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Do men and women arrive, stay, and respond differently to cognitive behavior group therapy for social anxiety disorder?

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1. Introduction

Social anxiety disorder (SAD) is a common and debilitating psychiatric disorder with an estimated lifetime prevalence rate of 12.1% (Kessler et al., 2005). It is characterized by a marked and persistent fear of one or more social situations (e.g., talking to a stranger or peer, going to a party) or performance activities (e.g., giving a speech) in which the person is exposed to unfamiliar people, and may face possible scrutiny by others (American Psychiatric Association, 2013). These difficulties in interpersonal interactions experienced by individuals with SAD result in significant impairment in almost all facets of daily life, including relationships, work, and studies (e.g., Aderka et al., 2012; Alden & Taylor, 2004).

Considering the large body of research on SAD, and despite accumulating data about gender differences in other disorders (e.g., agoraphobia: Bekker, 1996; specific phobias: Fredrikson, Annas, Fischer, & Wik, 1996; obsessive-compulsive disorder: Bogetto, Venturello, Albert, Maina, & Ravizza, 1999; generalized anxiety disorder: Vesga-López et al., 2008) there is a paucity of research directly examining gender differences in SAD. However, a recent review of the literature (Asher, Asnaani, & Aderka, 2017) found gender differences in a number of domains of SAD and stressed the need for more research on the topic as gender differences can have substantial clinical implications (Asher et al., 2017). The present study is focused on identifying and elucidating gender differences in pretreatment clinical severity, treatment seeking, attrition and treatment response in SAD.

Pretreatment clinical severity

Previous studies have converged to suggest that women are approximately 1.5 times more likely than men to have SAD (e.g., Kessler et al., 1994; MacKenzie & Fowler, 2013; Ohayon & Schatzberg, 2010). Along these lines, findings from a recent review of gender differences in SAD (Asher et al., 2017) indicated that women with SAD report greater clinical severity of the disorder. For instance, Turk et al. (1998) found that women who sought treatment for SAD reported more severe symptoms compared to men on a number of symptom measures (the Social Interaction Anxiety Scale, Social Phobia Scale, the Fear Questionnaire – Social Phobia subscale, and the Liebowitz Social Anxiety Scale – Performance Fear subscale). Similarly, a number of studies have demonstrated that women with SAD endorse a greater number of social fears compared to men with SAD (Turk et al., 1998; Xu et al., 2012). For example, data from the National Comorbidity Survey Replication (NCS-R), demonstrated that SAD involving 1–4 social fears is more common among men, whereas SAD involving a larger number of fears is more common in women (Ruscio et al., 2008).

Treatment seeking

Several studies have indicated that men with SAD may be more likely to seek treatment compared to women (e.g., Amies, Gelder, & Shaw, 1983; Solyom, Ledwidge, & Solyom, 1986). Along these lines, the DSM-5 states that men seem to be overrepresented in clinical samples and may display heightened help-seeking behavior (American Psychiatric Association, 2013). More recently, equal proportions of men and women seeking treatment for SAD have been found in naturalistic settings (e.g., Aderka, Hermesh, Marom, Weizman, & Gilboa-Schechtman, 2011; Marom, Gilboa-Schechtman, Aderka, Weizman, & Hermesh, 2009). These findings may actually indicate a greater propensity of men with SAD to seek treatment. Specifically, because a larger percentage of women meet criteria for SAD in the population than men, if an equal number of men and women seek treatment, then the proportion of men with SAD who seek treatment is larger than the proportion of women with SAD who seek treatment (Asher et al., 2017; Kessler et al., 1994; Although see Asher & Aderka, 2018 for conflicting findings). Albeit speculative, several studies have suggested that this gender difference in treatment seeking may be the result of gender roles and stereotypes (see Asher et al., 2017 for a review and discussion of this issue).

However, it is important to note a number of gaps in this literature. First, very few studies have directly focused on identifying gender differences in treatment seeking, and many studies have used procedures which limit our ability to examine gender differences. For instance, most clinical trials do not report the gender of individuals seeking treatment but only the gender of individuals included and randomized. This is consistent with CONSORT guidelines (Moher, Schulz, & Altman,

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2001) which stress the importance of reporting the number of individuals excluded at each stage of randomized trials but do not address demographic variables related to excluded individuals. Thus, naturalistic studies with minimal or no exclusion may facilitate the examination of gender differences in treatment seeking. Second, many previous studies have been underpowered to detect significant gender differences. For instance, most trials are sufficiently powered to detect differences between treatment arms but not between subgroups within these arms (e.g., Egbewale, 2015).

