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Psychology and Exercise

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ACUTE AND CHRONIC PSYCHOLOGICAL EFFECTS OF EXERCISE

Research evidence reveals that physical activity yields numerous health benefits [1−4]. There is also scholastic evidence linking regular exercise and/or sport with positive mental wellbeing [5−9], as well as lower psychophysiological reactivity to mental stress [10−13]. The acute psychological benefits of exercise on various measures of affect and state anxiety are consistently demonstrated in the literature [7,14−22]. Since a single bout of acute exercise yields immediate psychological benefits, it may be seen as a suitable non-pharmaceutical antidote to stress and various mood disorders, in addition to its other health benefits. It is therefore not surprising then that the American College of Sports Medicine (ACSM) launched the 'Exercise is Medicine' program initiative [23] to make physical exercise part of both prevention and treatment of various morbidities.

Research has confirmed that different forms of exercise can trigger positive psychological changes [16,24−27]. The mechanisms by which acute exercise leads to improved wellbeing are primarily based on the volume and/or the duration and intensity of exercise (as a mediator of the psychological effect). The most popular theories are the endorphin hypothesis [28], the amine hypothesis [28], and the thermogenic hypothesis [29]. However, most of these theories have been challenged, because it is now evident that the intensity of exercise has little or no role in the acute psychological benefits of exercise on feelings states [22,30,31]. A placebo mechanism, that complements other mechanisms, has recently been proposed [32].

Ekkekakis [30] reviewed over one hundred research papers and concluded that exercise performed at self-selected intensity triggers effects in wellbeing and may be appropriate from a public health perspective. In considering the duration of exercise, research has shown that a number of positive psychological changes occur even after brief 10-minute bouts of physical exercise [14,33,34]. Therefore, brief exercise bouts are sufficient for experiencing psychological benefits, in contrast to physiological effects that require greater volumes [35]. However, using a cluster randomized cross-over design, Sjögren [36] found that an average of 5 minutes training per working day decreased the prevalence of headache, neck, shoulder and low back symptoms, and alleviated the intensity of headaches, neck and low back pain among symptomatic office workers. The intervention also improved subjective physical wellbeing. Therefore, physical benefits— despite the possibility that they may occur via placebo effects— also occur after short bouts of exercise.

Long-term regular exercise also benefits one's psychological health. A recent review by Gogulla, Lemke, and Hauer [37] showed that most research reports claim that physical exercise results in a significant reduction of depression and fear of falling in healthy elderly participants. However, the evidence was not convincing in elderly people with cognitive impairment. The reviewed studies also suggested that high-intensity aerobic or anaerobic exercise appears to be the most effective in reducing depression, while Tai-Chi and multimodal training are more effective in reducing the fear of falling. Another recent study showed that physical exercise training helps in reducing symptoms of worry among generalized anxiety disordered patients [38]. In contrast to a waiting list

control group, the symptoms of worry decreased after 6 weeks of bi-weekly interventions in female participants in both aerobic exercise and resistance training exercise groups. Consequently, from a mental health perspective, both aerobic (endurance) and anaerobic (strength) exercises have beneficial long-term effects.

In an earlier review, Herring, Connor, and Dishman [39] concluded that exercise training significantly reduced anxiety symptoms when compared with no-treatment conditions. The authors noted that the exercise interventions that resulted in the largest anxiety improvements were those that (i) lasted not longer than 12 weeks, (ii) used exercise sessions lasting at least 30 minutes, and (iii) measured persistent anxiety lasting for more than 1 week. Milani and Lavie [40] showed that, apart from the anxiety-mediating effects of exercise, regular physical activity is also beneficial in the management of stress-related illnesses. The authors found that psychosocial stress is an independent risk factor for mortality in patients with coronary artery disease, and regular exercise training could effectively reduce its prevalence. In their study, the authors claimed that exercise training reduced mortality in patients with coronary artery disease, and that the observed effect may be mediated (at least in part) by the positive effects of exercise on psychosocial stress.

