognitive factors should be relatively weaker when the challenge posed by the exercise stimulus is either trivial or overwhelming. The studies conducted since then have produced conflicting results (Blanchard et al., 2002; McAuley et al., 2000; Tate, Petruzzello, & Lox, 1995; Treasure & Newbery, 1998).

Although the inconsistencies could be partly due to differences in the age and physical condition of the participants and the measures of affect, it is also possible that they are due to the arbitrarily selected levels of exercise intensity. Ekkekakis (2003) examined the results of a series of unpublished studies in which the intensity of exercise was determined in relation to the gas exchange threshold. These studies showed that self-efficacy was significantly related to affective responses when the intensity was proximal to the gas exchange threshold but not when it was significantly below or above it. In multiple regression analyses, in which both self-efficacy and physiological variables were entered as predictors of affective valence, self-efficacy accounted for the majority of the variance near the gas exchange threshold, whereas physiological variables were dominant when the intensity was higher and approached maximal capacity.

On the basis of these data, Ekkekakis (2003, 2005; Ekkekakis, Hall, & Petruzzello, 2005) formulated a new conceptual model of exercise-induced affective responses, named the dual-mode theory. A central thesis underpinning this conceptual model is that physical activity must be considered from an adaptational perspective. Thus, the dual-mode theory is based on the following core

assumptions (see Ekkekakis, Hall, & Petruzzello, 2005, for a more detailed discussion and references). (a) Physical activity has been an essential component of the conditions that shaped human evolution; (b) affective responses are manifestations of evolved psychological mechanisms, selected for their ability to promote health and well-being or to solve recurrent adaptational problems, with pleasure signifying utility and displeasure signifying danger (Cabanac, 1971, 1979; also see chapter 6); (c) affective responses, including those that originate in the body, depend on a hierarchically organized system involving multiple layers of control, from oligosynaptic, subcortical, and evolutionarily primitive pathways at the bottom and polysynaptic, evolutionarily recent, cortical pathways at the top; and (d) evolutionarily primitive functions show less interindividual variation, whereas functions that are evolutionarily recent show larger variability, as they are mostly shaped by individual developmental histories.

Based on these core assumptions, the dual-mode theory (Ekkekakis, 2003) posits that affective responses to exercise are the products of the continuous interplay between two general factors, namely (a) relevant cognitive processes originating primarily in the frontal cortex and involving such processes as appraisals of the meaning of exercise, goals, self-perceptions including self-efficacy, attributions, and considerations of the social context of exercise; and (b) interoceptive cues from a variety of receptors stimulated by exercise-induced physiological changes, which reach the affective centers of the brain via oligosynaptic subcortical pathways. The relative salience of these two factors is hypothesized to shift systematically as a function of exercise intensity. Specifically, cognitive factors should be dominant at intensities proximal to the aerobic-anaerobic transition, whereas interoceptive cues should gain salience when the intensity exceeds the aerobic-anaerobic transition and approaches an individual's maximal exercise capacity (see figure 7.1). The model, therefore, is consistent with extant findings showing that (a) the negative relationship between affective valence and physiological variables becomes increasingly stronger as the intensity of exercise increases and (b) the positive relationship between affective valence and self-efficacy appears to be strongest when the intensity approximates the aerobic-anaerobic transition but is weakened when the intensity approaches maximal levels.

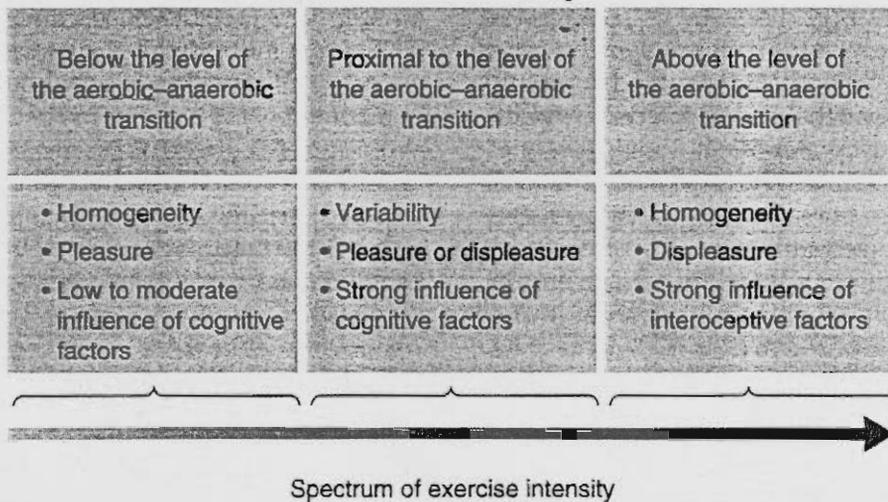


Figure 7.1 The dose-response model based on the dual-mode theory. Exercise intensity is considered in relation to the individually determined level of transition from aerobic metabolism to anaerobic supplementation. The model takes into account systematic patterns of change in the degree of interindividual variability and proposes a mechanistic basis for the type of affective responses that emerge at different intensities.

A Putative Neural Pathway

It is still not possible to take full advantage of the modern brain imaging technologies to examine the patterns of brain activity in response to vigorous exercise, due to problems associated with movement artifact (see chapter 3). This limitation makes examination of the dual-mode hypothesis at the neural level extremely difficult. Nevertheless, neuroscience has made considerable advances in delineating the neural network responsible for processing interoceptive cues, including those associated with exercise, and generating affective responses (see chapter 2). The extant information is sufficient to allow us to examine whether the predictions of the dual-mode theory are plausible at the level of the nervous system. This can be achieved through a review of evidence on whether neuroanatomical connections and neurophysiological mechanisms that would be needed for the model to function as hypothesized are in place.

To support the plausibility of the model, the neuroscientific evidence would have to indicate that

- the system that collects and relays interoceptive information to the brain is highly sensitive to variations in the intensity of internal signals;
- the processing of interoceptive signals in the brain is based on a hierarchically organized system that includes both corti-

cally mediated and noncortically mediated pathways; and

- there are appropriate gating mechanisms capable of redirecting interoceptive signals to the subcortical pathways when the intensity exceeds a certain critical threshold.

Before we present the relevant findings that support each of these statements, we must issue a caveat. Particularly in recent years, it has become increasingly clear that the amygdaloid complex (a part of the limbic system, situated in the anterior part of the temporal lobe; Sah et al., 2003) serves as a critical center for integrating affective information and assigning affective significance to sensory events (Aggleton & Young, 2000; LeDoux, 1986, 1989, 1996; Zald, 2003). Although, consistent with these accounts, the discussion here focuses on the amygdala, it is important to note that several other brain areas, such as the insula and cingulate cortex (Critchley et al., 2004), are activated by exercise and also appear to play important roles in the genesis of affective responses.

In humans, direct evidence of amygdala involvement during vigorous exercise is still lacking because of the aforementioned technical difficulties in obtaining brain imaging data free of movement artifacts. However, indirect evidence is available, in the form of studies showing that the amygdala is activated in response to noxious somatic stimuli (Bingel et al., 2002; Bornhövd et al., 2002; Derbyshire et al., 1997; Schneider et al.,

2001), blood pressure elevations (Harper et al., 2000) and drops (Henderson et al., 2004), and induced dyspnea and "air hunger" sensations (Brannan et al., 2001; Evans et al., 2002; Liotti et al., 2001). However, most of the relevant evidence comes from animal studies. A study in rats showed that glucose utilization was increased in response to exercise by 56% in the central nucleus, 33% in the lateral nucleus, and 18% in the medial nucleus of the amygdala (Vissing, Andersen, & Diemer, 1996). This was not a surprising finding. Anatomical studies had already established that the amygdala receives afferent connections from various brain regions known to be involved in cardiovascular, respiratory, and endocrine regulation during exercise, including the hypothalamus and the ventrolateral medulla (Aggleton, Burton, & Passingham, 1980; Cechetto, Ciriello, & Calaresu, 1983; Ciriello, Schultz, & Roder, 1994; Ottersen, 1980; Roder & Ciriello, 1993, 1995; Turner & Herkenham, 1991; Volz et al., 1990; Zardetto-Smith & Gray, 1995). Moreover, physiological studies had shown that neurons in the amygdala respond to spontaneous increases in blood pressure (Ben-Ari et al., 1973), stimulation of baro- and chemoreceptors (Cechetto & Calaresu, 1984, 1985; Knuepfer et al., 1995; Schütze et al., 1987), and stimulation of the carotid sinus and aortic depressor nerves (Cechetto & Calaresu, 1983). Furthermore, discharges in amygdala neurons exhibit timing relationships with the cardiac and respiratory cycles (Frysinger & Harper, 1989; Frysinger, Zhang, & Harper, 1988). The amygdala had also been shown to receive extensive somatosensory input (Romanski et al., 1993; Uwano et al., 1995) and to be involved in the processing of noxious somatosensory stimuli (Bernard & Besson, 1990; Bernard, Bester, & Besson, 1996; Bernard, Huang, & Besson, 1990, 1992; Bernard, Peschanski, & Besson, 1989; Romanski et al., 1993).

