Risk of Exercise Dependence in University Students:

A Subtyping Study Utilizing Latent Profile Analysis

by

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Abstract

Exercise dependence (ED) is a maladaptive pattern of exercise that increases risk of physical and psychological harm. There is a need to identify whether individuals symptomatic/at-risk for exercise dependence are a heterogenous group, and if so, to identify risk factors associated with different subtypes. This study sought to determine whether subtypes of individuals symptomatic/at-risk for exercise dependence can be distinguished on their alexithymia profiles, self-reported depressive symptoms, and eating disorder symptomatology. Latent profile analysis revealed two classes. One reported stronger affective than cognitive alexithymic traits, and limited feelings of personal distress in response to others' suffering. The other reported stronger cognitive than affective alexithymic traits along with elevated eating disorder symptomatology and concomitant mood disturbance. This class also reported heightened signs of personal distress. A follow up mediation analysis demonstrated that cognitive, but not affective, alexithymia mediated the relationship between empathy (personal distress, perspective taking, and empathic concern) and ED, with increased cognitive alexithymia predicting more severe ED. This study is unique as it is the first of its kind to explore the complex dynamics between alexithymic traits, empathy, and ED. Results from this subtyping research provide insights into underlying risk factors that may contribute to the development of ED, and may help to refine existing theories. The results may also inform subsequent research, targeted treatment methods, and psychoeducation programs for use with athletes, parents, and coaches.

Keywords: exercise dependence; alexithymia; empathy

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Dependence is a pathological condition that is often defined in the context of alcohol and drug use. It refers to the body's adaptation to substance use, which results in uncontrollable drug seeking and/or drug-using behaviour (Marian & Willard, 2014). It is also characterized by its impact on psychosocial functioning, namely, the prioritizing of the activity over social relationships and occupational functioning (Pridgeon & Grogan 2012). Dependence can lead to addiction--defined as behaviour that functions to produce both pleasure and escape from internal discomfort, and that is employed in a re-occurring, uncontrollable manner despite negative consequences (Goodman, 1990). As classified by the *Diagnostic and Statistical Manual of Mental Disorders-V* (DSM-V; American Psychiatric Association [APA], 2013*),* substance use addictions are diagnosed when there is evidence of impaired control, social impairment, risky use, and pharmacological criteria such as tolerance and withdrawal.

The concepts of dependence and addiction can be extended to behaviours other than substance use (Lejoyeux, Avril, Richoux, Embouazza, & Nivoli, 2008). Indeed, in the most recent edition of the DSM gambling disorder was added as a new, behavioural addiction category (APA, 2013). Feeding and eating disorders are included as a distinct category despite the fact that, as is the case with substance-use disorders, they are often accompanied by symptoms such as cravings and impulsive use (APA, 2013). Taken together, these observations provide support for the proposition that addiction does not just apply to substance-use disorders, but also to other categories of excessive and uncontrollable behaviour (Griffiths, 1996) that can be identified and diagnosed on the basis of six key symptoms: salience, mood modification,

tolerance, withdrawal, personal conflict, and relapse (Adams, 2009; Berczik et al., 2012; Szabo, 2010).

Many behavioural syndromes that are not specifically identified in the DSM-V clearly contain a pronounced addictive component (Goodman, 1990). In addition to overeating (Powell, 1986) and hypersexuality (Carnes, 1983), contemporary definitions incorporate exercise dependence (Glasser, 1976; Landolfi, 2013; Szabo, 1995). Indeed, in their recent literature review, Berczik et al. (2012, p. 412) concluded that exercise dependence is a "robust representative of behavioural addictions." Exercise dependence is an important area of study, given the relatively recent acceptance of behavioural addiction as a distinct disorder, and the potentially harmful effects of excessive exercise. In the current research, I examined associations between several factors that appear to be linked to exercise dependence. Investigations of this sort may inform future work exploring the development and maintenance of maladaptive patterns of exercise.

Exercise Dependence

Terminology. For most people, exercise serves as a healthy activity; its psychological and physiological benefits are almost undisputed (Hausenblas, Schreiber, & Smoliga, 2017; Szabo, 2010). Indeed, the *Canadian Physical Activity Guidelines for Adults 18-64 years* (Canadian Society for Exercise Physiology, 2011) recommends 150 minutes of moderate to intense aerobic activity per week. There are numerous documented benefits of exercise, including improvements to overall health (Landolfi, 2013) and decreased rates of depression (Berczik et al., 2012), Type II diabetes (Kriska, 2000), and cardiovascular disease (Blair, Kohl, & Paffenbarger, 1989). However, it is now well recognized that excessive exercise can lead to exercise dependence--a maladaptive pattern of exercise that increases risk of physical and

psychological harm (Allegre et al., 2007). Exercise dependence is characterized by feelings of loss of control that lead to excessive exercise; symptoms of overtraining such as fatigue and sleep disturbance, anaemia, or amenorrhea; and withdrawal symptoms characterized by restlessness, sadness, and irritability (Hausenblas et al., 2017).

Different research disciplines use different terms to describe this phenomenon, including *exercise addiction, obligatory exercise, exercise abuse,* and *compulsive exercise* (Berczik et al., 2012; Szabo, 2010). In this thesis, the term *dependence* will be used rather than *addiction.* This is deemed most appropriate because the term dependence is consistent with DSM criteria for substance use disorders, where tolerance and withdrawal are not *necessary* for a diagnosis (APA, 2013).

When William Glasser first introduced the term exercise dependence in 1976, he used it to refer to a *commitment to exercise* (Szabo, 2010). Unfortunately, he also described it as a "positive addiction" because he believed it was better to be addicted to a good thing than a bad thing (such as alcohol or drugs). As a result, the term has often been misused in the academic literature. The lack of a conceptual basis led to inconsistencies in definitions, assessment methods, and findings with regards to exercise dependence (Adams, 2009).

In recent years, researchers have attempted to create a clear distinction between commitment to exercise and addiction to exercise (Szabo, 2010). Hausenblas and Downs (2002) operationalized exercise dependence based on DSM-IV criteria for substance dependence in an effort to clearly distinguish between the two phenomena (see also Hausenblas et al., 2017; Szabo, 2010). Individuals characterized by a commitment to exercise derive satisfaction, enjoyment, and a sense of achievement from exercising. Committed exercisers control their physical activity and do not experience withdrawal symptoms. Addicted exercisers, on the other

hand, lose control over their physical activity, as evidenced by the fact that they will frequently visit a doctor or health care clinic because they continue to exercise despite minor injuries. They describe exercise as a cardinal aspect to their lives, and experience strong withdrawal symptoms when they are unable to exercise. In short, committed exercises are said to be distinguished from addicted exercisers in that they exercise for positive reinforcement (e.g., because they enjoy an aspect of exercise) rather than negative reinforcement (e.g., to avoid withdrawal symptoms) (Berczik et al., 2012). In addicted exercisers, avoidance or escape behaviours are strongly reinforced and highly resistant to extinction (Berczik et al., 2012).

Assessment. Existing exercise dependence assessment tools do not measure one's commitment to exercise. Moreover, they can only assess someone's *risk* for exercise dependence. They cannot be used to diagnose exercise dependence in a clinical population; as such, they should be regarded as surface-screening tools only (Berczik et al., 2012). Until validated cut-off scores are established, data from the administration of these instruments should be interpreted with caution (e.g., Blaydon & Lindner, 2002).

The Obligatory Exercise Questionnaire (OEQ; Ackard, Brehm, & Steffen, 2002), and the Exercise Dependence Questionnaire (EDQ; Ogeden, Veale, & Summers, 1997) have both been found to be psychometrically valid and reliable (Berczik et al., 2012). However, the Exercise Dependence Scale (EDS; Hausenblas & Downs, 2002) and the 6-item Exercise Addiction Inventory (EAI; Terry, Szabo, & Griffiths, 2004) are considered to be the most psychometrically valid screening tools for exercise dependence (Berczik et al., 2012). In the EDS, exercise dependence is conceptualized based on the DSM-IV criteria for substance dependence and operationalized as "a multidimensional maladaptive pattern of exercise, leading to clinically significant impairment or distress'' (Hausenblas & Downs, 2002, p. 4).

Prevalence. The true prevalence of exercise dependence remains unclear due to differences in how it is defined, inconsistent assessment methods, and small sample sizes. It is thought to present an issue for an estimated 0.3% to 0.5% of the general population and 3% of the exercising population (e.g., gym attendees) (Mónok et al., 2012). These observed low rates in the general population may be due to the inherent physical effort and commitment required, especially when compared to other addictions such as alcohol or drug addiction, for example (Berczik et al., 2012; Szabo, 2010). However, the true prevalence may be higher and context dependent; for instance, one study found the prevalence of exercise dependence to be almost twice as high among sport science students compared to the general exercising population (Szabo & Griffiths, 2007), and some studies have reported rates as high as 25% in runners (Slay, Hayaki, Napolitano, & Brownell, 1998) and 52% in triathletes (Blaydon et al., 2002).

Recent research suggests that rates of exercise dependence are also higher in competitive athletes than in non-competitive, leisure exercisers (de la Vega, Parastatidou, Ruiz-Barquin, & Szabo, 2016). However, this may be due to differences in how the phenomenon is conceptualized in these populations (de la Vega et al., 2016). Competitive athletes experience concurrent high levels of passion and dedication to sport, which may influence how they interpret the items included in exercise dependence assessment tools (de la Vega et al., 2016; Szabo, Griffiths, de la Vega, Mervo, & Demetrovics, 2015). As these tools fail to assess commitment to exercise, they may be inadequate for assessing the prevalence of exercise dependence in competitive athletes. New measures will need to be developed to obtain more accurate estimates of the prevalence of exercise dependence in both the general and sporting populations (Adams, 2009).

Etiology. Many theories regarding the root causes of exercise dependence have been put forth, yet consensus has not yet been reached (Adams, 2009). A literature review on exercise dependence conducted by Hamer and Karageorghis (2007) outlined the most significant psychophysiological explanations offered to date, including the *β-endorphin hypothesis*, the *sympathetic arousal hypothesis*, and the *affect regulation hypothesis.*

β-endorphin hypothesis. The β-endorphin hypothesis (a.k.a. the *runner's high hypothesis*; Berczik et al., 2012) suggests that the release of endogenous opioids (i.e., endorphins) during exercise inhibits pain perception and improves mood (Berczik et al., 2012; Griffiths, 1997; Hamer et al., 2007). The mood enhancement serves as a positive reinforcer for exercise. This hypothesis relies on the fact that endogenous opioids can be modified in a way that allows them to cross the blood-brain-barrier (Sforzo, 1988). Support for this hypothesis comes from Sforzo, Seeger, Pert, Pert, and Dotson (1986) who found an increase in the opioid-receptor binding sites in rats after exercise, and from Smith and Yancey (2003) who found opioid-tolerance and physical dependence as a result of chronic exercise in rats. However, the only study to my knowledge that has examined β-endorphin responses to exercise and exercise dependence in humans did not find a significant association (Pierce et al., 1993). It is important to note, however, that this study had limitations, such as a sample size of only eight participants and the use of a poorly validated measure of exercise dependence (Hamer et al., 2007). Qualitative research provides some support for the β-endorphin hypothesis, as exercise-dependent participants have described feeling "addicted" to the "shot of happiness hormones" that occurs after exercise (Pridgeon & Grogan, 2012). However, some researchers have dismissed the β-endorphin hypothesis altogether, given that most support for it is based on circumstantial evidence and the fact that there is no current scientific mechanism that exists

which would allow it to take place (Dietrich & McDaniel, 2004). Taken together, these mixed results suggest a need for research to further explore β-endorphin responses to exercise and exercise dependence in humans.

Although a clear link between the β -endorphin system and exercise dependence has yet to be demonstrated, recent research highlights the potential impact of the endocannabinoid system. The endocannabinoid system reduces the experience of pain (Richardson, 2000) and also impacts emotional and cognitive processes (Chaperon & Thiebot 1999; Diaz, 1997). Recent research suggests that exercise increases serum concentrations of endocannabinoids in both animals and humans (Dietrich & McDaniel, 2004; Sparling, Giuffrida, Piomelli, Rosskopf, & Dietrich, 2003). Indeed, a recent study examining the effects of exercise on anxiety and pain in mice demonstrated several noteworthy findings: (1) mice experienced less anxiety and pain after exercise (a.k.a. a runner's high) and (2) blockage of the endocannabinoid (but not the endorphin) system prevented this from occurring (Fuss et al., 2015). In humans, exercise dependent individuals have been found to have lower levels of anandamide compared to controls, both at baseline and during an exercise withdrawal period (Antunes et al., 2016). Interestingly, although acute exercise has been found to raise anandamide levels in humans in past research (e.g., Heyman et al., 2012; Sparling et al., 2003), one exercise session was not enough to raise these levels in exercise dependent individuals, further suggesting that exercise dependent individuals might exercise in an attempt to increase the activation of the endocannabinoid system and this "bliss molecule" (Antunes et al., 2016). Given research showing the effects of the endocannabinoid system on emotion, cognition, and reward (Chaperon & Thiebot 1999; Diaz, 1997; Glass, Dragunow & Faull, 1997) and psychological changes due to exercise (Dietrich & McDaniel, 2004), one can see how it may be linked to compulsive behaviour (Antunes et al.,

2016). However, more research is needed to determine (a) how the endocannabinoid system responds to exercise, and whether this varies depending on the nature of the exercise undertaken, and individuals' sex and age; and (b) what functional role the production and biding sites of endocannabinoids play in exercise (Dietrich & McDaniel, 2004).

Sympathetic arousal hypothesis. The sympathetic arousal hypothesis suggests that repeated exercise is maintained by hormonal changes that result from consistent training (Thompson & Blanton, 1987). Engaging in regular exercise may result in decreased sympathetic arousal at rest--in other words, humans may adapt to consistent aerobic activity. Because this "training effect" leads to lower basal heart rate and lower levels of arousal, which translates to feelings of lethargy at rest, people need to exercise more and more (and at a higher intensity) to continue to feel aroused and energetic (Berczik et al., 2012; Szabo, 2010). Little conclusive evidence exists for this speculation (Berczik et al., 2012), however it has been reported that coronary patients who partake in dynamic exercise experience a diminished sympathetic response at rest (Cousineau et al., 1977), and more recent research has shown decreased immune and autonomic functioning in individuals who develop pain, fatigue, and negative mood changes following one week of exercise deprivation (Glass et al., 2018). Taken together, these findings suggest that experiencing hypoactivity in the biological stress response systems *during rest* may prompt exercise-dependent individuals to exercise regularly to maintain arousal and suppress negative symptoms (Hamer et al., 2007).

Catecholamines are involved in regulation of the sympathetic nervous system, but it is important to note that the catecholamine dopamine also impacts both mood and the reward system (Berczik et al., 2012; Griffiths, 1997). Some suggest that chronic exercise may attenuate rewarding experiences associated with activities that would otherwise normally activate the

reward pathways (Adams, 2009). Interestingly, dysfunction in dopamine regulation has been associated with impulse control problems in behavioural addictions involving eating, shopping, and sex (Adams, 2009), and with pathological gambling (da Silva Lobo et al., 2007).

Affect regulation hypothesis. Finally, according to the affect regulation hypothesis exercise serves both to increase positive affect and to decrease negative affect (Berczik et al., 2012; Hamer et al., 2007) and these dual effects may lead some to use it as a means to cope with stress (Berczik et al., 2012; Szabo, 1995). Because exercise-dependent individuals experience larger improvements in mood after exercising than non-dependent exercisers and sedentary individuals (Rosa, Mello, Negrão, & Souza-Formigoni, 2004) along with relief from other negative symptoms (e.g., withdrawal symptoms such as irritability, guilt and anxiety), they may find that exercise acquires particularly strong negative reinforcement properties. According to Berczik et al. (2012), however, the effects on mood are temporary, and an increase in negative affect becomes more likely as the interval between two training sessions increases. Moreover, exercise-dependent individuals can become trapped in a vicious cycle of exercising to reduce negative feelings, but then experiencing a loss of control over the exercise--which, in itself, engenders more negative feelings.

Research on the effects of exercise deprivation on habitual exercisers supports the affect regulation hypothesis (Berlin, Kop, & Deuster, 2006; Glass et al., 2018). For example, Pridgeon and Grogan (2012) found that exercise-dependent individuals describe feeling “guilt” over not exercising. In addition, Antunes et al. (2016) found that, compared to a control group, individuals with exercise dependence showed increased depression, confusion, anger, and fatigue following a two-week exercise withdrawal period.

Are There Subtypes of Exercise Dependence?

Although the work described above suggests several possible mechanisms through which exercise dependence might arise, more research is needed. Studies aimed at subtyping may give us clues about which individuals may be most at risk, and lead to refinements of existing theories or the development of new ones. They may also inform our approach to intervention.

Currently, the literature distinguishes between two subtypes of exercise dependence. In *primary exercise dependence*, exercise is the primary problem and it manifests itself as a behavioral addiction (Berczik et al., 2012; Hausenblas et al., 2017). Here, exercising is the main objective; it may serve as an escape, though it is possible for the affected individual to be entirely unaware of this motivation (Berczik et al., 2012). In contrast, in *secondary exercise dependence* exercising is used as a means to an end, usually to control weight (Berczik et al., 2012; Hausenblas et al., 2017). Consequently, secondary exercise dependence may not necessarily represent an addiction at all (Adams, 2009), although it may contribute to the development of eating disorders (Cook & Hausenblas, 2008). Men are more frequently affected by primary exercise dependence whereas women are more frequently affected by secondary exercise dependence (Hausenblas et al., 2017).