MOTIVATION FOR EXERCISE BEHAVIOR: WHY DO PEOPLE EXERCISE?

Motivation for exercise could be physically or psychologically oriented. Physical motives include (i) being in better physical condition, (ii) having a better looking and healthier body, (iii) having greater strength and endurance, and/or (iv) facilitating weight loss. However, the work-for and achievement of a physical goal also inherently triggers psychological rewards. Individuals participate in physical activity for one or more specific reasons. The reason is often an intangible social reward that itself stems from psychological needs of the person, like being with old buddies or making new friends. The personal experience of the anticipated reward strengthens the exercise behavior. The key point here is that there is always an anticipated reward, and the degree of fulfillment of that reward strongly predicts the continuance of the exercise behavior. Behaviorists, adhering to one of the most influential schools of thought in the field of psychology, postulate that most human behavior can be understood and explained through reinforcement and punishment. The gist of the theory is the operant conditioning-based governance of behavior, which involves positive reinforcement, negative reinforcement, and punishment [41]. Positive reinforcement is a motivational incentive for engaging in an activity to gain a reward that is subjectively pleasant or desirable (e.g., increased muscle tone). The reward then becomes a motivational incentive that increases the likelihood that the behavior will reoccur. In contrast, negative reinforcement is a motivational incentive for doing something to avoid a noxious or unpleasant event (e.g., gaining weight). The avoidance or reduction of the noxious stimulus is the reward, which then increases the probability that the behavior will reoccur. Here, the behavior is essentially used as a coping mechanism by the individual. It should also be noted that while both positive and negative reinforcers increase the likelihood of engaging in the behavior [41], their mechanisms are different because in positive reinforcement there is a 'gain' following the action (e.g., feeling revitalized), whereas in behaviors motivated by negative reinforcement one attempts— for whatever reason—to 'avoid' or prevent something bad, unpleasant, and/or simply undesirable (e.g., feeling guilty or fat if a planned exercise session is missed). Punishment, on the other hand, refers to situations in which the imposition of some noxious or unpleasant stimulus or event (or alternately the removal of a pleasant or desired stimulus or event) reduces the probability of a given behavior reoccurring. In contrast to reinforcers, punishers suppress the behavior and, therefore, exercise or physical activity, reading or other desirable behaviors should never be used (by teachers, parents, or coaches) as punishment.

Habitual exercisers may be motivated by positive reinforcement associated with muscle gain. However, numerous exercisers are motivated by negative reinforcement (e.g., to avoid gaining weight). Every time a person undertakes behavior to avoid something negative, bad, or unpleasant, the motive behind that behavior is classified as negative reinforcement. In these situations, the person involved *has to do it* in contrast to *wants to do it*. In the punishment situation, the person has to do it in a similar way to negative reinforcement, with the difference that (unless we talk about rare instances of self-punishment) the source of obligation (i.e., one has to do it) comes from an outside source (e.g., a parent, a teacher, the law, etc.) rather than from the inside. It is very important to differentiate between imposed punishment and self-selected negative reinforcement in exercise behavior.

There are many examples in other sport areas where a behavior initially driven by positive reinforcement may turn into negatively reinforced behavior. For example, an outstanding football player who starts playing the game *for fun*, after being discovered as a talent and being offered a service contract in a team, becomes a professional player who upon signing the contract is *expected* to perform. Although the player

may still enjoy playing (especially when all goes well), the pressure or expectation to perform is the "has to do" new facet of football playing and the negatively reinforcing component of their sporting activity.

