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Randomized Clinical Trial Evaluating the Efficacy of a Brief Intervention Targeting Anxiety Sensitivity Cognitive Concerns

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Objective: Anxiety sensitivity (AS) is a well-established, malleable risk factor for anxiety and other forms of psychopathology. Structural evaluation models of AS suggest it can be decomposed into physical, social, and cognitive concerns, and emerging work indicates that these components may be differentially related to various adverse outcomes. In particular, AS cognitive concerns have been consistently linked with suicide. Prior work has also shown that brief interventions can effectively reduce overall AS, but these treatments tend to focus on its physical subcomponent. The aim of the current investigation was to design and evaluate the efficacy of an AS treatment more specifically focused on its cognitive component. ***Method:*** Non-treatment-seeking participants ($N = 108$) with elevated AS were randomly assigned to a 1-session intervention utilizing psychoeducation and interoceptive exposure techniques to target AS or a health information control intervention and assessed posttreatment and at 1-month follow-up. ***Results:*** The active treatment condition produced significantly greater reductions in AS at posttreatment. Group differences persisted at 1-month follow-up that were specific to AS cognitive concerns. Moreover, changes in cognitive AS mediated symptom change at follow-up including suicide outcomes. ***Conclusions:*** Despite the brevity of the treatment intervention, findings demonstrate that it resulted in substantial reductions in AS cognitive concerns that were linked with symptom improvement.

Keywords: anxiety, anxiety sensitivity, suicide, prevention

Anxiety disorders represent a highly prevalent form of psychopathology often resulting in substantial disability and economic burden (Greenberg et al., 1999; Kessler, Chiu, Demler, & Walters, 2005). It has been suggested that much of the burden associated with anxiety disorders could be avoided through prevention and early intervention (Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008). While there are many well-established cognitive behavioral treatments for anxiety disorders (Chambless & Ollendick, 2001), research focused on amelioration of anxiety risk factors remains in a nascent stage (Feldner & Zvolensky, 2004). Zvolensky, Schmidt, Bernstein, and Keough (2006) have suggested a translational framework to advance risk factor treatment research. They have emphasized the importance of utilizing basic research that has identified malleable anxiety risk factors in the development of efficacious preventative interventions. For example, genetic or biological parameters such as family history of anxiety and environmental factors such as a history of trauma, though related to anxiety outcomes, are fixed risks that cannot be changed (Cougle, Timpano, Sachs-Ericsson, Keough, & Riccardi, 2010; Cromer, Schmidt, & Murphy, 2007). On the other hand, some cognitive risk factors have been found to be malleable and therefore could serve as targets for the prevention of later adverse outcomes.

One promising and malleable risk factor is anxiety sensitivity (AS). AS, otherwise known as a "fear of fear," is a well-established individual difference variable reflecting a tendency to fear bodily sensations associated with anxious arousal (Reiss & McNally, 1985). Individuals high in AS fear anxious arousal because they believe there will be a negative physical, cognitive, and/or social consequence associated with these symptoms. For example, individuals high in AS may misinterpret benign bodily sensations such as heart palpitations as being indicative of a heart attack, whereas those low in AS will simply regard the sensations as uncomfortable. Unlike trait anxiety and worry, which manifest as general tendencies to react to a broad array of situations with anxiety, AS is more specifically focused on exaggerated reactivity to stress and anxiety symptoms (Rapee & Medoro, 1994; Zvolensky, Kotov, Antipova, Leen-Feldner, & Schmidt, 2005).

The extant literature has established AS as a multidimensional construct comprising three separate dimensions reflecting fears of the physical, cognitive, and social domains of anxiety (Taylor et al., 2007). The three dimensions of AS have been found to be differentially related to various outcomes. For example, the AS cognitive concerns subscale, which refers to beliefs that anxiety-related sensations have catastrophic psychological outcomes such as going crazy or losing control of mental processes, appears to be particularly relevant to posttraumatic stress disorder (PTSD; Lang,

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Kennedy, & Stein, 2002; Vujanovic, Zvolensky, & Bernstein, 2008), depression (Cox, Enns, & Taylor, 2001; Naragon-Gainey, 2010; Taylor, Koch, Woody, & McLean, 1996) and suicide (Capron, Cougle, Ribeiro, Joiner, & Schmidt, 2012; Capron, Fitch, et al., 2012).

The specific relationship between AS and suicide can be illuminated by considering the role AS plays in response to stress. The well-established AS literature highlights the idea that AS increases distress responses in the context of general stress and anxiety symptoms. An expansion of this idea would include the possibility that AS predisposes some to show increased distress in the context of aversive physical and cognitive mood symptoms. Thus, we recently proposed a depression–distress amplification model of AS (Capron, Norr, Macatee, & Schmidt, 2013) that provides a more specific mechanism for the development of suicide relative to similar models such as the feedback model proposed by Katz, Yaseen, Mojtabai, Cohen, and Galynker (2011). In the depression–distress amplification model, suicidal ideation is considered a symptom of depression corresponding to the severity of the depression. Just as AS increases distress responses in the context of uncomfortable physical sensations (Schmidt, Maner, & Zvolensky, 2007), the depression–distress amplification model posits that AS cognitive concerns amplify distress brought on by the uncomfortable sensations experienced in the context of emerging or existing dysphoria (e.g., lack of concentration, insomnia, anhedonia). Suicidal ideation emerges when the distress caused by the amplified depression reaches severe levels.

A separate line of evidence has revealed that AS is malleable through cognitive-behavioral interventions among patients with anxiety disorders. Several research investigations focused on panic disorder treatment have reported significant reductions in AS following treatment (Barlow, Craske, Cerny, & Klosko, 1989; Schmidt et al., 2000; Telch et al., 1993; Westling & Öst, 1999). This work has led to investigations specifically focused on the reduction of AS as a preventative intervention in nonclinical, at-risk samples. Successful preventative work on AS has included 2-hr psychoeducation groups (Feldner et al., 2008), single-day workshops (Gardenswartz & Craske, 2001), and 6-week exercise programs (Broman-Fulks & Storey, 2008).