Attrition

A number of studies have examined the effects of demographic characteristics on attrition during treatment for SAD (see review by Mululo, Menezes, Vigne, & Fontenelle, 2012). Gender has generally not been found to predict attrition in treatment for SAD either in children (e.g., Gonzalez, Weersing, Warnick, Scahill, & Woolston, 2011), or in adults (e.g., Hoyer et al., 2016). Similar null findings have been reported in cognitive behavior group therapy (CBGT) for SAD, in which men and women were found to drop out to a similar extent (Hofmann & Suvak, 2006). Thus, the literature on attrition is consistent in suggesting that gender does not predict drop-out rates in treatments for SAD.

Despite the convergence of findings, it is important to consider an important issue that may have hindered our ability to detect significant gender differences in attrition. Specifically, attrition is a (relatively) low-probability event and most studies report drop-out rates of 5–30% (e.g., Acarturk, Cuijpers, Van Straten, & De Graaf, 2009). As a result, examinations of gender differences in attrition may be statistically underpowered to detect significant differences and large sample sizes may be needed in order to conduct sufficiently powered examinations of gender differences in attrition.

Response to treatment

Studies examining gender differences in treatment outcome have yielded equivocal findings. For example, one study with a sample of 52 adults diagnosed with SAD indicated that male gender was associated with a poorer response to group plus video feedback cognitive-behavioral therapy (CBT; Chen et al., 2010). Similarly, a recent study also found that male gender was associated with poorer improvement in both CBT and acceptance and commitment therapy (ACT) for SAD (Craske et al., 2014). In contrast to these findings, other studies have found no gender differences in treatment outcome in CBGT for SAD (e.g., Otto et al., 2000) and in cognitive therapy (CT) for SAD (Hoyer et al., 2016). It is important to note that whereas some data from child samples also indicated no gender differences in response to CBT for SAD (e.g., Hudson et al., 2015), one study of children diagnosed with SAD (n = 384) indicated that female gender was associated with poorer response to CBT (Hudson et al., 2013). Thus, data on gender differences in response to treatment remain scarce and inconclusive.

Gaps in the literature

There are a number of gaps in the literature on gender differences in treatment for SAD. First, most studies which have examined gender differences in treatment for SAD have done so as part of a larger examination of potential predictors or moderators of outcome. The present study aims to address this gap and to primarily focus on gender differences in order to conduct a comprehensive analysis of the effects of gender in treatment for SAD. Second, much of our knowledge of gender differences in SAD has been derived from randomized controlled trials (RCTs). While the contribution of RCTs to our field has been invaluable and has generated a wealth of knowledge, some common practices in RCTs may limit our ability to examine gender differences. Specifically, not reporting the gender of excluded individuals and using small sample sizes that can cause examinations of gender differences to be underpowered may limit the ability to examine gender differences.

The present study

The aim of the present study was to address these gaps in the literature in order to increase our understanding of gender differences in SAD. To do so, we conducted an analysis of gender differences using a very large sample (n = 1010) of individuals receiving CBGT for SAD in an outpatient clinic. We used a large sample to ensure we have sufficient statistical power to examine gender differences in low-probability events such as attrition (see power analysis in section 2.6). We also used a naturalistic setting to enhance our ability to examine differences in patterns of treatment seeking. We hypothesized that consistent with the previous studies, women will demonstrate greater pretreatment clinical severity as indicated by higher SAD symptom ratings (Hypothesis 1). In addition, consistent with most of the literature, men would seek treatment more than women, evidenced by greater proportions of men in outpatient treatment (Hypothesis 2). Due to the null findings in the literature on gender differences in attrition, we did not expect to find gender differences in attrition. Finally, despite some inconsistencies in the literature, we hypothesized that men would evince diminished response to CBGT compared to women as this was found by two recent studies (Hypothesis 3).

2. Method

2.1. Participants

Participants (n = 1010) were outpatients who sought group treatment for SAD at a large outpatient clinic in Israel between 1999 and 2016. Of the total sample, 472 (46.7%) were women and 538 (53.3%) were men. The mean age for the total sample was 31.45 (SD = 8.82) and mean years of education was 14.03 (SD = 2.01). Of the total sample, 27.1% were married. 67.7% were single, 4.8% were divorced, and 0.5% were widowed. Comorbid diagnoses included panic disorder (7.2%), generalized anxiety disorder (15.7%), obsessive-compulsive disorder (16.9%), specific phobia (16.2%), major depressive disorder (20.7%), posttraumatic stress disorder (1.3%), eating disorders (4.1%), and substance use disorders (1.9%).