THEORIES AND MODELS ACCOUNTING FOR THE PSYCHOLOGICAL BENEFITS OF EXERCISE

The Sympathetic Arousal Hypothesis

Back in the 1980s, Thompson and Blanton [42] developed the Sympathetic Arousal Hypothesis on the basis of the factual information that regular exercise (especially aerobic exercise like running) if performed for a sustained period, resulted in decreased heart rate at rest. While heart rate is only a rough measure of the body's sympathetic activity (which is directed by the autonomic nervous system), it is, nevertheless, a sensitive measure and it is often used to mirror sympathetic activity. A lower resting heart rate after training results from the adaptation of the person to exercise. With repeated exercise, the person develops a more efficient cardiovascular system characterized by lower basal heart rate, lower sympathetic activity, and lesser arousal at rest. This new state of lowered arousal may induce relaxation, tranquility, and a positive engagement in the habituated exerciser [43].

The Cognitive Appraisal Hypothesis

A psychological explanation based on negative reinforced behavior stems from Szabo [44]. According to this model, some exercisers workout to escape from their psychological hardship [45]. They use exercise as a means of coping with stress. Once the person uses exercise for coping with hardship, the affected individual starts to depend on the adopted form of exercise, because every session brings the desired psychological effect. Therefore, the person experiences a form of psychological relief after exercise. When exercise is prevented for some reason, the exerciser loses the means of coping, and the lack of exercise triggers the opposite effect, that is negative psychological feeling states like irritability, guilt, anxiousness, sluggishness, etc. These feelings collectively are known as withdrawal symptoms experienced because of no- or reduced exercise. Avoidance of these symptoms is a negative reinforcer for exercise behavior.

The Affect Regulation Hypothesis

The affect regulation hypothesis posits that exercise has a dual effect on mood. First it increases the positive affect (defined as momentary psychological feeling states of somewhat longer duration than momentary emotions) and therefore contributes to an improved general mood state (defined as prolonged psychological feeling states lasting for several hours or even days). Second, exercise decreases the negative affect or the transient state of guilt, irritability, sluggishness, anxiety, etc. and therefore contributes to an improved general mood state [46].

The Thermogenic Regulation Hypothesis

This model is based on physiological evidence that physical exercise increases body temperature. A warm body temperature induces a relaxing state with concomitant reduction in anxiety (similar to sun-tanning, Turkish or warm bath, and sauna effects). Therefore, physical exercise reduces anxiety [47,48] via an increased state of physical relaxation. Lower levels of anxiety and states of relaxation are therefore positive reinforcers in exercise behavior. A relaxed body relaxes the mind and yields a positive subjective feeling state.

The Catecholamine Hypothesis

This hypothesis is driven by the observation that increased levels of catecholamines may be measured (in the peripheral blood circulation) after exercise [49]. Catecholamines, among other functions, are involved in the stress response and sympathetic responses to exercise. In light of the catecholamine hypothesis, it is speculated that central catecholaminergic activity is altered by exercise. Because central catecholamine levels are involved in regulating mood and affect and play an important role in mental dysfunctions like depression, the alteration of catecholamines by exercise may be an attractive explanation. However, to date, there is inconclusive evidence for this hypothesis. Indeed, it is unclear whether the peripheral changes in catecholamines have an effect on brain catecholamine levels or vice versa. Furthermore, the changes in brain catecholamine levels during exercise in humans are unknown, because direct measurement in the human brain is not possible.

The Endorphin Hypothesis

This model is attractive and popular in the literature because it is connected to the "runner's high" phenomenon (i.e., a pleasant feeling state associated with positive self-image, sense of vitality, control, and a sense of fulfillment reported by runners as well as by other exercisers after a certain amount and intensity of

exercise). This feeling has been associated with increased levels of endogenous opioids and catecholamines observed after exercise. The theory behind this model is that exercise leads to increased levels of endorphins in the brain, which act as internal psychoactive agents yielding a sense of euphoria. In fact, this hypothesis is analogous to substance or recreational drug addiction (e.g., heroin, morphine, etc.) with the exception that the psychoactive agent (beta endorphin) is endogenously generated from within the body during exercise rather than being exogenously generated from a substance outside the body.