To date, the largest AS-focused intervention was conducted by Schmidt and colleagues (2007). Participants (N = 404) with Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) scores 1.5 *SD* above the nonclinical mean (Schmidt & Joiner, 2002) were randomly assigned to either the anxiety sensitivity amelioration training (ASAT) condition or a control condition based on health and nutrition. The ASAT condition consisted of a 30-min computer PowerPoint presentation followed by 10 min with an experimenter. The presentation explored the following concepts: the nature of stress, AS, myths about the harmfulness of physiological arousal, and interoceptive exposure (IE). Results indicate that both conditions produced a reduction in AS; however, the ASAT condition produced a significantly larger reduction in AS than did the control condition (30% vs. 17%, respectively). The reduction in AS was primarily due to a reduction in the physical AS subfactor. The social subfactor showed a small but significant reduction, whereas the cognitive subfactor did not. In terms of the development of psychopathology, those in the ASAT condition showed a lower incidence of Axis I diagnoses (First, Spitzer, Gibbon, & Williams, 1994) during the 2-year follow-up period.

Recently, an augmented version of ASAT was developed in an attempt to increase its potency (Keough & Schmidt, 2012). The revised protocol, anxiety sensitivity education and reduction training (ASERT), included more interaction with a therapist, more intensive IE exercises, and more rigorous homework requirements. The level of overall AS reduction was substantial in the active ASERT group (close to 60% at 1-month follow-up), and unlike ASAT, ASERT produced significant reductions in all three AS components. A 6-month follow-up assessment indicated that the treatment group retained the majority of their AS reduction, whereas the control group retained their elevated AS scores.

There is now emerging evidence that AS can be effectively mitigated, even with brief one-session treatments that require minimal therapist or experimenter involvement. However, a number of limitations in this literature suggest important opportunities for additional work. First, all of the prior AS reduction interventions, including even the brief ASAT and ASERT protocols, have utilized an experimenter/facilitator. Development of completely autonomous computer-administered AS interventions would be extremely useful to increase dissemination, since such protocols could be delivered to anyone with computer access. Second, the vast majority of prior work on AS has focused on the physical concerns component of this construct. However, emerging evidence suggests that AS cognitive concerns may be particularly relevant to certain forms of psychopathology, such as suicide. Therefore, an important extension of this work would be to attempt to bolster elements of the preventative intervention in order to more specifically target the AS cognitive domain. Finally, only one prior study has examined the impact of an AS intervention on psychopathology (e.g., Schmidt, Eggleston, et al., 2007), and no prior studies have explored mechanisms through which an AS intervention would impact anxiety and related psychopathology.

The present study was designed to evaluate the potency of an AS reduction protocol more specifically focused on AS cognitive concerns. To accomplish this, the new intervention increased focus on exaggerated fears related to cognitive dyscontrol. The cognitive anxiety sensitivity treatment (CAST) intervention was designed along the same lines as ASAT and ASERT, but it was programmed using more sophisticated software to allow for the use of audio and video presentations, as well as certain interactive features. Thus, the protocol was designed to be autonomous to increase the ease of dissemination. The primary study hypothesis was that CAST, compared to a health information control condition delivered using a similar format, would yield greater reductions in overall AS, as well as the cognitive AS subfactor. It was also anticipated that AS risk reductions would be maintained across the 1-month follow-up period. While risk reduction was expected to be maintained during follow-up, neither the length of the follow-up period nor the size of the sample allowed for an examination of preventative benefits such as the incidence of new psychopathology. However, we hypothesized that changes in AS would mediate changes in symptoms during the follow-up interval.

Method

Participants

Participants (N = 108) were recruited from the general community. The primary inclusionary criterion was some evidence of

AS indicated by scoring at or above the community sample mean on the Anxiety Sensitivity Index–3 (ASI-3; Taylor et al., 2007). In terms of exclusionary criteria, similar to prior work (Schmidt & Joiner, 2002), participants were excluded if they demonstrated evidence of a significant medical illness that would prevent the completion of IE exercises. Such conditions include significant cardiovascular disease, respiratory disorders, renal disease, epilepsy, stroke, and uncontrolled hypertension or migraines. We obtained a medical release from and consulted with the participant's physician for those screened to have any medical conditions of this nature. Other exclusionary criteria included no evidence of serious suicidal intent that would indicate a need for hospitalization or immediate treatment, no evidence of current substance abuse, and no evidence of current or past psychotic-spectrum disorders or uncontrolled bipolar disorder. Since comprehension of the intervention is critical, participants were also English speakers. Only those over the age of 18 were recruited for participation in the proposed project. The ages of participants ranged from 18 to 87 ($M = 40.80$, $SD = 17.45$), and gender was fairly evenly distributed (46.2% males). The sample was primarily Caucasian (67.9%), with 20.8% African American, 3.8% Hispanic, 0.9% Asian, and 6.6% Other (e.g., biracial).

Assessments

Diagnostic interview.

Structured Clinical Interview for DSM–IV, Non-patient Version (SCID/NP). All psychiatric diagnoses were determined using the SCID/NP (First et al., 1994). The SCID/NP was administered by trained doctoral level therapists who completed extensive training in SCID/NP administration and scoring. The training included reviewing SCID/NP training tapes, observing live SCID/NP administrations, and conducting practice interviews with other trained individuals. Throughout the training process, all trainees received feedback until they demonstrated high levels of reliability. In addition, all SCID/NP results were reviewed by a licensed clinical psychologist to ensure accurate diagnoses. Percentage agreement between clinical interviewers for a random sample of approximately 15% of these SCID/NP interviews resulted in high interrater agreement (e.g., over 80% with a kappa of .77).

Self-report measures.

Anxiety Sensitivity Index–3 (ASI-3). The ASI-3 (Taylor et al., 2007) was used to measure AS as well as its subfactors (physical, social, and cognitive concerns). The measure has shown good psychometric properties (Taylor et al., 2007). In the current study, the subscales demonstrated good internal consistency (cognitive $\alpha = .94$, social $\alpha = .88$, and physical $\alpha = .89$).

