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Research paper

Physical activity and analogue anxiety disorder symptoms and status: Mediating influence of social physique anxiety

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ABSTRACT

Background: Associations between physical activity (PA) and subclinical anxiety disorder symptoms and status, and potential mediating effect of social physique anxiety (SPA), remain understudied. We examined associations between PA and analogue Generalized Anxiety Disorder (AGAD), analogue Social Anxiety Disorder (ASAD), and analogue Panic Disorder (APD) symptoms and status, the mediating effect of SPA, and sex-related differences.
Methods: Participants (n = 470, 23.2 ± 4.8 years, 298 female) completed the Psychiatric Diagnostic Screening Questionnaire, seven-day PA recall, and Social Physique Anxiety Scale. ANCOVA examined differences in SPA and anxiety disorder symptoms between PA levels. Logistic regression examined associations between PA and analogue anxiety disorder status. Mediation analyses estimated the effect of change in PA on analogue anxiety disorder odds when SPA was at its mean.
Results: AGAD, ASAD, and APD prevalence was 38.1%, 60.0%, and 15.1%, respectively. AGAD and ASAD symptoms, but not APD symptoms, were significantly lower among higher PA levels. PA was associated with lower odds of AGAD, ASAD, and APD; findings were not significant after adjustment. The pure indirect effect of SPA significantly accounted for 58.2% and 47.9% of the total effect of PA on AGAD and ASAD, respectively; findings were not significant after adjusting for depression. Females showed greater odds of AGAD, ASAD, and APD; mediation findings did not differ based on gender.
Limitations: Cross-sectional design, self-reported exposure and outcomes, and sample size are potential limitations.
Conclusion: Symptoms and odds of AGAD and ASAD status were lower among young adults with greater PA; SPA partially mediated associations and warrants experimental investigation.

1. Introduction

Anxiety disorders are prevalent, debilitating, and costly public health problems, affecting an estimated 264 million people and approximately twice as many females than males (WHO, 2017). Anxiety disorders are the sixth leading contributor to global disability (Baxter et al., 2013, 2014; Gustavsson et al., 2011; Remes et al., 2016), are frequently comorbid with other physical and mental illnesses (Vancampfort et al., 2017), and treatment success is limited (Boschen et al., 2009; Carpenter et al., 2018; De Vries et al., 2016).

Regular physical activity (PA) is a potentially low-cost, low-risk, alternative or augmentation therapy for anxiety disorders. Robust evidence has supported that regular PA and exercise have potential protective and therapeutic benefits for anxiety symptoms and disorders (Gordon et al., 2017; Herring et al., 2012; Herring et al., 2014a; Herring et al., 2010; McDowell et al., 2019). Despite this, those with elevated anxiety tend to be less physically active, with females consistently reporting lower PA levels than males (Hallgren et al., 2019; McDowell

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et al., 2018). Available evidence for the protective benefits of PA for anxiety disorders has largely focused on Generalized Anxiety Disorder (GAD), with recent meta-analytic evidence showing 45.7% lower odds of developing GAD among physically active older adults (McDowell et al., 2019). However, associations between PA and anxiety disorder symptoms and status remain understudied, particularly among younger adults who are at higher risk for development of anxiety symptoms and disorders (Kessler et al., 2007).

Moreover, there is continued interest in exploration of modifiable factors that may influence PA, anxiety disorders, and their associations to inform intervention development and personalized prescription of PA to improve mental health. Social Physique Anxiety (SPA), anxiety resulting from the prospect or presence of interpersonal evaluation of one's physique, is a construct related to body image and body esteem (Hart et al., 1989). SPA has been inversely associated with PA motivation and behaviour (Baillot et al., 2020; Brunet and Sabiston, 2009), particularly feelings of incompetence in PA contexts (Kowalski et al., 2001) and the social and evaluative nature of PA settings (Sabiston et al., 2014). SPA also has been negatively associated with mental health. For example, 693 patients with mental illness reported higher levels of social anxiety in PA contexts compared to 2888 healthy controls (De Herdt et al., 2013). Even among regularly physically active individuals without a mental illness, SPA has negatively influenced mental health, primarily through increased psychological inflexibility (Alcaraz-Ibáñez et al., 2017). Given associations between SPA and both PA and mental health, it is plausible that SPA accounts for variability in the association between PA and mental health outcomes. However, the potential moderating and/or mediating effect of SPA on the associations between PA and anxiety disorders remains understudied. Mediation analyses require that several assumptions are met, including that the exposure plausibly precedes the outcome, and establishing temporality in cross-sectional mediation designs can be difficult. However, there is strong epidemiological (McDowell et al., 2019) and experimental (Herring et al., 2012) evidence to support that this is the case for the PA—Anxiety Disorder relationship.