From the perspective of the dual-mode theory, the key question is how the exercise-induced interoceptive cues reach the amygdala. Are there both subcortical and cortical routes, and is the intensity of stimulation important in determining which pathway is involved? As noted in the previous section, exercise generates a great variety of interoceptive signals, such as increases in heart rate and blood pressure, tissue ischemia, acidosis (drops in pH), increased concentration of protons (H^+) dissociated from lactic acid, breathlessness associated with hypoxia or hypercapnia, and muscle pain. In patient populations, exercise can generate additional signals, such as stimulation

of articular nociceptors in arthritic joints, angina pectoris resulting from myocardial ischemia, or ischemic pain in leg muscles due to intermittent claudication.

All of these bits of information about the condition of the body are collected by primary afferent neurons that exist in virtually all tissues (also see chapter 2). Some of these, situated at strategic locations, also perform specialized functions, such as monitoring arterial pressure, the tension of respiratory gases, and changes in pH. Exercise appears to engage mainly two types of primary afferent neurons, both of small diameter and therefore having relatively slow conducting velocities, the thinly myelinated A δ (or Group III) and the unmyelinated C (or Group IV). The majority of A δ neurons are activated by deformations in contracting muscles and are, therefore, considered mechanoreceptors, whereas the majority of C neurons are activated by metabolic changes and are therefore considered chemo- or metaboreceptors (Kaufman & Forster, 1996; Mense, 1996).

Consistent with the notion that the interoceptive system is highly sensitive to variations in the intensity of stimulation, primary afferent neurons have different threshold properties and consequently respond to different levels of stimulation (Cervero, 1994; Cervero & Janig, 1992). One type is characterized by a low threshold and is therefore activated by innocuous intensities of stimulation. These receptors, which in the case of exercise-associated stimuli have been characterized as ergoreceptors, carry information necessary for basic regulatory adjustments and might be involved in relaying nonnoxious sensations. A second type of primary afferent neuron is characterized by a high threshold and is used to encode intensity by responding only to stimuli in the noxious range. These neurons are therefore characterized as nociceptors. Finally, a third type, called the "silent nociceptor," exhibits no response under normal circumstances but shows a large response in cases of prolonged stimulation involving the high-threshold neurons. Such cases may include acute tissue hypoxia or inflammation. The different thresholds of the three receptor types ensure that the intensity of stimulation, and therefore the extent of any deviations of physiological parameters from the normal range, will be properly and reliably encoded as this information enters the central nervous system.

A δ and C afferents enter the spinal cord mainly through the dorsal horn and synapse with spinal neurons in several laminae but primarily with

lamina I (Craig, 1996; also see chapter 2). In turn, lamina I neurons have several major targets in the brain. These include the nucleus of the solitary tract, the parabrachial nucleus of the pons, the periaqueductal gray, the ventrolateral medulla, and the sensory thalamus (Iwamoto et al., 1996).

After this point, from the perspective of cognitive theories of emotion, the only relevant pathway is the one that projects from the thalamus to the somatosensory and frontal cortices. Anatomical studies have shown that the amygdala receives extensive input from several cortical areas (McDonald, 1998). This pathway is consistent with a view of emotions as driven by cognitive appraisals (LeDoux, 1994, 1989). The somatosensory cortices enable the recognition and localization of the source of the stimulation, and the frontal cortex can analyze its significance and possible implica-

tions for survival, well-being, and the goals of the individual. However, LeDoux (1986, 1996) showed that the thalamocorticoamygdala pathway is not necessary for fear conditioning to acoustic stimuli and therefore that this may not be the only pathway involved in emotional responses. He demonstrated that a direct thalamoamygdala projection that bypasses the cortex could suffice. He named this direct projection the “low road,” as opposed to the “high road” via the cortex (LeDoux, 1996; see figure 7.2a). Speculating on the functional significance of this “low road,” LeDoux (1996) noted that it represents an “evolutionary relic” (p. 163) that, since it has continued to exist for millions of years despite the evolution of the more advanced “high road,” must still serve some useful function. According to LeDoux (1996), the capacity of the “low road” to activate the amygdala several mil-

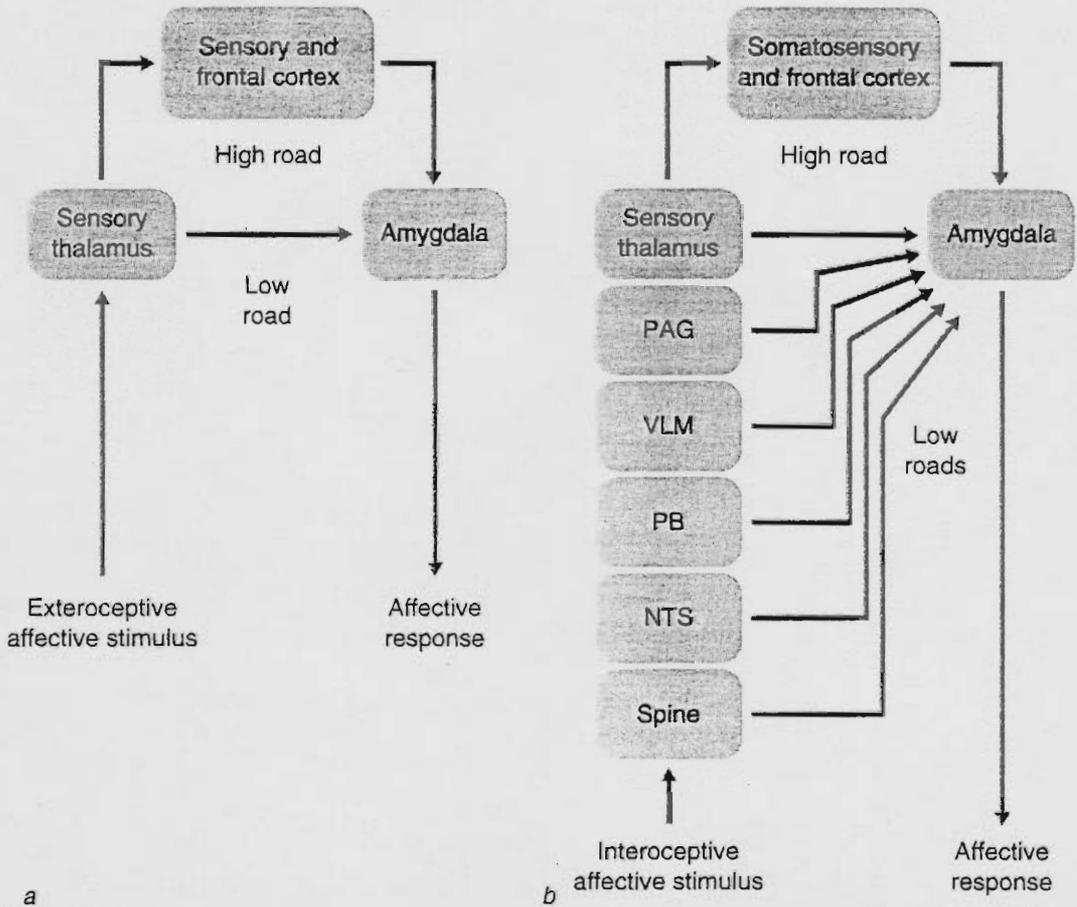


Figure 7.2 (a) The model of affective response generation proposed by LeDoux (1986, 1996) for exteroceptive stimuli. (b) A proposed modification specifically for exercise-associated interoceptive stimuli. In addition to the sensory thalamus, subcortical pathways to the amygdala originate in the spine, nucleus of the solitary tract (NTS), parabrachial nucleus (PB), ventrolateral medulla (VLM), and periaqueductal gray (PAG). The presence of multiple “low roads” in the case of interoceptive stimuli can be explained from an evolutionary standpoint, since in general, intense stimuli from the internal environment of the body probably have more immediate and potentially severe implications for survival and adaptation than exteroceptive stimuli.

liseconds more rapidly than the “high road” can make the difference between “the quick and the dead” (p. 163).