Beck Anxiety Inventory (BAI). The BAI was used to measure general anxiety symptomatology (Beck, Epstein, Brown, & Steer, 1988). The BAI has been widely used and shown to be both valid and reliable in clinical samples (coefficient alpha = .92) and nonclinical samples (coefficient alpha = .91; Beck et al., 1988; Borden, Peterson, & Jackson, 1991). The BAI demonstrated excellent internal consistency in the present investigation ($\alpha = .95$).

Beck Depression Inventory–II (BDI-II). The BDI-II was used to assess depressive symptomatology (Beck, Steer, & Brown, 1996). This measure has been shown to be valid and reliable among college and clinical samples (Endler, Rutherford, & Denisoff, 1999). Within the current study, the BDI-II demonstrated excellent internal consistency ($\alpha = .90$).

Beck Suicide Scale (BSS). The BSS was used to assess various behaviors and attitudes related to suicide risk including suicidal ideation, plans and/or preparations, and past attempts (Beck & Steer, 1991). Within the current investigation, the BSS demonstrated good internal consistency ($\alpha = .83$).

Depressive Symptom Inventory–Suicide Subscale (DSI-SS). The DSI-SS (Metalsky & Joiner, 1997) was used as an additional measure of suicidal ideation during the past 2 weeks. The frequency and intensity of suicidal thoughts are reported on a 4-point Likert type scale ranging from 0 to 3, with higher scores indicating more severe depressive symptoms. The DSI-SS has been shown to have good validity and psychometric properties (e.g., Joiner, Pfaff, Acres, & Johnson, 2002; Joiner & Rudd, 1996). Internal consistency in the current investigation was good ($\alpha = .85$).

Demographic and Medical Screening Questionnaire. This scale was created to collect data on the participants' gender, ethnicity, educational/occupational level, and current medical conditions and medications. It was administered during the screening appointment to ensure participant eligibility.

Procedure

Time points. All procedures were approved by the university's institutional review board. Participants who met study entry criteria presented for evaluation. Diagnostic assessment was based on an initial phone screening interview followed by a face-to-face structured clinical interview using the SCID/NP (First et al., 1994).

Screening appointment. Participants first read and signed informed consent that ensured confidentiality, thoroughly outlined their proposed study involvement, and emphasized that they could discontinue their participation at any time, for any reason, and at absolutely no penalty. They then completed the SCID/NP and the medical screening questionnaire. If the participant did not meet all entry criteria, they were debriefed, thanked for their time, and awarded any monetary compensation that they earned. Those who met entry criteria were randomly assigned, based on a random numbers table, to one of the two intervention conditions (see description of experimental conditions) and scheduled for their intervention appointment.

Intervention appointment. Participants completed the preintervention assessment measures followed by their assigned intervention and the postintervention questionnaires. Participants were then scheduled for their 1-month follow-up appointment.

Month 1: Follow-up appointment. Upon arrival at the laboratory offices, participants were directed to an individual testing room, where they completed the self-report questionnaires. Following this, individuals in the control condition were given the opportunity to receive the CAST protocol, which was delivered at that time or scheduled for a later time.

Description of experimental conditions.

Cognitive anxiety sensitivity treatment (CAST). The CAST intervention was programmed using Articulate Presenter and included audio and video along with some interactive features. This training intervention was developed to closely model the educational and behavioral techniques that are commonly employed in the treatment of individuals with anxiety disorders. Specifically, the educational component of this condition was

adapted from the AS intervention used by Schmidt, Eggleston, et al. (2007). The CAST intervention consists of 50 screens containing information. Audio narration runs throughout. There are four "quizzes" interspersed throughout the program in order to increase comprehension of important material. A quiz consists of one item (e.g., "Everyone has stress sensitivity") along with the option to respond true or false. Incorrect responses result in corrective information, and correct responses are reinforced (e.g., "That's right! You are correct"). The first few screens focus on providing a rationale for the intervention followed by psychoeducation describing the nature of stress and its effects on the body, including the potential for developing "stress sensitivity" that causes some to overreact to stress symptoms. The program is designed to dispel exaggerated thoughts regarding the immediate dangers of stress on the body. Unlike prior AS reduction programs, CAST specifically attends to sensations and feared consequences associated with elevated cognitive AS (e.g., "I will lose my mind"). Participants are taught that the physiological arousal associated with stress is not dangerous and that they may have developed a conditioned fear to those arousal sensations, which is indicated by their elevated AS score. A rationale for IE exercises, designed to correct the conditioned fear to these bodily sensations, is provided. These exercises involve repeated exposure to a feared bodily sensation until the fear dissipates. The program guides participants through a repeated hyperventilation IE exercise. Hyperventilation is an excellent choice for targeting AS cognitive concerns because many sensations that elicit these fears (e.g., light-headedness, derealization) are brought on during hyperventilation. First, the program demonstrates hyperventilation via a brief video. Next, participants complete a hyperventilation exercise in which they are instructed to follow along with a video in which the demonstrator is breathing at a rate of one breath every 2 s for a total of 60 s. After the trial, participants rate the level of distress/fear and intensity of sensations experienced during each exercise using a 10-point scale. The program graphically depicts responses to the exercise over time to provide feedback and increase awareness of the desired outcome, that is, extinction of the distress response. The program instructs participants to attempt to keep sensation intensity consistent across trials. The protocol also included examples of four other IE exercises that participants review. Participants are encouraged to practice these exercises on their own in order to further address their stress sensitivity. The duration of CAST is approximately 45 min.

Physical health education training (PHET). In the PHET condition, participants spend approximately the same amount of time (45 min) with a program that presents information regarding the importance and benefits of a healthy lifestyle and provides guidelines to achieve a healthy lifestyle. The program covers the following topics during the session: diet, alcohol, water consumption, exercise, sexual health, good hygiene, stress management, life organization, social support, positive outlook, and sleep.