THE "RUNNERS' HIGH" PHENOMENON AND THE ACUTE PSYCHOLOGICAL EFFECTS OF EXERCISE

"I believe in the runner's high, and I believe that those who are passionate about running are the ones who experience it to the fullest degree possible. To me, the runner's high is a sensational reaction to a great run! It's an exhilarating feeling of satisfaction and achievement. It's like being on top of the world, and truthfully ... there's nothing else quite like it!" —*Sasha Azevedo (http://www.runtheplanet.com/resources/ historical/runquotes.asp)*

For many decades, marathon runners, long-distance joggers, and even regular joggers have reported a feeling state of strong euphoria masking the fatigue and pain of physical exertion caused by very long sessions of exercise. This euphoria triggers a sensation of "flying", effortless movement, and has become a legendary goal referred to as "the zone" [50]. The existence of runner's high is subject of heated debate in scholastic circles. The question is whether a biochemical explanation for the runner's high exists, or it is a purely subjectively (psychologically) conceptualized and popularized terminology. Runners (and most if not all habitual exercisers) experience withdrawal symptoms when their exercise is prevented. The symptoms include guilt, irritability, anxiety, and other unpleasant feelings [44]. Research has shown that the human body produces its own opiate-like peptides, called endorphins. Like morphine, these peptides can cause dependence [51] and consequently may be the route of withdrawal symptoms. In general, endorphins are known to be responsible for pain and pleasure responses in the central nervous system. Morphine and other exogenous opiates bind to the same receptors that the body intended for endogenous opioids or endorphins, and since morphine's analgesic and euphoric effects are well documented, comparable effects for endorphins can be anticipated [52].

Research has been conducted to examine the effects of fitness levels, gender, and exercise intensity on endogenous opioid—mainly beta-endorphin—production during cycling, running on a treadmill, participating in aerobic dance, and running marathons. Research by Biddle and Mutrie [53] reported that aerobic exercise can cause beta-endorphin levels to increase five-fold compared with baseline levels. Fitness level of the research participants appears to be irrelevant as both trained and untrained individuals experienced an increase in beta-endorphin levels, although the metabolism of beta-endorphins appeared to be more efficient in trained athletes [54].

Goldfarb et al. [55] examined gender differences in beta-endorphin production during exercise. Their results did not show any gender differences in beta-endorphin response to exercise. Other studies have demonstrated that both exercise intensity and duration are factors in increasing beta-endorphin concentrations. For example, the exercise needs to be performed at above 60% of the individual's maximal oxygen uptake (VO_{2max}) [54] and for at least 3 minutes [56] to detect changes in endogenous opioids.

Researchers have further examined the correlation between exercise-induced increase in beta-endorphin levels and mood changes, using the Profile of Mood States (POMS) inventory [51]. Here, the POMS was administered to all participants before and after their exercise session. The participants gave numerical ratings to five negative categories of mood (i.e., tension, depression, anger, fatigue, and confusion) and one positive category (vigor). Adding the five negative affect scores and then subtracting from the total, the vigor score yields a "total mood disturbance" (TMD) score. In Farrell's study the TMD scores improved by 15 and 16 raw score units from the baseline, after participants exercised at 60% and 80% VO_{2max}. Quantitatively, mood improved about 50%, which corresponds to clinical observations that people's moods are elevated after vigorous exercise workouts. Using radioimmunoassay techniques, Farrell et al. [51] also observed two- to five-fold increase in plasma beta-endorphin concentrations as measured before and after exercise.