Inclusion criteria for study participation were: (a) a primary diagnosis of SAD; (b) a minimum 1-year duration of SAD; (c) stable pharmacotherapy during the 3 months prior to the beginning of treatment; (d) age between 18–60 years. Exclusion criteria were: (a) past or present psychosis; (b) engaging in another psychotherapeutic treatment during the study (with the exception of stable pharmacotherapy).

2.2. Procedure and treatment

Upon contacting the outpatient clinic, participants underwent the Mini International Neuropsychiatric Interview (MINI – Sheehan et al., 1998) to establish diagnoses. The MINI was administered by graduate students who received prior training in its administration. Diagnoses were also reviewed by an experienced clinician (S.M.). Reliability of the MINI diagnoses at the clinic was high (see Marom et al., 2009 for more details).

Treatment was conducted by an experienced PhD-level clinical psychologist and a co-therapist (a psychiatry/psychology resident). There were 18 weekly group sessions of 1.5 h duration. The treatment protocol was based on the protocols of Heimberg, Juster, Hope, & Mattia (1995; Heimberg and Becker, 2002) and on the theoretical writings of Clark and Wells (1995). The protocol included (a) psychoeducation about SAD; (b) exposure to feared social situations, (c) reduction of safety behaviors, (d) cognitive restructuring, (e) instruction on external focus of attention, and (f) social skills training. A detailed description of the treatment can be found in Marom et al. (2009). Importantly, treatment was found to be highly efficacious with a large

pre-post effect size of $d = 1.23$ (Marom et al., 2009).

2.3. Measures

2.3.1. Diagnoses

The Mini International Neuropsychiatric Interview version 5 (MINI; Sheehan et al., 1998) is a widely used structured interview assessing DSM-IV Axis I disorders. Each MINI diagnostic module consists of a series of screening items followed by questions about specific symptoms. Strong inter-rater reliability of the MINI was demonstrated with kappas ranging from 0.88–1.0 (Lecrubier et al., 1997). In addition, good concordance of the MINI diagnoses with the Structured Clinical Interview for DSM diagnoses (SCID; Spitzer, First, Gibbon, & Williams, 1990) was reported, with only a single kappa value below 0.5 (Sheehan et al., 1997).

2.3.2. Clinical measures administered at pre- and post-treatment

Social Anxiety Symptoms. The Liebowitz Social Anxiety Scale – Self Report version (LSAS – SR; Liebowitz, 1987) assesses 24 situations (13 performance situations and 11 social interaction situations) that individuals with SAD typically fear or avoid, such as going to a party, meeting strangers, and speaking up at a meeting. For each situation, fear and avoidance are rated separately on a scale of 0 to 3. The LSAS-SR has been shown to have high internal consistency (Cronbach's $\alpha = 0.95$), high 12-day test-retest reliability ($r = 0.83$) and strong convergent and discriminative validity (Baker, Heinrichs, Kim, & Hofmann, 2002). The self-report version has been shown to assess non-clinical as well as clinical levels of social anxiety reliably (Rytwinski et al., 2009).

Depressive symptoms. The Beck Depression Inventory – II (BDI-II; Beck, Steer, Ball, & Ranieri, 1996) contains 21-items assessing cognitive, affective and behavioral depressive symptoms. The BDI–II is a widely used measure and has demonstrated excellent reliability and validity (Beck, Steer, & Brown, 1996) in both clinical (Sprinkle et al., 2002) and non-clinical samples (Storch, Roberti, & Roth, 2004; Whisman, Perez, & Ramel, 2000).

Functional impairment. The Sheehan Disabilities Scale (SDS – Sheehan, 1983) was used to assess impairment in work, social and family areas. The SDS evaluates the degree of impairment in work, social and family areas, on a 10 point Likert scale. Reliability and construct validity of the SDS are high (Leon, Olfson, Portera, Farber, & Sheehan, 1997) and it has strong correlations with symptoms of social anxiety, depressive symptoms, and quality of life among individuals with social phobia (Hambrick, Turk, Heimberg, Schneier, & Liebowitz, 2004).