Data Analytic Procedure

A latent difference score approach, using growth curve modeling (Mara et al., 2012; Mun, von Eye, & White, 2009) was used to model the effects of the intervention on changes in ASI-3 levels from preintervention to postintervention as well as on changes from postintervention to Month 1 follow-up. This approach was used over more traditional methods such as analysis of variance for several reasons. This approach reduced the number of analyses, as a single model could be used to examine differences from pre- to postintervention and from postintervention to Month 1 follow-up. In addition, growth curve modeling has more power to detect treatment effects than does traditional analysis of variance-based methods, is robust to nonnormality, and provides additional information, such as individual variability and fit statistics, not available in traditional methods (Curran, West, & Finch, 1996; B. O. Muthén & Curran, 1997).

Conceptually, our models were similar to the proposed conditional model of Mun et al. (2009) using the postintervention as the referent point (i.e., intercept). Models were identified by fixing the residual variances in change from pre- to postintervention (Change 1) and from postintervention to Month 1 follow-up (Change 2). Mara et al. (2012) demonstrated improved modeling power when baseline performance was covaried out. Therefore, baseline ASI-3 levels were also included as a covariate. Figure 1 is an illustration of the model that was used. Models were fit in Mplus Version 5.1 (L. K. Muthén & Muthén, 2008) using full information maximum likelihood and the Yuan-Bentler scaled chi-squared index (Y-B χ^2) to adjust standard errors for nonnormality and nonindependence. Overall model fit was primarily determined by the Y-B χ^2. A nonsignificant value indicated that the model provided good fit to the data (Kline, 2011). The comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA) were also examined. In general, CFI values greater than .95 indicate good fit. RMSEA values below .05 indicate good fit, although RMSEA tends to overly reject models as poor-fitting at small sample sizes (Hu & Bentler, 1999; MacCallum, Browne, & Sugawara, 1996). Whereas additional model fit statistics are provided, a nonsignificant χ^2 value is generally considered the best test of model fit, as it is the only *statistic* of model fit, and all other fit indices are based on this statistic (Barrett, 2007; Kline, 2011). To examine differences between conditions, parameters were restricted to equality and the χ^2 difference test was used to compare the restricted model to the unconstrained model. A significant difference indicated that the model parameters varied significantly between conditions. Effect sizes were evaluated for the difference in change from pre- to

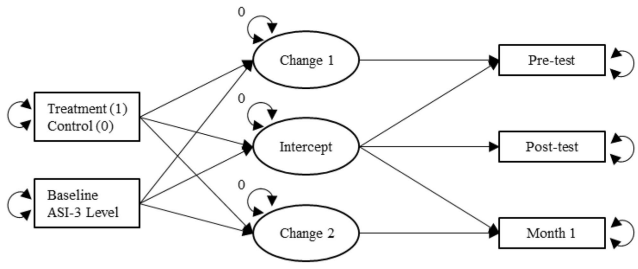

Figure 1. Latent difference score model for Anxiety Sensitivity Index–3 (ASI-3) scale and subscale models from preintervention to postintervention (i.e., Change 1) and from postintervention to Month 1 follow-up (i.e., Change 2).

postintervention. Effect sizes were adapted from the formula provided by Feingold (2009) for *d*, for which the difference in change from pre- to postintervention between CAST and PHET was divided by the pooled standard deviation.

Although the intervention was targeted toward amelioration of ASI-3 levels, the effect of the intervention on BAI, BDI-II, BSS, and DSI-SS scores, collected at the Month 1 follow-up, was examined as well. It was expected that the effect of treatment would influence psychopathology through Month 1 ASI-3 cognitive concerns subscale scores. Mediation models were conducted using maximum-likelihood estimation and bias-corrected bootstrapped confidence intervals (CIs) for indirect effects, with 5,000 bootstrap samples to provide consistent and replicable results (Preacher & Hayes, 2008). The bootstrap method is preferable to other mediation approaches because the asymmetric CIs can provide an optimal balance between power and Type I error.

Results

Sample and Preliminary Analysis

The sample was divided roughly equally between individuals assigned to the CAST (treatment) condition (n = 55, 52%) and individuals assigned to the PHET (control) condition (n = 51, 48%) at baseline (see Figure 2 CONSORT diagram). There was one individual from each condition who did not complete the intervention and was therefore not included in any analyses. The intervention was conducted on 54 individuals in the CAST con-