However, Farrell et al.'s research is inconclusive. First, only six well-trained endurance athletes were studied, and the six showed large individual variations in beta-endorphin response to submaximal treadmill exercise. Second, the exercise-induced changes in mood scores were not statistically significantly different between pre- and post-exercise scores. Third, no significant relationship between mood measures obtained with the POMS inventory and plasma beta-endorphin levels was found. Therefore, the obtained results do not conclusively prove that beta-endorphins cause mood elevations. However, a more questionable issue—also recognized by Farrell et al.—is that the beta-endorphin measure in the experiment comes from

plasma, which means that this type of beta-endorphin is located in the periphery. Because of its chemical makeup, beta-endorphin cannot cross the blood brain barrier (BBB). Hence, plasma beta-endorphin fluctuations do not reflect beta-endorphin fluctuations in the brain. Some researchers have speculated that endogenous opiates in the plasma may act centrally and therefore can be used to trace CNS activity [53]. At this time, such models concerning beta-endorphins only rely on circumstantial evidence that two opioids (i.e., met-enkephalin and dynorphin) show a modification mechanism that might possibly transport them across the BBB [52]. Unfortunately, direct measurement of changes in brain beta-endorphins involves cutting open the brain and employing radioimmunoassay techniques on brain slices. Animal studies, using rats, have been performed and they have shown an increase in opioid receptor binding after exercise [57].

In humans, to work around this problem, researchers proposed that naloxone could be useful in testing whether beta-endorphins play a role in CNS-mediated responses like euphoria and analgesia. Since it is a potent opioid receptor antagonist, it competes with beta-endorphin to bind to the same receptor. Thus, injection of naloxone into humans should negate the euphoric and analgesic effects produced by exercise, if indeed beta-endorphin facilitates such effects. Such research has found that naloxone decreases the analgesic effect reportedly caused by runner's high, but other researchers who have conducted similar experiments remain divided about these results. As for naloxone's effects on mood elevation, Markoff, Ryan, and Young [58] observed that naloxone did not reverse the positive mood changes induced by exercise.

Mounting evidence demonstrates that beta-endorphins are not necessary for the euphoria experienced by exercisers. Harte, Eifert, and Smith [59] noted that, although exercise produces both positive emotions and a rise in beta-endorphin levels, the two are not necessarily connected. Indeed, physically undemanding activities like watching comedy programs or listening to music produce elevations in mood identical to those resulting from exercise [60,61], although accompanying elevations in beta-endorphins were not be observed after watching comedy programs [62] or music [63]. Similarly, Harte et al. [59] found that both running and meditation resulted in significant positive changes in mood. In addition to taking mood measures, Harte et al. have also measured plasma beta-endorphin levels of the participants. As expected, those in the meditation group did not show a rise in beta-endorphin levels, despite reported elevations in mood. Such results seem to further question the link between mood improvement and changes in beta-endorphin levels following exercise.

Answering the improved mood and increased beta-endorphin levels connection question inversely, experiments were carried out in which beta-endorphin was directly injected into the bloodstream of healthy participants. The results failed to show any changes in mood [53]. On the other hand, beta-endorphin injections had a positive effect on clinically depressed patients [53]. Furthermore, electroconvulsive therapy, used to treat patients with depression, also increased plasma beta-endorphin levels.

The lack of beta-endorphin release during meditation, and the lack of mood alteration after beta-endorphin injection, call for attention on factors that influence beta-endorphin levels. In an effort to consolidate peripheral beta-endorphin data with the central nervous effects, researchers have realized that the peripheral opioid system requires further investigation. Taylor et al. [64] proposed that, during exercise, acidosis is the trigger of beta-endorphin secretion in the bloodstream. Their results showed that blood pH level strongly correlated with beta-endorphin level (i.e., acidic conditions raise the concentration of beta-endorphin; buffering the blood attenuates this response). The explanation behind such observations is that acidosis increases respiration and stimulates a feedback inhibition mechanism in the form of beta-endorphin. The latter interacts with neurons responsible for respiratory control, and beta-endorphin therefore serves the purpose of preventing hyperventilation [64]. How then is this physiological mechanism connected to CNS-mediated emotional responses? Sforzo [52] noted that, since opioids have inhibitory functions in the CNS, if a system is to be activated through opioids, at least one other neural pathway must be involved. Thus, instead of trying to establish how peripheral amounts of beta-endorphin act on the CNS, researchers could develop an alternate physiological model demonstrating how the emotional effects of opioids may be activated through the inhibition of peripheral sympathetic activity [52].