2.4. Statistical analyses

Our analyses focused on gender differences in presentation of SAD and response to treatment. Measures of a continuous nature were compared between men and women using one-way analysis of variance (and η^2 as a measure of effect size). Measures of categorical or proportionate nature were compared between groups using χ^2 tests (and Cramer's *V*s as a measure of effect size). To examine whether men and women differed in their proportions of treatment-seeking we used a one-sample, two-tailed binomial test. This test compares the observed proportions of men and women in the sample with the expected 50–50 proportion assumed if no gender differences exist. To examine gender differences in response to treatment we used Hierarchical Linear Modeling (HLM) with repeated measurements before and after treatment (Level 1 units) nested within individuals (Level 2 units). According to the guidelines of Tabachnik and Fidell (2013) we did not include a third level of analysis (the group level) because the intraclass correlation suggested that variance between groups was very small (< 5%). In our 2-level model, we examined whether Gender (male vs. female), Time (pretreatment vs. posttreatment) and their interaction (Gender*Time) predicted outcome measures.

2.5. Power analyses

We conducted a power analysis for detecting significant differences between men and women in attrition. Using a χ^2 test with our sample size ($n = 1010$) and an alpha level of 0.05, power to detect a small (0.1) effect size was 0.88 and above the commonly used cutoff for sufficient power (0.80; Cohen, 1988). Power for all additional analyses was found to be greater than 0.8.

3. Results

3.1. Sociodemographic measures

Among individuals who sought treatment for SAD, men were more likely to be single [$\chi^2_{(1)} = 6.35$, $p = 0.01$; Cramer's $V = 0.08$; $n_{women} = 300$, 63.7%, $n_{men} = 382$, 71.1%], whereas women were more likely to be divorced [$\chi^2_{(1)} = 5.04$, $p = 0.03$; Cramer's $V = 0.07$; $n_{women} = 30$, 6.4%, $n_{men} = 18$, 3.4%]. However, there was no gender difference found in the percentage of men and women that were married [$\chi^2_{(1)} = 1.8$, $p = 0.18$; Cramer's $V = 0.08$; $n_{women} = 137$, 50.2%, $n_{men} = 136$, 49.8%], or widowed [$\chi^2_{(1)} = 2.24$, $p = 0.14$; Cramer's $V = 0.05$; $n_{women} = 4$, 0.8%, $n_{men} = 1$, 0.2%]. Men and women did not differ in their employment status (i.e., employed vs. not employed) at the time of intake [$\chi^2_{(1)} = 2.92$, $p = 0.09$; Cramer's $V = 0.05$; $n_{women} = 373$, 79.4%, $n_{men} = 447$, 83.6%], nor were there gender differences in years of education [$F_{(1,1001)} = 1.06$, $p = 0.3$, $\eta^2 = 0.001$]. Similarly, no gender differences in age of men and women at the time of intake were found [$F_{(1,\ 1005=0.41)}$, $p = 0.52$, $\eta^2 < 0.001$].

3.2. Pretreatment clinical severity

Women demonstrated significantly higher levels of SAD symptoms compared to men as measured by the LSAS (see Table 1). This difference stemmed primarily from the anxiety subscale of the measure. Specifically, whereas men and women did not significantly differ in their level of social avoidance as indicated by the LSAS, women reported significantly higher levels of social anxiety. Based on DSM-IV

Table 1
Clinical measures at intake for men and women seeking treatment for SAD.

	Men			Women			*df*	*F*	η^2	*p*
	M	*SD*	*N*	*M*	*SD*	*N*				
LSAS-SAD symptoms	74.91	24.27	396	78.90	23.89	343	1, 737	5.04	0.01	**0.03**
LSAS-Avoidance	35.20	13.36	398	36.43	12.38	343	1, 739	1.49	0.002	0.22
LSAS-Anxiety	39.99	12.51	398	42.40	12.63	344	1, 740	6.77	0.01	< **0.01**
BDI-Depressive symptoms	13.98	9.37	340	15.51	9.35	400	1, 738	4.94	0.01	**0.03**
SDS-Work	6.85	2.35	510	7.37	2.17	437	1, 945	12.27	0.01	< **0.001**
SDS-Social life	7.30	2.50	513	7.10	2.67	444	1, 955	1.41	0.001	0.24
SDS-Family	3.90	2.97	511	3.28	3.06	442	1, 951	10.01	0.01	< **0.01**