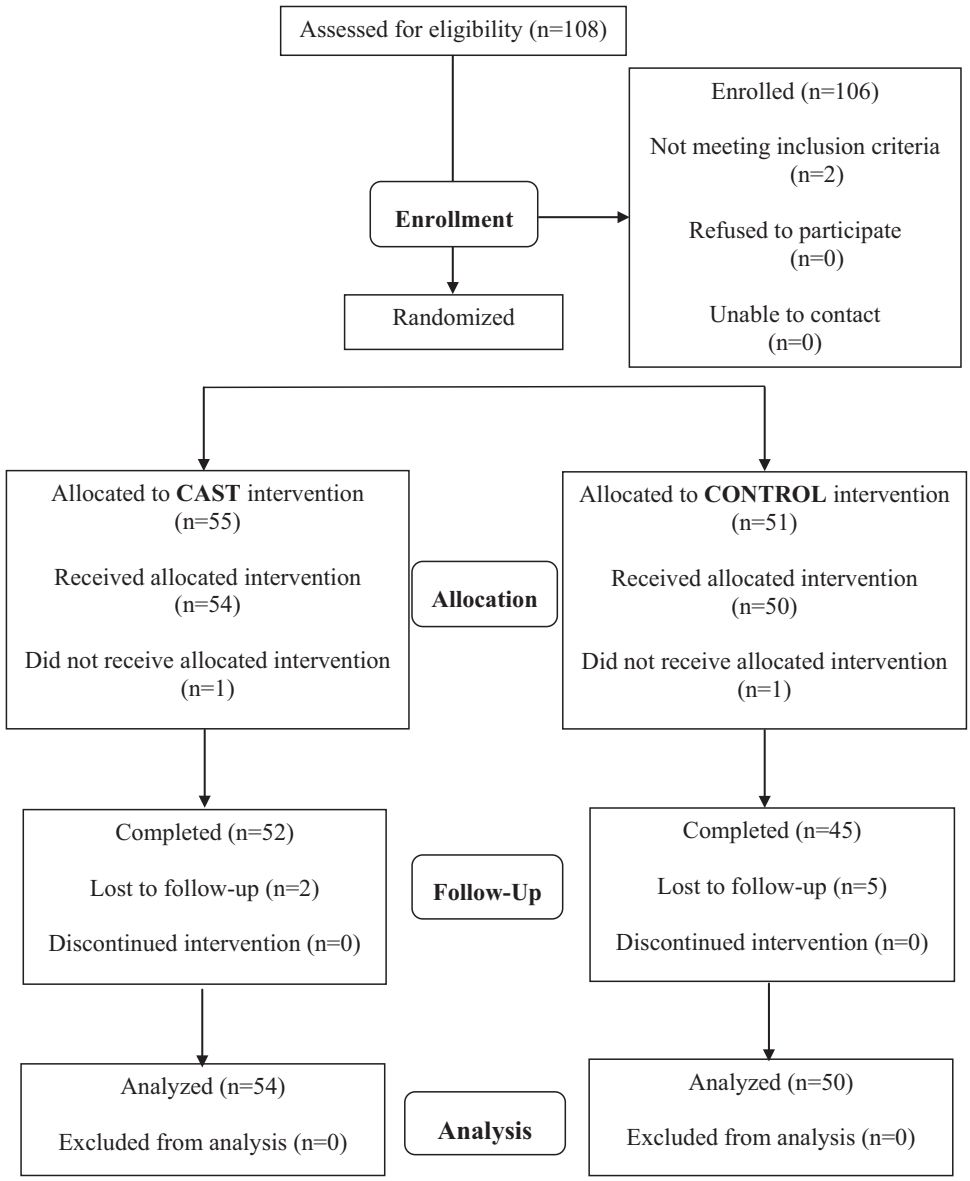

Figure 2. CONSORT chart of participants detailing patient flow, assignment, and dropout. CONSORT = consolidated standards of reporting trials.

dition and 50 individuals in the PHET condition. There were nine individuals, three from the CAST and six from the PHET conditions, who did not have Month 1 follow-up information available. No differences were found between individuals who completed Month 1 follow-up and individuals who did not for age, $F(1, 102) = 0.32$, $p > .05$; sex, $\chi^2 = 0.97$ (1, $N = 104$), $p > .05$; or race, $\chi^2 = 2.64$ (4, $N = 104$), $p > .05$, or for preintervention ASI-3 total score, $F(1, 102) = 0.00$, $p > .05$; physical concerns subscale score, $F(1, 102) = 0.62$, $p > .05$; cognitive concerns subscale score, $F(1, 102) = 0.87$, $p > .05$; or social concerns subscale score, $F(1, 102) = 0.08$, $p > .05$.

Means of study variables, primary diagnoses, and demographic variables by treatment condition are provided in Table 1. Comparing variables across CAST and PHET conditions indicated that individuals in the CAST condition had significantly higher scores at preintervention than did individuals in the PHET condition on the ASI-3 physical concerns subscale. There were no other significant differences.

Table 1
Descriptive Statistics for Preintervention Measures by Treatment Condition

Variable	CAST		PHET		
	M	*SD*	*M*	*SD*	*F*
ASI-3					
Total	34.04	16.84	28.76	15.33	2.78
Physical concerns	10.56	6.41	7.86	6.08	4.82*
Cognitive concerns	10.85	7.26	10.34	6.86	.14
Social concerns	12.63	6.00	10.56	6.15	3.02
BAI	22.15	14.37	19.78	13.01	.77
BDI-II	24.11	13.00	24.14	11.90	.00
BSS	2.09	4.09	1.62	3.31	.42
DSI-SS	.65	1.34	.63	1.36	.01
Age	40.00	17.93	41.67	17.04	.16
	%		%		χ^2
Primary disorders					
Panic disorder	9		2		2.09
Specific phobia	2		2		0.003
Social anxiety disorder	9		16		1.06
OCD	6		2		0.82
PTSD	17		16		0.01
Generalized anxiety	11		8		0.29
Anxiety NOS	6		8		0.25
MDD	9		16		1.06
Alcohol use disorders	0		6		0.00
Other	8		6		0.08
Sex					0.18
Male	48		44		
Female	52		56		
Race					2.94
Caucasian	70		64		
African American	20		22		
Latino	2		6		
Asian	0		2		
Other	7		6		

Note. CAST $N = 54$, PHET $N = 50$ (for DSI-SS: CAST $N = 51$, PHET $N = 48$). CAST = cognitive anxiety sensitivity treatment; PHET = physical health education training; ASI-3 = Anxiety Sensitivity Index–3; BAI = Beck Anxiety Index; BDI-II = Beck Depression Index–II; BSS = Beck Suicide Scale; DSI-SS = Depressive Symptom Inventory–Suicidality Subscale; OCD = obsessive compulsive disorder; PTSD = posttraumatic stress disorder; NOS = not otherwise specified; MDD = major depressive disorder. ASI-3 measures are from preintervention. All other data are from baseline.
* $p < .05$.

Latent Difference Score Models of ASI-3 Total and Subscales

Unconstrained latent difference score models for ASI-3 total scores, physical concerns, cognitive concerns, and social concerns subscale scores were fit using multigroup analysis. The concordant baseline ASI-3 score or subscale score was centered and included as a predictor. Model fit indices are provided in Table 2. The fully unconstrained ASI-3 physical concerns and cognitive concerns models initially demonstrated significant χ^2 values indicating model misfit. Modification indices supported adding correlated residuals between postintervention and Month 1 ASI-3 subscale score. Given that it makes sense empirically for these subscales to be correlated over time, correlated residuals were included in the models. All models demonstrated good model fit, as demonstrated by a nonsignificant χ^2. There was an elevated RMSEA value for the ASI-3 cognitive concerns model. However, all other fit indices demonstrated good fit, and there is some evidence that the RMSEA performs poorly in models with few degrees of freedom (e.g., Hu & Bentler, 1999; Kenny, 2010). Therefore, it was determined that the latent difference score models of ASI-3 total and subscale scores fit the data well.