While the "runner's high" phenomenon has not been empirically established as a fact, and beta-endorphins' importance in this event is questionable, other studies have shown how peripheral beta-endorphins affect centrally-mediated behavior. Electroacupuncture used to treat morphine addiction by diminishing cravings and relieving withdrawal symptoms, caused beta-endorphin levels to rise [65]. Since exercise also increases beta-endorphin levels in the plasma, McLachlan et al. [65] investigated whether exercise could lower exogenous opiate intake. Rats were fed morphine and methadone for several days and then randomly divided into two groups of exercisers and non-exercisers. At that time, voluntary exogenous opiate intake was recorded to see if the exercise

would affect the consumption of opiate in exercising rats. The results showed that, while opiate consumption had increased in both groups, exercising rats did not consume as much as non-exercising animals, and the difference was statistically significant [65]. These findings suggest that exercise decreases craving.

In conclusion, the connection between beta-endorphins and runner's high is an elegant explanation but without sufficient empirical support. It is likely that the intense positive emotional experience, to which athletes, runners, and scientists refer as the runner's high, is evoked by several mechanisms acting jointly. Szabo [60] has shown that, while exercise and experiencing humor are equally effective in decreasing negative mood and increasing positive mood, the effects of exercise last longer than those of humor. These results are evidence for the involvement of more than one mechanism in mood alterations after physically active and relatively passive interventions.

THE DARK SIDE OF PHYSICAL ACTIVITY: EXERCISE ADDICTION

Beside the many advantageous effects of physical training, excessive exercise also has the potential to have adverse effects on both physical and mental health, and to lead to exercise addiction. Currently, exercise addiction is not cited within any officially recognized medical or psychological diagnostic frameworks. However, it is important, on the basis of the known and shared symptoms with related morbidities, that the dysfunction receives attention in a miscellaneous category of other or unclassified disorders. Based on symptoms with diagnostic values, exercise addiction could potentially be classified within the category of *behavioral addictions* [66–69]. Despite increased usage of the term 'exercise addiction', several incongruent terminologies are still in use for this phenomenon [70]. The most popular is arguably exercise dependence [71,72]. Others refer to the phenomenon as obligatory exercising [73] and exercise abuse [74], while in the media the condition is often described as compulsive exercise [75].

The Symptoms of Exercise Addiction

Regarding the symptoms, exercise addiction is characterized by six common symptoms of addiction: salience, mood modification, tolerance, withdrawal symptoms, personal conflict, and relapse [76–78]. However, it is important to clarify whether exaggerated exercise behavior is a primary problem in the affected person's life or emerges as a secondary problem in consequence of another psychological dysfunction. In the former case, the dysfunction is classified as primary exercise addiction because it manifests itself as a form of behavioral addiction. In the latter case it is termed as secondary exercise addiction because it co-occurs as a consequence of another dysfunction, typically with eating disorders such as anorexia nervosa or bulimia nervosa [79–81]. In the former, the motive for over-exercising is typically geared towards avoiding something negative [78], although the affected individual may be totally unaware of their motivation. It is a form of escape response to a source of disturbing, persistent, and uncontrollable stress. However, in the latter, excessive exercise is used as a means of achieving weight loss (in addition to very strict dieting). Thus, secondary exercise addiction can have a different etiology than primary exercise addiction. Nevertheless, it should be highlighted that many symptoms and consequences of exercise addiction are similar whether it is a primary or secondary exercise addiction. The distinguishing feature between the two is that in primary exercise addiction the exercise is the objective, whereas in secondary exercise addiction weight loss is the objective, while exaggerated exercise is one of the primary means in achieving the objective.