Whereas some researchers have suggested that the distinction between primary and secondary exercise dependence is meaningful, others assert that there is not enough evidence to assume the existence of primary exercise dependence (Blaydon et al., 2002). Moreover, due to the lack of psychopathological symptoms in primary exercise dependence (e.g., Blaydon, Lindner, & Kerr, 2004), Bamber, Cockerill, & Carroll (2000) have argued that primary exercise dependence is not harmful enough to warrant a diagnosis.

Other approaches to subtyping may prove to be more useful. To my knowledge, only one study has utilized latent profile analysis (LPA) to identify potential subgroups of exercise

dependence. Magee and colleagues (2016) conducted an LPA on 345 adult Ironman participants, using the seven subscales of the Exercise Dependence Scale-21 (EDS-21; Hausenblas et al., 2002b). Follow-up ANOVAS and chi-square tests of independence were used to identify group differences between profiles in relation to eating patterns and psychological distress. A model with five latent classes was found, which were labelled as asymptomatic, time committed, low psychological dependence, symptomatic, and at-risk. Furthermore, the symptomatic and at-risk groups displayed greater unhealthy eating patterns and psychological distress than the other groups. These results support the hypothesis that there are individual differences in the nature and severity of exercise dependence, and co-occurring psychological disorders.

The focus in the study by Magee and colleagues (2016) was on elite athletes. As outlined in more detail below, a key goal of the current research was to determine if subgroups of individuals symptomatic or at-risk for exercise dependence could be identified in a general sample of university students through the use of LPA. To justify the selection of variables I included in my LPA, I turn now to a discussion of comorbidity, and of personality characteristics that have been associated with exercise dependence in past research.

Comorbidity. Exercise dependence is positively related to anxiety and obsessive compulsiveness (Spano, 2001). However, the relationship between exercise dependence and depression is not yet clear. It is important to explore possible links with depression, given the existing evidence that depression often co-occurs with other forms of addictive behaviour (Adams, 2009). For example, depression has been shown to be significantly associated with pathological gambling (e.g., Rizeanu, 2013), to be causally influenced by alcohol-use disorder (Boden & Fergusson, 2011), and to be three to four times more likely to occur in individuals

with substance-use disorder than in members of the general population (Lai, Cleary, Sitharthan, & Hunt, 2015).

The relationship between depression and exercise dependence may be complex. For instance, although previous studies have demonstrated that regular aerobic exercise is associated with lower levels of depression in members of the general public (e.g., Da Silva et al., 2012), Weinstein, Maayan, and Weinstein (2015) found that depression was elevated in compulsive (vs. non-compulsive) exercisers, and in professional (vs. recreational) athletes. Lichtenstein, Nielsen, Gudex, Hinze, and Jørgensen (2018) also found that regular exercisers with exercise dependence reported more depression than regular exercisers who did not exhibit exercise dependence. Other research suggests that depression may pose a more severe problem for exercise-dependent individuals who have an eating disorder, compared to both exercise dependent individuals without an eating disorder and healthy controls (Bamber et al., 2000). These findings suggest that including measures of both mood and eating disorder severity may improve one's ability to identify meaningful subgroups of individuals with exercise dependence; for this reason, the LPA conducted in the present study included these variables.

Personality characteristics and exercise dependence. Due to the lack of longitudinal studies on individuals with exercise dependence, its course is not known (Berczik et al., 2012). However, because personality characteristics usually influence the acquisition, development, and maintenance of addiction, exploring links between these characteristics and exercise dependence may prove useful (Berczik et al., 2012; Lichtenstein, Christiansen, Elklit, Bilenberg, & Støving, 2014). In this regard, past research suggests that exercise dependence is positively related to both perfectionism (Hagan & Hausenblas, 2003) and obsessive (but not harmonious) passion (de la Vega et al., 2016; Paradis, Cooke, Martin, & Hall, 2013). Risk for exercise dependence is also

elevated in individuals scoring high in narcissism, impulsiveness, and extraversion, and low in agreeableness (Hausenblas et al., 2017; Lichtenstein, et al., 2014; Spano, 2001). Research also suggests that self-esteem (Hausenblas et al., 2017; Szabo, 2010) and personality traits such as straightforwardness, altruism, modesty, and compliance are negatively correlated with exercise dependence. As noted by Lichtenstein et al. (2014), these latter findings suggest that those with exercise dependence may be at an elevated risk for experiencing interpersonal difficulties. These same authors also posit that personality traits such as excitement seeking and activity may contribute to exercise dependence by impacting one's motivation to exercise by promoting goal setting and aspiring to high standards.

The importance of certain personality traits in exercise dependence has been consistently demonstrated in the literature. Given this, an important question addressed in the current study was whether including measures of the personality trait alexithymia in my LPA would help me to identify meaningful subgroups of individuals with exercise dependence. As discussed below, alexithymia may be linked to exercise dependence if its leads to attempts to increase one's physiological arousal, and/or if it interferes with emotion regulation or promotes maladaptive coping.

Alexithymia. Alexithymia is a multidimensional psychological construct characterized by deficits in identifying, verbalizing, and regulating one's emotional state (Goerlich-Dobre et al., 2015; Panaite & Bylsma, 2012). Coined by Sifneos et al. in 1973, the term alexithymia is derived from the Greek words *a* (lack), *lexis* (word), and *thymos* (emotion) (van't Wout, Aleman, Bermond, & Kahn, 2007). Symptoms extend to having difficulties separating feelings from bodily sensations, experiencing an emptiness of feelings and a lack of positive emotions, having difficulties in interpersonal communication, and showing a lack of imaginative abilities (van't

Wout et al., 2007; Vorst & Bermond, 2001). Individuals with alexithymia may appear to not experience any emotion at all due to their difficulties in realizing and expressing their emotions (Barlow et al., 2015). Their poor emotion understanding and communication is thought to limit their empathic skills, hamper their interpersonal relationships, and have a significant, negative impact on their personal lives (Barlow et al., 2015).

Alexithymia has an estimated prevalence rate of 10% in the population (Salminen, Saarijarvi, Aarela, Toikka, & Kauhanen, 1999). Although not a clinical disorder itself, it has been linked to various neurological, psychological, and medical conditions, including (but not limited to) depression, somatoform disorders, anxiety disorders, schizophrenia, chronic pain, and cardiovascular problems (Goerlich-Dobre et al., 2015; Panayiotou & Constantinou, 2017; Salminen et al., 1999). Thus, alexithymia is seen as a risk factor for decreased mental and physical health (Panayiotou et.al, 2017). Furthermore, it has been linked to a wide range of addictive behaviours (e.g., Bossard & Miller, 2009; Morie et al., 2017; Noel et al., 2017; Taylor, Parker, & Bagby, 1990), so much so that it is now considered influential in the development of addiction (Morie et al., 2017).

Alexithymia has been suggested to relate to exercise dependence through its impact on emotion regulation (Taylor, Bagby, & Parker, 1991); specifically, some individuals with alexithymia may turn to exercise to help them self-regulate in the face of overwhelming negative emotions (Taylor, Bagby, & Parker, 1999). Emotion regulation refers to the ability to influence which emotions one experiences, and when and how one experiences and expresses them (Gross, 1999). It is considered an adaptive ability, as individuals who are better able to identify and verbalize their emotions during periods of stress have better daily health outcomes than those who cannot (Zautra, Smith, Afleck, & Tennen, 2001). Alexithymia may impede effective

emotion regulation (Connelly & Denney, 2007) because difficulties identifying, verbalizing, and analyzing one's emotions interfere with reappraisal (van der Meer, van't Wout, & Aleman, 2009). Indeed, emotion regulation skills have been shown to be weak in alexithymic individuals (Feldmanhall, Dalgleish, & Mobbs, 2013). They may excessively exercise to achieve levels of emotion and emotion regulation that are perceived as not being accessible to them in their everyday lives (Barlow, Woodman, & Hardy, 2013; Cazenave, Le Scanff, & Woodman, 2007). Similar factors may drive some individuals with alexithymia to engage in high-risk sport or other risk-taking behaviours (Barlow et al., 2015).

There is some evidence to suggest that individuals with alexithymia show atypical physiological responses to emotional stimuli; however, the literature is mixed with regards to the direction and magnitude of this effect. One of the earliest models of alexithymia was the *stress-alexithymia hypothesis* (Martin & Pihl, 1985), which states that individuals with alexithymia experience physiological hyperarousal during stress. Because they are unable to identify the emotional basis of this arousal they experience prolonged exposure to the stressor (Martin & Pihl, 1985), which can lead to behavioural problems (Chrousos, 1998). Indeed, research has found that the disclosure of stressful events is associated with reduced arousal (Pennebaker, Hughes, & O'Heeron, 1987) and fewer physician visits for illness (Pennebaker, Colder, & Sharp, 1990). Further support for this hypothesis comes from the fact that alexithymia is found in a disproportionately high percentage of people with anxiety and other stress-sensitive disorders (Bogdanov et al., 2013). However, while individuals with alexithymia have been found to exhibit higher baseline physiological arousal *at rest* (Bogdanov et al., 2013; Martin & Pihl, 1985), most studies have found either lower (e.g., Linden, Lenz, & Stossel, 1996) or comparable (e.g., Stone

& Nielson, 2001) physiological reactivity between individuals with and without alexithymia during times of *stress* (Eastabrook, Lanteigne, & Hollenstein, 2013).

In contrast to the stress-alexithymia hypothesis, the *hypoarousal theory* (Donges & Suslow, 2017) postulates that individuals with alexithymia experience blunted physiological arousal to emotion information, and thus are slower to identify and verbalize their emotions. For example, Gaigg, Cornell, and Bird (2016) found that self-reported alexithymia was significantly correlated with reduced skin conductance responses while viewing emotion-inducing pictures, suggesting that alexithymia impairs the interaction between physiological arousal and emotional awareness.

In their work, Pollatos and colleagues (2011) found that hypoarousal was accompanied by increased anxiety, a phenomenon they referred to as decoupling. Eastabrook, et al. (2013) went on to show that individuals with alexithymia had greater physiological arousal *at baseline only*, and experienced comparable levels of physiological arousal but greater self-consciousness during a speech task. They argued that alexithymia interferes with the ability to regulate the physiological experience and external expression of emotion. In light of the above, it is plausible that individuals with lower baseline physiological arousal may exercise in an attempt to increase arousal, consistent with the sympathetic arousal hypothesis (Thompson & Blanton, 1987). Furthermore, these individuals might have limited insight into the physiological costs of exercise (e.g., pain, injury), which might encourage excessive exercise.

The variability in reported physiological reactivity in individuals with alexithymia may be due to cross-study differences in group selection, physiological measures, and type of emotional stimulation (Bogdanov et al., 2013). However, failure to account for the possibility that there may be subtypes of alexithymia that respond differently to emotional stimuli may also

play a role. Recent subtyping studies have focused on the idea that there may be two dimensions of alexithymia: a cognitive dimension (i.e. difficulties in identifying, analyzing, and verbalizing feelings) and an affective dimension (i.e. difficulties in emotionalizing and fantasizing) (Bermond, Bierman, Cladder, Moormann, & Vorst, 2010). According to these authors, differences in the extent to which these dimensions are expressed result in several distinct subtypes, with different neural bases (Bermond et al., 2007; Moormann et al. 2008). Type I alexithymia is characterized by deficits in both cognitive and affective dimensions, Type II involves impairment in the cognitive dimension only, Type III involves impairment in the affective dimension only, Type IV (a.k.a. "lexithymia") is characterized by no impairment in either dimension, Type V (a.k.a. "modal") is characterized by average scores on both dimensions, and individuals who fail to meet criteria for the above five subtypes are classified as having a "mixed" profile (Goerlich-Dobre et al., 2015; Moormann et al., 2008).

Support for the idea that the cognitive and affective dimensions are distinct and that subtypes of alexithymia exist is mixed. For example, a cluster analysis by Bagby et al. (2009) failed to identify meaningful subtypes. More recently, Preece, Becerra, Allan, Robinson, and Dandy (2017) compared different models of alexithymia statistically and found that the best fitting solution did not include difficulties emotionalizing or fantasizing. Other studies, however, have found psychometric support for the two higher order factors of alexithymia (e.g., Bermond et al., 2007; Vorst & Bermond, 2001). Theoretically, Preece et al. (2017) suggest that the lack of differentiation between positive vs. negative emotional reactivity among items in the emotionalizing scale might contribute to these inconsistencies. In line with research demonstrating that different subtypes of alexithymia are risk factors for different personality disorders (Moormann et al., 2008; van der Meer et al., 2009), the current study was designed to

determine whether the cognitive and affective dimensions of alexithymia differentially relate to exercise dependence.

Alexithymia and exercise. There is only a limited literature examining alexithymia in athletes. Jodat, Ghasempour, and Tavakoli (2015) found that student athletes scored lower than non-athletes on all scales of the Farsi version of the Toronto Alexithymia Scale (TAS-20; Bagby, Taylor, & Parker, 1994)—a widely-used self-report measure of alexithymia. Iacolino, Pellerone, Formica, Lombardo, and Tolini (2017) found opposite results: athletes scored higher than non-athletes on the TAS-20, specifically on the externally-oriented thinking style subscale and on the total score. The discrepant results from these two studies may reflect differences in study populations; indeed, athletes were older and much more heavily involved in sport in the latter study.

One study, conducted in 2007 by Allegre, Souville, Noel-Jorand, Pellegrin, and Therme, found a relationship between the *intensity* of exercise swimmers engaged in and the degree to which their verbal discourse could be characterized as alexithymic. These authors also observed that expert swimmers displayed more severe alexithymic verbal discourse than amateur swimmers (i.e., little expression of feelings and emotions, proportionately more action words, and distancing of themselves from what they experienced). Though both a small sample ($N = 20$) and the exploratory nature of the study must be kept in mind, these findings offer some support for the suggestion that those who engage in intense exercise differ from those who do not in at least one aspect of alexithymia. It could be that, in elite athletes, alexithymia reflects a coping mechanism employed to deal with the hardship and stress involved in high performance sport (Allegre et al., 2007). Indeed, in studies conducted on athletes, Type II alexithymia was associated with severe levels of anxiety (Moormann, Bermond, & Albach, 2004).

The current study explores links between alexithymia and exercise dependence in a university sample. This is of interest as the transition from high school to university can be a particularly challenging time for students (Clark, 2005). Emotion regulation skills, which have been shown to be weak in alexithymic individuals, are thought to be vital in coping (Feldmanhall et al., 2013). It is possible, therefore, that students displaying high levels of alexithymic traits would be more likely to utilize exercise as a way to cope with stress.

To my knowledge, only one study has directly examined the relationship between alexithymia and exercise dependence. Bossard and Miller (2009) found that alexithymia was positively related to exercise dependence ($r = 0.41$). Subjects included men and women, aged 18-40 years, who completed the TAS-20, the Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983), the Exercise Dependence Scale (EDS-21; Hausenblas et al., 2002), and the Beck Depression Inventory (BDI; Beck & Beck, 1972). In this sample, 40% of individuals with exercise dependence were classified as alexithymic.

Unfortunately, the measure of alexithymia used in the Bossard and Miller (2009) study (the TAS-20) assesses only the cognitive dimension of alexithymia, characterized by difficulties identifying and describing one's feelings, and an externally-oriented thinking style (Bermond, Oosterveld, & Vorst, 2015). As such, it is not known if the affective dimension of alexithymia (which reflects atypical emotionalizing and fantasizing abilities) is related to exercise dependence. It is important to consider the possibility that there may be subtypes of individuals with alexithymia that differ with regard to the relative strength of cognitive and affective alexithymic traits (Bermond et al., 2007; Goerlich-Dobre et al., 2015). Taking both dimensions into consideration may help to clarify the nature of the relationship between alexithymia and exercise dependence.

Are There Links Between Empathy and Exercise Dependence?

Atypical empathy has been implicated in numerous addictions, including behavioural addictions (e.g., Ferrari, Smeraldi, Bottero, & Politi, 2014; Maurage et al., 2011; Tomei, Besson, & Grivel, 2017). However, it is not known how empathy relates to exercise dependence, if at all. Given this, the second goal of this study was to examine the relationship between empathy and exercise dependence. A subgoal was to determine whether cognitive and/or affective alexithymia mediated any observed relationship between these two variables, given reported links between alexithymia and atypical empathy (e.g., Bernhardt & Singer, 2012; Bernhardt et al., 2013; Moriguchi et al., 2007; Silani et al., 2008).

Empathy underlies good social functioning and interpersonal relationships, allowing individuals to effectively navigate the social world (Anderson & Keltner, 2002; Ferrari et al., 2014). It is viewed as a positive human trait (Bird & Viding, 2014) and is defined as a process wherein the perception of another's state generates a similar state in the subject (Hoffman, 2000). Thus, it is the ability of an individual to take on the subjective feelings and emotions of another. This definition is relatively uncontroversial and widely accepted throughout the academic field (Bird & Viding, 2014).