criteria, a greater proportion of women met criteria for the generalized subtype of SAD (n_{women} = 426, 90.6%, n_{men} = 460, 86%), whereas a greater proportion of men met criteria for the specific subtype [$\chi^2_{(1)}$ = 5.2, p = 0.02; Cramer's V = 0.07; n_{women} = 44, 9.4%, n_{men} = 75, 14%]. In addition, men reported a slightly later onset (M = 10.4, SD = 7.05) of SAD compared to women [M = 9.56, SD = 6.15; $F_{(1,958)}$ = 3.78, p = 0.05, η^2 = 0.004]. At intake, women reported significantly higher levels of depressive symptoms compared to men as measured by the BDI (see Table 1). Whereas women reported significantly greater impairment in work at intake as measured by the SDS, men reported significantly greater impairment in their family life (see Table 1). No significant gender differences emerged for overall social impairment as measured by the SDS. There were no gender differences found in psychiatric comorbidity, except for women being more likely to suffer from phobias [$\chi^2_{(1)}$ = 5.3, p = 0.02; Cramer's V = 0.07; n_{women} = 140, 29.7%, n_{men} = 125, 23.3%], and eating disorders [$\chi^2_{(1)}$ = 16.79, p < 0.001; Cramer's V = 0.13; n_{women} = 32, 6.8%, n_{men} = 9, 1.7%] compared to men.

3.3. Treatment seeking

Of our total sample (n = 1010), 472 individuals (46.7%) were women and 538 (53.3%) were men. A one-sample, two-tailed binomial test indicated that the proportion of treatment seeking men and women with SAD was significantly different from the expected equal proportion of 50% (p = 0.04). This indicates that men have higher proportions of treatment seeking compared to women.

Men and women were as likely to have received psychotherapy in the past [$\chi^2_{(1)}$ = 0.76, p = 0.38; Cramer's V = 0.03; n_{women} = 338, 72.8%, n_{men} = 370, 70.3%]. They were also as likely to receive medication at the time of intake [$\chi^2_{(1)}$ = 1.74, p = 0.19; Cramer's V = 0.04; n_{women} = 138, 30.4%, n_{men} = 178, 34.4%]. However, men were more likely than women to have received medication in the past [$\chi^2_{(1)}$ = 6.58, p = 0.01; Cramer's V = 0.08; n_{women} = 192, 41.4%, n_{men} = 260, 49.5%].

3.4. Attrition

Of the entire sample, 256 individuals (25.3%) dropped out of treatment. Men dropped out of treatment significantly more than women [$\chi^2_{(1)}$ = 4.78, p = 0.03; Cramer's V = 0.07; n_{women} = 106, 24%, n_{men} = 150, 30.4%].

3.5. Response to treatment

In order to examine response to treatment, we calculated a series of HLM analyses with Gender, Time, and their interaction as predictors (Table 2 presents results for all outcome measures). We found a significant Time effect for all outcome measures suggesting that treatment was effective in reducing social anxiety, depression, and functional impairment. We found a significant Gender*Time interaction for LSAS and the BDI but not for the SDS subscales. Specifically, the results indicated that slopes of change for women (B_{LSAS} = −24.13;[1] B_{BDI} = −5.94[1]) were significantly more negative compared to those for men (B_{LSAS} = −21.83[1]; B_{BDI} = −5.18[1]) suggesting greater improvement for women with SAD compared to men with SAD in these measures. Importantly, the possibility that these findings are due to regression to the mean cannot be ruled out as women reported higher levels of SAD symptoms at intake.

To further examine gender differences in response to treatment we also calculated the reliable change index (RCI; Jacobson and Truax, 1991) from pre- to post-treatment for each participant. We found that in our sample, reliable change was observed for 104 men (19.62%) and 119 women (25.48%). This difference was significant [$\chi^2_{(1)}$ = 4.91, p = 0.03; Cramer's V = 0.07] indicating that more women than men experienced reliable change.

Table 2
Gender differences in response to treatment for SAD.

Effect	*B*	*SE*	*df*	*t*	*p*	95% CI
LSAS						
Gender	1.33	0.97	530	1.37	0.172	−0.58 to 3.24
Time	−11.49	0.42	530	−27.23	< **0.001**	−12.32 to −10.66
Gender*Time	−1.15	0.42	530	−2.73	**0.007**	−1.98 to −0.32
BDI						
Gender	0.40	0.35	505.05	1.14	0.253	−0.29 to 1.10
Time	−2.78	0.18	490.12	−15.83	< **0.001**	−3.12 to −2.43
Gender*Time	−0.38	0.18	490.12	−2.17	**0.031**	−0.72 to −0.04
SDS – work subscale						
Gender	0.31	0.09	529.09	3.49	**0.001**	0.14 to 0.49
Time	−1.06	0.06	512.36	−16.85	< **0.001**	−1.18 to −0.94
Gender*Time	−0.001	0.06	512.36	−0.02	0.982	−0.12 to 0.12
SDS – social subscale						
Gender	−0.14	0.11	526.63	−1.27	0.206	−0.35 to 0.07
Time	−0.98	0.06	502.12	−16.76	< **0.001**	−1.09 to −0.86
Gender*Time	−0.09	0.06	502.12	−1.52	0.130	−0.20 to 0.03
SDS – family subscale						
Gender	−0.16	0.15	533.85	−1.08	0.283	−0.44 to 0.13
Time	−0.39	0.11	520.66	−3.62	< **0.001**	−0.61 to −0.18
Gender*Time	0.10	0.11	520.66	0.93	0.351	−0.11 to 0.32

Note. LSAS = Liebowitz Social Anxiety Scale; BDI = Beck Depression Inventory; SDS = Sheehan Disabilities Scale. Numbers in bold represent statistically significant differences.

