Frequent exercise: A healthy habit or a behavioral addiction?

Wan-Jing Chen

Health Management Centre, Sichuan Academy of Medical Sciences & Sichuan Provincial People's Hospital, Chengdu, Sichuan 610072, China

Received 1 August 2016
Available online 20 December 2016

Abstract

It is well known that regular physical activity helps improve overall health and fitness and reduces the risk of many chronic diseases. However, excessive exercise might be harmful. Exercise addiction (EA) is a pattern of uncontrolled exercise that involves a craving for overwhelming exercise with addictive attributes. So far, little is known about this unique behavioral addiction. The aim of the current study is to introduce the diagnosis and assessment of EA, and to summarize several developing theoretical models. Eating disorders, body image disorder, low self-esteem, and high narcissism are related to high risk of EA. The paper also discusses the distinction between EA and highly involved physical activity.

Keywords: Exercise addiction; Behavior addiction; Physical activity; Theoretical model

Introduction

Regular physical exercise has been proved to promote psychological and physical health and to improve quality of life. However, indulging in uncontrollable excessive exercise may bring about adverse effects, increasing susceptibility to sport injuries or social-occupational dysfunction. For example, overtraining increases the risk of acute exercise injuries (nausea and emesis, hypoglycemia, apopsychia, chest distress, chest pain, arrhythmia, and even sudden death). It can also cause chronic musculoskeletal pain and injury and lead to a malfunction of the human immune system. This phenomenon is referred to as “exercise addiction (EA).” EA is conceptualized as a loss of control over one's exercise behavior, which further becomes a compulsion in which the symptoms of a classical addiction are manifested. In the 5th version of Diagnostic and Statistical Manual of Mental Disorders (DSM-5), EA is recommended for classification under behavior addiction, but it is not listed as a mental dysfunction due to insufficient peer-reviewed evidence. Several other terms are also used for describing EA, for instance, exercise dependence, compulsive exercise, obligatory exercise, and exercise abuse. This paper discusses the diagnostic criteria and various theoretical models of EA. It also discusses the underlying drivers and co-occurring disorders in order to distinguish frequent recreational exercise and competitive sports from EA.
Exercise as an addition: diagnosis, high-risk rate and assessment

The idea of EA was first introduced in 1970 by Baekeland,\textsuperscript{10} when he examined the effect of exercise deprivation on sleep. Subjects were paid to participate in the study, but they expressed a strong desire to continue the exercise even without further monetary compensation. Since then, much attention has been paid to describe this as an addictive behavior.

Researchers have defined EA using several different models. Based on the theoretical model of behavioral addictions, the following should be considered as key components of EA: (1) salience, considering exercise as the most important thing in life; (2) mood modification, regarding exercise as a coping strategy to unexpected events and to regulate emotions; (3) tolerance, individual increases the amount of exercise to reduce craving; (4) withdrawal, manifested by anhedonia, irritability, depression, and anxiety when the individual suddenly reduces or stops exercise; the person may also have difficulties in the performance of professional or social activities; (5) relapse, individual has the tendency to revert to earlier patterns of exercise.\textsuperscript{11–13} Similarly, Hausenblas and Downs\textsuperscript{15,16} defined EA based on the criteria of substance dependence in DSM-IV-TR, which is aligned with the core components of behavior addiction criteria. Accordingly, the key components were tolerance, withdrawal, lack of control, intention effects, spending a lot of time exercising, reduction in other activities, and continuance, that is, continuing to exercise despite knowing that it is causing physical, psychological, and/or social problems.\textsuperscript{17}

Preliminary studies have found varying incidence of high risk for EA. It is relatively rare in the general population, ranging from 0.3–0.5% to 3\%\textsuperscript{18,19} However, the figure varies greatly among regular exercisers and professionals. Mónok et al\textsuperscript{18} found that the incidence of high risk for EA was 0.9–3.2\%. Similarly, Szabo and Griffiths\textsuperscript{20} estimated that 3.6\% of habitual exercisers were at the risk of EA, while the figure was much higher among sport university students (6.9\%). However, several other studies reported stunningly higher rates of exercisers being at risk of EA, namely 22–50\%.\textsuperscript{21–23}

The highly inconsistent prevalence rates may be related to the heterogeneity of measurement tools. The most commonly used and well-validated assessment instruments are the Exercise Addiction Inventory (EAI)\textsuperscript{24} and Exercise Dependence Scale (EDS).\textsuperscript{16} The varying results yielded in studies may be explained by their different frameworks: EAI is based on the diagnosis of mental disorders (6 items) and EDS on addiction symptoms, that is, tolerance, withdrawal, intention effects, lack of control, time, reduction in other activities, and continuance (21 items).\textsuperscript{4,25} Although EAI and EDS are commonly used in clinics, they cannot be used to make a definite diagnosis for EA owing to lack of empirical research. Further, interpretations may differ across different genders and cultures.\textsuperscript{26} Most importantly, an intense involvement in sports or exercise may influence either the interpretation or the scoring of the instruments utilized.\textsuperscript{26} Overestimation may be even more pertinent when the self-report instrument or qualitative interview is applied to individuals suffering from eating disorders.