Thus, the objectives of this cross-sectional investigation were to: a) quantify the associations between PA and subclinical, or analogue Generalized Anxiety Disorder (AGAD), analogue Social Anxiety Disorder (ASAD), and analogue Panic Disorder (APD) symptoms and status, b) examine SPA as a mediator of these associations, and c) explore potential sex-related differences.

Based on the current literature, we hypothesised that: a) active young adults would report significantly lower AGAD, ASAD, and APD symptoms and lower odds of AGAD, ASAD, and APD statuses; b) SPA would significantly mediate associations between PA and AGAD, ASAD, and APD symptoms and statuses; and, c) associations between PA and AGAD, ASAD, and APD symptoms and status and the mediating influence of SPA on these associations would be stronger among females than males.

2. Methods

2.1. Participants recruitment

This study protocol was approved by the University's Research Ethics Board. Four hundred eighty-six participants were recruited from the surrounding area via posters, emails, and word of mouth to complete an electronically-administered physical activity and mood survey as part of the screening process for an on-going randomized controlled trial. Inclusion criteria included age of 18–40 years, completion of the online battery of questionnaires, and willingness for data to be analysed. Participants were not compensated for participation. Sixteen respondents outside the age range were excluded, resulting in a sample size of 470 for the study reported herein (Gordon et al., 2020).

2.2. Exposure, outcomes, and covariates

2.2.1. Analogue anxiety disorder symptoms and statuses

AGAD, ASAD, and APD symptoms and status were assessed with the 10-item GAD, 15-item SAD, and 8-item PD subscales of the Psychiatric Diagnostic Screening Questionnaire, respectively (Zimmerman and Mattia, 2001b). AGAD, ASAD, and APD status were classified using validated cut-scores of $\geq$6 on the PDSQ-GAD and $\geq$4 for both the PDSQ-SAD and PDSQ-PD subscales (Zimmerman and Mattia, 2001a). The PDSQ-GAD, PDSQ-SAD, and PDSQ-PD have sensitivity/specificity of 95/41%, 91/69%, and 91/63%, respectively. Internal consistency in the current sample was Cronbach's $\alpha = 0.87$ (ICC: 0.87, 95%CI: 0.85, 0.88), Cronbach's $\alpha = 0.88$ (ICC: 0.87, 95%CI: 0.84, 0.89), and Cronbach's $\alpha = 0.84$ (ICC: 0.84, 95%CI: 0.81, 0.86) for PDSQ-GAD, PDSQ-SAD, and PDSQ-PD, respectively.

2.2.2. Physical activity

PA was assessed using a seven-day Physical Activity Recall (Blair et al., 1985). Participants self-reported time engaged in sleep, moderate, hard, and very hard physical activities over the prior seven days. Values of 1, 1.5, 4, 6, and 10 metabolic equivalents (METs; i.e., the ratio of energy expenditure in kilocalories divided by resting energy expenditure in kilocalories) were assigned to sleep, light, moderate, hard, and very hard activities, respectively. Estimated energy expenditure was expressed as kilocalories per week. Because weight was self-reported, and self-reports of weight are often biased, weight was not factored into MET estimates; rather, standard procedures for MET estimates and estimates of overall energy expenditure from the seven-day physical activity recall were used. Based on thresholds validated among university students (Dishman and Steinhardt, 1988), estimated energy expenditure levels of <245 kcal/wk were classified as low-active, 245 kcal/wk to <280 kcal/wk were classified as moderately-active, and $\geq$280 kcal/wk were classified as highly-active.

2.2.3. Social physique anxiety

SPA was measured with the 12-item Social Physique Anxiety Scale (SPAS) (Hart et al., 1989). The SPAS examines the extent to which people become anxious when others evaluate their physiques; it has demonstrated strong psychometric properties, including high internal consistency and content and logical validity (Hart et al., 1989). The SPAS demonstrated strong internal consistency in the current sample (Cronbach's $\alpha = 0.93$; ICC: 0.91, 95%CI: 0.89, 0.93). Approximately 1% of the sample ($n = 5$) were missing SPA data and were excluded from analyses that included SPA.