4. Discussion

The present study examined gender differences in a large sample (n = 1010) of individuals receiving CBGT for SAD in an outpatient clinic. We used a large sample to ensure we had sufficient statistical power to examine gender differences even in low-probability events such as attrition. We conducted a comprehensive examination of gender differences in pretreatment clinical severity, and in multiple aspects of treatment such as treatment seeking, attrition, and response to treatment. Our main findings indicated that women had greater pretreatment severity of SAD compared to men. However, men sought treatment to a greater extent than women and also dropped out of treatment to a greater extent than women. Finally, women responded to treatment more than men (i.e., improved more during treatment).

Consistent with our first hypothesis, our findings indicated that treatment seeking women with SAD report greater clinical severity compared to treatment seeking men with SAD. Specifically, women reported greater severity of SAD symptoms and depressive symptoms, and were more likely to meet criteria for the generalized subtype of SAD compared to men who were more likely to meet criteria for the specific subtype of SAD. This finding is consistent with previous studies that found greater severity of social fears, and a greater number of social fears among women with SAD (Asher & Aderka, 2018; Crome, Baillie, & Taylor, 2012; Turk et al., 1998; Xu et al., 2012; and see Asher et al., 2017 for review).

Gender differences in clinical severity of SAD may stem from several factors. One such potential factor is behavioral inhibition (BI; Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984), a heritable temperamental trait that may predispose individuals to SAD (e.g., Biederman et al., 2001; and see Schneier & Goldmark, 2015 for a review). BI has been shown to be greater and more stable among girls compared to

[1] Slopes reported here are twice the value of B values reported in Table 2 because all variables were effects-coded prior to analyses (i.e., values of −1 for men and +1 for women). Thus, pre-to-post slopes are represented by a change of 2 units of Time (from −1 to +1).

boys (Essex, Klein, Slattery, Goldsmith, & Kalin, 2010; Hirshfeld et al., 1992) and may contribute to a greater risk for SAD and to higher levels of social anxiety among women. Along these lines a few studies have found higher levels of arousal among women with SAD, as indicated by autonomic parameters (Alvares et al., 2013; Grossman, Wilhelm, Kawachi, & Sparrow, 2001). For example, a recent study found that women with SAD had higher heart rate at rest and lower heart rate variability (HRV) compared to non-socially-anxious women, and importantly, these differences were not observed among men (Alvares et al., 2013). The authors suggested that these findings reflect an enhanced sensitivity among women to the effects of SA on parasympathetic nervous system reactivity. This sensitivity may also underlie gender differences in SAD clinical severity.

The gender differences in clinical severity of SAD found in the present study may also be related to general vulnerability factors for anxiety disorders. For instance, negative affectivity, a temperamental trait that may predispose individuals to anxiety disorders has been found to be elevated among girls compared to boys (Craske, 2003). In addition, women have been found to be more likely to suffer childhood sexual and physical abuse compared to men and these types of abuse may contribute to the development and severity of anxiety disorders (e.g., Mancini, Van Ameringen, & Macmillan, 1995; Stein, Walker, Anderson, & Hazen, 1996).

Finally, gender differences in SAD severity may be understood through the lens of gender theories such as self-construal theory (Cross & Madson, 1997; see Cross, Hardin, & Gercek-Swing, 2011, for a review of empirical evidence). According to this theory men tend to construct and maintain an independent self-construal in which others are represented as separate from the self, whereas women tend to construct and maintain an interdependent self-construal, in which others are represented as part of the self (Markus & Kitayama, 1991). Importantly, this does not mean that all women are highly interdependent and all men are highly independent, but rather that gender differences are found on average between men and women in self-construals (see Cross et al., 2011, for a review). Because women may construe their self as being interdependent to a greater extent than men, they may experience more anxiety regarding the consequences of interpersonal interactions and this may be reflected in greater SAD severity.