Theoretical models: how exercise addition develops

The lack of understanding of this exercise paradox calls for theoretical research. After exercising, individuals usually experience euphoric feelings. This may be due to the release of hormones and chemical reactions in the human body.\textsuperscript{27} During exercise, endorphins released by the pituitary gland block the feeling of pain and induce pleasure. Physical activities also stimulate the production of dopamine, and an increased level of dopamine is associated with feelings of happiness and pleasure. In addition, the level of serotonin, a neurotransmitter accounting for euphoria and good appetite, is also increased during regular exercise.\textsuperscript{27,28} It also enhances energy levels and alertness. These “happiness hormones” may play a role in reducing stress levels and therefore may have an indirect connection to EA.

Several models have been proposed to explain EA. The Sympathetic Arousal Hypothesis\textsuperscript{29} is a physiological model suggesting that an organism’s adaptation to habitual exercise may lead to addiction. It states that regular exercise leads to a decreased sympathetic arousal at rest. When individuals feel physically lethargic and tired, and psychologically feel low and negative, they have an urge to increase their arousal levels, which leads to continuing exercise workout. However, this hypothesis does not explain why sympathetic adaptation to exercise is universal, and only a small percentage of exercisers become addicted.\textsuperscript{17}

The second model proposed by Szabo,\textsuperscript{30} the Cognitive Appraisal Hypothesis, views exercise as a means of coping with stress. Thus, reducing or stopping exercise also means losing coping mechanisms, and this leads to individuals being vulnerable to actual
and perceived stress and experiencing psychological hardship (withdrawal symptoms). As a result, individuals become eager to resume the previous pattern of exercise. However, this model only explains the maintenance of the addiction, but not its onset.31

There is also a Four Phase Model for EA, which argues that there are four stages of addiction, from the primary “recreational exercise” to finally “exercise addiction”.32 Each phase is broken into three components (motivation, consequences, and frequency/control). According to the model, in the initial phase (Phase 1—Recreational Exercise), individuals experience pleasure of the physical activities with few negative consequences except for sore muscles or minor strains. Gradually, this leads to a highly engaged, rigid, and indiscriminant behavior pattern wherein exercising becomes the primary or sole coping mechanism for hardships (Phase 2—At-risk Exercise, Phase 3—Problematic Exercise). Finally, the individuals become addicted to exercise (Phase 4—EA). This model explains the onset of EA, but does not specify whether the distress progresses slowly or just suddenly appears. It also does not explain under what conditions individuals adopt exercise as a mechanism coping with life stress.31

The fourth model from the biopsychosocial perspective states that in elite athletes, specific biological triggers such as body mass index (BMI) and social factors interact with psychological ones to trigger EA.33 The social factors include coach, teammate, parental or peer pressure, socio-cultural pressure, etc., and the psychological factors are self-esteem and training beliefs. This model states that elite athletes do not exercise to release stress but to achieve higher sport goals.

Nevertheless, there is also research investigating the possible role of interleukin-6 (IL-6) in EA from the psychobiological point of view.34 IL-6, secreted by T-cells and macrophages, acts as both pro-inflammatory and anti-inflammatory myokine, and operates to stimulate immune response and fight infections. This theoretic model argues that exercise can temporarily reduce negative emotions and create a sense of enjoyment, but it can co-currently lead to excessive release of IL-6 and over-activate neuroendocrine pathways that are related to behavioral and mental disturbances.34 This is because of its important mediation role in acute response including stress, depression, and anxiety. Subsequently, an increased level of IL-6 generates cytokine-induced sickness behaviors, causing negative mental state.35 Some individuals may therefore resort to exercise as a means to relieve discomfort, which in turn increases IL-6 level in the body and causes an “exercise-increased IL-6” loop. However, the limitation of this model is that it does not explain that some individuals may reach for other means of escape such as substance abuse.