2.2.4. Symptoms of major depressive disorder

To facilitate sensitivity analyses, symptoms of Major Depressive Disorder (MDD), or depressive symptoms, were quantified with the 21-item PDSQ-MDD subscale (Zimmerman and Mattia, 2001b), which has sensitivity/specificity of 90%/67% and demonstrated adequate internal consistency of Chronbach's $\alpha = 0.83$ (ICC: 0.80, 95%CI: 0.77, 0.83).

2.3. Statistical analyses

Statistical analyses were conducted using SPSS Version 26.0 (Armonk, NY: IBM Corp.) and Stata 14.0 (StataCorp for Windows). ANCOVA adjusted for age and gender, with Bonferroni-corrected *post-hoc* tests, examined differences in SPA and anxiety disorder symptoms between PA levels. The magnitude of differences were quantified using Cohen's d; positive effect sizes represented improved outcomes. Effects of 0.2, 0.5, and 0.8 were appraised as small, moderate, and large, respectively. Logistic regression examined associations between PA level and analogue anxiety disorder status. Crude models and models adjusted for age, gender, and smoking status are reported.

Mediation analyses were performed using the Stata command *med4way* to estimate the effect of change in PA level (from low-active to

highly-active) on odds of AGAD, ASAD, and APD when SPA was at its mean and covariates were adjusted in the model (Discacciati et al., 2019; VanderWeele, 2015). This mediation method has been applied in epidemiological studies where exposure and mediator variables are measured concurrently (Hallgren et al., 2020). The overall effect of the exposure on the outcome, in the presence of the mediator with which the exposure may interact, is decomposed into four components: how much of the effect is due to (1) the controlled direct effect (neither mediation nor interaction), (2) reference interaction (the interaction only), (3) mediated interaction (both mediation and interaction), and (4) the pure indirect effect (mediation only; see Table 1). When statistically significant, the pure indirect effect indicated a potential mediating effect of SPA. Causal effects were estimated for a change in PA from low-active to highly-active and at the mean level of the covariates. The controlled direct effect was computed fixing SPA at its mean. As sensitivity analyses, mediation models were rerun, additionally controlling for symptoms of Major Depressive Disorder.

3. Results

Table 2 presents participant characteristics. Prevalence rates of AGAD, ASAD, and APD in the current sample were 38.1%, 60.0%, and 15.1%, respectively. ANOVA showed that age significantly differed between PA levels ($F_{(2, 469)} = 5.04$, $p \leq 0.007$); highly-active participants were significantly younger than low-active participants (mean difference=−1.81, $p \leq 0.006$; d = 0.38, 95%CI: 0.13, 0.62).

3.1. Sex-Related differences in exposure & outcomes

Independent samples *t*-tests showed that, compared to males, females reported significantly greater SPA ($t_{(463)=}6.61$, $p<0.001$; $d = 0.64$, 95%CI: 0.44, 0.83) and symptoms of GAD ($t_{(468)=}5.32$, $p<0.001$; $d = 0.51$, 95%CI: 0.32, 0.70), SAD ($t_{(468)=}2.69$, $p \leq 0.007$; $d = 0.26$, 95%CI: 0.07, 0.45), and PD ($t_{(429.67)=}3.34$, $p \leq 0.001$; $d = 0.30$, 95%CI: 0.11, 0.49), and significantly lower estimated energy expenditure ($t_{(284.34)=-}3.46$, $p \leq 0.001$; $d = 0.33$, 95%CI: 0.14, 0.52). Logistic regression showed that females had significantly greater odds of AGAD (OR=2.52, 95%CI: 1.67, 3.81, $p<0.001$), ASAD (OR=1.59, 95%CI: 1.09, 2.33, $p<0.02$), and APD (OR=2.67, 95%CI: 1.44, 4.95, $p \leq 0.002$).