In a recent epidemiological study on gender differences in SAD, rates of comorbid generalized anxiety disorder, posttraumatic stress disorder, and substance use disorders were found to differ between men and women with SAD (Asher & Aderka, 2018). However, such differences were not found in our sample. This discrepancy may be due to the fact that the present study examined a treatment-seeking sample, whereas previous studies reporting gender differences in comorbid diagnoses were epidemiological studies. Future studies are needed in order to shed light on this issue.

According to our second hypothesis, we found significantly more men with SAD compared to women with SAD in our treatment seeking sample. This is consistent with the overrepresentation of men in clinical samples suggested by the DSM-5 (American Psychiatric Association, 2013). This heightened help seeking behavior among men with SAD is especially interesting considering that women have been found to seek treatment more than men for other disorders (e.g., Shear, Feske, & Greeno, 2000, for anxiety disorders in general; Vesga-López et al., 2008, for generalized anxiety disorder; Goodwin, Koenen, Hellman, Guardino, & Struening, 2002, for obsessive-compulsive disorder; Angst, Gamma, Gastpar, Lépine, Mendlewicz, & Tylee, 2002, for depression). Thus, our findings indicate that SAD may be unique among the anxiety disorders and depression in patterns of treatment seeking among men and women.

One possible explanation for this divergent pattern is that among individuals with SAD, treatment (which is a form of social interaction) can be a trigger for social anxiety. Thus, if women have greater symptom severity, it is possible that they seek treatment to a lesser extent compared to men due to their elevated social fears. This can explain both the gender difference in treatment seeking in SAD (i.e., more men seek treatment) as well as the pattern of treatment seeking in SAD in relation to other anxiety disorders. Specifically, that the interaction with the therapist may be a trigger for anxiety and avoidance among individuals with SAD more so than for individuals with other disorders. Importantly, this explanation is speculative and much future research is needed to enhance our understanding of the reasons behind treatment seeking behavior in SAD.

An alternative explanation for men's enhanced help seeking behavior may be related to traditional social roles or gender stereotypes. Gender stereotypes depict men's ideal self as assertive, active and dominant (Eagly & Wood, 1991; Eagly, Wood, & Diekman, 2000) – traits which are opposite to patterns of behavior in SAD (e.g., submissive; Walters & Hope, 1998; Walters & Inderbitzen, 1998; Weeks, Heimberg, & Heuer, 2011). Thus, men may experience a greater discrepancy between their ideal and actual selves compared to women (Higgins, 1987, 1996) and this may lead to elevated treatment seeking. Along these lines, Zimmerman, Morrison, & Heimberg (2015) recently found that submissive behaviors mediated the relationship between SA and shame in men with SAD but not women with SAD, even after controlling for depression. Thus, it is possible that compared to women, men with SAD experience elevated shame as a result of not conforming with social roles. However, this explanation should be considered in caution, in light of significant changes which have been observed in gender roles across the world over the past 2 decades (e.g., Bolzendahl & Myers, 2004; Cotter, England, & Hermsen, 2008; Cotter, Hermsen, & Vanneman, 2011; England et al., 2004).

We expected that men and women would not differ in patterns of attrition. In contrast to our expectation, we found that men who sought treatment for SAD dropped out of treatment to a greater extent compared to women. This represents a divergence from previous studies that did not find gender differences in attrition among individuals with SAD (e.g., Hoyer et al., 2016) and among individuals with other anxiety disorders (posttraumatic stress disorder: Blain, Galovski, & Robinson, 2010; obsessive compulsive disorder: Aderka, Anholt, et al., 2011). However, one potential explanation for this discrepancy in findings is that previous studies may have been statistically underpowered to detect significant gender differences in low-probability events such as attrition (e.g., 15 drop-outs in Otto et al., 2000). An additional explanation for men's higher levels of attrition may be that attending treatment may inadvertently *increase* the discrepancy between male gender stereotypes of autonomy and independence (which may serve as an ideal self) and actual help-seeking behavior (which may be perceived as indicating weakness or dependency). Thus, it is possible that men may find attending treatment more distress-inducing than women as it is stereotypically seen as being dependent. Along these lines, a recent experimental study found that activating dependency stereotypes resulted in reduced engagement in help-seeking behaviors (Wakefield, Hopkins, & Greenwood, 2013).