Parameter estimates for the latent difference score models are provided in Table 3. Mean scores from preintervention to Month 1 follow-up were plotted for treatment and control conditions separately for all models. Intercept values provided in Table 3 are essentially postintervention means, controlling for baseline ASI-3 scores. The ASI-3 total score was reduced by 10.40 ($p < .001$) from pre- to postintervention in the CAST condition and was not significantly reduced in the PHET condition, a result that was significantly different between conditions (χ^2 difference = 18.44, $p < .001$), with a *d* of 0.57. The ASI-3 physical concerns score was significantly reduced by 4.38 ($p < .001$) in the CAST condition, which was significantly different from the nonsignificant reduction in the PHET condition (χ^2 difference = 20.45, $p < .001$), with a *d* of 0.64. The ASI-3 cognitive concerns score was significantly reduced by 3.38 ($p < .001$) in the CAST condition, which was also significantly different from the nonsignificant reduction in the PHET condition (χ^2 difference = 5.37, $p < .05$), with a *d* of 0.38. In the cognitive concerns model, the intercept was also significantly lower in the CAST condition compared to the intercept in the PHET condition (χ^2 difference = 4.32, $p < .05$), indicating that not only was there a significant reduction in ASI-3 cognitive concerns in the CAST condition compared to the reduction in ASI-3 cognitive concerns in the PHET condition, but also this difference resulted in significantly lower cognitive concerns scores postintervention for those in the CAST condition compared to cognitive concerns scores for those in the PHET condition. The ASI-3 social concerns score was significantly reduced by 2.62 ($p < .001$) in the CAST condition, which was significantly different from the nonsignificant reduction in the PHET condition (χ^2 difference = 13.42, $p < .001$), with a *d* of 0.41. Across all models, there were no significant changes from ASI-3 scores from postintervention to Month 1 follow-up.

To examine whether the differences in ASI-3 scores were significant at Month 1 follow-up, latent difference score models with the intercept centered at Month 1 follow-up were examined. There were no significant differences between conditions at Month 1 follow-up for ASI-3 total scores, physical concerns scores, or social concerns scores. However, there was a significant difference between conditions for ASI-3 cognitive concerns (χ^2 difference = 11.31, $p < .001$) such that individuals in the CAST condition had a lower score at Month 1 follow-up than did individuals in the PHET condition.[1]

Mediation Analyses Examining the Effects of Treatment on Anxiety, Depression, and Suicidality

Baseline and Month 1 BSS and DSI-SS scores were log-transformed prior to analysis to account for the skewed nature of these variables. Mediation analysis was conducted examining the effect of treatment through Month 1 ASI-3 total scores, physical concerns, cognitive concerns, and social concerns on Month 1 BAI, BDI-II, BSS, and DSI-SS scores. Independent models were included for each ASI-3 score as a mediator for BAI, BDI-II, BSS, and DSI-SS scores. Baseline BAI, BDI-II, BSS, and DSI-SS were included as control variables in their respective model. Baseline ASI-3 scores were included as control variables in their respective ASI-3 model as well. Table 4 contains effects and 95% bootstrapped confidence intervals (CIs). Analyses for ASI-3 total score, physical concerns, and social concerns as mediators revealed no significant mediation effects. Significant mediation effects were found such that ASI-3 cognitive concerns mediated the relations between treatment condition and BAI, BDI-II, BSS, and DSI-SS.

Discussion

The primary aim of the current investigation was to examine the potency of a risk amelioration intervention focused on the cognitive aspects of AS (i.e., fears of mental dyscontrol). At the same time, we sought to increase the autonomy of the intervention by utilizing an interactive computer program that did not require therapist or experimenter participation. Findings indicated that despite the health information control condition being a somewhat active control group, the CAST group demonstrated significantly greater reductions in AS immediately following treatment. Unlike the recent trial (Keough & Schmidt, 2012), which resulted in reductions across all three subscales over time, participants in the CAST group reported a significantly lower AS, specific to the cognitive subscale, across the Month 1 follow-up period. Consistent with expectation, this was the first AS intervention trial demonstrating that changes to one AS domain (cognitive concerns) are possible and that changes to cognitive AS mediate changes in suicide parameters over time.

While it may be argued that reductions across all AS domains are likely to be beneficial for the prevention of anxiety psychopathology (Schmidt, Eggleston, et al., 2007), data from some prior work suggest that treatments specifically focused on AS cognitive concerns may be ideal for addressing suicide risk. Whereas in-

Table 2
Model Fit Statistics for Latent Difference Score Models of ASI-3 Scale and Subscales Testing Intercept and Change Parameters for Equality

Model	Y-B χ^2	*df*	*p*	CFI	RMSEA	Y-B χ^2 Δ
ASI-3 total	13.77	7	.06	.98	.14	
Intercept equal	16.80	8	.03	.98	.15	3.15
Change 1 equal	29.45	8	.00	.93	.23	18.44***
Change 2 equal	14.40	8	.07	.98	.12	0.21
ASI-3 physical concerns	11.99	6	.06	.98	.14	
Intercept equal	14.50	7	.04	.97	.14	2.61
Change 1 equal	30.46	7	.00	.90	.25	20.45***
Change 2 equal	12.09	7	.10	.98	.12	0.004
ASI-3 cognitive concerns	9.25	6	.16	.99	.10	
Intercept equal	13.09	7	.07	.98	.13	4.32*
Change 1 equal	14.67	7	.04	.97	.15	5.37*
Change 2 equal	9.53	7	.22	.99	.08	0.03
ASI-3 social concerns	7.57	7	.92	1.00	.04	
Intercept equal	7.67	8	.94	1.00	.00	0.23
Change 1 equal	17.89	8	.89	.97	.15	13.42***
Change 2 equal	9.73	8	.92	.99	.06	2.21

Note. ASI-3 = Anxiety Sensitivity Index–3; Y-B χ^2 = Yuan-Bentler scaled chi-square; CFI = comparative fit index; RMSEA = root-mean-square error of approximation.
* $p < .05$. *** $p < .001$.