Finally, the latest model, the Interactional Model,31 is similar to the Pragmatics, Attraction, Communication and Expectation (PACE) model for addictions.36 According to this model, a number of personal factors interact with the environment to determine the primary motivation for exercise activity. These factors may be personal value, social image, and previous exercise experience and life situations.31 The motivations may lead to two aims: aiming to gain health and reduce stress levels (therapeutic-orientation) or to enhance performance and be more productive (mastery-orientation). However, when an idiographic life stressor emerges—whether it is an on-going one, which the person can no longer deal with, or a suddenly appearing one, it causes psychological pain that individuals may not be able to manage. When individuals explore means to cope with the situation, their choice is determined by conscious and subconscious interactions between personal thoughts, environmental factors and the past exercise behaviors. At this point, some of the mastery-orientated exercisers shift to therapeutic-orientation to escape from the mental stress. Finally, the more individuals gain from exercise, the more likely they use it as a coping strategy to deal with unexpected events in life.31 This model explains why EA emerges suddenly and sometimes only a small percentage, and not all exercisers are at high risk.

**Distinguishing exercise addition from healthy frequent exercise**

One of the most difficult issues is to distinguish healthy physical activity from EA. Regular exercise is beneficial to human body, for example, reducing the risk of developing diabetes and high blood pressure, helping build and maintain healthy bones, muscles, and joints, and improving mental health. However, highly engaged behavior shares some attributes of an addiction: frequent thoughts, feeling good when engaging in the activity, and tolerance.37,38 For example, in the EAI questionnaire, both EA individuals and elite athletes would tick “yes” on its 6 items, but their interpretations differ.26 For example, regarding the item “exercise is the most important thing in my life”, the interpretation of an exercise-addicted person might be that he/she
cannot manage his/her life without exercise, whereas from the perspective of athletes, they might just want to achieve their sport goals and become stronger and better. It is reasonable to assume that the astonishingly high prevalence of EA in the regular exercise population is partly caused by their confusingly different interpretations. To avoid possible misunderstandings, it is important to investigate if individuals have underlying disorders such as eating disorders and self-image or self-esteem problems that are related to EA.

Risk factors for exercise addition

The literature has shown that EA is usually related to eating disorders and body image disorders. Specifically, individuals with anorexia (AN) or bulimia nervosa (BN) tend to adopt excessive exercise as a compensation to manage weight and relieve guilt. It is estimated that around 39–48% of individuals with eating disorders also suffer from EA. Moreover, Jones et al have reported that young women with normal BMI but unsatisfied with their physiques are at high risk of developing EA. While men get involved in sport exercise due to social and competitive vigor-related factors, women exercise aiming to burn calories and get fit and thin. One study indicated that shopping addiction is also common in addicted exercisers because they share similar appearance-related motivations.

Some studies have investigated if low self-esteem and narcissism make individuals more vulnerable to EA. By applying the EAI, the Narcissistic Personality Inventory (NPI), and the Coopersmith Self-Esteem Inventory (SEI) to 150 regular gym exercisers, Bruno et al found that the high EA risk group reported significantly higher score on NPI and lower score on SEI than the low EA risk group. This confirms the role of low self-esteem and high narcissism in the development of EA as predictive factors. The idea that narcissism is a key factor in addiction has been proposed since Freud. Specifically, the fulfillment of inner narcissism is mediated by repetitive behaviors in order to assure omnipotence and provide self-protection against the potential lack of satisfaction or admiration. Low self-esteem was also detected in a study on an Italian population with EA. Indeed, low self-esteem is one of the characteristics of the “addictive personality.” Since exercise builds physique and increases individuals’ self-confidence, it becomes a coping strategy for unexpected life events. Lack of self-worth can keep individuals addicted.

Finally, social physique anxiety (anxiety related to the public presentation of one's image) can be a high risk factor for the development of EA. This means that when a person identifies himself/herself as an exerciser, he/she experiences a high degree of social physique anxiety, and this pressure makes him/her to exercise more rigidly and even develop addiction. Therefore, the careful identification of EA requires a multidisciplinary approach. It is not simply a behavior of frequent intense exercising but a behavior that involves a person's physical and psychological well-being, his/her eating habits, his/her self-esteem, and body image as well as coping strategies for stress.

Conclusion

There is a growing body of literature on EA. However, the risk rates vary greatly among studies. This shows methodological and conceptual limitations. Self-report instruments only provide a risk score and cannot be used to make a definite diagnosis because of inconsistent interpretations related to the studied sample. Some core attributes of EA (e.g., tolerance and withdrawal) overlap with the behaviors of committed athletes and leisure gym exercisers. Similar to other addictions, EA may reflect an escape from stress along with an accessible way to overcome both internal and external criticisms. Therefore, more research is warranted to investigate EA and its co-occurring disorders, as well as their possible interactions.