Further broadly accepted, though not considered unequivocal, is the multifaceted nature of empathy, namely its division into cognitive and affective dimensions (Davis, 1980, 1983; Ferrari et al., 2014; Goerlich-Dobre et al., 2015; Tomei et al., 2017). Davis (1996) has asserted that these dimensions represent "two distinctly separate" abilities (p. 9). The cognitive empathic dimension includes the ability to understand what another person thinks or feels (i.e. perspective taking) and the ability to project oneself into fictional situations (through fantasizing) (Goerlich-Dobre et al., 2015). These abilities are considered cognitive because they can occur without one

experiencing a corresponding emotional response. The affective dimension is comprised of a person's automatic emotional response to another's suffering (empathic concern) and the feelings of distress this engenders (personal distress). These responses fall into the affective dimension because they do not require a cognitive understanding of the other individual's experience.

There are, of course, individual differences in cognitive and affective empathic abilities. For example, Baron-Cohen (2002) has suggested that females are generally better able to empathize than males. Smith (2006) has suggested that this may be because males show a greater imbalance in the strength of their cognitive and affective empathic abilities than females. Smith's *empathy imbalance theory* (2009) also predicts that such imbalances may characterize various clinical disorders. In support of this theory, some individuals working in the field of addictions have described impaired affective empathy with intact cognitive empathy in individuals with alcohol-dependence (Maurage et al., 2011) and drug addiction (Ferrari et al., 2014). Conversely, Tomei and colleagues (2017) found impaired cognitive empathy and *heightened* personal distress (an aspect of affective empathy characterized by strong emotional reactivity) in a sample of 78 participants with problem gambling. Given these observations, in the present study, I gathered information about both cognitive and affective empathic abilities using the Interpersonal Reactivity Index (IRI; Davis, 1980).

Although the findings mentioned above support the distinctiveness of the cognitive and affective dimensions of empathy, some authors posit that it may be more useful to think of these as interacting rather than separate components (Lamm & Majdandzic, 2015)—particularly as some studies have found the same neural activation in both affective and cognitive routes (e.g., Lamm, Decety & Singer, 2011). Others point out that the Personal Distress and Fantasy

subscales of the IRI tap into processes beyond just empathy, including self-oriented processes such as imagination and emotional self-control (Baron-Cohen & Wheelwright, 2004). In contrast, high-level empathic skills such as perspective-taking and empathic concern are largely other-focused, and require awareness of the self-other distinction.

Alexithymia and empathy. Alexithymia has been associated with atypicalities in empathy (Bernhardt & Singer, 2012; Bernhardt et al., 2013; Bird & Viding, 2014; Moriguchi et al., 2007; Silani et al., 2008), including experiencing heightened personal distress in response to others' suffering (Guttman & Laporte, 2002; Moriguchi et al., 2006; Patil & Silani, 2014). This is particularly evident in research on individuals with eating disorders such as anorexia nervosa (e.g., Beadle, Paradiso, Salerno, & McCormick, 2013). Indeed, in a study examining empathy for pain Brewer et al. (2018) found that co-occurring alexithymia *explained* increased personal distress, particularly in individuals with eating disorders compared to healthy controls, when controlling for both the eating disorder diagnosis and depression. Interestingly, alexithymia has also been associated with atypical structure in neural regions supporting empathy (Bernhardt & Singer, 2012; Decety, 2011; Lamm, Rütgen, & Wagner, 2017) including the anterior insula (AI) and anterior cingulate cortex (ACC) (Bernhardt & Singer, 2012; Moriguchi et al., 2007; Singer, Critchley, & Preuschoff, 2009), which have been linked to individual differences in personal distress (Banissy, Kanai, Walsh, & Rees, 2012; Cheetham, 2009; Eres, Decety, Louis, & Molenberghs, 2015).

Other work suggests that some individuals scoring high on alexithymia show empathic deficits (Decety & Moriguchi, 2007; Preston & de Waal, 2002), particularly in the areas of perspective taking and empathic concern (Berthoz, Wessa, Kedia, Wicker, & Grezes, 2008; Bird & Viding, 2014; Goerlich-Dobre et al., 2015). As noted above, impaired awareness of the self-

other distinction may contribute to these difficulties. Problems in these aspects of empathy could also reflect another feature common in alexithymia, namely poor emotion regulation. Indeed, it has been suggested that emotion regulation is necessary for individuals to engage in empathic concern because it allows them to decrease the personal distress they feel in response to another's suffering, and increase feelings of compassion (Eisenberg et al., 1994).

Given the above, it is possible that some alexithymic individuals exercise to excess as a way to cope with high levels of personal distress and due to problems with emotion regulation. However, it is also possible that some individuals with alexithymia—namely those showing emotional blunting and/or empathic deficits that result in social difficulties—turn to exercise to help them cope with feelings of loneliness and social isolation (see Maurage et al., 2011 for a similar argument regarding factors contributing to alcohol abuse). In these cases, alexithymic traits and accompanying empathic deficits may not only contribute to the establishment and maintenance of exercise dependence, but be exacerbated by it, due to the limitations that engaging in excessive exercise can place on social interaction (Ferrari et al., 2014).

Whatever the mechanism, the research described above suggests that alexithymia may underlie observed links between atypicalities in empathy and exercise dependence. If this is the case, then assessing whether cognitive and affective alexithymic traits fully or partially mediate any observed relationship between empathy and exercise dependence would be informative.

The Present Study

The first goal of the current study was to explore links between the cognitive and affective dimensions of alexithymia and exercise dependence, through the use of LPA. Specifically, my goal was to determine whether subgroups of individuals who are symptomatic or at-risk for exercise dependence could be discriminated based, in part, on unique "alexithymia

profiles." Despite the classification of exercise dependence into primary and secondary subtypes, it is presently unclear whether subgroups of exercise dependence exist that are meaningfully associated with varying levels of psychological functioning. Furthermore, the current classification of exercise dependence, as proposed by Hausenblas and Downs (2002), is based on DSM-IV criteria for substance-use disorders and relies on total number of criteria met, which can overlook meaningful subtypes (Chamberlain, Stochl, Redden, Odlaug, & Grant, 2017). Given that individuals with problem gambling tend to be a heterogeneous group (Carragher et al., 2011; Chamberlain et. al, 2017; Cunningham-Williams & Hong, 2007), differing in severity across a number of factors (Carragher et al., 2011), it was considered plausible that subgroups of individuals with exercise dependence might also systematically differ from one another. I also expected that any observed latent classes might show different sex distributions, given that primary exercise dependence is more frequent among men whereas secondary exercise dependence is more frequent among women (Hausenblas et. al, 2017).

As noted earlier, it has been suggested that people can be classified as belonging to distinct subtypes of alexithymia based on the cognitive and affective alexithymic traits they endorse (Bermond et al., 2007; Goerlich-Dobre et al., 2015; Moormann et al., 2008). According to Bermond and colleagues (2010), subtypes I, II, and IV (lexithymic) are well-supported in the literature, but the evidence for Type III (weak cognitive but strong affective traits) is still equivocal. Given this, I predicted that either a two-class or a three-class model would be found, and that the classes would be differentiated not only on the basis of the cognitive and affective alexithymic traits they endorse, but also on the strength of self-reported symptoms of eating disorder and depression.

Based on past research, those characterized by strong cognitive alexithymic traits (Types I and II) might be expected to utilize exercise as a means of emotion regulation, as these individuals tend to be stress-prone (Moormann et al., 2008) and to use emotional suppression rather than (more effective) cognitive reappraisal strategies to self-regulate (van der Meer et al., 2009). This might place them at higher risk for depression, given that those who are less able to verbalize their emotions during stressful times have worse daily health outcomes than those who cannot (Zautra et al., 2001). These individuals might also be expected to report more symptoms of an eating disorder, given past reports linking depression and eating disorders to exercise dependence (Bamber et al., 2000).

In contrast to the above, individuals with an alexithymia profile characterized by strong affective traits (e.g., Type III) are said to be stress resistant, but susceptible to social problems associated with empathic deficits and manipulative tendencies. They may utilize exercise to increase arousal levels, or for self-serving purposes. Narcissism, which can characterize these individuals (Moormann et al., 2008), is correlated with both exercise dependence and social difficulties (Lichtenstein et al., 2014). This group might also be expected to have lower levels of depression and symptoms of an eating disorder, given that Type III alexithymia is associated with psychological well-being (Moormann et al., 2008).

The second goal of the current study was to investigate the relationship between empathy and exercise dependence. This was of interest given past research linking atypical empathy in other behavioural addictions (e.g., Tomei et al., 2017). Based on the literature reviewed above, I expected that deficits in perspective-taking and empathic concern, and/or atypical emotional reactivity (reflected in unusually strong or weak personal distress), might be associated with exercising to excess. My subgoal was to determine if cognitive and/or affective alexithymic traits

might mediate any observed relationships. I predicted that difficulties in perspective-taking and empathic concern (Berthoz et al., 2008; Bird & Viding, 2014; Goerlich-Dobre et al., 2015) and heightened personal distress (Guttman & Laporte, 2002; Moriguchi et al., 2006; Patil & Silani, 2014), might be more evident in exercise dependent individuals scoring high on cognitive alexithymic traits, and that deficits in self-focused aspects of empathy (personal distress, fantasy) might be most evident in exercise dependent individuals reporting strong affective alexithymic traits (Beadle et al., 2013; Brewer et al., 2018; Guttman & Laporte, 2002; Moriguchi et al., 2006; Patil & Silani, 2014; Tomei et al., 2017).

Method

Participants and Procedures

This study was open to any individual registered in PSYC 1200 (Introduction to Psychology) at the University of Manitoba who had previously provided complete data on the Problem Gambling Severity Index (Ferris & Wynne, 2001) and the IRI as part of the Fall 2017 mass testing survey (N = 1904); only data from the latter measure were used in the current study. The final sample (after exclusions, see below) included 600 individuals. Recruitment was done by inviting eligible individuals (via email) to complete the study via Qualtrics, an online survey system. As part of the Qualtrics survey, those who consented to take part entered identifying information that was used to match their survey responses to the data gathered previously in the 2017 mass pre-test. All participants completed the EDS-21, the Eating Disorder Examination-Questionnaire Short (EDE-QS; Gideon et al., 2016), the Patient Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, 2001), and the Bermond Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001) via Qualtrics, in that order. Participants gave informed

consent and received credit toward a course requirement. The protocol was approved by the Psychology/Sociology Research Ethics Board at the University of Manitoba.

Materials

Exercise Dependence Scale-21 (Hausenblas et al., 2002b). Exercise dependence was assessed using the EDS-21, a self-report measure that screens for exercise dependence in a way consistent with DSM-IV criteria. It is comprised of seven subscales tapping into the following symptoms: (1) tolerance (e.g., *I continually increase my exercise intensity to achieve the desired effects/benefits*); (2) withdrawal (e.g., *I exercise to avoid feeling irritable*); (3) intention effect (e.g., *I exercise longer than I intend*); (4) lack of control (e.g., *I am unable to reduce how long I exercise*); (5) time (e.g., *I spend a lot of time exercising*); (6) reductions in other activities (e.g., *I would rather exercise than spend time with family/friends*); and (7) continuance (e.g., *I exercise despite recurring physical problems*). Participants indicated their responses to each of 21-items using a Likert scale ranging from 1 (*never*) to 6 (*always*). Based on their responses, individuals are categorized into one of three categories: *at risk for exercise dependence, non-dependent symptomatic*, and *non-dependent asymptomatic*. An individual is considered at risk for exercise dependence if they score above a 5 on at least three of the seven subscales; nondependent-symptomatic if they score in the 3 to 4 range on at least three of the seven subscales; and nondependent-asymptomatic if they score in the 1 to 2 range on 5 or more of the 7 subscales (Hausenblas et al., 2002b). The EDS-21 also differentiates between individuals who have or do not have physiological dependence, as evidenced by symptoms of tolerance or withdrawal (Hausenblas et al., 2002b). The EDS-21 has good psychometrics, including good concurrent validity with the EAI: $r = 0.81$ (Szabo & Griffiths, 2007), and high internal consistency,

reliability, and convergent validity with other extreme physical activity measures (Hausenblas et. al, 2002a).

Eating Disorder Examination Questionnaire – Short (EDE-QS; Gideon et al., 2016). The EDE-QS was given in order to quantify current eating disorder symptoms. The EDE-QS is a brief, self-report screening instrument based on the past week. It cannot be used to identify individuals with an eating disorder. In the first part of the survey, participants report on the frequency with which various thoughts/behaviours occurred in the preceding seven days. Responses are made to each of 10-items using a Likert scale ranging from 0 (*0 days*) to 3 (*6-7 days*). In the second part of the survey, participants report on the extent to which their weight or shape has influenced how they judge themselves (1 item), and how satisfied they have been with their weight (1 item) over the past seven days. Responses to these 2-items are made using a Likert scale ranging from 0 (*Not at all*) to 3 (*Markedly*). A total score was computed by calculating the mean score of the 12 items.

Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001). Depressive symptoms were assessed with the PHQ-9, given the substantial co-morbidity seen between exercise dependence and mood disturbance (e.g., Adams, 2009). The PHQ-9 is a brief, self-report tool designed to assess the severity of depressive symptoms, based on DSM-IV criteria. Participants indicated their responses to each of 9-items using a Likert scale ranging from 0 (*Not At All*) to 3 (*Nearly Every Day*). A total score was computed by calculating the total score of the 9 items. Based on their responses, individuals were assigned to one of four categories (*mild, moderate, moderately severe*, and *severe depression*) based on cut points of 5, 10, 15 and 20 respectively.

Bermond-Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001). The BVAQ was administered to assess both cognitive and affective alexithymic traits. The BVAQ is a 40-item self-report questionnaire that is comprised of five subscales (eight items each) tapping into the following traits: (1) emotionalizing (e.g., *When something unexpected happens, I remain calm and unmoved*); (2) fantasizing (e.g., *I have few daydreams and fantasies*); (3) identifying emotions (e.g., *When I am tense, it remains unclear from which of my feelings this comes*); (4) verbalizing emotions (e.g., *I find it difficult to express my feelings verbally*); and (5) analyzing emotions (e.g., *I hardly ever consider my feelings*). Participants responded to each item using a 5-point Likert scale ranging from 1 (*This definitely applies*) to 5 (*This in no way applies*). Subscale scores were derived by reverse-coding alexithymia contra-indicative items and then summing scores for the eight items in each subscale. A cognitive composite score was found by summing the Identifying, Verbalizing, and Analyzing subscales, and an affective composite score was found by summing the Emotionalizing and Fantasizing subscales.

Previous studies have confirmed the five-factor structure and psychometric validity of the BVAQ (Berthoz, Ouhayoun, Perez-Diaz, Consoli, & Jouvent, 2000, Vorst & Bermond, 2001). In the current study I found acceptable-to-high internal validity for each of the five subscales: $\alpha = .78$ for Identifying, $\alpha = .87$ for Verbalizing, $\alpha = .71$ for Analyzing, $\alpha = .72$ for Emotionalizing and $\alpha = .76$ for Fantasizing. Cronbach's alphas for the cognitive and affective composite scores were .87 and .75, respectively. There is a strong correlation between total (summed) scores across the three cognitive subscales of the BVAQ and total scores on the TAS-20, indicating that these scales measure the same (cognitive) dimension of alexithymia (van't Wout et. al, 2007).

Five subtypes of alexithymia have been proposed in the literature (Moormann et al., 2008; see Table 1). Four of these subtypes are found by using the 30th and 70th percentiles as cut-

points to represent extremes on either the cognitive or affective dimension, or both. The fifth subtype, referred to as "modals," includes individuals who score between the 30th and 70th percentile on both dimensions. Finally, individuals who do not meet criteria for any of the proposed five subtypes are referred to as having a "mixed" profile.

Table 1
Criteria to Create 5 Subtypes of Alexithymia plus a Mixed Subtype

	Label	Criteria
Type I	Both	Affective dimension > 70, Cognitive dimension > 70
Type II	Cognitive	Affective dimension < 30, Cognitive dimension > 70
Type III	Affective	Affective dimension > 70, Cognitive dimension < 30
Type IV	Lexithymic	Affective dimension < 30, Cognitive dimension < 30
Type V	Modal	Affective dimension = 30-70 and Cognitive dimension = 30-70
Mixed	Mixed	Do not meet above criteria

Note: Numbers represent percentiles

Interpersonal Reactivity Index (IRI; Davis, 1980). The IRI is a psychometrically sound assessment of empathy that consists of four subscales: Perspective Taking (PT), Fantasy (F), Empathic Concern (EC), and Personal Distress (PD). These subscales possess good internal consistency, with Cronbach's α coefficients ranging from .71 to .77, and good test-retest reliability, with coefficients ranging from .61 to .81 (Davis, 1980). Each subscale is comprised of seven items that are scored on a 5-point Likert Scale from 0 ("*Does not describe me well*") to 4 ("*Describes me very well*") resulting in a possible score of 0 to 28 per subscale (Davis, 1980).

Attention Checks: Conscientious Responders Scale (CRS; Marjanovic, Struthers, Cribbie, & Greenglass, 2014). The five item CRS provides a measure of conscientious (honest and accurate) responding (Marjanovic, et al., 2014); it can be used to detect poor effort in individuals responding to surveys and to increase the validity of survey research. The nature of the items allows one to determine objectively whether particular instructions are followed or not, which has been shown to correctly distinguish between conscientious and random responding

with greater than 93% accuracy (Marjanovic, et al., 2014). Including such attention checks is recommended by Meade and Craig (2012) as a practical way to decrease careless responding. The five items comprising the CRS were dispersed throughout the other measures.