Table 3
Model Parameters for Latent Difference Score Model of ASI-3 Controlling for Baseline ASI-3 Across CAST and PHET Conditions

	CAST		PHET	
Parameters	*M*	*SE*	*M*	*SE*
ASI-3 total				
Intercept	23.64***	1.56	27.51***	1.77
Intercept variance	17.92**	6.92	39.16***	10.30
Change 1	10.40***	1.74	1.26	1.59
Change 2	−0.25	1.57	−1.25	1.45
ASI-3 physical concerns				
Intercept	6.18***	0.63	7.49***	0.59
Intercept variance	2.92	1.71	5.75**	1.91
Change 1	4.38***	0.74	0.37	0.53
Change 2	−0.20	0.71	−0.23	0.46
ASI-3 cognitive concerns				
Intercept	7.47***	0.73	9.65***	0.70
Intercept variance	3.37	1.79	7.53***	1.97
Change 1	3.38***	0.83	0.69	0.71
Change 2	−0.27	0.66	−0.09	0.66
ASI-3 social concerns				
Intercept	10.01***	0.53	10.43***	0.73
Intercept variance	3.24***	1.04	7.19***	1.73
Change 1	2.62***	0.61	0.13	0.55
Change 2	0.19	0.59	−0.99	0.57

Note. ASI-3 = Anxiety Sensitivity Index–3; CAST = cognitive anxiety sensitivity treatment; PHET = physical health education training.
** $p < .01$. *** $p < .001$.

[1] Given the elevated RMSEA in the reported models, results were verified by examining analyses of covariance on difference scores from pre- to postintervention, controlling for baseline ASI-3 scores as well as difference scores from postintervention to Month 1 follow-up, controlling for baseline ASI-3 scores. There were no differences in the pattern of findings using this approach.

Table 4
Mediation Models of Treatment Condition and ASI-3 Total Score and Subscale Score Models on Psychopathology

	ASI-3 total			Physical concerns			Cognitive concerns			Social concerns		
		95% CI			95% CI			95% CI			95% CI	
Variable	*B*	LL	UL	*B*	LL	UL	*B*	LL	UL	*B*	LL	UL
Beck Anxiety Index												
Baseline	**0.46**	**0.27**	**0.66**	**0.52**	**0.35**	**0.68**	**0.55**	**0.38**	**0.74**	**0.59**	**0.38**	**0.79**
Condition	0.06	−3.53	3.55	−0.07	−3.77	3.52	−0.37	−4.39	3.33	−1.48	−5.35	2.17
Month 1 ASI-3 score	**0.37**	**0.20**	**0.54**	**0.86**	**0.39**	**01.34**	**0.64**	**0.31**	**0.95**	0.42	−0.04	0.93
Indirect effect	−1.41	−3.76	0.13	−1.21	−3.46	0.06	**−1.31**	**−3.22**	**−0.13**	−0.19	−1.76	0.42
Beck Depression Index–II												
Baseline	**0.45**	**0.26**	**0.70**	**0.59**	**0.39**	**0.80**	**0.49**	**0.31**	**0.70**	**0.66**	**0.44**	**0.90**
Condition	1.96	−2.52	6.09	1.56	−3.08	5.90	1.90	−2.48	5.97	1.52	−6.08	7.59
Month 1 ASI-3 score	**0.31**	**0.11**	**0.48**	0.49	−0.02	0.95	**0.62**	**0.28**	**0.98**	0−.32	−2.75	2.06
Indirect effect	−1.19	−3.35	0.06	−0.68	−2.55	0.07	**−1.27**	**−3.20**	**−0.15**	−0.78	−6.86	5.21
Beck Suicide Scale												
Baseline	**0.66**	**0.48**	**0.85**	**0.69**	**0.50**	**0.87**	**0.64**	**0.47**	**0.82**	**0.68**	**0.50**	**0.88**
Condition	−0.09	−0.33	0.14	−0.10	−0.36	0.14	−0.07	−0.30	0.15	−0.11	−0.35	0.12
Month 1 ASI-3 score	0.01	−0.001	0.02	0.004	−0.02	0.03	**0.02**	**0.004**	**0.04**	0.01	−0.01	0.03
Indirect effect	−0.02	−0.10	0.002	−0.01	−0.07	0.02	**−0.04**	**−0.13**	**−0.01**	−0.003	−0.04	0.01
DSI-SS												
Baseline	**0.40**	**0.16**	**0.67**	**0.44**	**0.18**	**0.71**	**0.40**	**0.16**	**0.67**	**0.39**	**0.16**	**0.68**
Condition	−0.01	−0.21	0.20	−0.02	−0.23	0.20	−0.01	−0.21	0.20	−0.03	−0.23	0.19
Month 1 ASI-3 score	**0.01**	**0.001**	**0.02**	0.01	−0.01	0.03	**0.02**	**0.01**	**0.04**	0.01	0.00	0.03
Indirect effect	−0.03	−0.10	0.00	−0.01	−0.07	0.01	**−0.04**	**−0.13**	**−0.003**	−0.01	−0.05	0.01

Note. ASI-3 = Anxiety Sensitivity Index–3; DSI-SS = Depressive Symptom Inventory–Suicidality Subscale; CI = confidence interval; LL = lower limit; UL = upper limit. All significant results are in bold. Effects of treatment condition and baseline ASI-3 scores are omitted from models.

creased cognitive concerns appear to elevate risk, potentially through amplified responses to depressed mood distress (Capron, Norr, Macatee, & Schmidt, 2013), elevated physical concerns may reduce suicide risk for some individuals given the anticipated physical distress that would likely occur during a suicide attempt (Capron, Cougle, et al., 2012; Capron, Kotov, & Schmidt, 2013). While these more complex relations among AS dimensions and suicide outcomes require further study, these data indicate that CAST shows promise as a suicide prevention intervention.