LeDoux’s interpretation of the functional significance of the “low road” as an alternative non-cognitive pathway for the elicitation of affective responses, which questions the long-held belief in social-cognitive psychology that cognitive appraisal is a prerequisite for such responses, has met with criticism. Emotion theorists have argued that LeDoux’s model essentially begs the invocation of a homunculus at the subcortical level. Clore and Ortony (2000), for example, noted that, for the amygdala to be activated via the “low road” when a snake is seen on the ground, as LeDoux had suggested, some recognition and evaluation of the adaptational significance of the snake must take place. However, by most definitions, these functions constitute cognitive processing.

Here, we use LeDoux’s basic idea of a system that involves both a cortically mediated and a noncortically mediated route for the activation of the amygdala and, therefore, both a cognitively mediated and a noncognitively mediated mode of eliciting affective responses. However, our model differs from LeDoux’s in an important way that avoids the homunculus problem. Although LeDoux’s model pertained to exteroceptive stimuli, such as auditory and visual cues, which must undergo some—even rudimentary—recognition and interpretation before they can acquire adaptational significance, most of the adaptational significance of interoceptive stimuli, such as those induced by exercise, is already encoded in their intensity. This fundamental property (intensity) can be “interpreted” by as basic and simple a mechanism as a neural gate that allows only impulses exceeding a certain threshold to flow through. From an evolutionary perspective, the intensity of interoceptive stimulation has critical survival value (Schneirla, 1959), and consequently “responses to stimulus intensity are highly conserved throughout the neuraxis” (Coghill et al., 1999, p. 1940). According to Damasio (1999),

The range of possible states of the internal milieu and of the viscera is tightly limited. This limitation is built into the organism specifications since the range of states that is compatible with life is small. The permissible range is indeed so small and the need to respect its limits so absolute for survival that organisms spring forth equipped with an automatic regulation system to ensure that

life threatening deviations do not occur or can be rapidly corrected. (p. 141)

Another difference between the structures of the systems that process exteroceptive and interoceptive stimuli is also very important. Unlike what happens with the singular thalamo-amygdala pathway that functions as the “low road” in LeDoux’s model, interoceptive stimuli are forwarded to the amygdala by a multitude of subcortical routes, or several “low roads” (see figure 7.2b). Specifically, interoceptive information can reach the amygdala via the thalamus (Ottersen & Ben-Ari, 1979; Turner & Herkenham, 1991), the nucleus of the solitary tract (Ottersen, 1981; Ricardo & Koh, 1978; Zardetto-Smith & Gray, 1990), the parabrachial nucleus (Bernard, Alden, & Besson, 1993; Bernard, Peschanski, & Besson, 1989; Ma & Peschanski, 1988; Ottersen, 1981), the periaqueductal gray (Rizvi et al., 1991), and a direct spinal projection (Burstein & Potrebic, 1993; Cliffer, Burstein, & Giesler, 1991; Giesler, Katter, & Dado, 1994; Menétrey & de Pommery, 1991). Furthermore, as noted earlier, the amygdala also receives projections from other subcortical areas involved in cardiovascular, respiratory, and endocrine regulation, including the ventrolateral medulla and the hypothalamus.

A remaining important issue pertains to the presence of appropriate intensity-sensitive gating mechanisms that would permit the activation of the amygdala through the aforementioned subcortical pathways once the intensity of exercise exceeds the level of the aerobic-anaerobic transition and the barrage of interoceptive stimuli from throughout the body intensifies (see the summary of the peripheral physiological changes that accompany the aerobic-anaerobic transition in the previous section). Based on what is presently known, such gating can occur at least at the following four points. First, the sensory thalamus is the primary gate that can control information flow to both the cortex and the amygdala (McCormick & Bal, 1994). Second, the nucleus of the solitary tract, a major recipient of interoceptive information from the spinal cord and a major source of projections to the amygdala, has been proposed to serve a gating function for various modalities of interoceptive afferents (Mifflin, Spyer, & Withington-Wray, 1988b; Toney & Mifflin, 2000; Zhang & Mifflin, 2000). Its inhibitory effect is, at least in part, under the control of GABA-ergic (gamma-aminobutyric acid) projections from the central nucleus of the amygdala (Saha, Batten, & Henderson, 2000) and

the hypothalamus (Jordan, Mifflin, & Spyer, 1988; Mifflin, Spyer, & Withington-Wray, 1988a). Third, the parabrachial nucleus, a recipient of interoceptive information from the spinal cord and nucleus of the solitary tract and also a major source of projections to the amygdala, has been postulated to serve as a modulator of this information, under the influence of input from the periaqueductal gray (Krout, Jansen, & Loewy, 1998). Fourth, the lateral nucleus of the amygdala, the entry point of most sensory input to the amygdala, can also function as a gating mechanism, regulating information flow through the rest of the amygdaloid complex (Lang and Paré, 1998; Paré et al., 2003; Royer, Martina, & Paré, 1999).

Theoretically, these gates make it possible for interoceptive impulses to be directed toward the cortex or directly toward the amygdala, depending on their intensity. Preliminary data are consistent with this scenario. Imaging studies in humans have shown that, in general, "as the stimulus rating increases in intensity, an increasing number of brain regions become active" (Derbyshire et al., 1997, p. 438). This suggests that certain intensity-sensitive gating mechanisms must exist that permit the flow of information to additional areas as the intensity rises. Specifically regarding the amygdala, an imaging study in humans involving thermal stimuli ranging from innocuous to painful showed that the amygdala is activated once a painful threshold is exceeded and that, thereafter, additional increases in stimulus intensity are reflected by linear increases in amygdala activation (Bornhövd et al., 2002). In rats, somatosensory stimulation using an air puff resulted in a mean latency of 61 msec for the response of the lateral nucleus of the amygdala (Uwano et al., 1995), whereas the mean value using an electric foot shock was only 17 msec (Romanski et al., 1993). This difference suggests that the two stimuli reached the lateral nucleus through different pathways, with the more innocuous one likely having followed the longer and, therefore, slower thalamocorticoamygdala route and the more intense one likely having reached the target via one or more of the shorter and faster subcortical routes.

In summary, there appears to be enough evidence to consider the dual-mode theory plausible at the neural level. The data we reviewed indicate that

- a. the intensity of interoceptive information is a feature of paramount adaptational importance that is well preserved from the level

of the primary afferents to the higher levels of the brain,

- b. interoceptive information reaches the amygdala (and other areas involved in affective responses) via both cortical and subcortical routes, and
- c. appropriate gating mechanisms exist at critical locations that could permit the flow of interoceptive cues either to the cortical or to the subcortical routes depending on their intensity.

Therefore, as the dual-mode theory postulates, once the level of the aerobic-anaerobic transition is exceeded and a multitude of peripheral physiological cues initiate a positively accelerated response, it seems reasonable to suggest that the flow of this information to the amygdala will be primarily via the previously described subcortical pathways. Following the rationale of LeDoux's (1986, 1989, 1994, 1996) model, this would suggest that, when the intensity of exercise exceeds the level of the aerobic-anaerobic transition, the role of cortically mediated cognitive influences on affective responses would be attenuated and these responses would more directly reflect the perturbed physiological condition of the body.

Conclusions

The following conclusions can be drawn from the data summarized in this chapter. First, contrary to the widely popularized view that exercise, in general, makes people feel better, substantial evidence indicates that the relationship between exercise and affective responses is complex, with affective responses, under certain conditions, being negative rather than positive. In fact, the positive affective responses are limited to (a) during and after low-intensity and self-paced exercise and (b) recovery from vigorous exercise. In contrast, exercise intensity that exceeds the level of the aerobic-anaerobic transition (or the level of intensity associated with the onset of other symptoms, such as muscular, skeletal, or cardiorespiratory) is associated with declines in affective valence.

Second, a midrange intensity (i.e., not "too low," not "too high") is not necessarily the optimal stimulus for positive affective change. It is only the recovery from such activity that brings about unified positive responses. During exercise performed at a midrange intensity, there appears to be great

interindividual variability, with some individuals experiencing positive and others experiencing negative changes in affective valence.