Results

Data cleaning and management

The sample (N = 696) was recruited from first year university students enrolled in the Introduction to Psychology course at the University of Manitoba. These individuals participate in research for partial course credit and, as such, data quality can be a concern. In an attempt to decrease the impact of careless responding and increase the validity of participant responses, I followed Meade and Craig's (2012) recommendations, including having participants identify themselves by name and email address, and identifying and removing duplicate entries (n = 29), treating the first entry as the most likely to be accurate. Next, I excluded participants who had failed to complete the consent form (n = 1), those who had not completed the mass testing (and for whom I had no IRI data; n = 31), and those who omitted at least one subscale of any given questionnaire in its entirety (n = 11). In addition, modeling Marjanovic and colleagues (2014), I excluded those who failed to correctly answer three or more of the attention checks in the CRS (n = 16). I also examined the time participants took to complete the study. According to Huang, Curran, Keeney, Poposki, and DeShon (2012), it is unlikely that a participant can answer a single survey item in less than two seconds (DeSimone, Harms, & DeSimone, 2015). Given that the questionnaire contained 88 items, a minimum acceptable completion time of 176 seconds was established; all remaining participants met this criterion. Finally, modeling Meade and Craig (2012) and Maniaci and Rogge (2014), I also excluded "clear outliers" on the high end of the distribution who took longer than one hour to complete the study (likely due to their leaving the

survey and then returning; $n = 8$). After exclusions due to one or more of the criteria outlined above I was left with a final sample of $N = 600$.

Across the 18 subscales, there was between 0.0 and 0.3% missing data. Using Little's MCAR Test, it was found that all data were missing randomly, $p \geq .23$. Therefore, expectation maximization was used to estimate missing data.

Characteristics of the Full Sample

Demographic variables. There was a small amount of missing data for the following demographic variables: age (1.0%), ethnicity (0.8%), first language (0.2%), born in Canada (0.2%), and biological sex (3.2%). The mean age of respondents was 18.9 years (*SD* 2.9, range 17-46). The majority identified as female (65.7%), were born in Canada (73.9%), and indicated English as a first language (74.3%). Approximately half of the full sample (53.6%) identified as White/European, 28.3% as Filipino or Asian/South East Asian, and 17.9% as First Nation/Métis or Other.

Study variables. In the full sample, scores on the EDE-QS covered virtually the full range, going from 0 to 2.92 (see Table 2). As noted above, the EDE-QS is intended for screening purposes only. As such, there are no established diagnostic cut-off scores for this instrument.

Based on their responses to the PHQ-9, 170 participants in the full sample (28.3%) reported low to mild depressive affect, 211 (35.2%) reported moderate depressive affect, 118 (19.7%) reported moderately severe depressive affect, and 101 (16.8%) reported severe depressive affect. The prevalence of moderately severe or severe depressive affect was consistent with that reported in systematic reviews examining the prevalence of depression among university students (e.g., Ibrahim, Kelly, Adams & Glazebrook, 2013).

Table 2
Characteristics of the Full Sample on Measures of Exercise Dependence, Eating Disorder Severity, Depressive Affect, Symptoms of Cognitive and Affective Alexithymia, and Empathy (N = 600)

	M (SD)	Minimum	Maximum
EDS Total	51.8 (19.5)	21	126
EDE-QS Total	0.7 (0.6)	0	2.9
PHQ-9 Total	8.7 (5.8)	0	27
BVAQ Cognitive Total	62.8 (14.1)	25	100
BVAQ Affective Total	39.6 (8.7)	19	67
Perspective Taking	42.4 (8.6)	19	68
Fantasy	43.5 (11.5)	13	70
Personal Distress	34.7 (9.7)	7	65
Empathic Concern	50.5 (9.7)	11	70

Note: EDS: Exercise Dependence Scale; EDE-QS: Eating Disorder Examination Questionnaire-Short; PHQ-9: Patient Health Questionnaire; BVAQ: Bermond-Vorst Alexithymia Questionnaire

Scores on the cognitive and affective alexithymia subscales were generally somewhat higher than expected (Salminen et al., 1999); however, university students are reported to be at higher risk for alexithymia than members of the general population (e.g., Lyvers, Duric, & Thorberg, 2014). The reason(s) for this are unclear. A descriptive analysis using the criteria outlined in Table 1 confirmed that all five subtypes of alexithymia and a large group showing a "mixed" profile were evident in my sample. Specifically, 10.5% of individuals displayed a Type I profile, 8.0% displayed a Type II profile, 6.8% displayed a Type III profile, 10.5% displayed a Type IV (lexithymic) profile, 18.3% displayed a Type V (modal) profile, and finally 45.8% displayed a mixed profile. This distribution of subtypes was similar to that reported by Rood (2003) in a student sample.

As classified by the EDS, 221 participants (36.8%) were rated as "asymptomatic," 359 (59.8%) as "symptomatic," and 20 (3.3%) as "at-risk" for exercise dependence. The prevalence

of "at-risk" individuals was slightly higher than that seen in past research conducted in a general population (Mónok et al., 2012), but consistent with research conducted with habitual exercisers (Griffiths, Szabo & Terry, 2005; Szabo & Griffiths 2007). Due to the low number of "at-risk" participants, they were combined with "symptomatic" participants for all subsequent analyses.

Latent Profile Analysis

I utilized LPA to explore the multidimensionality of exercise-dependent behaviour in university students (Goodman, 1974). This analysis was restricted to the 379 individuals scoring in the symptomatic/at-risk range on the EDS-21. The LPA was conducted using Mplus Version 6.0 (Muthén & Muthén, 1998-2010).

LPA allows for participants to be categorized into two or more distinct groups based on individual characteristics (Collins & Lanza, 2010)—an element that is relatively lacking in current exercise dependence research. Total scores from the EDS-21, EDE-QS, and PHQ-9, along with the cognitive and affective composite scores from the BVAQ, were entered into the LPA model. All of these are theoretically relevant variables (Wurpts & Geiser, 2014).

As reported by Nylund, Asparouhov, and Muthén (2007), an LPA model yields two types of estimated parameters: (1) the probability of endorsing specific items from the above questionnaires, and (2) the probability that any participant will be in any latent class. In LPA, no *a priori* assumptions are made regarding the number of subgroups present (Carragher et al., 2011). Therefore, modeling Carragher and colleagues (2011), I began with one subgroup, and increased that number by one until the best fitting model was found (Shiu-Yee et. al., 2018)—that is, the model that minimized the Bayesian Information Criterion (BIC; Schwarz, 1978) and maximized the entropy statistic (Ramaswamy, DeSarbo, Reibstein, & Robinson, 1993).

Table 3 presents the fit statistics for the estimated model solutions assuming two to six classes. Five latent models were tested. Although the 4-Class and 5-Class models fit the data well and approached significance, I decided to retain the 2-Class model for two reasons. Firstly, the Lo, Mendell, and Rubin (2001) likelihood ratio test (LMR-LRT) statistic indicated that only the 2-Class model was significant. Secondly, a 2-Class model is consistent with extant literature suggesting two subtypes of exercise dependence (Bamber et al., 2002; Berczik et al., 2012; Hausenblas et al., 2017) as well as only two types of alexithymia associated with psychological morbidity (i.e., subtypes I and II; Bermond et al., 2010).

Table 3

Selected Fit Indices for 2- to 6-Class Latent Profile Models Among University Students Symptomatic or At-Risk for Exercise Dependence (N = 379)

Model	BIC	Change in BIC	Entropy	LMR-LRT
2-Class	**11866.15**	**0**	**0.811**	**0.01**
3-Class	11817.03	-49.12	0.867	0.48
4-Class	11797.61	-19.42	0.871	0.06
5-Class	11757.23	-40.38	0.841	0.07
6-Class	11753.07	-4.16	0.810	0.21

Note: BIC: Bayesian Information Criterion; LMR-LRT: Lo, Mendell, and Rubin (2001) likelihood ratio test

Class 1 was comprised of 300 participants, 59% of whom were female. Class 2 was comprised of 79 participants, 75.9% of whom were female. The two classes differed with regard to sex distribution, $X^2(2) = 11.83$, $p = .003$.

Characteristic profiles for each class with regard to the LCA input variables and the seven symptoms of exercise dependence are shown in Figures 1 and 2, respectively. In each figure, scores for each class are plotted using Z-scores (computed using data from the full sample, $N = 600$) in order to place the variables on a common scale. Data from the asymptomatic participants are included for comparison purposes.

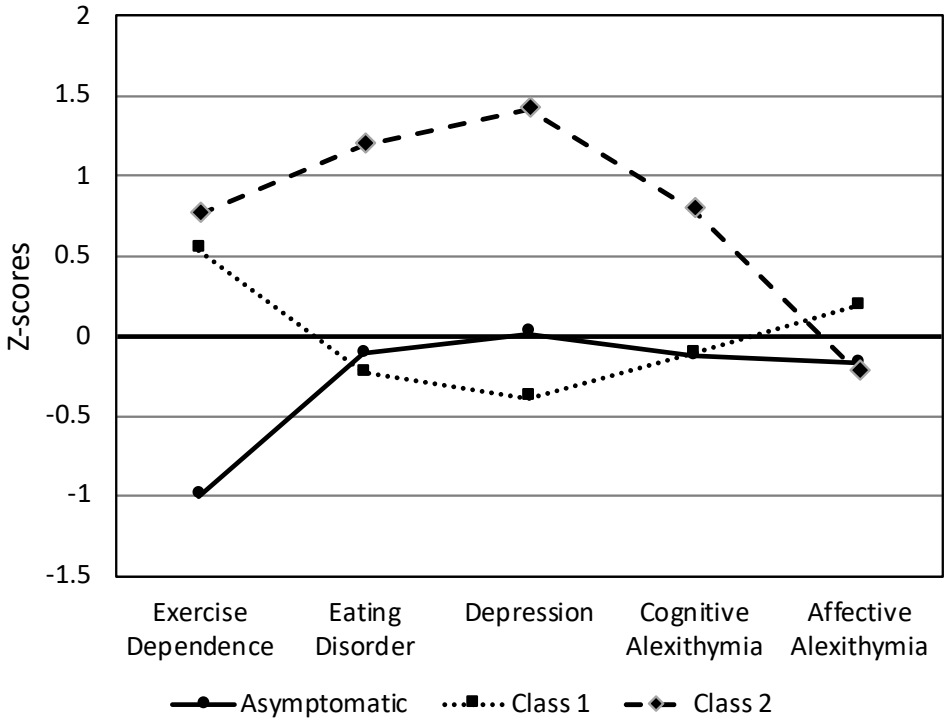

Figure 1. Plotted are mean Z scores for each of the five input variables, for each class. Means for the asymptomatic group are included for comparison.

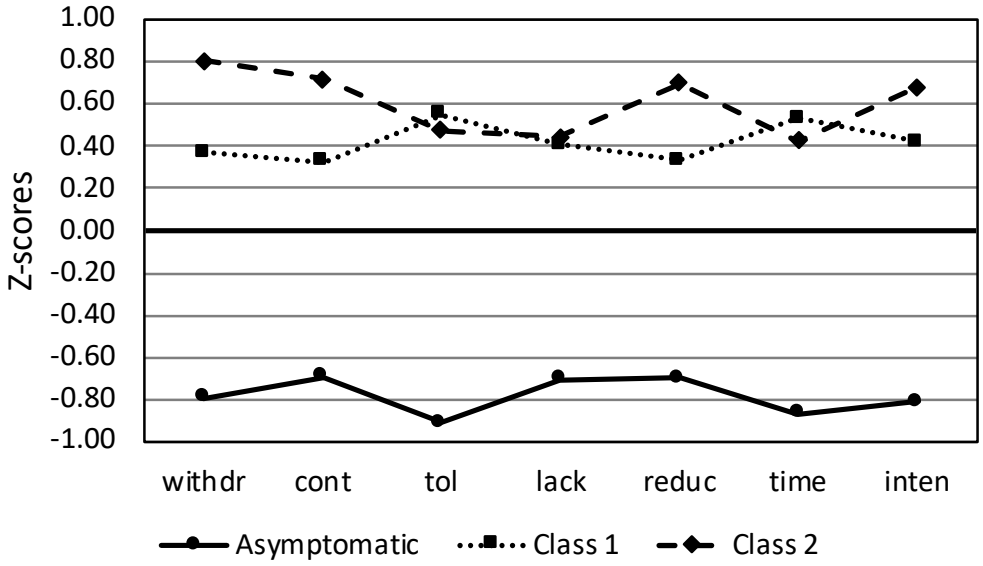

Figure 2. Exercise dependence symptom profiles (withdr: withdrawal; cont: continuance; tol: tolerance; lack: lack of control; reduc: reductions in other activities; intent: intention effects). Plotted are mean Z scores for each class; means for asymptomatic individuals are included for comparison purposes.

A series of Welch's tests were performed to explore how well the observed latent classes could be distinguished from one another on the basis of the LPA input variables (Table 4), and on the individual symptoms of exercise dependence (Table 5). Class 1 scored higher on all seven symptoms of exercise dependence [$F > 244.7$, $p < .001$ in all cases] and was significantly less depressed [$F(1,328) = 25.8$, $p < .001$] than the asymptomatic group. Class 2 had higher EDS-21 total scores, and reported more withdrawal symptoms, continuance symptoms, reductions in other activities, and intention effects, compared to Class 1. Class 2 also reported significantly more severe symptoms of depression, eating disorder, and cognitive alexithymia, but significantly weaker signs of affective alexithymia, than Class 1. Together, these observations suggest that: (a) despite creating significant functional disruption, exercise was not associated with depression in Class 1; and (b) problems regulating both mood and eating were prevalent in Class 2.

Table 4

Group Differences between Class 1 and Class 2 on measures of Exercise Dependence, Eating Disorder Severity, Depressive Affect, and Symptoms of Cognitive and Affective Alexithymia (N = 379)

	Class 1	Class 2					
	M (SD)	M (SD)	Lower Bound 95% CI	Upper Bound 95% CI	*F*	*p*	*g*
EDS Total	62.2 (13.8)	66.7 (16.7)	61.7	64.6	4.81	.030	0.31
EDE-QS Total	0.6 (0.5)	1.5 (0.7)	0.7	0.8	125.33	<.001	1.64
PHQ-9 Total	6.4 (3.6)	16.9 (4.1)	8.0	9.2	433.21	<.001	2.83
BVAQ Cognitive Total	61.2 (13.1)	73.9 (13.9)	62.4	65.3	53.63	<.001	0.96
BVAQ Affective Total	41.2 (8.8)	37.6 (8.3)	39.6	41.4	11.52	.001	0.41

Note: EDS: Exercise Dependence Scale; EDE-QS: Eating Disorder Examination Questionnaire-Short; PHQ-9: Patient Health Questionnaire; BVAQ: Bermond-Vorst Alexithymia Questionnaire

Table 5

Group Differences between Class 1 and Class 2 on Symptoms of Exercise Dependence (N = 379)

	Class 1	Class 2					
	M (SD)	M (SD)	Lower Bound 95% CI	Upper Bound 95% CI	*F*	*p*	*g*
Tolerance	12.6 (3.1)	12.3 (3.4)	12.2	12.9	0.60	.442	0.09
Withdrawal	10.1 (3.4)	11.8 (3.2)	10.2	10.8	17.01	<.001	0.60
Intention Effect	8.6 (3.1)	9.6 (3.7)	8.5	9.2	4.41	.038	0.31
Lack of Control	7.3 (2.9)	7.5 (3.3)	7.1	7.7	0.09	.770	0.07
Time	9.1 (3.0)	8.8 (3.6)	8.7	9.4	0.70	.405	0.10
Reduc	6.6 (2.6)	7.6 (3.2)	6.5	7.1	6.46	.012	0.36
Continuance	7.8 (3.6)	9.2 (3.6)	7.7	8.4	9.51	.003	0.39

Note: Reduc: Reductions in Other Activities

To get a sense of how the two classes compared with regard to the proportion of individuals showing "clinically significant" difficulties in different areas, I categorized scores using established cut-offs (see Methods section) or, in the case of scores on the EDE-QS, as falling into a given quartile based on scores in the full sample (N = 600). The resulting distributions in each class were then compared using a series of chi-square tests. Where cell counts were less than 5, visual inspection was used to compare the distributions. The first analysis showed that Class 2 had a larger proportion of individuals who were classified as "at-risk" for exercise dependence than Class 1 (10% vs. 4% respectively), X^2 (1) = 4.70, p = .03, η^2 = .11 (see Figure 3).