Direct comparisons across AS intervention trials are complicated by a host of factors, including different follow-up time points, AS measures, levels of baseline AS, and intensity of treatment. Remaining cognizant of the imperfect nature of such a comparison, it is still useful to evaluate the efficacy of the current trial in light of AS intervention trials that have preceded it. To assist comparison between trials, percentile decreases in AS score were calculated as well as Cohen's *d*s to provide an effect of treatment relative to the control condition. The reduction in total AS scores seen in the current study was 32%, with a *d* of 0.57 at posttreatment, and this remained at 32% ($d = 0.59$) at follow-up. This level of amelioration is comparable to the original ASAT trial, which evidenced a 30% overall reduction and a similar effect size (Schmidt, Eggleston, et al., 2007). Other AS intervention protocols show similar reductions of 34% ($d = 0.29$; Feldner et al., 2008), 43% ($d = 0.20$; Gardenswartz & Craske, 2001), and 38% ($d = 1.91$; Broman-Fulks & Storey, 2008), although it should be noted that the effect size in Broman-Fulks and Storey (2008) should be interpreted with caution given their small sample size (*n*s = 12 for treatment and control conditions) and somewhat anomalous finding of increases in AS at the end of treatment among their control group. The reductions in AS in the current intervention are somewhat less than our more intensive AS treatment (Keough & Schmidt, 2012), which yielded a 28% reduction, with a *d* of 0.75, at posttreatment that increased to a 58% reduction, and a *d* of 1.46, at Month 1 follow-up.

With regard to AS cognitive concerns specifically, the current protocol reduced these fears by 34% ($d = 0.38$) from pretreatment to posttreatment and 32% ($d = 0.32$) from pretreatment to Month 1 follow-up. This was superior to our original trial, which yielded a reduction of 27% in cognitive concerns. In that trial, the effect size estimate actually favored the control condition ($d = -0.11$), but this was most likely due to the low base rate of AS cognitive concerns in this sample (i.e., preintervention means of 1.1 for the treatment condition and 1.4 for the control condition; Schmidt, Eggleston, et al., 2007). In our more intensive AS reduction protocol, we also reported a comparable reduction of 29% in cognitive concerns ($d = 0.36$) at posttreatment, although the reduction from pretreatment to Month 1 follow-up (58%, $d = 1.46$) was greater (Keough & Schmidt, 2012). It should be noted that CAST required significantly less time to complete than did any of these other interventions, and it was the only intervention to date designed to be completed via computer with no clinician support. Therefore, we suggest that in terms of balancing efficacy and efficiency, CAST is well positioned relative to these previous AS amelioration trials.

Unlike many prior AS reduction studies, the current study used a community sample that included those with current and past Axis I psychopathology. Thus, this trial would not be considered a primary prevention intervention, at least for the majority of the sample. Indeed, the severity of psychopathology in the current group was considerable, with 78.8% (82/104) meeting for a current Axis I condition. This strikingly high rate of psychopathology for

individuals selected for AS cognitive concerns highlights the association between elevated cognitive AS and a range of psychopathology. In a recent trial with college students, the AS reduction intervention worked comparably for those with and without an Axis I diagnosis (Keough & Schmidt, 2012). CAST also yielded a comparable rate of AS reduction regardless of the presence of Axis I psychopathology. For those in the treatment condition, the change from pre- to postintervention on the AS total score was 8.41 for those without an Axis I diagnosis ($n = 13$) and 11.29 for those with an Axis I diagnosis ($n = 41$). Similarly, the change from pre- to postintervention on the AS cognitive subscale was 2.96 for those without an Axis I diagnosis ($n = 13$) and 3.52 for those with an Axis I diagnosis ($n = 41$). This pattern of effects suggests that CAST may have benefits as a primary or secondary prevention intervention (Feldner et al., 2008).

This study should be considered in light of its limitations and opportunities for subsequent research. The primary limitation was the short follow-up, which did not allow us to assess for the effects on diagnoses, including the remission of Axis I psychopathology. In particular, we are interested in the effects of CAST on suicide risk. A much larger sample, followed at considerably longer intervals, would have been needed to adequately evaluate effects on suicide attempts or completed suicide. However, we feel that the follow-up period is adequate to see preliminary evidence of clinical significance. Appropriate follow-up periods should be considered in the context of the treatment being delivered. The "dose" of CAST is so low (<1 hr, no clinician) that showing significant reductions after Month 1 demonstrates individuals were positively affected. We should also note that similar interventions have demonstrated a maintenance of AS reductions for up to 2 years (Schmidt, Eggleston, et al., 2007). Another limitation is the high rate of Axis I psychopathology in the current sample, which precludes an evaluation of CAST as a primary prevention intervention. The reliance on self-report measures to assess the mediation effects of AS on psychopathology is a further limitation that could be ameliorated if there were a longer follow-up, allowing for examination of Axis I psychopathology development.

This investigation provides important information regarding the amelioration of a well-established anxiety risk factor linked to a wide range of psychopathology including suicide (Capron, Blumenthal, et al., 2012; Capron, Fitch, et al., 2012; Capron, Gonzalez, Parent, Zvolensky, & Schmidt, 2012; Capron, Norr, Zvolensky, & Schmidt, 2014). While there are many well-established psychological and pharmacological treatments for anxiety disorders, many individuals receive treatment after years of impairment or receive no treatment at all (Wang, Berglund, et al., 2005; Wang, Lane, et al., 2005). Among the primary factors that limit patient participation in treatment are access and cost (Schmidt & Keough, 2010). For anxiety disorders, empirically supported therapy sessions with a highly trained therapist remains the therapeutic gold standard, but it is clear that this model is not sufficiently meeting society's profound need. For example, returning military veterans with mental health needs report stigma as the primary barrier to seeking mental health services (Hoge et al., 2004). Programs like CAST that can be delivered over the Internet, and are completely private, eliminate stigma concerns. The fact that CAST shows a moderate effect ($d = 0.57$) in reducing AS, while addressing these dissemination and accessibility challenges indicates it has potential for both specific populations (e.g., military) and also in stepped-care approaches. In sum, preventative interventions such as CAST have significant public health potential by addressing these long-standing and common barriers to treatment.

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