Measurement of Exercise Addiction

In measuring exercise addiction, two popular scales are worth noting. The Exercise Dependence Scale (EDS) [82–84] conceptualizes compulsive exercise on the basis of the DSM-IV criteria for substance abuse or addiction [85], and empirical research shows that it is able to differentiate between at-risk, dependent, and non-dependent athletes, and also between physiological and non-physiological addiction. The EDS has seven subscales: (i) tolerance, (ii) withdrawal, (iii) intention effect, (iv) lack of control, (v) time, (vi) reduction of other activities, and (vii) continuance. To generate a quick and easily administrable tool for surface screening of exercise addiction, Terry, Szabo and Griffiths [86] developed the 'Exercise Addiction Inventory' (EAI), a short six-item instrument aimed at identifying the risk of exercise addiction. The EAI assesses the six common symptoms of addictive behaviors mentioned above: (i) salience, (ii) mood modification, (iii) tolerance, (iv) withdrawal symptoms, (v) social conflict, and (vi) relapse. Both measures have been psychometrically investigated and proved to be reliable instruments [87].

Epidemiology of Exercise Addiction

Studies of exercise addiction prevalence have been carried out almost exclusively on American and British

samples of regular exercisers. In five studies carried out among university students, Hausenblas and Downs [84] reported that between 3.4% and 13.4% of their samples were at high risk of exercise addiction. Griffiths, Szabo, and Terry [88], reported that 3.0% of a British sample of sport science and psychology students were identified as at-risk of exercise addiction. These research-based estimates are in concordance with the argument that exercise addiction is *relatively* rare [89,90] especially when compared with other addictions [91]. Nevertheless, given the severity of the problem, even a tenth of positive diagnoses among the high-risk cases may be large (i.e., 0.3% is 30/10,000 cases).

Among those who are also professionally connected to sport, the prevalence may be even higher. For example, Szabo and Griffiths [92] found that 6.9% of British sport science students were at risk of exercise addiction. However, in other studies where more-involved exercisers were studied, much higher estimates have generally been found. Blaydon and Lindner [93] reported that 30.4% of triathletes could be diagnosed with primary exercise addiction, and a further 21.6% with secondary exercise addiction. In another study, 26% of 240 male and 25% of 84 female runners were classified as "obligatory exercisers" [94]. Lejoyeux et al. [95] found that 42% of clients of a Parisian fitness room could be identified as exercise addicts. Recently, he reported lower rates of just under 30% [96]. However, one study that surveyed 95 'ultra-marathoners' (who typically run 100 km races) reported only three people (3.2%) as at-risk for exercise addiction [97]. Gender, however, can have a moderating effect on ideal-weight goals and exercise dependence symptoms [98]. It is evident that, besides differences in the applied measures and criteria, these appreciable differences in the estimates may be attributable to the sample selection, small sample size, and the sampling method. With the exception of the study by Lejoyeux et al. [95] that applied consecutive sampling, all the aforementioned studies used convenience sampling.

To date, the only national representative study is the one carried out by Mónok et al. [87] on a Hungarian adult population aged 18−64 years (N = 2,710), and assessed by both the EAI and the EDS. According to their results 6.2% (EDS) and 10.1% (EAI) of the population were characterized as nondependent-symptomatic exercisers, while the proportions of the at-risk exercisers were 0.3% and 0.5%, respectively.

CONCLUSIONS

This chapter reviewed physical exercise (both acute and chronic) and showed that it can have advantageous and disadvantageous effects. Long-term exercising at an optimal level can significantly contribute to physical and psychological health, whereas, in some cases, excessive exercisers can develop exercise addiction that can have various harmful effects. Similarly to other behaviors that can also become addictive, it was demonstrated that exercising also has the potential to develop over-engagement that might lead to negative consequences. The task and responsibility of researchers and healthcare promoters is to communicate clearly on these issues. More specifically, they should promote exercising as a behavior to improve health but also draw attention to the possible harms related to over-exercising and addiction.

Acknowledgements

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