Visual inspection revealed clear differences across the two classes in the distributions of scores on the EDE-QS (see Figure 3). Specifically, about a quarter of individuals in Class 1 scored in the first quartile (27.0%), whereas only 1.3% of individuals in Class 2 fell in this range. Furthermore, while only 15.0% of individuals in Class 1 scored in the fourth quartile, the vast majority of individuals in Class 2 (73.4%) fell in this range. Individuals in Class 2 also reported

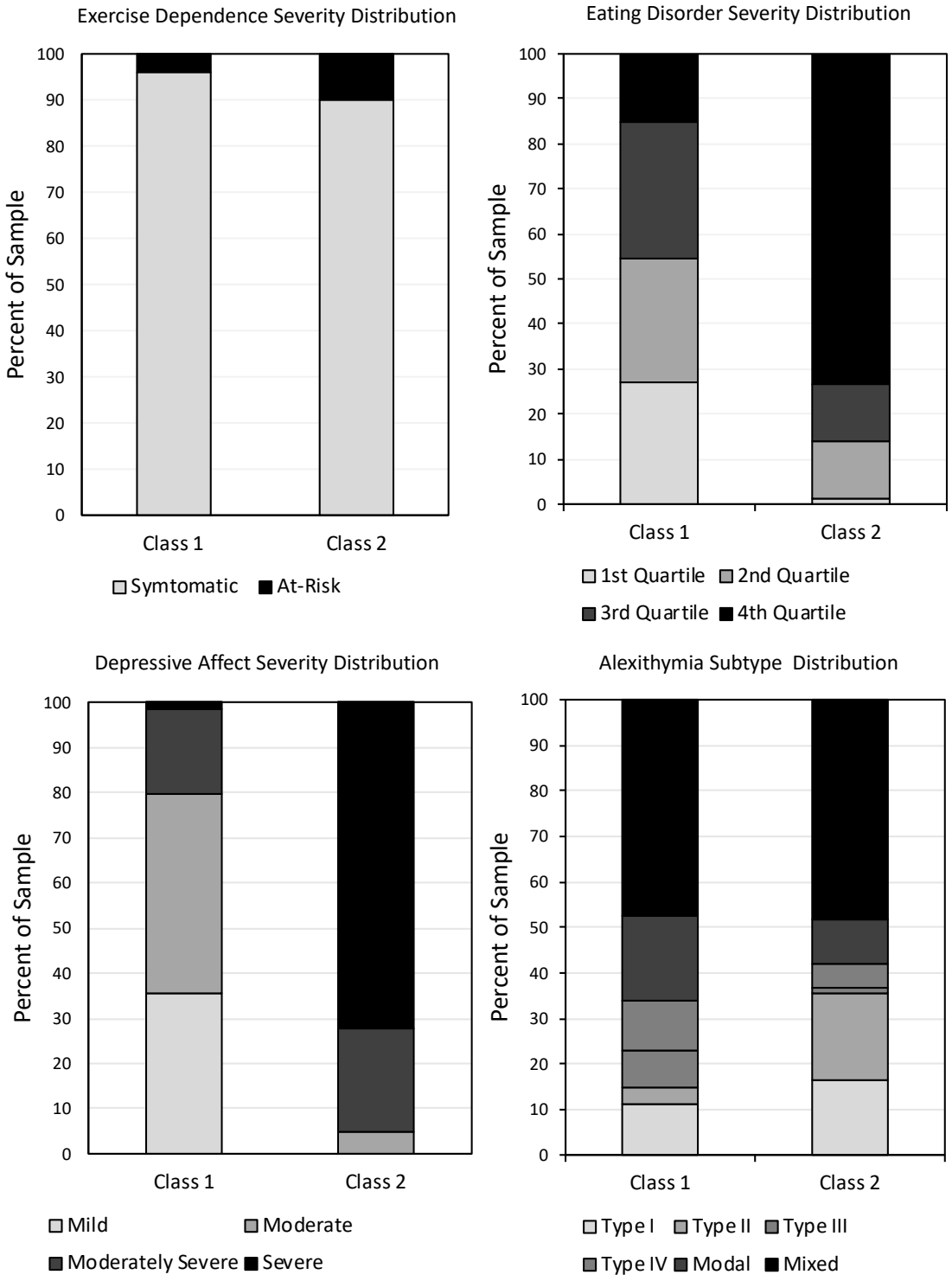

Figure 3. Percentage of each class exhibiting different characteristics of exercise dependence (upper left quadrant), eating disorder severity (upper right quadrant), depressive symptoms (lower left quadrant), and alexithymia (lower right quadrant).

more frequent use of exercise to control their weight, shape or body fat, or to burn off calories, as evidenced by their responses to Question 8 of the EDE-QS, $F(1, 377) = 34.8$, $p < .001$, $g = 0.84$.

The two classes differed dramatically with respect to the proportion of individuals scoring in the mild, moderate, moderately severe, and severe range for depressive affect (see Figure 3). Specifically, about a third of individuals in Class 1 experienced mild depressive affect (35.3%), whereas no individuals in Class 2 fell in this range. Furthermore, while only 1.33% of individuals in Class 1 scored in the severe range for depressive affect, the vast majority of individuals in Class 2 (72.2%) met criteria for this cutoff.

Visual inspection also revealed clear differences across the two subgroups with respect to the proportion of individuals meeting criteria for the five subtypes of alexithymia and the "mixed" profile (see Figure 3). Most striking was the fact that Class 2 had a larger proportion of individuals classified as Type II alexithymic (19% vs. 3.7%), but a smaller proportion of individuals classified as Type III alexithymic (1% vs. 8%) compared to Class 1. In the scatterplot shown in Figure 4, I present individual participants' scores on the two scales (expressed as *Z* scores). The lower left quadrant includes individuals reporting weak cognitive and affective alexithymic traits; extreme scores in this quadrant meet criteria for lexithymia. As can be seen by examining the distribution of points in the remaining quadrants, individuals in Class 1 (open circles) frequently reported stronger affective than cognitive traits (scores falling below the diagonal), while the majority of those in Class 2 (closed circles) showed the opposite pattern.

Links Between Empathy and Exercise Dependence

Figure 5 shows empathy profiles of the two latent classes, plotted using *Z* scores computed for each scale separately using data from the full sample ($N = 600$). Data from the asymptomatic class are again included for comparison purposes.

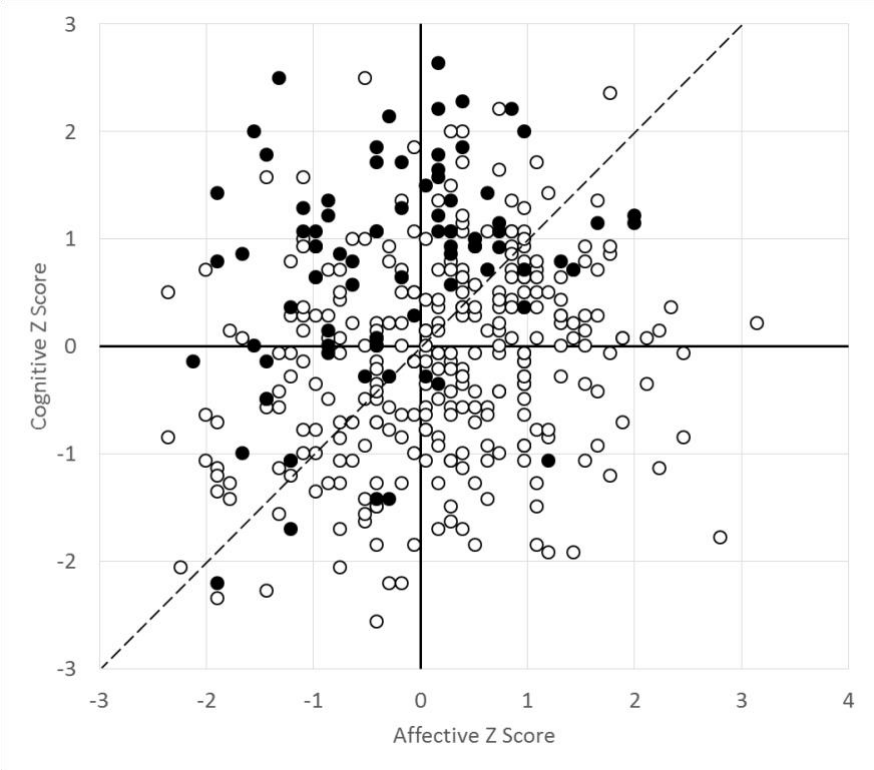

Figure 4. Scatterplot showing scores for participants in Class 1 (open circles) and Class 2 (closed circles) on the Cognitive and Affective scales of the BVAQ (expressed as *Z* scores).

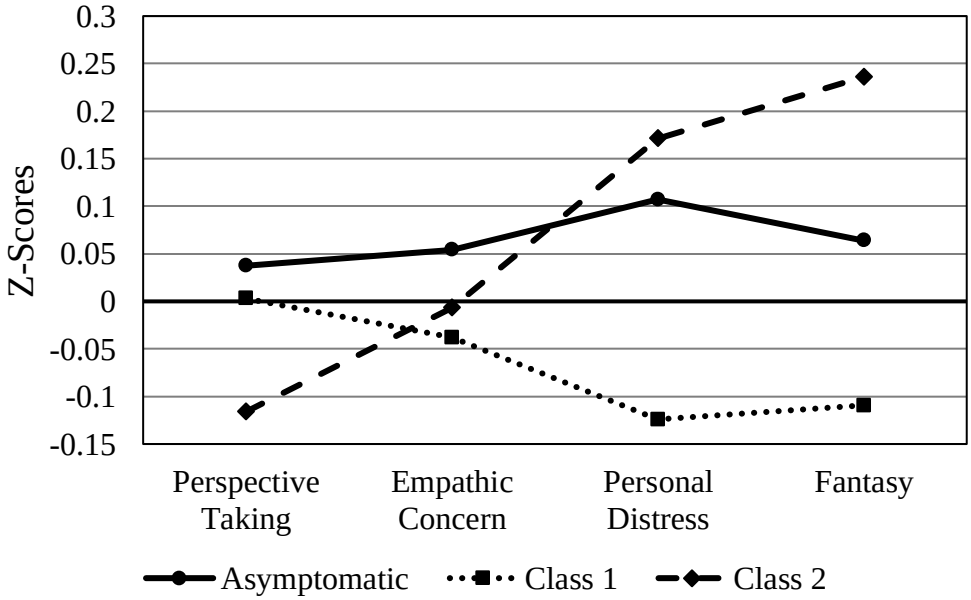

Figure 5. Mean scores for each class on the four subscales of the Interpersonal Reactivity Index, plotted as Z scores. Means for asymptomatic individuals are included for comparison purposes.

Welch's tests were performed to explore the relationship between class membership and self-reported empathic abilities. Perspective Taking and Empathic Concern did not significantly differ across classes. However, the two classes did differ with respect to Fantasy ($F = 6.90$, $p = .01$, $g = 0.35$) and Personal Distress ($F = 5.08$, $p = .03$, $g = 0.31$), with individuals in Class 1 reporting being less able to empathize with a fictional character (M = 42.22 vs. 46.20) and experiencing lower levels of personal distress in response to others' suffering (M = 33.47 vs. 36.34) than those in Class 2. Supplementary tests confirmed that Class 1 also scored significantly lower than the asymptomatic group on these two scales ($F > 3.89$, $p < .049$ in both cases). Together, these results suggest a relative weakness in these aspects of empathy in Class 1, and a relative strength in Class 2.

To explore whether the cognitive and affective dimensions of alexithymia differentially mediated the relationship between empathy and exercise dependence, I performed a mediation analysis using Mplus Version 6.0 (Muthén & Muthén, 1998-2010). Here, the four dimensions of empathy were evaluated as correlated mediators, as were cognitive and affective alexithymia scores. Several well-established fit statistics were used to determine whether the model was a good fit: Comparative Fit Index (CFI) ≥ 0.95, Root Mean Square Error of Approximation (RMSEA) ≤ 0.05, and Standardized Root Mean Square Residual (SRMR) $\leq .08$. Examination of these statistics indicated a good fit to the data ($\chi^2_{(4)} = 6.64$, $p = .16$, $CFI = 0.98$, $RMSEA = 0.04$, 90% CI [.00, .10], $SRMR = 0.02$). As illustrated in Figure 6, there was a significant indirect effect of perspective taking ($B = -.025$, 95% CI [-.391, -.027]), empathic concern ($B = -.026$, 95% CI -.399, -.038]), and personal distress ($B = .039$, 95% CI [139, .483]) on exercise dependence through cognitive alexithymia. Importantly, the relationships between cognitive alexithymia and both personal distress and exercise dependence were *positive*. Thus, cognitive alexithymia

(which was more prevalent in Class 2) mediated the link between experiencing *heightened* personal distress and risk of exercise dependence. In contrast, cognitive alexithymia mediated the link between experiencing *decreased* perspective taking and empathic concern and risk of exercise dependence. Although affective alexithymia was negatively associated with empathic concern, personal distress, and fantasy, it was not a mediator of exercise dependence. These results suggest that past reports linking deficits in affective empathy to alcohol-dependence (Maurage et al., 2011) and drug addiction (Ferrari et al., 2014) may need to be reassessed, taking individual differences in cognitive and affective alexithymia into account.

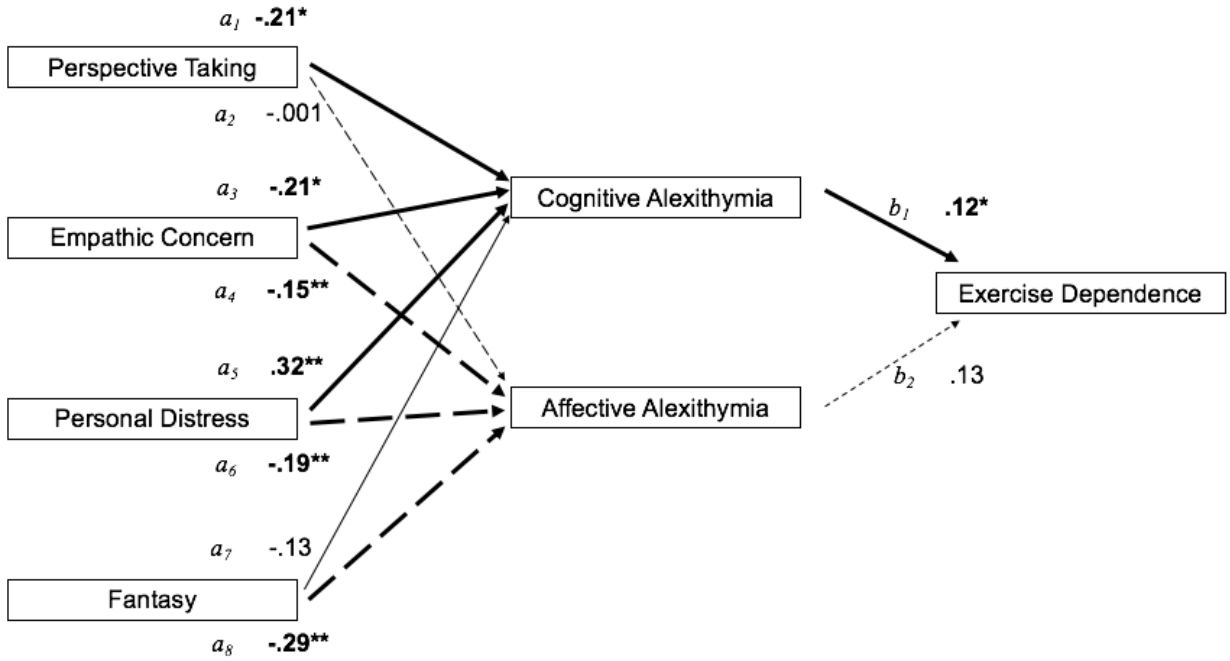

Figure 6. Mediation model testing the indirect effects of cognitive and affective alexithymia on the relationship between empathy and exercise dependence on individuals symptomatic/at-risk for exercise dependence (N = 379). The four dimensions of empathy were included as correlated mediators. Cognitive and alexithymia were also included as correlated mediators.
* $p < .05$; ** $p < .01$.
Note. Solid lines indicate the cognitive alexithymia pathway. Hashed lines indicate the affective alexithymia pathway. Bold lines indicate a significant effect.

Discussion

The first aim of this study was to determine if individuals who are symptomatic/at-risk for exercise dependence are a heterogeneous group, consistent with other behavioural addictions like problem gambling (Carragher et al., 2011; Chamberlain et. al, 2017; Cunningham-Williams & Hong, 2007). This was found to be the case, as two latent classes emerged that could be differentiated on the cognitive and affective alexithymic traits that they endorsed, as well as eating disorder severity and depressive affect.

Class 1 individuals were characterized by lower severity across most factors, including exercise dependence, symptoms of disordered eating, depression, and cognitive alexithymia. The prevalence of individuals at-risk for exercise dependence in Class 1 (4%) was consistent with what has been reported in the literature for a general exercising population (Mónok et al., 2012). This class most resembled Bamber et al.'s (2000) class of exercisers reporting primary exercise dependence. Compared to the asymptomatic group, Class 1 obtained similar mean scores on symptoms of eating disorder severity and cognitive alexithymia and *lower* mean scores on symptoms of depression. Class 2, in contrast, reported stronger signs of exercise dependence, and more severe symptoms of depression and eating disorder than Class 1. As discussed below, they could most accurately be described as displaying secondary exercise dependence. Together, the results suggest that, consistent with previous research (e.g., Bamber et al., 2000), depression may pose a more severe problem for exercise-dependent individuals who report more severe symptoms of eating disorder (i.e., Class 2), compared to those who report fewer symptoms of eating disorder (i.e., Class 1) and/or healthy controls (Bamber et al., 2000). Although females were over-represented in my sample, it is important to note that Class 1 had a higher proportion of males than Class 2, consistent with previous reports suggesting that men are more likely to be

affected by primary exercise dependence than secondary exercise dependence (Hausenblas et al., 2017).