3.2. Differences in symptom severity and SPA by PA level

ANCOVA adjusted for age and gender showed that symptoms of GAD ($F_{(2, 465)}=3.03$, $p \leq 0.049$) and SAD ($F_{(2, 465)}=3.06$, $p \leq 0.048$) and SPA ($F_{(2, 460)}=3.90$, $p \leq 0.021$) significantly differed between PA levels. Bonferonni-corrected *post-hoc* tests showed that GAD symptoms were significantly greater among low-active compared to highly-active (mean difference=0.98, $p \leq 0.043$; $d = 0.36$, 95%CI: 0.12, 0.60). SAD symptoms were non-significantly greater among low-active compared to highly-active (mean difference=1.20, $p \geq 0.064$; $d = 0.30$, 95%CI: 0.06, 0.55). SPA was significantly greater among low-active compared to moderately-active (mean difference=3.49, $p \leq 0.048$; $d = 0.24$, 95%CI: −0.01, 0.50) and highly-active (mean difference=3.65, $p \leq 0.027$; $d = 0.33$, 95%CI: 0.09, 0.58). PD symptoms did not significantly differ between PA levels ($F_{(2, 465)}=2.34$, $p \geq 0.098$).

Table 1
Explanation of the decomposition of potential effects.

Total effect	The overall effect of physical activity on mental health
Controlled direct effect	The direct effect of physical activity on mental health that was not explained by social physique anxiety
Reference interaction	The effect of physical activity on mental health due to the interaction with social physique anxiety
Mediated interaction	The effect of physical activity on mental health due to both mediation and interaction with social physique anxiety
Pure indirect effect	The effect of physical activity on mental health that was 'transferred' through social physique anxiety

Table 2
Characteristics of Included Participants; Mean (SD) unless otherwise stated.

	Males (n = 172)	Females (n = 298)	Total (N = 470)
Age (years)	23.5 (5.3)	23.1 (4.5)	23.2 (4.8)
PDSQ-GAD	3.1 (3.2)	4.8 (3.3)***	4.2 (3.3)
PDSQ-SAD	4.7 (4.2)	5.8 (4.2)**	5.4 (4.2)
PDSQ-PD	0.9 (1.7)	1.5 (2.1)**	1.3 (2.0)
Comorbid GAD-SAD (n, %)	38 (22.1%)	114 (38.3%)***	152 (32.3%)
Comorbid GAD-PD (n,%)	11 (6.4%)	48 (16.1%)**	59 (12.6%)
Comorbid SAD-PD (n,%)	14 (8.1%)	46 (15.4%)*	60 (12.8%)
SPA	34.0 (11.2)	41.2 (11.2)***	38.6 (11.7)
PA (kcals/wk)	297.4 (61.9)	279.9 (46.8)**	286.3 (53.4)

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ for comparison between males and females. Abbreviations: PDSQ-GAD=Psychiatric Diagnostic Screening Questionnaire-Generalized Anxiety Disorder subscale; PDSQ-SAD=Psychiatric Diagnostic Screening Questionnaire-Social Anxiety Disorder subscale; PDSQ-PD=Psychiatric Diagnostic Screening Questionnaire-Panic Disorder subscale; SPA=Social Physique Anxiety; PA=Physical activity; kcals=kilocalories; wk=week.

3.3. Associations between PA level and analogue anxiety disorder status

3.3.1. Analogue GAD

In crude logistic regression models, moderately-active and highly-active showed ~28% (OR=0.72, 95%CI: 0.43, 1.20, $p>0.20$) and ~42% (OR=0.58, 95%CI: 0.35, 0.94, $p \leq 0.027$) lower odds of AGAD, respectively. After adjustment for age, gender, and smoking status, moderately-active and highly-active showed ~29% (OR=0.71, 95%CI: 0.42, 1.20, $p>0.20$) and ~37% (OR=0.63, 95%CI: 0.38, 1.06, $p>0.07$) lower odds of AGAD, respectively. Gender and smoking were significant covariates; females and smokers showed ~163% (OR=2.63, 95%CI: 1.72, 4.00, $p<0.001$) and 151.0% (OR=2.51, 95%CI: 1.40, 4.50, $p \leq 0.002$) greater odds of AGAD, respectively.