Third, the patterns of interindividual variability in affective responses appear to be systematic and dependent on the intensity of exercise. Specifically, homogeneity emerges during exercise of low intensity (below the aerobic-anaerobic transition), when most responses are positive, and during exercise of high intensity (above the aerobic-anaerobic transition), when most responses are negative. On the other hand, variability emerges during activity performed near the level of the aerobic-anaerobic transition.

Fourth, the influence of cognitive factors (e.g., self-efficacy) on affective responses to exercise appears to be modulated by the intensity of exercise and is gradually reduced once the intensity exceeds the level of the aerobic-anaerobic transition (or symptom onset). Conversely, the influence of peripheral physiological cues, such as those associated with respiration and blood lactate concentration (or symptom-related nociception), is gradually increased and becomes dominant as the intensity exceeds the level of the aerobic-anaerobic transition (or symptom onset).

This last point has significant implications for interventions aimed at helping exercisers cope with the unpleasant sensations particularly during the critical early stages of exercise involvement. Based on the dual-mode theory, the effectiveness of some of the popular cognitive techniques for coping with such sensations might be limited to intensities below or near the aerobic-anaerobic transition (or symptom onset). Such techniques include attentional dissociation (i.e., "turn your attention away from your body"), cognitive restructuring (i.e., "think of these unpleasant symptoms as something positive, as signs of improvement"), and bolstering one's sense of physical self-efficacy (i.e., "believe that you can do it"). Insistence on the part of an exercise leader on these techniques, when the intensity exceeds a critical level that renders them ineffective, might lead to persistent negative affective responses to exercise, frustration, reduction of intrinsic motivation for exercise, and eventually dropout.

Directions for Future Investigation

From the standpoint of basic research, perhaps the most important condition for substantial progress

is the abandonment of the exclusive focus on the positive affective responses to exercise. It should be clear that the affective responses to exercise can be complex and multifaceted and, therefore, driven by a variety of underlying mechanisms.

Future basic studies should seek to replicate and thus establish the reliability of the recent dose-response findings reviewed here. That the aerobic-anaerobic transition is the turning point toward a decline in affective valence has now been found in three different laboratories (Acevedo et al., 2003; Bixby, Spalding, & Hatfield, 2001; Hall, Ekkekakis, & Petruzzello, 2002) and replicated in one (Ekkekakis, Hall, & Petruzzello, 2001, 2004). That there are systematic intensity-dependent changes in the patterns of interindividual variation in affective valence responses has been examined in only one laboratory (Ekkekakis, Hall, & Petruzzello, 2005). Although several laboratories have found that the magnitude of the negative relationship between affective valence and physiological variables increases as the intensity of exercise increases (e.g., Acevedo, Rinehardt, & Kraemer, 1994; Ekkekakis, 2003; Hardy & Rejeski, 1989), the relationship of affective valence with cognitive variables across different levels of exercise intensity remains unclear (Blanchard et al., 2002; McAuley & Courneya, 1992; McAuley et al., 2000; Tate, Petruzzello, & Lox, 1995; Treasure & Newbery, 1998). As noted earlier, this is possibly due to the fact that different laboratories have defined exercise intensity in different ways, using arbitrarily selected percentages of maximal capacity. Preliminary evidence indicates that clarity might be established once the intensity is defined in relation to the level of the aerobic-anaerobic transition (Ekkekakis, 2003). What other cognitive variables, besides self-efficacy, are related to affective responses during exercise is also a question that warrants attention. In general, as basic research on the dose-response relationship moves forward, it is necessary to consider the methodological issues that were outlined here (also see Ekkekakis & Petruzzello, 1999, 2000).

Additional steps are also clearly needed in deciphering the neural underpinnings of the affective responses to various intensities of exercise. The putative model we presented here appears reasonable based on the extant evidence but remains speculative. Given the current technical difficulty with testing the model in a human brain imaging study, priority should be given to animal research. As noted, only one known study (Vissing, Andersen, & Diemer, 1996) has directly examined

the acute response of the amygdala to exercise. This appears to be the unfortunate consequence of the fact that most neuroanatomical and neurophysiological studies of exercise responses focus on cardiovascular and respiratory regulation and thus rarely venture into areas beyond the brainstem. It is also important to reiterate that, although here we focused on the amygdala, the role of other areas, including the insula, the cingulate, the prefrontal cortex, and the nucleus accumbens, should be investigated as well.

From the standpoint of practical applications, the data and conceptual model presented here raise some interesting possibilities for future applied research. First, there is the possibility of using the significant decline in affective valence that accompanies the transition to anaerobic metabolism as a practical aid in self-monitoring and self-regulating exercise intensity (Acevedo et al., 2003; Ekkekakis, Hall, & Petruzzello, 2004). Research has shown that novice exercisers, on average, intuitively select an intensity corresponding to the level of the aerobic-anaerobic transition (Lind, Joens-Matre, & Ekkekakis, 2005). It is interesting to ponder whether increasing the awareness of affective changes could be used as a guide for helping those individuals who inadvertently select intensities either significantly below or significantly above this level to improve their self-regulatory skills.

Applied studies should also examine the range of effectiveness of cognitive techniques aimed at helping novice exercisers cope with the unpleasant affective responses to exercise (e.g., attentional dissociation, cognitive restructuring, self-efficacy), particularly during the critical early stages of exercise participation. We have suggested here that the effectiveness of these techniques might be limited to intensities below or near the level of the aerobic-anaerobic transition. Intensities that exceed this level are probably not only ineffective at conferring any additional fitness or health benefits to exercisers, but will also raise the risk of muscular and skeletal injuries, cardiac complications, and so on. Therefore, the proper solution would not be to learn to tolerate these intensities but rather to develop the appropriate skills to detect and avoid them. Techniques based on the principles of biofeedback, aimed at increasing interoceptive acuity specifically for the physiological, perceptual, and affective symptoms characteristic of the aerobic-anaerobic transition, might be helpful in this regard (Ekkekakis & Petruzzello, 2002b).

Finally, the long-term goal should be to examine the intensity-affect-adherence hypothesized causal chain in its entirety. There are presently no studies that have manipulated exercise intensity, examined the impact of these manipulations on adherence and dropout, and tested the mediational role of affective responses. This is clearly a significant void. To repeat our statement in the introduction, in the long run, people generally choose to do what makes them feel good and avoid what makes them feel bad. Therefore, it is possible that improving our understanding of the relationship between exercise intensity and affective responses might be one of the keys to solving the critical public health problem of physical inactivity.

References

- Acevedo, E.O., Gill, D.L., Goldfarb, A.H., & Boyer, B.T. (1996). Affect and perceived exertion during a two-hour run. *International Journal of Sport Psychology, 27*, 286-292.
- Acevedo, E.O., Kraemer, R.R., Haltom, R.W., & Tryniecki, J.L. (2003). Perceptual responses proximal to the onset of blood lactate accumulation. *Journal of Sports Medicine and Physical Fitness, 43*, 267-273.
- Acevedo, E.O., Rinehardt, K.F., & Kraemer, R.R. (1994). Perceived exertion and affect at varying intensities of running. *Research Quarterly for Exercise and Sport, 65*, 372-376.
- Aggleton, J.P., Burton, M.J., & Passingham, R.E. (1980). Cortical and subcortical afferents to the amygdala of the rhesus monkey (*macaca mulatta*). *Brain Research, 190*, 347-368.
- Aggleton, J.P., & Young, A.W. (2000). The enigma of the amygdala: On its contribution to human emotion. In R.D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 106-128). New York: Oxford University Press.
- Ben-Ari, Y., Le Gal la Salle, G., & Champagnat, J. (1973). Amygdala unit activity changes related to a spontaneous blood pressure increase. *Brain Research, 52*, 394-398.
- Bernard, J.F., Alden, M., & Besson, J.M. (1993). The organization of the efferent projections from the pontine parabrachial area to the amygdaloid complex: A phaseolus vulgaris leucoagglutinin (PHA-L) study in the rat. *Journal of Comparative Neurology, 329*, 201-229.
- Bernard, J.F., & Besson, J.M. (1990). The spino(trigemin)ontoamygdaloid pathway: Electrophysiological evidence for an involvement in pain processes. *Journal of Neurophysiology, 63*, 473-490.