Class 1: Primary Exercise Dependence

The low levels of depression observed in Class 1 may be related to the fact that individuals in this class showed lower cognitive and higher affective alexithymia scores than those in Class 2. Indeed, Class 1 had a higher proportion of individuals classified as Type III alexithymic than Class 2 (8% vs. 1%)—a profile that some researchers (i.e., Moormann et al., 2008) associate with psychological well-being. Bamber and colleagues (2000) concluded that, due to the absence of comorbid personality and mood disorders, primary exercise dependence should not be operationalized as a form of "pathology." Others have argued that there is not enough evidence to suggest that primary exercise dependence exists at all (Blaydon et al., 2002). My results, however, suggest that primary exercise dependence not only exists, but that it can have debilitating consequences. Individuals in Class 1 reported stronger symptoms of exercise dependence--including withdrawal, continuance (exercising despite injury), and loss of control--than asymptomatic participants. Thus, while this class may not be at risk for co-occurring mood or eating disorders, their excessive exercise may put them at risk for other problems that interfere with daily life. It is also possible (though not tested here) that they are at risk for other behavioural addictions, such as problem gambling, compulsive buying, internet addiction, and work addiction (Villella et al., 2011).

It is important to note that Class 1 may have included some individuals with a commitment, rather than an addiction, to exercise. If so, it seems likely that the proportion of committed exercisers was small. I say this because individuals in Class 1 reported greater severity on *all seven symptoms* of exercise dependence than asymptomatic individuals, including

greater loss of control—a feature said to distinguish addicted from committed exercisers. Future research is needed to illuminate the differences between committed exercisers and those with primary exercise dependence. It is possible that committed exercisers may transition to primary exercise dependence, or that being committed to exercise or showing primary exercise dependence puts one at risk for transitioning to secondary exercise dependence over time. Longitudinal research will need to be performed to examine these possibilities and to determine what the risk factors are.

It seems plausible that individuals in Class 1 exercise primarily to receive positive reinforcement. Particularly suggestive is the fact that they scored *lower* on depression that the asymptomatic group, perhaps indicating that exercise has a mood enhancing effect for individuals in this group. Recent work highlights the potential role that the endocannabinoid system might play in generating a "runner's high" (Antunes et al., 2016; Dietrich & McDaniel, 2004), but more research is needed. Class 1 may also exercise to increase arousal/energy levels, consistent with both the sympathetic arousal hypothesis (Thompson & Blanton, 1987) and the hypoarousal theory (Donges & Suslow, 2017).

Individuals in Class 1 showed a weakness in two self-focused aspects of empathy: fantasizing abilities and personal distress. In this respect, they resembled individuals with alcohol-dependence (Maurage et al., 2011) and drug addiction (Ferrari et al., 2014). Results from the mediation analysis suggest that deficits in perspective taking, empathic concern, and personal distress predict affective alexithymia; however, affective alexithymia does not predict exercise dependence. Although these empathic deficits do not appear to be contributing to exercise dependence through affective alexithymia, they may certainly be contributing to social impairment. Social impairment, in turn, may increase risk of primary exercise dependence.

Given that I did not include a measure of social functioning in the current study, future research will need to be performed to examine these possibilities.

Class 2: Secondary Exercise Dependence

The prevalence of individuals at-risk for exercise dependence in Class 2 (10%) was higher than what has been reported in the literature for a general exercising population (Mónok et al., 2012). In addition to scoring higher on exercise dependence than those in Class 1, Class 2 individuals were characterized by stronger symptoms of disordered eating, depression, and cognitive alexithymia. This class most resembled Bamber et al.'s (2000) class of exercisers reporting secondary exercise dependence. Indeed, approximately 74% showed severe symptoms of eating disorder, and many reported the frequent use of exercise to control their weight, shape, or body fat, or to burn off calories. Adams (2009) has argued that secondary exercise dependence might not represent an addiction at all. However, I found that individuals in Class 2 scored higher than those in Class 1 on four of seven symptoms of exercise dependence, including withdrawal, intention effects, reduction in other activities, and continuance, suggesting that the exercise was interfering with their daily functioning and increasing their risk of injury, above and beyond what was seen even in those with primary exercise dependence. This appears to be in line with the operationalization of addiction in the DSM-V, in that it involves a behaviour being employed uncontrollably despite negative consequences (APA, 2013). I argue, on these grounds, that secondary exercise dependence represents a true behavioural addiction--one associated with a high risk of comorbid eating disorder pathology and depression.

Consistent with previous work linking alexithymia to various addictive behaviours (e.g., Bossard et al., 2009; Morie et al., 2017; Noel et al., 2017; Taylor et al., 1990), my results support the idea that cognitive alexithymia may be influential in the development or maintenance of

secondary exercise dependence. Those who struggle to verbalize their emotions during stressful times have poorer daily health outcomes than those who do not (see also Zautra et al., 2001). This may be partly due to associated increases in the amount of personal distress individuals with secondary exercise dependence experience. Indeed, others have also noted a link between cognitive alexithymia and increased personal distress (Guttman & Laporte, 2002; Moriguchi et al., 2006; Patil & Silani, 2014), especially in those with eating disorders (e.g., Beadle et al., 2013; Brewer et al., 2018). Increased personal distress has also been implicated in problem gambling (Tomei et al., 2017).

Results of the mediation analysis demonstrate the influence of diminished perspective-taking skills and levels of empathic concern, coupled with heightened personal distress, on cognitive alexithymia, which increases risk for exercise dependence. This mediation pathway supports the idea that some individuals may excessively exercise due to problems with emotion regulation. Indeed, individuals who cannot effectively reduce their feelings of distress struggle to adopt another's point of view (Decety and Moriguchi, 2007, Eisenberg et al., 1994). Interestingly, an empathy profile characterized by weak perspective-taking skills and heightened personal distress frequently characterizes individuals suffering from depression (Cusi, MacQueen, Spreng, & McKinnon, 2011; Wilbertz, Brakemeier, Zobel, Härter, & Schramm, 2010).

Given the results presented above, it seems plausible that individuals in Class 2 exercise primarily for negative reinforcement—specifically, to combat overwhelming negative emotions (see Taylor et al., 1991, 1999). Their maladaptive use of exercise may reflect generalized problems with emotion regulation that are linked with cognitive alexithymia (Connelly & Denney, 2007; Moormann et al., 2008); these problems may limit their ability to engage in

strategies such as cognitive appraisal. In this case, the affect regulation hypothesis of exercise dependence (Hamer et al., 2007) seems to provide the best explanation for secondary exercise dependence.

Theoretical and Clinical Implications

The literature on problem drinking describes motivational alcohol-use models, whereby individuals drink to regulate both positive and negative emotions (Cooper, Frone, Russell, & Mudar, 1995; Cox & Klinger, 1988). I have argued above that individuals with primary exercise dependence (Class 1) might exercise primarily for positive reinforcement, whereas those with secondary exercise dependence (Class 2) might exercise primarily for negative reinforcement. I also suggested that different theories of exercise addiction could explain these different motivations. Clinicians and researchers should explore the motivations that drive exercise-use in the moment in more detail, as this may provide important clues about the specific factors driving excessive exercise in particular individuals.

Clinically, it is important to recognize that certain individuals with exercise dependence are more likely to display comorbid psychopathology (Blaydon et. al, 2004). Specifically, individuals with secondary exercise dependence, who exercise to control their weight or body shape and exhibit other signs of eating disorder, also reported more severe depressive affect and cognitive alexithymic traits than those with primary exercise dependence. This suggests a need to increase emotional awareness in individuals with secondary exercise dependence. Emotion focused therapy (Greenberg, 2002) could help these individuals more fully understand their emotions, reducing the need to participate in excessive exercise (Noel et al., 2017). Alternatively, mindfulness-based interventions that aim to bring awareness to bodily sensations and accompanying emotions have been found to be effective in reducing cognitive alexithymia in

elite athletes (Haase et al., 2015), possibly due to enhanced awareness and identification of bodily sensations (i.e., interoception; Ernst et al., 2014). Indeed, a recent meta-analysis found a significant effect (with a moderate effect size) of mindfulness-based treatment on cognitive alexithymia, compared to a control group (Norman et al., 2019). (See Leweke, Bausch, Leichsenring, Walter, & Stingl, 2009, for further discussion of the impact of cognitive alexithymia on response to different treatment approaches).

Other strategies may be more beneficial for individuals with primary exercise dependence, who have difficulty fantasizing and emotionalizing and may experience emotional blunting (as reflected in low scores on personal distress). For example, certain approaches that utilize role playing or that target nonverbal communication could be beneficial in fostering an increase in empathy (Beresnevaite, 2000; Levant, Halter, Hayden, & Williams, 2009; Tulipani et al., 2010).

As noted by Lichtenstein et al. (2014), past research highlighting negative correlations between exercise dependence and straightforwardness, altruism, modesty, and compliance suggest that some individuals with exercise dependence may be at an elevated risk for experiencing interpersonal difficulties. These may be exacerbated in individuals displaying narcissism, which has also been linked to exercise dependence (Spano, 2001). Whether these features are more common in those with primary or secondary exercise dependence is unclear. Treatment may need to address these problems. It may also need to address anxiety disorders that may vary by alexithymia subtype (e.g., Moormann et al., 2008; Spano, 2001). It could be the case that individuals in Class 1 (who may utilize exercise as a form of self-serving behaviour) would score high on narcissistic traits, while those in Class 2 (who have problems with emotion regulation) would be more likely to experience anxiety and panic attacks. In any case, including

a focus on interpersonal functioning in therapy may help individuals displaying these characteristics to maintain healthier relationships and gain more adaptive coping skills (Ogrodniczu, Sochting, Piper, & Joyce, 2012).

In addition to the above, the current research leads to testable hypotheses concerning which interventions are *unlikely* to be effective for certain individuals. For example, individuals with affective alexithymia who are characterized as having a poor fantasy life may be unlikely to benefit from "possible selves" interventions that have been found to influence behaviour change in both academic (Hock, Deshler, & Schumaker, 2006) and physical settings (Strachan, Marcotte, Giller, Brunet, & Schellenberg, 2017).

As a final point here, it is important to note that there was a large group of asymptomatic individuals in our original sample who reported rates of depressive affect that were similar to those seen in symptomatic/at-risk individuals. This suggests the presence of at least two groups of individuals who experience depressive symptoms: those who exercise and those who do not. Learning more about the characteristics of these subgroups, and how best to support them, should be an important focus of future research. Between 2012 and 2014, 44.4% of university students in Canada reported difficulty functioning due to depression, and 13.0% reported contemplating suicide in the past 12 months (American College Health Association, 2013). Some studies report that these numbers are only rising (e.g., Fier & Brzezinksi, 2010).

Limitations

There are a number of limitations to consider when interpreting the results of this study. Firstly, I did not ask participants to identify themselves as athletes or non-athletes. This may have important implications, given studies that suggest that current exercise dependence tools

may overestimate the prevalence of exercise dependence in competitive athletes (Szabo et al., 2015).

Secondly, the cross-sectional nature of the study does not provide any information on how or if class membership may change over time. Furthermore, due to the correlational nature of the design, causality cannot be inferred. It is therefore recommended that longitudinal research be conducted to investigate the evolution of different subtypes of exercise dependence.

Finally, it is important to note that the existence of alexithymia "subtypes" is considered equivocal, and that there may be a more theoretically sound way to group participants in the LPA. For example, Preece and colleagues (2017) suggest a three-factor model comprised of two higher order dimensions: difficulty appraising emotions and difficulty attending to emotions. It is therefore recommended that further research examine the psychometric validity of the BVAQ as a tool for examining alexithymic traits.

Conclusion

This study provides empirical support for the view that individuals symptomatic/at-risk for exercise dependence are a heterogeneous group that differ in severity in a number of ways. The results have important implications for theories regarding the etiology of exercise addiction, and for clinical practice.

References

Adams, J. (2009). Understanding Exercise Dependence. *Journal of Contemporary Psychotherapy*, *39*(4), 231. doi:10.1007/s10879-009-9117-5

Allegre, B., Souville, M., Noel-Jorand, M. C., Pellegrin, L., & Therme, P. (2007). Intensive Physical Activity and Alexithymia: Results from Swimmers' Discourse Analysis. *Psychological Reports*, *100*(3), 1129–1139. doi:10.2466/pr0.100.4.1129-1139

American College Health Association. (2013). American College Health Association—National College Health Assessment II: Canadian reference group executive summary spring 2013.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders*. doi:10.1176/appi.books.9780890425596.dsm16

Anderson, C., & Keltner, D. (2002). The role of empathy in the formation and maintenance of social bonds. *Behavioral and Brain Sciences*, *25*(1), 21–22. doi:10.1017/S0140525X02230010

Antunes, Leite, Lee, Barreto, Santos, et al. (2016). Exercise deprivation increases negative mood in exercise-addicted subjects and modifies their biochemical markers. *Physiology & Behavior*, *156*, 182–190. doi:10.1016/j.physbeh.2016.01.028

Bagby, R. M., Taylor, G. J., & Parker, J. D. A. (1994). The twenty-item Toronto Alexithymia scale-II. Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research*, *38*(1), 33–40. doi:10.1016/0022-3999(94)90006-X

Bagby, R. M., Quilty, L. C., Taylor, G. J., Grabe, H. J., Luminet, O., et al. (2009). Are there subtypes of alexithymia? *Personality and Individual Differences*, *47*, 413–418.

Bamber, D., Cockerill, I. M., & Carroll, D. (2000). The pathological status of exercise

dependence. *British Journal of Sports Medicine*, *34*(2), 125–132. doi:10.1136/bjsm.34.2.125

Banissy, M. J., Kanai, R., Walsh, V., & Rees, G. (2012). Interindividual differences in empathy are reflected in human brain structure. *NeuroImage*, *62*, 2034–2039.

Barlow, M., Woodman, T., Chapman, C., Milton, M., Stone, D., Dodds, T., & Allen, B. (2015). Who Takes Risks in High-Risk Sport?: The Role of Alexithymia. *Journal of Sport and Exercise Psychology*, *37*(1), 83–96. doi:10.1123/jsep.2014-0130

Barlow, M., Woodman, T., & Hardy, L. (2013). Great expectations: Different high-risk activities satisfy different motives. *Journal of Personality and Social Psychology*, *105,* 458–475. doi.org/10.1037/a0033542

Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, *6*, 248-254.

Baron-Cohen, S., & Wheelwright, S. (2004). The Empathy Quotient: An Investigation of Adults with Asperger Syndrome or High Functioning Autism, and Normal Sex Differences. *Journal of Autism and Developmental Disorders*, *34*(2), 163–175. doi.org/10.1023/B:JADD.0000022607.19833.00

Beadle, J. N., Paradiso, S., Salerno, A., & McCormick, L. M. (2013). Alexithymia, emotional empathy, and self-regulation in Anorexia Nervosa. *Annals of Clinical Psychiatry: Official Journal of the American Academy of Clinical Psychiatrists*, *25*(2), 107–120.

Beck, A. T., & Beck, R. W. (1972). Screening depressed patients in family practice. A rapid technic. *Postgraduate Medicine*, *52*(6), 81–85.

Berczik, K., Szabó, A., Griffiths, M. D., Kurimay, T., Kun, B., Urbán, R., & Demetrovics, Z. (2012). Exercise Addiction: Symptoms, Diagnosis, Epidemiology, and Etiology.

Substance Use & Misuse, 47(4), 403–417. doi:10.3109/10826084.2011.639120

Beresnevaite, M. (2000). Exploring the benefits of group psychotherapy in reducing alexithymia in coronary heart disease patients: a preliminary study. *Psychotherapy and Psychosomatics*, *69*, 117-22.

Berlin, A. A., Kop, W. J., Deuster, P. A. (2006). Depressive mood symptoms and fatigue after exercise withdrawal: the potential role of decreased fitness. *Psychosomatic Medicine*, *68*, 224–230.

Bermond, B., Bierman, D. J., Cladder, M. A., Moormann, P. P., & Vorst, H. C. M. (2010). The cognitive and affective alexithymia dimensions in the regulation of sympathetic responses. *International Journal of Psychophysiology*, *75*(3), 227–233. doi:10.1016/j.ijpsycho.2009.11.004

Bermond, B., Clayton, K., Liberova, A., Luminet, O., Maruszewski, T., Ricci Bitti, P. E., & Wicherts, J. (2007). A cognitive and an affective dimension of alexithymia in six languages and seven populations. *Cognition and Emotion*, *21*, 1125–1136.

Bermond, B., Oosterveld, P., Vorst, H. (2015). Measures of Alexithymia. *Measures of Personality and Social Psychological Constructs*, 227-256. doi:10.1016?B978-0-12-386915-9.00009-7.

Bernhardt, B. C., & Singer, T. (2012). The neural basis of empathy. *Annual Review of Neuroscience*, *35*, 1–23.

Bernhardt, B. C., Valk, S. L., Silani, G., Bird, G., Frith, U., & Singer, T. (2013). Selective Disruption of sociocognitive structural brain networks in autism and alexithymia. *Cerebral Cortex, 24*, 3258–3267.