3.3.2. Analogue SAD

In crude logistic regression models, moderately-active and highly-active showed ~32% (OR=0.68, 95%CI: 0.40, 1.16, $p>0.15$) and ~35% (OR=0.65, 95%CI: 0.39, 1.07, $p>0.08$) lower odds of ASAD, respectively. After adjustment for age, gender, and smoking status, moderately-active and highly-active showed ~33% (OR=0.67, 95%CI: 0.39, 1.14, $p>0.14$) and ~34% (OR=0.67, 95%CI: 0.40, 1.12, $p>0.12$) lower odds of ASAD. Gender was a significant covariate; females showed ~61% greater odds of ASAD (OR=1.61, 95%CI: 1.09, 2.38, $p<0.02$).

3.3.3. Analogue PD

In crude logistic regression models, moderately-active and highly-active showed 37% (OR=0.63, 95%CI: 0.33, 1.21, $p>0.16$) and ~51% (OR=0.49, 95%CI: 0.26, 0.93, $p<0.03$) lower odds of APD. After adjustment for age, gender, and smoking status, moderately-active and highly-active showed ~38% (OR=0.62, 95%CI: 0.32, 1.23, $p>0.17$) and ~50% (OR=0.50, 95%CI: 0.26, 0.98, $p \leq 0.044$) lower odds of APD. Gender and smoking status were significant covariates; females and smokers showed 194% (OR=2.94, 95%CI: 1.52, 5.56, $p \leq 0.001$) and 300% (OR=4.00, 95%CI: 2.07, 7.74, $p<0.001$) greater odds of APD, respectively.

3.4. Mediation

Tables 3, 4, and 5 show output of mediation analysis for AGAD, ASAD, and APD, respectively. The pure indirect effect significantly accounted for 58.2% ($p \leq 0.021$) and 47.9% ($p \leq 0.012$) of the total effect of PA on AGAD and ASAD, respectively, and non-significantly accounted for 21.3% ($p = 0.170$) of the total effect of PA on APD. These associations were not statistically significant in sensitivity analyses controlling for depressive symptoms (Supplementary Tables 1–3).

Table 3
Decomposition of the overall effect of physical activity on analogue generalized anxiety disorder.

	β	95% CI	P-value
Total effect	−0.368	−0.805 to 0.070	0.099
Controlled direct effect	−0.140	−0.428 to 0.147	0.338
Reference interaction	−0.041	−0.559 to 0.476	0.876
Mediated interaction	0.028	−0.202 to 0.258	0.810
Pure indirect effect	−0.214	−0.396 to −0.033	**0.021**

Adjusted for age, sex, and smoking.

Table 4
Decomposition of the overall effect of physical activity on analogue social anxiety disorder.

	β	95% CI	P-value
Total effect	−0.555	−0.941 to −0.169	**0.005**
Controlled direct effect	−0.092	−0.304 to 0.120	0.395
Reference interaction	−0.366	−0.776 to 0.044	0.080
Mediated interaction	0.169	−0.048 to 0.386	0.127
Pure indirect effect	−0.266	−0.473 to −0.059	**0.012**

Adjusted for age, sex, and smoking.

Table 5
Decomposition of the overall effect of physical activity on analogue panic disorder.

	β	95% CI	P-value
Total effect	−0.493	−0.846 to −0.139	**0.006**
Controlled direct effect	−0.458	−0.855 to −0.060	**0.024**
Reference interaction	0.094	−0.283 to 0.470	0.626
Mediated interaction	−0.024	−0.213 to 0.166	0.807
Pure indirect effect	−0.105	−0.255 to 0.045	0.170

Adjusted for age, sex, and smoking.

Mediation analyses stratified by gender did not reveal any sex-related differences (Supplementary Tables 4–6).

4. Discussion

Largely consistent with hypotheses, these cross-sectional findings showed that symptoms of GAD and SAD, but not symptoms of PD, significantly differed by PA levels, with lower anxiety disorder symptoms reported among higher levels of self-reported PA. Further, PA was associated with lower odds of AGAD, SAD, and PD, but findings were not statistically significant after adjusting for age, gender, and smoking status. Mediation analyses showed that 58.2% and 47.9% of the total effects of PA on AGAD and ASAD, respectively, were mediated by SPA. However, these findings were no longer statistically significant after more fully adjusting for depressive symptoms. Though females showed significantly greater odds of AGAD, ASAD, and APD, mediation findings did not differ based on gender. To the authors' knowledge, this is the first study to demonstrate the mediating effect of SPA on associations between PA and any anxiety disorder, findings that may have critical implications for physical activity intervention among young adults with elevated anxiety disorder symptoms. These findings should be interpreted with some caution, particularly given that cross-sectional mediation designs can be problematic due to likelihood of biased estimates (Maxwell and Cole, 2007; VanderWeele, 2016; Winer et al., 2016). These points are discussed in more detail in the following sections.