- Bernard, J.F., Bester, H., & Besson, J.M. (1996). Involvement in the spino-parabrachio-amygdaloid and -hypothalamic pathways in the autonomic and affective emotional aspects of pain. *Progress in Brain Research, 107*, 243-255.
- Bernard, J.F., Huang, G.F., & Besson, J.M. (1990). Effects of noxious somesthetic stimulation on the activity of neurons of the nucleus centralis of the amygdala. *Brain Research, 523*, 347-350.
- Bernard, J.F., Huang, G.F., & Besson, J.M. (1992). Nucleus centralis of the amygdala and the globus pallidus ventralis: Electrophysiological evidence for an involvement in pain processes. *Journal of Neurophysiology, 68*, 551-569.
- Bernard, J.F., Peschanski, M., & Besson, J.M. (1989). A possible spino(trigemino)-ponto-amygdaloid pathway for pain. *Neuroscience Letters, 100*, 83-88.
- Bingel, U., Quante, M., Knab, R., Bromm, B., Weiller, C., & Büchel, C. (2002). Subcortical structures involved in pain processing: Evidence from single-trial fMRI. *Pain, 99*, 313-321.
- Bixby, W.R., Spalding, T.W., & Hatfield, B.D. (2001). Temporal dynamics and dimensional specificity of the affective response to exercise of varying intensity: Differing pathways to a common outcome. *Journal of Sport and Exercise Psychology, 23*, 171-190.
- Blanchard, C.M., Rodgers, W.M., Courneya, K.S., & Spence, J.C. (2002). Moderators of the exercise/feeling-state relationship: The influence of self-efficacy, baseline, and in-task feeling states at moderate- and high-intensity exercise. *Journal of Applied Social Psychology, 32*, 1379-1395.
- Bornhövd, K., Quante, M., Glauche, V., Bromm, B., Weiller, C., & Büchel, C. (2002). Painful stimuli evoke different stimulus-response functions in the amygdala, prefrontal, insula and somatosensory cortex: A single-trial fMRI study. *Brain, 125*, 1326-1336.
- Boyd, A.E., Gamber, S.R., Mager, M., & Lebovitz, H.E. (1974). Lactate inhibition of lipolysis in exercising man. *Metabolism, 23*, 531-542.
- Brannan, S., Liotti, M., Egan, G., Shade, R., Madden, L., Robillard, R., Abplanalp, B., Stofer, K., Denton, D., & Fox, P.T. (2001). Neuroimaging of cerebral activations and deactivations associated with hypercapnia and hunger for air. *Proceedings of the National Academy of Sciences, 98*, 2029-2034.
- Burstein, R., & Potrebic, S. (1993). Retrograde labeling of neurons in the spinal cord that project directly to the amygdala or the orbital cortex in the rat. *Journal of Comparative Neurology, 335*, 469-485.
- Cabanac, M. (1971). Physiological role of pleasure. *Science, 173*, 1103-1107.
- Cabanac, M. (1979). Sensory pleasure. *Quarterly Review of Biology, 54*, 1-29.
- Cechetto, D.F., & Calaresu, F.R. (1983). Response of single units in the amygdala to stimulation of buffer nerves in cat. *American Journal of Physiology, 244*, R646-R651.
- Cechetto, D.F., & Calaresu, F.R. (1984). Units of the amygdala responding to activation of carotid baro- and chemoreceptors. *American Journal of Physiology, 246*, R832-R836.
- Cechetto, D.F., & Calaresu, F.R. (1985). Central pathways relaying cardiovascular afferent information to amygdala. *American Journal of Physiology, 248*, R38-R45.
- Cechetto, D.F., Ciriello, J., & Calaresu, F.R. (1983). Afferent connections to cardiovascular sites in the amygdala: A horseradish peroxidase study in the cat. *Journal of the Autonomic Nervous System, 8*, 97-110.
- Cervero, F. (1994). Sensory innervation of the viscera: Peripheral basis of visceral pain. *Physiological Reviews, 74*, 95-138.
- Cervero, F., & Janig, W. (1992). Visceral nociceptors: A new world order? *Trends in Neurosciences, 15*, 374-378.
- Ciriello, J., Schultz, C.G., & Roder, S. (1994). Collateral axonal projections from ventrolateral medullary non-catecholaminergic neurons to central nucleus of the amygdala. *Brain Research, 663*, 346-351.
- Cliffer, K.D., Burstein, R., & Giesler, G.J. (1991). Distributions of spinothalamic, spinohypothalamic, and spinotelencephalic fibers revealed by anterograde transport of PHA-L in rats. *Journal of Neuroscience, 11*, 852-868.
- Clore, G.L., & Ortony, A. (2000). Cognition in emotion: Always, sometimes, or never? In R.D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 24-61). New York: Oxford University Press.
- Coghill, R.C., Sang, C.N., Maisog, J.M., & Iadarola, M.J. (1999). Pain intensity processing within the human brain: A bilateral, distributed mechanism. *Journal of Neurophysiology, 82*, 1934-1943.
- Craig, A.D. (1996). An ascending general homeostatic afferent pathway originating in lamina I. *Progress in Brain Research, 107*, 225-242.
- Critchley, H.D., Wiens, S., Rothstein, P., Öhman, A., & Dolan, R.J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience, 7*, 189-195.
- Damasio, A.R. (1995). Toward a neurobiology of emotion and feeling: Operational concepts and hypotheses. *Neuroscientist, 1*, 19-25.
- Damasio, A.R. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.
- Derbyshire, S.W.G., Jones, A.K.P., Gyulai, F., Clark, S., Townsend, D., & Firestone, L.L. (1997). Pain processing during three levels of noxious stimulation produces differential patterns of central activity. *Pain, 73*, 431-445.
- Dishman, R.K., & Buckworth, J. (1996). Increasing physical activity: A quantitative synthesis. *Medicine and Science in Sports and Exercise, 28*, 706-719.