Conflicts of interest

The author declares that she has no conflicts of interest.

References

1. World Health Organization (WHO). Physical Activity. http://www.who.int/mediacentre/factsheets/fs385/en/. Accessed November 20, 2016.
2. Shepherd C. Pacing and exercise in chronic fatigue syndrome. J Physiother. 2001;87:395–396.
3. Herczeg K. Szabó A, Griffiths MD, et al. Exercise addiction: symptoms, diagnosis, epidemiology, and etiology. Subst Use Misuse. 2012;47:403–417.
4. Szabo A. Addiction to Exercise: A Symptom or a Disorder? New York, NY: Nova Science Publishers Inc; 2010.
5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Arlington, VA: American Psychiatric Publishing Inc; 2013.
6. Adams J. Understanding exercise dependence. J Contemp Psychother. 2009;39:231–240.
7. Murray SB, Maguire S, Russell J, Touyz SW. The emotional regulatory features of bulimic episodes and compulsive exercise in muscle dysmorphia: a case report. *Eur Eat Disord Rev.* 2012;20:68–73.

8. Thome JL, Espelage DL. Obligatory exercise and eating pathology in college females: replication and development of a structural model. *Eat Behav.* 2007;8:334–349.

9. Calogero RM, Pedrotty KN. The practice and process of healthy exercise: an investigation of the treatment of exercise abuse in women with eating disorders. *Eat Disord.* 2004;12:273–291.

10. Baekeland F. Exercise deprivation. Sleep and psychological re-actions. *Arch Gen Psychiatry.* 1970;22:365–369.

11. Brown RIF. A theoretical model of the behavioural addictions—applied to offending. In: Hodge JE, McMurran M, Hollin CR, eds. *Addicted to Crime.* 1st ed. Chichester, England: John Wiley & Sons; 1997:13–65.

12. Brown RIF. Gaming, gambling and other addictive play. In: Apter MJ, Kerr JH, eds. *Adult Play: A Reversal Theory Approach.* New York, NY: Garland Science Publisher; 1991:101–118.

13. Aidman EV, Woollard S. The influence of self-reported exercise addiction on acute emotional and physiological responses to brief exercise deprivation. *Psychol Sport Exerc.* 2003;4:225–236.

14. Allegre B, Souville M, Therme P, Griffiths MD. Definitions and measures of exercise dependence. *Addict Res Theory.* 2006;14:631–646.

15. Hausenblas HA, Downs DS. How much is too much? The development and validation of the exercise dependence scale. *Psychol Health,* 2002;17:387–404.

16. Downs DS, Hausenblas HA, Nigg CR. Factorial validity and psychometric examination of the Exercise Dependence Scale-Revised. *Meas Phys Educ Exerc Sci.* 2009;8:183–201.

17. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Text Revision. Washington, DC: American Psychiatric Publishing Inc; 2000.

18. Mönk K, Berzík K, Urbán R, et al. Psychometric properties and concurrent validity of two exercise addiction measure: a population wide study. *Psychol Sport Exerc.* 2012;13:739–746.

19. Sussman S, Lisha N, Griffiths M. Prevalence of the addictions: a problem of the majority or the minority? *Evalu Health Prof.* 2011;34:3–56.

20. Szabo A, Griffiths MD. Exercise addiction in British sport science students. *Int J Ment Health Addict.* 2007;5:25–28.

21. Lejoyeux M, Avril M, Richoux C, Embouaza H, Nivoli F. Prevalence of exercise addiction and other behavioral addictions among clients of a Parisian fitness room. *Compr Psychiatry.* 2008;49:353–358.

22. Anderson SJ, Basson CJ, Geils C, Farman R. Personality style and mood states associated with a negative addiction to running. *Sports Med.* 1997;4:6–11.

23. Allegre B, Therme P, Griffiths M. Individual factors and the context of physical activity in exercise dependence: a prospective study of ‘ultra-marathoners’. *Int J Ment Health Addict.* 2007;5:233–243.

24. Terry A, Szabo A, Griffiths M. The exercise addiction inventory: a new brief screening tool. *Addict Res Theory.* 2004;12:489–499.

25. Griffiths MD, Urbán R, Demetrovics Z, et al. A cross-cultural re-evaluation of the Exercise Addiction Inventory (EAI) in five countries. *Sports Med Open.* 2015:1:5.

26. Szabo A, Griffiths MD, de La Vega Marcos R, Mervó B, Demetrovics Z. Methodological and conceptual limitations in exercise addiction research. *Yale J Biol Med.* 2015;88:303–308.