Anxiety disorders are highly prevalent among young adults, with global prevalence rates among this age group of ~5.1% (Baxter et al., 2013). However, the prevalence of potentially meaningful subclinical symptom severity (i.e., AGAD, ASAD, APD) is largely unknown. Herein, the prevalence rates of AGAD, ASAD, and APD were quite high at 38.1%, 60.0%, and 15.1%, respectively. Given that there is no standard, prescribed treatment paradigm for such subclinical symptoms (i.e., it is unlikely that a clinician would/should prescribe a frontline therapy to an individual without a clinical diagnosis), it is imperative to identify accessible, potentially low-cost and low-risk alternative therapeutic options that may be 'prescribed' or 'self-prescribed' at large-scale. Regular PA is one such potentially low-cost, accessible, alternative therapies for which there are few, if any, negative side effects, unlike most frontline treatments.

Herein, highly-active individuals reported significantly lower symptoms of GAD and SAD compared to low-active individuals; the magnitude of these differences exceeded 0.25SD at 0.36SD and 0.30SD for GAD and SAD, respectively. However, symptoms of PD did not significantly differ between PA levels. Inconsistent findings for PD may have resulted from the relatively low prevalence of PD in this sample. Additionally, these inconsistent findings may highlight that etiological underpinnings of these anxiety disorders and/or age of onset are differentially associated with PA behavior. For example, although this sample was comprised of young adults, the sample age range includes ages beyond the typical age of onset for SAD, within the range for PD, and before the typical age of onset for GAD. Though speculative, it is plausible that, because the age of onset of PD is within the current age range, emerging PD symptoms may have resulted in avoidance of exercise due to similarities of sensations experienced during exercise and those characteristic of panic attacks (i.e., increased heart rate, perspiration, and respiration; also the premise of exercise as exposure therapy for PD), which may have reduced variability in PA. The differences in findings across anxiety disorders support future investigations of putative mechanisms underlying PA—Anxiety relationships.

Further, PA was associated with 29%−50% reductions in odds across analogue GAD, SAD, and PD statuses; however, in transparency, these findings were not statistically significant after adjustment for age, gender, and smoking status (these covariates are discussed later). The magnitude of these associations was consistent with previous associations between PA and symptoms of GAD (r=−0.07) and SAD (r=−0.12) reported among ~1000 US young adult women (Herring et al., 2014b) these odds reductions are also comparable to recent meta-analytic evidence of associations between PA and reduced odds of any anxiety disorder (33.7%) and GAD (45.6%) (McDowell et al., 2019). Collectively, these and previous findings support that PA interventions to combat anxiety disorders and symptoms may be particularly important, especially among young adults with elevated symptoms indicative of a potential anxiety disorder; intervening with PA at this point in the severity spectrum may prevent/reduce future anxiety-related burden (McDowell et al., 2019).

To this end, because less than 25% of the US population meets recommended levels of PA (Bennie et al., 2020), there is a need to identify modifiable variables that may help to improve engagement in, maintenance of, and response to PA, particularly among those with subclinical or clinical impairments in mental health. Previous evidence has supported inverse associations of PA and SPA (Brunet and Sabiston, 2009; McAuley et al., 2002), and the protective effects of PA on anxiety disorders (McDowell et al., 2019). Although SPA is highly comorbid with other mental health disorders like eating disorders (Pallister and Waller, 2008), less is known regarding associations between SPA and anxiety disorders or how SPA may influence associations between PA and anxiety disorder symptoms.