- Ekkekakis, P. (2003). Pleasure and displeasure from the body: Perspectives from exercise. *Cognition and Emotion, 17*, 213-239.
- Ekkekakis, P. (2005). The study of affective responses to acute exercise: The dual-mode model. In R. Stelter & K.K. Roessler (Eds.), *New approaches to exercise and sport psychology* (pp. 119-146). Oxford, United Kingdom: Meyer & Meyer Sport.
- Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2001). Intensity of acute exercise and affect: A critical reexamination of the dose-response relationship [abstract]. *Medicine and Science in Sports and Exercise, 33*, S50.
- Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2004). Practical markers of the transition from aerobic to anaerobic metabolism during exercise: Rationale and a case for affect-based exercise prescription. *Preventive Medicine, 38*, 149-159.
- Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2005). Variation and homogeneity in affective responses to physical activity of varying intensities: An alternative perspective on dose-response based on evolutionary considerations. *Journal of Sports Sciences, 23*, 477-500.
- Ekkekakis, P., Hall, E.E., Van Landuyt, L.M., & Petruzzello, S.J. (2000). Walking in (affective) circles: Can short walks enhance affect? *Journal of Behavioral Medicine, 23*, 245-275.
- Ekkekakis, P., & Petruzzello, S.J. (1999). Acute aerobic exercise and affect: Current status, problems, and prospects regarding dose-response. *Sports Medicine, 28*, 337-374.
- Ekkekakis, P., & Petruzzello, S.J. (2000). Analysis of the affect measurement conundrum in exercise psychology: I. Fundamental issues. *Psychology of Sport and Exercise, 2*, 71-88.
- Ekkekakis, P., & Petruzzello, S.J. (2002a). Analysis of the affect measurement conundrum in exercise psychology: IV. A conceptual case for the affect circumplex. *Psychology of Sport and Exercise, 3*, 35-63.
- Ekkekakis, P., & Petruzzello, S.J. (2002b). Biofeedback in exercise psychology. In B. Blumenstein, M. Bar-Eli, & G. Tenenbaum (Eds.), *Brain and body in sport and exercise: Biofeedback application in performance enhancement* (pp. 77-100). Chichester, England: Wiley.
- Evans, K.C., Banzett, R.B., Adams, L., McKay, L., Frackowiak, R.S.J., & Corfield, D.R. (2002). BOLD fMRI identifies limbic, paralimbic, and cerebellar activation during air hunger. *Journal of Neurophysiology, 88*, 1500-1511.
- Favero, T.G., Zable, A.C., Bowman, M.B., Thompson, A., & Abramson, J.J. (1995). Metabolic end products inhibit sarcoplasmic reticulum Ca^{2+} release and [3H]ryanodine binding. *Journal of Applied Physiology, 78*, 1665-1672.
- Fox, K.R. (1999). The influence of physical activity on mental well-being. *Public Health and Nutrition, 2*, 411-418.
- Frynsinger, R.C., & Harper, R.M. (1989). Cardiac and respiratory correlations with unit discharge in human amygdala and hippocampus. *Electroencephalography and Clinical Neurophysiology, 72*, 463-470.
- Frynsinger, R.C., Zhang, J., & Harper, R.M. (1988). Cardiovascular and respiratory relationships with neuronal discharge in the central nucleus of the amygdala during sleep-waking states. *Sleep, 11*, 317-332.
- Gauvin, L., & Brawley, L.R. (1993). Alternative psychological models and methodologies for the study of exercise and affect. In P. Seraganian (Ed.), *Exercise psychology: The influence of physical exercise on psychological processes* (pp. 146-171). New York: Wiley.
- Giesler, G.J., Katter, J.T., & Dado, R.J. (1994). Direct spinal pathways to the limbic system for nociceptive information. *Trends in Neurosciences, 17*, 244-250.
- Goldsmith, S.R., Iber, C., McArthur, C.D., & Davies, S.F. (1990). Influence of acid-base status on plasma catecholamines during exercise in normal humans. *American Journal of Physiology, 258*, R1411-R1416.
- Hall, E.E., Ekkekakis, P., & Petruzzello, S.J. (2002). The affective beneficence of vigorous exercise revisited. *British Journal of Health Psychology, 7*, 47-66.
- Hardy, C.J., & Rejeski, W.J. (1989). Not what, but how one feels: The measurement of affect during exercise. *Journal of Sport and Exercise Psychology, 11*, 304-317.
- Harper, R.M., Bandler, R., Spriggs, D., & Alger, J.R. (2000). Lateralized and widespread brain activation during transient blood pressure elevation revealed by magnetic resonance imaging. *Journal of Comparative Neurology, 417*, 195-204.
- Helal, J.N., Guezennec, C.Y., & Goubel, F. (1987). The aerobic-anaerobic transition: Re-examination of the threshold concept including an electromyographic approach. *European Journal of Applied Physiology, 56*, 643-649.
- Henderson, L.A., Richard, C.A., Macey, P.M., Runquist, M.L., Yu, P.L., Galons, J.-P., & Harper, R.M. (2004). Functional magnetic resonance signal changes in neural structures to baroreceptor reflex activation. *Journal of Applied Physiology, 96*, 693-703.
- Iwamoto, G.A., Wappel, S.M., Fox, G.M., Buetow, K.A., & Waldrop, T.G. (1996). Identification of diencephalic and brainstem cardiorespiratory areas activated during exercise. *Brain Research, 726*, 109-122.
- Jones, D.A., Ainsworth, B.E., Croft, J.B., Macera, C.A., Lloyd, E.E., & Yusuf, H.R. (1998). Moderate leisure-time physical activity: Who is meeting the public health recommendations? A national cross-sectional study. *Archives of Family Medicine, 7*, 285-289.
- Jordan, D., Mifflin, S.W., & Spyer, K.M. (1988). Hypothalamic inhibition of neurones in the nucleus tractus

- solitarius of the cat is GABA mediated. *Journal of Physiology*, 399, 389-404.
- Katch, V., Weltman, A., Sady, S., & Freedson, P. (1978). Validity of the relative percent concept for equating training intensity. *European Journal of Applied Physiology*, 39, 219-227.
- Kaufman, M.P., & Forster, H.V. (1996). Reflexes controlling circulatory, ventilatory and airway responses to exercise. In L.B. Rowell & J.T. Shephard (Eds.), *Handbook of physiology, Exercise: Regulation and integration of multiple systems* (pp. 381-447). New York: Oxford University Press.
- Kirkcaldy, B.C., & Shephard, R.J. (1990). Therapeutic implications of exercise. *International Journal of Sport Psychology*, 21, 165-184.
- Knuepfer, M.M., Eismann, A., Schütze, I., Stumpf, H., & Stock, G. (1995). Response of single neurons in amygdala to interoceptive and exteroceptive stimuli in conscious cats. *American Journal of Physiology*, 268, R666-R675.
- Krout, K.E., Jansen, A.S.P., & Loewy, A.D. (1998). Periaqueductal gray matter projection to the parabrachial nucleus in rat. *Journal of Comparative Neurology*, 401, 437-454.
- Lang, E.J., & Paré, D. (1998). Synaptic responsiveness of interneurons of the cat lateral amygdaloid nucleus. *Neuroscience*, 83, 877-889.
- LeDoux, J.E. (1986). Sensory systems and emotion: A model of affective processing. *Integrative Psychiatry*, 4, 237-248.
- LeDoux, J.E. (1989). Cognitive-emotional interactions in the brain. *Cognition and Emotion*, 3, 267-289.
- LeDoux, J.E. (1994). The degree of emotional control depends on the kind of response system involved. In P. Ekman & R.J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 270-272). New York: Oxford University Press.
- LeDoux, J.E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Simon & Schuster.
- Lee, J.Y., Jensen, B.E., Oberman, A., Fletcher, G.F., Fletcher, B.J., & Raczynski, J.M. (1996). Adherence in the Training Levels Comparison Trial. *Medicine and Science in Sports and Exercise*, 28, 47-52.
- Lind, E., Joens-Matre, R.R., & Ekkekakis, P. (2005). What intensity of physical activity do previously sedentary middle-aged women select? Evidence of a coherent pattern from physiological, perceptual, and affective markers. *Preventive Medicine*, 40, 407-419.
- Liotti, M., Brannan, S., Egan, G., Shade, R., Madden, L., Abplanalp, B., Robillard, R., Lancaster, J., Zamarripa, F.E., Fox, P.T., & Denton, D. (2001). Brain responses associated with consciousness of breathlessness (air hunger). *Proceedings of the National Academy of Sciences*, 98, 2035-2040.
- Ma, W., & Peschanski, M. (1988). Spinal and trigeminal projections to the parabrachial nucleus in the rat: Electron-microscopic evidence of a spino-ponto-amygdalian somatosensory pathway. *Somatosensory Research*, 5, 247-257.
- McAuley, E., Blissmer, B., Katula, J., & Duncan, T.E. (2000). Exercise environment, self-efficacy, and affective responses to acute exercise in older adults. *Psychology and Health*, 15, 341-355.
- McAuley, E., & Courneya, K.S. (1992). Self-efficacy relationships with affective and exertion responses to exercise. *Journal of Applied Social Psychology*, 22, 312-326.
- McCann, D.J., Mollé, P.A., & Caton, J.R. (1995). Phosphocreatine kinetics in humans during exercise and recovery. *Medicine and Science in Sports and Exercise*, 27, 278-387.
- McCormick, D.A., & Bal, T. (1994). Sensory gating mechanisms of the thalamus. *Current Opinion in Neurobiology*, 4, 550-556.
- McDonald, A.J. (1998). Cortical pathways to the mammalian amygdala. *Progress in Neurobiology*, 55, 257-332.
- Menétrey, D., & de Pommery, J. (1991). Origins of spinal ascending pathways that reach ventral areas involved in visceroreception and visceronociception in the rat. *European Journal of Neuroscience*, 3, 249-259.
- Mense, S. (1996). Group III and IV receptors in skeletal muscle: Are they specific or polymodal? *Progress in Brain Research*, 113, 83-100.
- Mifflin, S.W., Spyer, K.M., & Withington-Wray, D.J. (1988a). Baroreceptor inputs to the nucleus tractus solitarius in the cat: Modulation by the hypothalamus. *Journal of Physiology*, 399, 369-387.
- Mifflin, S.W., Spyer, K.M., & Withington-Wray, D.J. (1988b). Baroreceptor inputs to the nucleus tractus solitarius in the cat: Postsynaptic actions and the influence of respiration. *Journal of Physiology*, 399, 349-367.
- Nagata, A., Muro, M., Moritani, T., & Yoshida, T. (1981). Anaerobic threshold determination by blood lactate and myoelectric signals. *Japanese Journal of Physiology*, 31, 585-597.
- Ojanen, M. (1994). Can the true effects of exercise on psychological variables be separated from placebo effects? *International Journal of Sport Psychology*, 25, 63-80.
- Ottersen, O.P. (1980). Afferent connections to the amygdaloid complex of the rat and cat: II. Afferents from the hypothalamus and the basal telencephalon. *Journal of Comparative Neurology*, 194, 267-289.
- Ottersen, O.P. (1981). Afferent connections to the amygdaloid complex of the rat with some observations in the cat: III. Afferents from the lower brain stem. *Journal of Comparative Neurology*, 202, 335-356.