Berthoz, S., Ouhayoun, B., Perez-Diaz, F., Consoli, S., & Jouvent, R. (2000). Comparison of the

psychometric properties of two self-report questionnaires measuring Alexithymia; Confirmatory factor analysis of the 20-item Toronto Alexithymia Scale and the Bermond-Vorst Alexithymia Questionnaire. *European Review of Applied Psychology*, *50*(4), 359–368. doi:1035400009766990.0020

Berthoz, S., Wessa, M., Kedia, G., Wicker, B., & Grezes, J. (2008). Cross-cultural validation of the empathy quotient in a French-speaking sample. *The Canadian Journal of Psychiatry*, *53*(7), 469-477.

Bird, G., & Viding, E. (2014). The self to other model of empathy: Providing a new framework for understanding empathy impairments in psychopathy, autism, and alexithymia. *Neuroscience and Biobehavioral Reviews*, *47*, 520–532.

Blair, S. N., Kohl, H. W., & Paffenbarger, R. S. (1989). Physical fitness and all-cause mortality: a prospective study of healthy men and women. *JAMA*, *262*(17), 2395–2401.

Blaydon, M. J., & Lindner, K. J. (2002). Eating Disorders and Exercise Dependence in Triathletes. *Eating Disorders*, *10*(1), 49–60. doi:10.1080/106402602753573559

Blaydon, M. J., Lindner, K. J., & Kerr, J. H. (2004). Metamotivational characteristics of exercise dependence and eating disorders in highly active amateur sport participants. *Personality and Individual Differences*, *36*(6), 1419–1432.

Boden, J. M. & Fergusson, D. M. (2011). Alcohol and depression. *Addiction, 106,* 906-914. doi:10.1111/j.1360-0443.2010.03351

Bogdanov, V., Bogdanova, O., Gorlov, D., Gorgo, Y. D., Joris, M., Mykola, S., Jean, C., Hugo, C. (2013). Alexithymia and Empathy Predict Changes in Autonomic Arousal During Affective Stimulation. *Cognitive & Behavioral Neurology. 26(3), 121-132.*

Bossard, J., & Miller, W. C. (2009). Alexithymia Is Related To Disordered Eating, Exercise

Dependence, And Depression In Young Adults: 2371: Board #16 May 29 9:00 AM - 10:30 AM. *Medicine & Science in Sports & Exercise*, *41*(5). Retrieved from http://journals.lww.com/acsmmsse/Fulltext/2009/05001/Alexithymia_Is_Related_To_Disordered_Eating,.2571.aspx

Brewer, R., Cook, R., Cardi, V., Treasure, J., Catmur, C., & Bird, G. (2018). Alexithymia explains increased personal distress in individuals with and without eating disorders. *Quarterly Journal of Experimental Psychology.* doi:10.1177/1747021818816051

Canadian Society for Exercise Physiology. (2011). *Canadian Physical Activity Guidelines.* Retrieved from http://csep.ca/CMFiles/Guidelines/CSEP_PAGuidelines_0-65plus_en.pdf

Carnes, P. (1983). Out of the Shadows: Understanding Sexual Addiction, CompCare, New York, NY.

Cazenave, N., Le Scanff, C., & Woodman, T. (2007). Psychological profiles and emotional regulation characteristics of women engaged in risk-taking sports. *Anxiety, Stress, and Coping*, *20*, 421–435. doi:10.1080/10615800701330176

Chamberlain, S. R., Stochl, J., Redden, S. A., Odlaug, B. L., & Grant, J. E. (2017). Latent class analysis of gambling subtypes and impulsive/compulsive associations: Time to rethink diagnostic boundaries for gambling disorder? *Addictive Behaviors*, *72*, 79–85. doi:10.1016/j.addbeh.2017.03.020

Chaperon, F. & Thiebot, M. H. (1999). Behavioral effects of cannabinoid agents in animals. *Critical Review of Neurobiology*, *13*, 243–81.

Cheetham, M. (2009). Virtual milgram: Empathic concern or personal distress? Evidence from functional MRI and dispositional measures. *Frontiers in Human Neuroscience*, *3*, 29.

Chrousos, G.P. (1997). Stressors, stress, and neuroendocrine integration of the adaptive response.

Stress of Life: From Molecules to Man. New York, New York Academy of Sciences, 311-335.

Clark, M. R. (2005). Negotiating the Freshman Year: Challenges and Strategies Among First-Year College Students. *Journal of College Student Development, 46*(3), 296-316.

Collins, L., & Lanza, S. (2010). Latent Class and Latent Transition Analysis with Applications in the Social, Behavioral, and Health Sciences. *Wiley Series in Probability and Statistics*.

Cook, B. J., & Hausenblas, H. A. (2008). The role of exercise dependence for the relationship between exercise behavior and eating pathology: Mediator or moderator? *Journal of Health Psycholo*gy, *13*(4), 495–502. doi:10.1177/1359105308088520

Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to Regulate Positive and Negative Emotions: A Motivational Model of Alcohol Use. *Journal of Personality and Social Psychology, 69*(5), 990-1005.

Connelly, M., & Denney, D. R. (2007). Regulation of emotions during experimental stress in alexithymia. *Journal of Psychosomatic Research*, *62*, 649-656.

Cox, W. M., & Klinger, E. (1988). A Motivational Model of Alcohol Use. *Journal of Abnormal Psychology*, *97*(2), 168-180.

Cousineau, D., Ferguson, R. J., de Champlain, J., Gauthier, P., Cote, P., & Bourassa, M. (1977). Catecholamines in coronary sinus during exercise in man before and after training. *Journal of Applied Physiology*, *43*(5), 801–806. doi:10.1152/jappl.1977.43.5.801

Cunningham-Williams, R., & Hong, S. L. (2007). A Latent Class Analysis (LCA) of Problem Gambling Among a Sample of Community-Recruited Gamblers. *The Journal of Nervous and Mental Disease, 195*(11), 939-947.

Cusi, A. M., MacQueen, G. M., Spreng, R. N., & McKinnon, M. C. (2011). Altered empathic

responding in major depressive disorder: relation to symptom severity, illness burden, and psychosocial outcome. *Psychiatry Research, 188*(2), 231-236.

da Silva Lobo, D. S., Vallada, H. P., Knight, J., Martins, S. S., Tavares, H., Gentil, V., et al. (2007). Dopamine genes and pathological gambling in discordant sib-pairs. *Journal of Gambling Studies*, *23*(4), 421–433. doi:10.1007/s10899-007-9060-x.

Da Silva, M. A., Singh-Manoux, A., Brunner, E. J., Kaffashian, S., Shipley, M. J., Kivimäki, M. & Nabi, H. (2012). Bidirectional association between physical activity and symptoms of anxiety and depression: The Whitehall II study. *European Journal of Epidemiology*, *27*, 537–546.

de la Vega, R., Parastatidou, I. S., Ruiz-Barquin, R., & Szabo, A. (2016). Exercise addiction in athletes and leisure exercisers: The moderating role of passion. *Journal of Behavioral Addictions*, *5*(2), 325.

DeSimone, J. A., Harms, P. D., & DeSimone, A. J. (2015). Best practice recommendations for data screening. *Management Department Faculty Publications*. 124.

Davis, M. H. (1980). A multidimensional approach to individual differences in empathy. *JSAS Catalog of Selected Documents in Psychology*, *10*, 85.

Davis, M. H. (1983). Measuring individual differences in empathy: evidence for a multidimensional approach. *Journal of Personality and Social Psychology, 44*(1), 113-126.

Davis, M. H. (1996). *Empathy: A social psychological approach.* Boulder, CO: Westview Press.

Decety, J. (2011). Dissecting the neural mechanisms mediating empathy. *Emotion Review*, *3*, 92–108. doi:10.1177/ 1754073910374662

Decety, J., Moriguchi, Y. (2007). The empathic brain and its dysfunction in psychiatric populations: implications for intervention across different clinical conditions. *BioPsychoSocial Medicine*. *1*(1), 22. doi:10.1186/1751-0759-1-22

Diaz, J. (1997). How drugs influence behavior: a neurobehavioral approach. Upper Saddle River, NJ: Prentice Hall.

Dietrich, A., & McDaniel, W. F. (2004). Endocannabinoids and exercise. *British Journal of Sports Medicine*, *38*, 536-541.

Donges, U. & Suslow, T. (2017). Alexithymia and automatic processing of emotional stimuli: a systematic review. *Reviews in the Neurosciences*, *28*(3), 247-264. https://doi.org/10.1515/revneuro-2016-004

Eastabrook, J. M., Lanteigne, D. M., & Hollenstein, T. (2013). Decoupling between physiological, self-reported, and expressed emotional responses in alexithymia. *Personality and Individual Differences*, *55*(8), 978–982. doi:10.1016/j.paid.2013.08.001

Eisenberg, N., Fabes, R. A., Murphy, B., Karbon, M., Maszk, P., Smith, M. (1994). The relations of emotionality and regulation to dispositional and situational empathy-related responding. *Journal of Personality and Social Psychology*, *66*(4), 776–797.

Eres, R., Decety, J., Louis, W. R., & Molenberghs, P. (2015). Individual differences in local gray matter density are associated with differences in affective and cognitive empathy. *NeuroImage*, *117*, 305–310.

Ernst, J., Böker, H., Hättenschwiler, J., et al. (2014). The association of interoceptive awareness and alexithymia with neurotransmitter concentrations in insula and anterior cingulate. *Social Cognitive and Affective Neuroscience*, *9*, 857–863. doi:10.1093/scan/nst058

Feldmanhall, O., Dalgleish, T., Mobbs, D. (2013). Alexithymia decreases altruism in real social

decisions. *Cortex, 49*(3), 899-904. doi:10.1016/j.cortex.2012.10.015

Ferrari, V., Smeraldi, E., Bottero, G., & Politi, E. (2014). Addiction and empathy: a preliminary analysis. *Neurological Sciences, 35*(6), 855–859. doi:10.1007/s10072-013-1611-6

Ferris J., & Wynne H.J. (2001). *The Canadian Problem Gambling Index Final Report*. Ottawa, ON: Canadian Centre on Substance Abuse.

Fier, S. M., & Brzezinski, L. G. (2010). Facilitating the high school to college transition for students with psychiatric disabilities: Information and strategies for school counselors. *Journal of School Counseling, 8*(10), 280–287.

Fuss, J., Steinle, J., Bindila, L., Auer, M. K., Kirchherr, H., Lutz, B., & Gass, P. (2015). A runner's high depends on cannabinoid receptors in mice. *Proceedings of the National Academy of Sciences, 14*, 996. doi:10.1073/pnas.1514996112

Gaigg, S. B., Cornell, A. S. F., & Bird, G. (2016). The psychophysiological mechanisms of alexithymia in autism spectrum disorder. *Autism*, *22*(2), 227–231. doi:10.1177/1362361316667062

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*(2), 15-34. doi:10.1002/1098-108X(198321)2:2<15::AID-EAT2260020203>3.0.CO;2-6

Gideon, N., Hawkes, N., Mond, J., Saunders, R., Tchanturia, K., & Serpell, L. (2016). Development and Psychometric Validation of the EDE-QS, a 12 Item Short Form of the Eating Disorder Examination Questionnaire (EDE-Q). *PLOS ONE, 11*(5).

Glasser, W. (1976). Positive Addictions, Harper & Row, New York, NY.

Glass, M., Dragunow, M., & Faull, R. L. (1997). Cannabinoid receptors in the human brain: a

detailed anatomical and quantitative autoradiographic study in the fetal, neonatal and adult human brain. *Neuroscience, 77*, 299-318. doi:10.1016/S0306-4522(96)00428-9

Glass, J. M., Lyden, A. K., Petzke, F., Stein, P., Whalen, G., Ambrose, K., et al. (2018). The effect of brief exercise cessation on pain, fatigue, and mood symptom development in healthy, fit individuals. *Journal of Psychosomatic Research*, *57*(4), 391–398. doi:10.1016/j.jpsychores.2004.04.002

Goerlich-Dobre, K. S., Lamm, C., Pripfl, J., Habel, U., & Votinov, M. (2015). The left amygdala: A shared substrate of alexithymia and empathy. *NeuroImage*. *122*, 20–32. doi:10.1016

Goerlich-Dobre, K. S., Votinov, M., Habel, U., Pripfl, J. and Lamm, C. (2015), Neuroanatomical profiles of alexithymia dimensions and subtypes. *Human Brain Mapping*, *36*, 3805–3818. doi:10.1002/hbm.22879

Goodman, A. (1990). Addiction: definition and implications. *British Journal of Addiction*, *85*(11), 1403–1408.

Goodman, L. A. (1974) Exploratory latent structure analysis using both identifiable and unidentifiable models. *Biometrika*, *6*, 215-231.

Griffiths, M. (1996). Behavioural addiction: an issue for everybody? *Employee Counselling Today*, *8*(3), 19–25. doi:10.1108/13665629610116872

Griffiths, M. (1997). Exercise Addiction: A Case Study. *Addiction Research*, *5*(2), 161–168. doi:10.3109/16066359709005257

Griffiths, M., Szabo, A. & Terry, A. (2005). The exercise addiction inventory: a quick and easy screening tool for health practitioners. *British Journal of Sports Medicine, 39*(30).

Gross, J. J. (1999). Emotion regulation: Past, present, future. *Cognition and Emotion, 13*, 551-

573.

Guttman, H., & Laporte, L. (2002). Alexithymia, empathy, and psychological symptoms in a family context. *Comprehensive Psychiatry*, *43*, 448–455.

Haase, L., May, A. C., Falahpour. M., et al. (2015). A pilot study investigating changes in neural processing after mindfulness training in elite athletes. *Frontiers in Behavioural Neuroscience*, *9*.

Hagan, A. L. & Hausenblas, H. A. (2003). The relationship between exercise dependence symptoms and perfectionism. *American Journal of Health Studies*, *18*(2/3), 133-137.

Hamer, M., & Karageorghis, C. I. (2007). Psychobiological mechanisms of exercise dependence. *Sports Medicine, 37*(6), 477–484.

Hausenblas, H. A., & Downs, D. S. (2002). Exercise dependence: a systematic review. *Psychology of Sport and Exercise*, *3*(2), 89–123. doi:10.1016/S1469-0292(00)00015-7

Hausenblas, H. A., & Downs, D. S. (2002). How Much is Too Much? The Development and Validation of the Exercise Dependence Scale. *Psychology & Health*, 17(4), 387–404. doi:10.1080/0887044022000004894

Hausenblas, H. A., Schreiber, K., & Smoliga, J. M. (2017). Addiction to exercise. *BMJ, 357*. doi:10.1136/bmj.j1745

Heyman, E., Gamelin, F. X., Goekint, M., Piscitelli, F., Roelands, B., Leclair, E., Di Marzo, V. & Meeusen, R. (2012). Intense exercise increases circulating endocannabinoid and BDNF levels in humans possible implications for reward and depression. *Psychoneuroendocrinology*, *37*(6), 844-851. doi:10.1016/j.psyneuen.2011.09.017

Hock, M. F., Deshler, D. D., Schumaker, J. B. (2006). Enhancing student motivation through the pursuit of possible selves. C. Dunkel, J. Kerpelman (Eds.), *Possible selves: Theory,*

research and applications, Nova, Hauppauge, NY, pp. 205-221.

Hoffman, M. L. (2000). Empathy and Moral Development: Implications for Caring and Justice. Cambridge University Press.

Huang, J. L., Curran, P. G., Keeney, J., Poposki, E. M., & DeShon, R. P. (2012). Detecting and deterring insufficient effort responding to surveys. *Journal of Business Psychology*, *27*, 99–114.

Iacolino, C., Pellerone, M., Formica, I., Lombardo, E. M. C., & Tolini, G. (2017). Alexithymia, Body Perception and Dysmorphism: A Study Conducted on Sportive and Non-Sportive Subjects. *Clinical Neuropsychiatry*, *14*(6), 400-406.

Ibrahim, A. K., Kelly, S. J., Adams, C. E., & Glazebrook, C. (2013). A systematic review of studies of depression prevalence in university students. *Journal of Psychiatric Research*, *47*(3), 391–400. doi:10.1016/j.jpsychires.2012.11.015

Jodat, H., Ghasempour, A., & Tavakoli, A. (2015). Comparison of Alexithymia in Athlete and Non-Athlete Students. *Annals of Applied Sport Science*, *3*(3), 17-24.

Kriska A. (2000). Physical activity and the prevention of type 2 diabetes mellitus: how much for how long*? Sports Medicine, 29*(3), 147–151.

Kroenke, K., Spitzer, R., & Williams, W. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*, 606-616.

Lai, H. M. X., Cleary, M., Sitharthan, T., & Hunt, G. E. (2015). Prevalence of comorbid substance use, anxiety and mood disorders in epidemiological surveys, 1990–2014: A systematic review and meta-analysis. *Drug and Alcohol Dependence*, *154*, 1–13. doi:10.1016/j.drugalcdep.2015.05.031

Lamm, C., Decety, J., Singer, T. (2011). Meta-analytic evidence for common and distinct neural

networks associated with directly experienced pain and empathy for pain. *Neuroimage*, *54*, 2492-2502.