Herein, SPA was associated with both PA and anxiety disorder symptoms and status; importantly, associations between PA and AGAD and ASAD status were partially mediated by SPA. These findings are consistent with previous evidence showing that physical self-esteem and self-concept, other body-related social-cognitive factors, mediated associations between PA and anxiety disorder symptoms among ~1000 US young adult women (Herring et al., 2014b). However, when mediation models were more fully adjusted for symptoms of Major Depressive Disorder (MDD), the reported associations were no longer statistically significant. Given the likelihood of overlapping etiological factors

among the anxiety disorders investigated here and MDD (i.e., monoaminergic dysregulation), it is plausible that inclusion of these outcomes together in analytical models could bias (i.e., overestimate or underestimate) the 'true' association with PA if, indeed, those etiological factors are among the mechanisms that underpin salutary benefits of PA for a given condition. Additionally, temporality, or temporal ordering, is difficult to establish in cross-sectional mediation designs. However, there is a strong epidemiological literature to show that PA is associated with lower prevalence and incidence of anxiety disorders (McDowell et al., 2019), and exercise training has significantly improved outcomes among adults with anxiety disorders (Herring et al., 2012), providing support that the exposure of PA plausibly precedes the outcome of anxiety disorders.

It also is plausible that young adults may use PA strategically to enhance their physical appearance (Thompson and Chad, 2002), thereby improving SPA and, subsequently, symptoms of SAD and GAD. Though a previous report showed significant associations between perceptions of appearance and symptoms of GAD and SAD, PA was not significantly associated with perceptions of appearance (Herring et al., 2014b). As stated earlier, PA is associated with reduced odds of anxiety disorders and protects against the development of anxiety disorders (McDowell et al., 2019); thus, PA involvement may protect against the development of SPA as well.

Conversely, SPA may predispose individuals to aversion to PA. Given that SPA is negatively associated with self-efficacy (Marquez and McAuley, 2001) and motivation (Thøgersen-Ntoumani and Ntoumanis, 2007), it is plausible that young adults with elevated SPA may have less motivation for PA. However, previous evidence showed that individuals with elevated SPA were not deterred from the performance of regular PA (Kowalski et al., 2001). The present findings highlight both the need for further consideration of SPA as a modifiable factor to enhance engagement in and maintenance of PA to potentially improve anxiety symptoms and disorders, and the need for future research into the extent to which SPA may influence anxiety response to acute and chronic exercise.

Young adult women reported significantly lower physical activity, greater severity of GAD, SAD, and PD symptoms and SPA, and showed 167%, 159%, and 59% greater odds of APD, AGAD, and ASAD, respectively. Based on a minimally important difference of 0.5SD (Norman et al., 2003), the magnitude of differences between GAD symptom severity ($d = 0.51$) and SPA ($d = 0.64$) between females and males is potentially clinically meaningful. There appeared to be no difference in the mediating effects of SPA on associations between PA and anxiety disorders between males and females. However, given the clearly worse PA, anxiety disorder, and SPA profile among women, future research in large samples of women, particularly those with anxiety symptomatology, is needed.

Consistent with previous evidence showing both significant associations between smoking and anxiety symptoms and disorders (Moylan et al., 2012) and smoking status as a significant covariate in models of associations between PA and incident GAD (McDowell et al., 2018), smoking was a significant covariate in all models of PA and AGAD and APD. Smokers showed 151% and 300% greater odds of AGAD and APD, respectively. Given previous evidence of positive effects of regular PA and exercise on smoking cessation among adults with high anxiety sensitivity (Smits et al., 2016), PA interventions among smokers with subclinical or clinical anxiety symptomatology may be particularly important.

5. Limitations

The current findings should be interpreted with some caution in light of potential limitations. As stated earlier, cross-sectional mediation designs can be problematic due to likelihood of biased estimates (Maxwell and Cole, 2007; VanderWeele, 2016; Winer et al., 2016). Though the rigor of the counterfactual approach to the mediation analyses was a potential strength, temporal ordering is difficult to establish in cross-sectional mediation designs. Self-reported PA may be subject to under-/over-reporting biases, and, though the anxiety disorder subscales of the PDSQ demonstrated adequate internal consistency, the relatively low level of specificity is a potential limitation of these subscales. Finally, a larger sample would have provided additional statistical power to detect differences across anxiety disorder and PA statuses.

6. Conclusions

Notwithstanding potential limitations, the present findings support that higher physical activity is associated with lower anxiety disorder symptoms and odds of subclinical/analogue anxiety disorder status, particularly for GAD and SAD. Social physique anxiety appears to partially mediate associations between physical activity and GAD and SAD, and should be investigated/manipulated as a plausible mechanism to enhance the influence of regular physical activity on GAD and SAD.

Authors' contributions

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Declaration of Competing Interest

All authors have no conflicts of interest.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2020.12.163.

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