- Ottersen, O.P., & Ben-Ari, Y. (1979). Afferent connections to the amygdaloid complex of the rat and cat: I. Projections from the thalamus. *Journal of Comparative Neurology*, *187*, 401-424.
- Paré, D., Royer, S., Smith, Y., & Lang, E.J. (2003). Contextual inhibitory gating of impulse traffic in the intra-amygdaloid network. *Annals of the New York Academy of Sciences*, *985*, 78-91.
- Parfitt, G., & Eston, R. (1995). Changes in ratings of perceived exertion and psychological affect in the early stages of exercise. *Perceptual and Motor Skills*, *80*, 259-266.
- Parfitt, G., Eston, R., & Connolly, D. (1996). Psychological affect at different ratings of perceived exertion in high- and low-active women: A study using a production protocol. *Perceptual and Motor Skills*, *82*, 1035-1042.
- Parfitt, G., Markland, D., & Holmes, C. (1994). Responses to physical exertion in active and inactive males and females. *Journal of Sport and Exercise Psychology*, *16*, 178-186.
- Perri, M.G., Anton, S.D., Durning, P.E., Ketterson, T.U., Sydeman, S.J., Berlant, N.E., Kanasky, W.F., Newton, R.L., Limacher, M.C., & Martin, A.D. (2002). Adherence to exercise prescriptions: Effects of prescribing moderate versus higher levels of intensity and frequency. *Health Psychology*, *21*, 452-458.
- Pronk, N.P., Crouse, S.F., & Rohack, J.J. (1995). Maximal exercise and acute mood response in women. *Physiology and Behavior*, *57*, 1-4.
- Ricardo, J.A., & Koh, E.T. (1978). Anatomical evidence of direct projections from the nucleus of the solitary tract to the hypothalamus, amygdala, and other forebrain structures in the rat. *Brain Research*, *153*, 1-26.
- Riley, M., Maehara, K., Pórszász, J., Engelen, M.P.K.J., Barstow, T.J., Tanaka, H., & Wasserman, K. (1997). Association between the anaerobic threshold and the break-point in the double product/work rate relationship. *European Journal of Applied Physiology*, *75*, 14-21.
- Rizvi, T.A., Ennis, M., Behbehani, M.M., & Shipley, M.T. (1991). Connections between the central nucleus of the amygdala and the midbrain periaqueductal gray: Topography and reciprocity. *Journal of Comparative Neurology*, *303*, 121-131.
- Roder, S., & Ciriello, J. (1993). Innervation of the amygdaloid complex by catecholaminergic cell groups of the ventrolateral medulla. *Journal of Comparative Neurology*, *332*, 105-122.
- Roder, S., & Ciriello, J. (1995). Convergence of ventrolateral medulla and aortic baroreceptor inputs onto amygdala neurons. *Brain Research*, *705*, 71-78.
- Romanski, L.M., Clugnet, M.C., Bordi, F., & LeDoux, J.E. (1993). Somatosensory and auditory convergence in the lateral nucleus of the amygdala. *Behavioral Neuroscience*, *107*, 444-450.
- Royer, S., Martina, M., & Paré, D. (1999). An inhibitory interface gates impulse traffic between the input and output stations of the amygdala. *Journal of Neuroscience*, *19*, 10575-10583.
- Russell, J.A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology*, *39*, 1161-1178.
- Sah, P., Faber, E.S.L., Lopez De Armentia, M., & Power, J. (2003). The amygdaloid complex: Anatomy and physiology. *Physiological Reviews*, *83*, 803-834.
- Saha, S., Batten, T.F.C., & Henderson, Z. (2000). A GABAergic projection from the central nucleus of the amygdala to the nucleus of the solitary tract: A combined anterograde tracing and electron microscopic immunohistochemical study. *Neuroscience*, *99*, 613-626.
- Saklofske, D.H., Blomme, G.C., & Kelly, I.W. (1992). The effects of exercise and relaxation on energetic and tense arousal. *Personality and Individual Differences*, *13*, 623-625.
- Schneider, F., Habel, U., Holthusen, H., Kessler, C., Posse, S., Müller-Gärtner, H.-W., & Arndt, J.O. (2001). Subjective ratings of pain correlate with subcortical-limbic blood flow: An fMRI study. *Neuropsychobiology*, *43*, 175-185.
- Schneirla, T.C. (1959). An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. In M.R. Jones (Ed.), *Nebraska symposium on motivation* (vol. 7, pp. 1-42). Lincoln, NE: University of Nebraska Press.
- Schütze, I., Knuepfer, M.M., Elsmann, A., Stumpf, H., & Stock, G. (1987). Sensory input to single neurons in the amygdala of the cat. *Experimental Neurology*, *97*, 499-515.
- Shinohara, M., & Moritani, T. (1992). Increase in neuromuscular activity and oxygen uptake during heavy exercise. *Annals of Physiological Anthropology*, *11*, 257-262.
- Spriet, L.L., Lindinger, M.I., McKelvie, R.S., Heigenhauser, G.J.F., & Jones, N.L. (1989). Muscle glycogenolysis and H⁺ concentration during maximal intermittent cycling. *Journal of Applied Physiology*, *66*, 8-13.
- Tanaka, H., Kiyonaga, A., Terao, Y., Ide, K., Yamauchi, M., Tanaka, M., & Shindo, M. (1997). Double product response is accelerated above the blood lactate threshold. *Medicine and Science in Sports and Exercise*, *29*, 503-508.
- Tate, A.K., Petruzzello, S.J., & Lox, C.L. (1995). Examination of the relationship between self-efficacy and affect at varying levels of aerobic exercise intensity. *Journal of Applied Social Psychology*, *25*, 1922-1936.
- Thayer, R.E. (1987). Energy, tiredness, and tension effects of a sugar snack versus moderate exercise. *Journal of Personality and Social Psychology*, *52*, 119-125.
- Toney, G.M., & Mifflin, S.W. (2000). Sensory modalities conveyed in the hindlimb somatic afferent input to the nucleus tractus solitarius. *Journal of Applied Physiology*, *88*, 2062-2073.

- Treasure, D.C., & Newbery, D.M. (1998). Relationship between self-efficacy, exercise intensity, and feeling states in a sedentary population during and following an acute bout of exercise. *Journal of Sport and Exercise Psychology, 20*, 1-11.
- Turner, B.H., & Herkenham, M. (1991). Thalamoamygdaloid projections in the rat: A test of the amygdala's role in sensory processing. *Journal of Comparative Neurology, 313*, 295-325.
- United States Department of Health and Human Services. (1996). *Physical activity and health: A report of the Surgeon General*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion.
- United States Department of Health and Human Services. (2000). *Healthy people 2010*. Washington, DC: Author.
- Urhausen, A., Weiler, B., Coen, B., & Kindermann, W. (1994). Plasma catecholamines during endurance exercise of different intensities as related to the individual anaerobic threshold. *European Journal of Applied Physiology, 69*, 16-20.
- Uwano, T., Nishijo, H., Ono, T., & Tamura, R. (1995). Neuronal responsiveness to various sensory stimuli, and associative learning in the rat amygdala. *Neuroscience, 68*, 339-361.
- Van Landuyt, L.M., Ekkekakis, P., Hall, E.E., & Petruzzello, S.J. (2000). Throwing the mountains into the lakes: On the perils of nomothetic conceptions of the exercise-affect relationship. *Journal of Sport and Exercise Psychology, 22*, 208-234.
- Vissing, J., Andersen, M., & Diemer, N.H. (1996). Exercise-induced changes in the local cerebral glucose utilization in the rat. *Journal of Cerebral Blood Flow and Metabolism, 16*, 729-736.
- Volz, H.P., Rehbein, G., Triepel, J., Knuepfer, M.M., Stumpf, H., & Stock, G. (1990). Afferent connections of the nucleus centralis amygdalae: A horseradish peroxidase study and literature survey. *Anatomy and Embryology, 181*, 177-194.
- Wasserman, K. (1978). Breathing during exercise. *New England Journal of Medicine, 298*, 780-785.
- Watson, D., Clark, L.A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.
- Wells, J.G., Balke, B., & Van Fossan, D.D. (1957). Lactic acid accumulation during work: A suggested standardization of work classification. *Journal of Applied Physiology, 10*, 51-55.
- Weltman, A., Wood, C.M., Womack, C.J., Davis, S.E., Blumer, J.L., Alvarez, J., Sauer, K., & Gaesser, G.A. (1994). Catecholamine and blood lactate responses to incremental rowing and running exercise. *Journal of Applied Physiology, 76*, 1144-1149.
- Zald, D.H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Reviews, 41*, 88-123.
- Zarretto-Smith, A.M., & Gray, T.S. (1990). Organization of peptidergic and catecholaminergic efferents from the nucleus of the solitary tract to the rat amygdala. *Brain Research Bulletin, 25*, 875-887.
- Zarretto-Smith, A.M., & Gray, T.S. (1995). Catecholamine and NPY efferents from the ventrolateral medulla to the amygdala in the rat. *Brain Research Bulletin, 38*, 253-260.
- Zhang, J., & Mifflin, S.W. (2000). Subthreshold aortic nerve inputs to neurons in nucleus of the solitary tract. *American Journal of Physiology, 278*, R1595-R1604.