27. Roberts AM. Health junkie: hormones and chemicals released due to exercise. http://www.popular.com/fitness/Hormones-Released-After-Working-Out-19252431/. Accessed October 16, 2016.

28. Hackney AC, Lane AR. Exercise and the regulation of endocrine hormones. *Prog Mol Biol Transl Sci.* 2015;135:293–311.

29. Thompson JK, Banton P. Energy conservation and exercise dependence: a sympathetic arousal hypothesis. *Med Sci Sports Exerc.* 1987;19:91–99.

30. Szabo A. The impact of exercise deprivation on well-being of habitual exercisers. *Aust J Sci Med Sport.* 1995:27:68–75.

31. Egorov AY, Szabo A. The exercise paradox: an interactional model for a clearer conceptualization of exercise addiction. *J Behav Addict.* 2013;2:199–208.

32. Freimuth M, Moniz S, Kim SR. Clarifying exercise addiction: differential diagnosis, co-occurring disorders, and phases of addiction. *Int J Environ Res Public Health.* 2011;8:4069–4081.

33. McNamara J, McCabe MP. Striving for success or addiction? Exercise dependence among elite Australian athletes. *J Sports Sci.* 2012;30:755–766.

34. Hamer M, Karageorghis CI. Psychobiological mechanisms of exercise dependence. *Sports Med.* 2007;37:477–484.

35. Cohen S, Doyle WJ, Skoner DP. Psychological stress, cytokine production, and severity of upper respiratory illness. *Psychosom Med.* 1999;61:175–180.

36. Sussman S, Leventhal A, Bluthenthal RN, Freimuth M, Forster M, Ames SL. A framework for the specificity of addictions. *Int J Environ Res Public Health.* 2011;8:3399–3415.

37. Charlton JP. A factor-analytic investigation of computer ‘addiction’ and engagement. *Br J Psychol.* 2002:93:329–344.

38. Jones JM, Bennett S, Olmsted MP, Lawson ML, Rodin G. Disordered eating attitudes and behaviours in teenage girls: a school-based study. *CMAJ.* 2001;165:547–552.

39. Bratland-Sanda S, Sundgot-Borgen J, Ro O, Rosenvinge JH, Hoffart A, Martinsen EW. “I’m not physically active — I only go for walks”: physical activity in patients with longstanding eating disorders. *Int J Eat Disord.* 2010;43:88–92.

40. Cook B, Hausenblas H, Crosby RD, Cao L, Wonderlich SA. Exercise dependence as a mediator of the exercise and eating disorders relationship: a pilot study. *Eat Behav.* 2015;16:9–12.

41. Bamber DJ, Cockerill IM, Rodgers S, Carroll D. Diagnostic criteria for exercise dependence in women. *Br J Sports Med.* 2003;37:393–400.

42. Klein DA, Bennett AS, Schebendach J, Foltin RW, Devlin MJ, Walsh BT. Exercise “addiction” in anorexia nervosa: model development and pilot data. *CNS Spectr.* 2004;9:531–537.

43. McCreary DR, Sasse DK. An exploration of the drive for muscularity in adolescent boys and girls. *J Am Coll Health.* 2000;48:297–304.

44. Vocks S, Hechler T, Rohrig S, Legenbauer T. Effects of a physical exercise session on state body image: the influence of pre-experimental body dissatisfaction and concerns about weight and shape. *Psychol Health.* 2009:24:713–728.

45. Modolo VB, Antunes HK, Gimenez PR, Santiago ML, Tufik S, Mello MT. Negative addiction to exercise: are there differences between genders? *Clinics (Sao Paulo).* 2011;66:255–260.

46. Freimuth M. Addicted? Recognizing Destructive Behavior Before It’s Too Late. Lanham, MD: Rowman & Littlefield Publishers, Inc; 2008.
47. Bruno A, Quattrone D, Scimeca G, et al. Unraveling exercise addiction: the role of narcissism and self-esteem. *J Addict*. 2014;2014:987841.

48. Freud S. *The Complete Letters of Sigmund Freud to Wilhelm Fliess, 1887–1904*. Cambridge, MA: Harvard University Press; 1985.

49. Guidi J, Clementi C, Grandi S. Psychological distress and personality characteristics among individuals with primary exercise dependence. *Riv Psychiatr*. 2013;48:121–129.

50. Cook B, Karr TM, Zunker C, et al. The influence of exercise identity and social physique anxiety on exercise dependence. *J Behav Addict*. 2015;4:195–199.

51. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol*. 1986;51:1173–1182.

52. Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D. How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *Am J Psychiatry*. 2001;158:848–856.

Edited by Pei-Fang Wei