In line with our third hypothesis, we found that women with SAD reported greater reductions in SAD and depressive symptoms during treatment compared to men with SAD. This is consistent with two recent studies which have also found an enhanced therapeutic response among women with SAD compared to men (Chen et al., 2010; Craske et al., 2014; although see Otto et al., 2000 and Hoyer et al., 2016 for null findings on gender differences). Interestingly, previous studies which found such heightened responsivity to treatment among women have mostly interpreted this finding as stemming from selection bias rather than as a representative and replicable finding (e.g., Craske et al., 2014). However, to the best of our knowledge there are now three independent studies including the present study which demonstrate that women respond to treatment for SAD more than men. In addition, there are no studies among adults with SAD with the opposite pattern (i.e., men responding to a greater extent than women) suggesting that this may be a replicable finding in the SAD literature.

One potential reason for women's elevated response to treatment is

that men dropped out of treatment more than women. This differential pattern of attrition may affect response to treatment analyses and bias them in favor of women. However, previous studies which reported such gender differences did not have gender differences in attrition (e.g., Craske et al., 2014). Nevertheless, this explanation should be considered and our findings should be taken with caution. In a similar vein, it is important to note that our findings regarding greater response to treatment among women with SAD may also be due to the fact that women reported higher levels of SAD symptoms at the time of intake. Thus, we cannot rule out the possibility that their greater improvement during treatment may be the result of regression to the mean.

If our findings do not stem from these methodological issues, one explanation that can be considered is that men may have greater tendency to avoid inner experiences compared to women (Spendelow & Joubert, 2018), and this may hinder their ability to progress and improve in treatment (e.g., their response to exposure). Another possible explanation is that men's social fears are more specific and less generalized compared to women's social fears (Xu et al., 2012). This is also in line with our findings regarding men's greater likelihood to meet criteria for the specific subtype of SAD. Thus, it is possible that group treatment was not sufficiently tailored and focused on men's specific fears (e.g., romantic relationships; family life). Importantly, these explanations are speculative and much future research is needed before firm conclusions can be drawn.

Effect sizes for gender differences found in the present study were small in magnitude. This is consistent with the literature on gender differences in other anxiety disorders and depression. For instance, Vesga-López and colleagues (2008) as well as Angst et al. (2002) examined gender differences in generalized anxiety disorder and depression (respectively) in large epidemiological samples and found gender differences in prevalence, severity, comorbidity, disability and treatment seeking and the vast majority of these effects were small in magnitude. Despite the small magnitude, our findings may be clinically significant. For instance, gender differences in diagnostic status indicated that men are 1.5 times more likely to receive the DSM-IV specifier for circumscribed SAD (i.e., SAD limited to a small number of situations). This finding may shed light on the way SAD differentially manifests among men and women who seek treatment, and may inform therapeutic interventions (e.g., exposure hierarchies). Similarly, men were found to drop out of treatment 1.4 times more than women, which is of high clinical significance and may stress the need to enhance engagement with treatment for men more than women.

The present study has several limitations. First, our findings (e.g., women report greater severity of social anxiety) are based on self-report and may be vulnerable to reporting bias. While this concern cannot be completely obviated, findings from studies using implicit physiological data are consistent with our findings. For instance, women with SAD have been found to experience heightened physiological arousal in response to social stimuli, whereas men were not (e.g., Alvares et al., 2013). Second, the dataset of this study included DSM-IV diagnoses rather than current DSM-5 diagnoses. Thus, generalizability to current definitions should be done with caution. However, it is important to note that differences between the versions in SAD diagnosis are very modest. Third, as this study examined gender differences in group treatment for SAD, conclusions may not necessarily generalize to individual treatment. Fourth, as discussed above, differential attrition in men and women as well as regression to the mean remain alterative explanations for the finding that women respond to treatment more than men.

Despite these limitations, our findings can have implications for both assessment and treatment for SAD. First, despite reporting similar levels of impairment overall, women reported higher levels of SAD symptoms than men. If such findings continue to accrue, clinical researchers can consider using different symptom thresholds to screen for SAD and suggest a probable diagnosis of men and women. Currently, the published cutoff score for screening for SAD using the LSAS is 60 for a probable diagnosis of generalized SAD (Rytwinski et al., 2009). However, this value assumes that the same threshold applies to men and women. Our findings, taken together with many previous studies (see Asher and Aderka, 2017 for a review) suggest that using different thresholds to suggest a probable diagnosis may be considered. In addition, our findings regarding gender differences in attrition may suggest that greater resources should be devoted to attrition prevention strategies for men as they are at greater risk for attrition. Finally, utilizing strategies to enhance treatment seeking among women may also help more women with SAD receive treatment and potentially improve.

To sum, our study found gender differences in clinical severity, treatment seeking, attrition, and response to treatment in individuals with SAD. These differences can inform the literature on SAD and may have clinical implications for assessment and treatment for the disorder.

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