PSYCHOBIOLOGY OF PHYSICAL ACTIVITY

Edmund O. Acevedo, PhD, FACSM

University of Mississippi

Panteleimon Ekkekakis, PhD, FACSM

Iowa State University

EDITORS



Human Kinetics

Library of Congress Cataloging-in-Publication Data

Psychobiology of physical activity / [edited by] Edmund O. Acevedo, Panteleimon Ekkekakis.

p.; cm.

Includes bibliographical references and index.

ISBN 0-7360-5536-3 (hard cover)

1. Exercise--Physiological aspects. 2. Exercise--Psychological aspects. 3. Sports--Physiological aspects. 4. Sports--Psychological aspects. 5. Cognitive psychology. 6. Psychobiology.

[DNLM: 1. Motor Activity. 2. Brain--physiology. 3. Exercise. WE 103 P974 2006] I. Acevedo, Edmund O. II. Ekkekakis, Panteleimon, 1968-

QP301.P79 2006

612'.044--dc22

2005024460

ISBN-10: 0-7360-5536-3

ISBN-13: 978-0-7360-5536-9

Copyright © 2006 by Edmund O. Acevedo and Panteleimon Ekkekakis

All rights reserved. Except for use in a review, the reproduction or utilization of this work in any form or by any electronic, mechanical, or other means, now known or hereafter invented, including xerography, photocopying, and recording, and in any information storage and retrieval system, is forbidden without the written permission of the publisher.

The Web addresses cited in this text were current as of July 1, 2005, unless otherwise noted.

Acquisitions Editor: Michael S. Bahrke, PhD; **Developmental Editor:** Judy Park; **Assistant Editors:** Kim Thoren, Sandra Merz Bott, and Kevin Matz; **Copyeditor:** Joyce Sexton; **Proofreader:** Anne Rogers; **Indexer:** Bobbi Swanson; **Permission Manager:** Carly Breeding; **Graphic Designer:** Fred Starbird; **Graphic Artist:** Dawn Sills; **Photo Manager:** Sarah Ritz; **Cover Designer:** Keri Evans; **Art Manager:** Kelly Hendren; **Illustrator:** Mic Greenberg; **Printer:** Sheridan Books

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

Human Kinetics

Web site: www.HumanKinetics.com

United States: Human Kinetics

P.O. Box 5076

Champaign, IL 61825-5076

800-747-4457

e-mail: humank@hkusa.com

Canada: Human Kinetics

475 Devonshire Road Unit 100

Windsor, ON N8Y 2L5

800-465-7301 (in Canada only)

e-mail: orders@hkcanada.com

New Zealand: Human Kinetics

Division of Sports Distributors NZ Ltd.

P.O. Box 300 226 Albany

North Shore City

Auckland

0064 9 448 1207

e-mail: info@humankinetics.co.nz

Europe: Human Kinetics

107 Bradford Road

Stanningley

Leeds LS28 6AT, United Kingdom

+44 (0) 113 255 5665

e-mail: hk@hkeurope.com

Australia: Human Kinetics

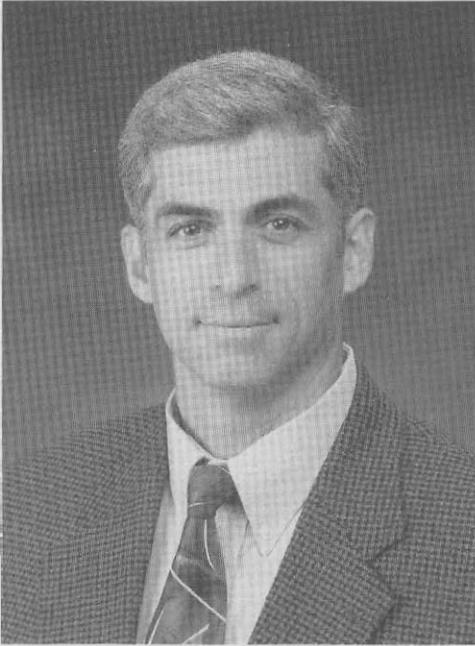
57A Price Avenue

Lower Mitcham, South Australia 5062

08 8277 1555

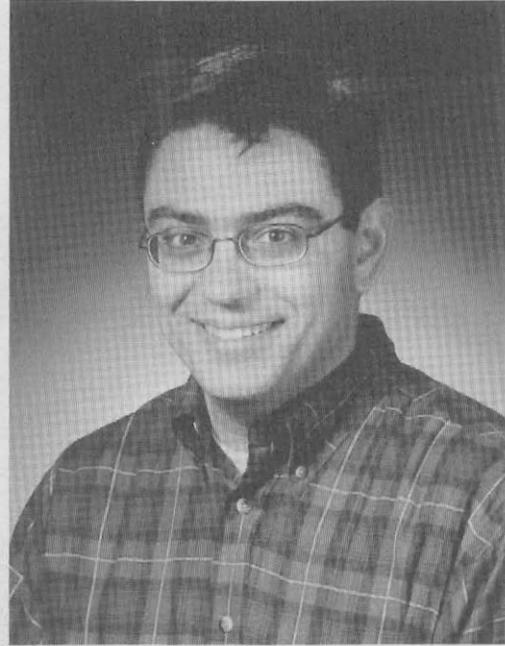
e-mail: liaw@hkaustralia.com

About the Editors



Edmund O. Acevedo, PhD, is an associate professor in the Department of Health, Exercise Science, and Recreation Management at the University of Mississippi and serves as director of the Applied Physiology Laboratory. For 20 years he has conducted research in the area of psychobiology of physical activity with a focus on the psychobiology of stress during physical activity. He has published in numerous journals and books and has made scientific presentations throughout the world.

Dr. Acevedo is a fellow of the American College of Sports Medicine and a certified exercise specialist. He has served as a reviewer for nine different journals in exercise and sport psychology, psychobiology, and exercise physiology. He is a member of the American Physiological Society, American Psychological Association, North American Society for the Psychology of Sport and Physical Activity, and Sigma Xi, the Scientific Research Society. In addition, he is a certified consultant of the Association for the Advancement of Applied Sport Psychology and has consulted with athletes and coaches at the professional, collegiate, and youth sport levels.



Panteleimon Ekkekakis, PhD, is an assistant professor of exercise and sport psychology at the Department of Health and Human Performance at Iowa State University. His research focuses on the affective responses that accompany acute exercise of different intensities, their underlying psychobiological mechanisms, and their implications for exercise adherence over the long haul. His publications span the areas of theoretical and affective psychology, psychometrics, psychophysiology, exercise science, preventive and behavioral medicine, and obesity.

Dr. Ekkekakis is a regular reviewer for many scientific journals, is a psychology section editor for *Research Quarterly for Exercise and Sport*, and serves on the editorial board of *Psychology of Sport and Exercise*. He is a fellow of the American College of Sports Medicine and a member of the North American Society for the Psychology of Sport and Physical Activity.