Lamm, C., & Majdandžić, J. (2015). The role of shared neural activations, mirror neurons, and morality in empathy – A critical comment. *Neuroscience Research*, *90*, 15–24. doi:10.1016/j.neures.2014.10.008

Lamm, C., Rütgen, M., & Wagner, I. C. (2017). Imaging empathy and prosocial emotions. *Neuroscience Letters*. Advance online publication. doi:10.1016

Landolfi, E. (2013). Exercise Addiction. *Sports Medicine*, *43*(2), 111–119. doi:10.1007/s40279-012-0013-x

Lejoyeux, M., Avril, M., Richoux, C., Embouazza, H., & Nivoli, F. (2008). Prevalence of exercise dependence and other behavioral addictions among clients of a Parisian fitness room. *Comprehensive Psychiatry*, *49*, 353-358, doi:10.1016/j.comppsych.2007.12.005

Levant, R. F., Halter, M. J., Hayden, E. W., & Williams, C. M. (2009). The efficacy of alexithymia reduction treatment: a pilot study. *The Journal of Mens Studies*, *17*, 75-84.

Leweke, F., Bausch, S., Leichsenring, F., Walter, B., & Stingl, M. (2009). Alexithymia as a predictor of outcome of psychodynamically oriented inpatient treatment. *Psychotherapy Research*, *19*(3), 323-331. doi: 10.1080/10503300902870554

Lichtenstein, M. B., Christiansen, E., Elklit, A., Bilenberg, N., & Støving, R. K. (2014). Exercise addiction: A study of eating disorder symptoms, quality of life, personality traits and attachment styles. *Psychiatry Research*, *215*(2), 410–416. doi:10.1016/j.psychres.2013.11.010

Lichtenstein, M. B., Nielsen, R. O., Gudex, C., Hinze, C. J., & Jørgensen, U. (2018). Exercise addiction is associated with emotional distress in injured and non-injured regular

exercisers. *Addictive Behaviors Reports*, *8*, 33–39. doi:10.1016/j.abrep.2018.06.001

Linden, W., Lenz, J.W., Stossel, C. (1996). Alexithymia, defensiveness and cardiovascular reactivity to stress. *Journal of Psychosomatic Research*, *41*, 575-583.

Lo, Y., Mendell, N. R., & Rubin, D. B. (2001). Testing the number of components in a normal Mixture. *Biometrika*, *88*, 767–778.

Lyvers, M., Duric, N., & Thorberg, F. A. (2014). Caffeine Use and Alexithymia in University Students. *Journal of Psychoactive Drugs*, *46*(4), 340-346. https://doi.org/10.1080/02791072.2014.942043

Magee, C. A., Buchanan, I., & Barrie, L. (2016). Profiles of exercise dependence symptoms in Ironman participants. *Psychology of Sport and Exercise*, *24*, 48–55. doi:10.1016/j.psychsport.2016.01.005

Maniaci, M., & Rogge, R. (2014). Caring about carelessness: Participant inattention and its effects on research. *Journal of Research in Personality*, *48*, 61-83.

Marian, N., & Willard, O. (2014). Dependence. *Drugs in American Society: an Encyclopedia of History, Politics, Culture, and the Law*, *3*, 248.

Marjanovic, Z., Struthers, C. W., Cribbie, R., & Greenglass, E. R. (2014). The conscientious responders scale: A new tool for discriminating between conscientious and random responders. *SAGE Open*, 1-10. doi:10.1177/2158244014545964

Martin, J. B., & Pihl, R. O. (1985). The Stress-Alexithymia Hypothesis: Theoretical and empirical considerations. *Psychotherapy and Psychosomatics*, *43*, 169-176.

Maurage, P., Grynberg, D., Noel, X., Joassin, F., Philippot, P., Hanak, C., Verbanck, P., Luminet, O., De Timary, P., & Campanella, S. (2011). Dissociation between affective and cognitive empathy in alcoholism: a specific deficit for the emotional dimension.

Alcoholism: Clinical and Experimental Research, 35(9), 1662–1668.

Meade, A., & Craig, B. (2012). Identifying Careless Responses in Survey Data. *Psychological Methods. 17*(3), 437-455.

Mónok, K., Berczik, K., Urbán, R., Szabo, A., Griffiths, M. D., Farkas, J., et al. (2012). Psychometric properties and concurrent validity of two exercise addiction measures: A population wide study. *Psychology of Sport and Exercise*, *13*(6), 739–746. doi:10.1016/j.psychsport.2012.06.003

Moormann, P., Bermond, B., & Albach, F. (2004). The Reality Escape Model: the intricate relationship between alexithymia, dissociation, and anesthesia in victims of child sexual abuse. In I. Nyklihek, A. J. J. M. Vingerhoets, & L. Temoshok (Eds.), *Emotional expression in health*, 82-89. Hove, UK: Brunner Routledge.

Moormann, P., Bermond, B., Vorst, H. M., Bloemendaal, A. T., Teijn, S., & Rood, L. (2008). New Avenues in Alexithymia Research: The Creation of Alexithymia Types. *Emotion Regulation*. 27–42.

Morie, K. P., Yip, S. W., Nich, C., Hunkele, K., Carroll, K., & Potenza, M. N. (2017). Alexithymia and addiction: A review and preliminary data suggesting neurobiological links to reward/loss processing. *Drug and Alcohol Dependence, 171*, 149–150. doi:10.1016/j.drugalcdep.2016.08.413

Moriguchi, Y., Decety, J., Ohnishi, T., Maeda, M., Mori, T., Nemoto, K., et al. (2007). Empathy and judging other's pain: An fMRI study of alexithymia. *Cerebral Cortex*, *17*, 2223–2234.

Moriguchi, Y., Ohnishi, T., Lane, R.D., Maeda, M., Mori, T., Nemoto, K. (2006). Impaired self-awareness and theory of mind: An fMRI study of mentalizing in alexithymia.

Neuroimage. *32*(3), 1472–82.

Muthén, L .K., & Muthén, B. O. (1998-2010). Mplus User's Guide, version 6. Muthén & Muthén, Los Angeles.

Noël, X., Saeremans, M., Kornreich, C., Bechara, A., Jaafari, N., & Fantini-Hauwel, C. (2017). On the Processes Underlying the Relationship Between Alexithymia and Gambling Severity. *Journal of Gambling Studies*. doi:10.1007/s10899-017-9715-1

Norman, H., Marzano, L., Coulson, M., et al. (2019). Effects of mindfulness-based interventions on alexithymia: a systematic review. *Evidence-Based Mental Health*, *22*, 36-43.

Nylund, K. L., Asparouhov, T., & Muthén, B.O. (2007). Deciding on the number of classes in latent class analysis and growth mixture modeling: a Monte Carlo simulation study. *Structural Equation Modeling, 14*, 535–569.

Ogrodniczuk, J. S., Sochting, I., Piper, W. E., & Joyce, A. S. (2012). A naturalistic study of alexithymia among psychiatric outpatients treated in an integrated group therapy program. *Journal of Psychology and Psychotherapy*, *85*(3), 278-291. doi:10.1111/j.2044-8341.2011.02032.x

Panaite, V. & Bylsma, L. M. (2012). Alexithymia. *Encyclopedia of Human Behavior*, 92–99. doi:10.1016/B978-0-12-375000-6.00018-5

Panayiotou, G., & Constantinou, E. (2017). Emotion dysregulation in alexithymia: Startle reactivity to fearful affective imagery and its relation to heart rate variability. *Psychophysiology*, *54*(9), 1323–1334. doi:10.1111/psyp.12887

Paradis, K. F., Cooke, L. M., Martin, L. J., & Hall, C. R. (2013). Too much of a good thing? Examining the relationship between passion for exercise and exercise dependence. *Psychology of Sport and Exercise*, *14*(4), 493-500.

http://dx.doi.org/10.1016/j.psychsport.2013.02.003

Patil, I., & Silani, G. (2014). Reduced empathic concern leads to utilitarian moral judgments in trait alexithymia. *Frontiers in Psychology*, *5*, 501.

Pennebaker, J. W., Colder, M., Sharp, L. K. (1990). Accelerating the coping process. *Journal of Personality and Social Psychology, 58*, 528-537.

Pennebaker, J. W., Hughes, C. F., O'Heeron, B. C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. *Journal of Personality and Social Psychology*, *52*, 781-793.

Pierce, E. F., Eastman, N. W., Tripathi, H. L., et al. (1993). β-Endorphin response to endurance exercise: relationship to exercise dependence. *Perceptual and Motor Skills*, *77*, 767–70.

Pollatos, O., Werner, N. S., Duschek, S., Schandry, R., Matthias, E., Traut-Mattausch, E. et al. (2011). Differential effects of alexithymia subscales on autonomic reactivity and anxiety during social stress. *Journal of Psychosomatic Research*, *70*, 525-533. doi:10.1016/j.jpsychores.2010.12.003

Powell, J. (1986). Excessive appetites: a psychological view of addictions. *Behaviour Research and Therapy*, *24*(5), 608. doi:10.1016/0005-7967(86)90048-3

Preece, Becerra, Allan, Robinson, & Dandy. (2017). Establishing the theoretical components of alexithymia via factor analysis: Introduction and validation of the attention-appraisal model of alexithymia. *Personality and Individual Differences, 119*, 341-352.

Preston, S. D., & de Waal, F. B. (2002). Empathy: its ultimate and proximate bases. *Behavioral and Brain Sciences*, *25*, 1–20.

Pridgeon, L., & Grogan, S. (2012). Understanding exercise adherence and dropout: an interpretative phenomenological analysis of men and women's accounts of gym

attendance and non-attendance. *Qualitative Research in Sport, Exercise and Health*, *4*(3), 382–399. doi:10.1080/2159676X.2012.712984

Ramaswamy, V., DeSarbo, W., Reibstein, D., & Robinson, W. (1993). An empirical pooling approach for estimating marketing mix elasticities with PIMS data. *Marketing Science, 12*, 103–124.

Richardson, J. D. (2000). Cannabinoids modulate pain by multiple mechanisms of action. *The Journal of Pain*, *1*, 2–14.

Rizeanu, S. (2013). Pathological Gambling and Depression. *Procedia - Social and Behavioral Sciences*, *78*, 501–505. doi:10.1016/j.sbspro.2013.04.339

Rosa, D. A., Mello, M. T. de, Negrão, A. B., & Souza-Formigoni, M. L. O. (2004). Mood Changes after Maximal Exercise Testing in Subjects with Symptoms of Exercise Dependence. *Perceptual and Motor Skills*, *99*(1), 341–353. doi:10.2466/pms.99.1.341-353

Salminen, J. K., Saarijarvi, S., Aarela, E., Toikka, T., Kauhanen, J. (1999): Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *Journal of Psychosomatic Researcb*, *46*(75).

Schwarz, G. E. (1978). Estimating the dimension of a model. *Annals of Statistics*, *6*(2), 461–464.

Sforzo, G. A. (1988). Opioids and exercise: An update. *Sports Medicine, 7*, 109–124.

Sforzo, G. A., Seeger, T. F., Pert, C. B., Pert, A., & Dotson, C. O. (1986). In vivo opioid receptor occupation in the rat brain following exercise. *Medicine and Science in Sports and Exercise, 18*(4), 380–384.

Shiu-Yee, K., Brincks, A. M., Feaster, D. J., Frimpong, J. A., Nijhawan, A., Mandler, R. N., et al. (2018). Patterns of Substance Use and Arrest Histories Among Hospitalized HIV Drug

Users: A Latent Class Analysis. *AIDS and Behavior*. doi:10.1007/s10461-017-2024-y

Silani, G., Bird, G., Brindley, R., Singer, T., Frith, C., & Frith, U. (2008). Levels of emotional awareness and autism: An fMRI study. *Social Neuroscience, 3*, 97–112.

Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences*, *13*, 334–340.

Slay, H. A, Hayaki, J., Napolitano, M. A., & Brownell, K. D. (1998). Motivations for running and eating attitudes in obligatory versus nonobligatory runners. *International Journal of Eating Disorders, 357*, 267-275.

Smith, A. (2006). Cognitive empathy and emotional empathy in human behavior and evolution. *The Psychological Record*, *56*, 3–21.

Smith, A. (2009). The empathy imbalance hypothesis of autism: a theoretical approach to cognitive and emotional empathy in autistic development. *The Psychological Record, 59*, 489–510.

Smith, M. A., & Yancey, D. L. (2003). Sensitivity to the effects of opioids in rats with free access to exercise wheels: μ-opioid tolerance and physical dependence. *Psychopharmacology*, *168*(4), 426–434. doi:10.1007/s00213-003-1471-5

Spano, L. (2001). The relationship between exercise and anxiety, obsessive-compulsiveness, and narcissism. *Personality and Individual Differences*, *30*(1), 87-93.

Sparling, P. B., Giuffrida, A., Piomelli, D., Rosskopf, L. & Dietrich, A. (2003). Exercise activates the endocannabinoid system. *Neuroreports*, *14*, 2209-2211. doi:10.1097/01.wnr.0000097048.56589.47

Stone, L. A., & Nielson, K. A. (2001). Intact Physiological Response to Arousal with Impaired Emotional Recognition in Alexithymia. *Psychotherapy and Psychosomatics*, *70*(2), 92–

102.

Strachan, S. M., Marcotte, M. M. E., Giller, T. M. T., Brunet, J., & Schellenberg, B. J. I. (2017). An online intervention to increase physical activity: Self-regulatory possible selves and the moderating role of task self-efficacy. *Psychology of Sport and Exercise*, *31*, 158–165. doi:10.1016/j.psychsport.2016.05.001

Szabo, A. (1995). The impact of exercise deprivation on well- being of habitual exercisers. *Australian Journal of Science and Medicine in Sport*, *27*, 68–75.

Szabo, A., & Griffiths, M. D. (2007). Exercise Addiction in British Sport Science Students. *International Journal of Mental Health and Addiction*, *5*(1), 25–28. doi.org:10.1007/s11469-006-9050-8

Szabo, A. (2010). *Addiction to exercise: A symptom or a disorder?* New York, NY: Nova Science Publishers.

Szabo, A., Griffiths, M. D., de la Vega, R., Mervo´, B., & Demetrovics, Z. (2015). Methodological and conceptual limitations in exercise addiction research. *Yale Journal of Biology and Medicine*, *88*, 303–308.

Taylor, G. J., Bagby, R. M., & Parker, J. D. (1991). The alexithymia construct. A potential paradigm for psychosomatic medicine. *Psychosomatics*, *32*(2), 153–164.

Taylor, G. J., Bagby, R. M., & Parker, J. D. (1999). Disorders of affect regulation: Alexithymia in medical and psychiatric illness. Cambridge University Press.

Taylor, G. J., Parker, J. D., & Bagby, R. M. (1990). A preliminary investigation of alexithymia in men with psychoactive substance dependence. *American Journal of Psychiatry*, *147*, 1228-1230.

Thompson, J. K., & Blanton, P. (1987). Energy conservation and exercise dependence: a

sympathetic arousal hypothesis. *Medicine and Science in Sports and Exercise*, *19*, 767-770.

Tomei, A., Besson, J., & Grivel, J. (2017). Linking empathy to visuospatial perspective-taking in gambling addiction. *Psychiatry Research*, *250*, 177–184. doi:10.1016/j.psychres.2016.12.061

Tulipani, C., Morelli, F., Spedicato, M. R., Maiello, E., Todarello, O., & Porcelli, P. (2010). Alexithymia and cancer pain: the effect of psychological intervention. *Psychotherapy and Psychosomatics*, *79*, 156-63.

van der Meer, L., van't Wout, M., Aleman, A. (2009): Emotion regulation strategies in patients with schizophrenia. *Psychiatry Res*, 170, 108–113.

van't Wout, M., Aleman, A., Bermond, B., & Kahn, R. S. (2007). No words for feelings: alexithymia in schizophrenia patients and first-degree relatives. *Comprehensive Psychiatry*, *48*(1), 27–33. doi:10.1016/J.COMPPSYCH.2006.07.003

Villella, C., Martinotti, G., Di Nicola, M., Cassano, M., La Torre, G., et al. (2011). Behavioural Addictions in Adolescents and Young Adults: Results from a Prevalence Study. *Journal of Gambling Studies*, *27*, 203-214.

Vorst H. & Bermond B. (2001) Validity and reliability of the Bermond–Vorst Alexithymia Questionnaire. *Personality and Individual Differences*, *30*, 413–434. doi:10.1016/S0191-8869(00)00033-7

Weinstein, A., Maayan, G., & Weinstein, Y. (2015). A study on the relationship between compulsive exercise, depression and anxiety. *Journal of Behavioral Addictions*, *4*(4), 315–318. doi:10.1556/2006.4.2015.034

Wilbertz, G., Brakemeier, E. L., Zobel, I., Härter, M., & Schramm, E. (2010). Exploring

preoperational features in chronic depression. *Journal of Affective Disorders*, *124*(3), 262-269.

Wurpts, I. C., & Geiser, C. (2014). Is adding more indicators to a latent class analysis beneficial or detrimental? Results of a Monte-Carlo study. *Frontiers in Psychology*, *5*, 920. doi:10.3389/fpsyg.2014.00920

Zautra, A., Smith, B., Affleck, G., Tennen, H. (2001). Examinations of chronic pain and affect relationships: applications of a dynamic model of affect. *Journal of Consulting and Clinical Psychology*, *69*, 786 – 795.