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Test of an Adolescent Anxiety Sensitivity Amelioration Program (AASAP) for At-Risk Youth

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Test of an Adolescent Anxiety Sensitivity Amelioration Program (AASAP) for At-Risk Youth

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy in Psychology

by

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Abstract

Adult research supports the effectiveness of targeting the malleable vulnerability factor of anxiety sensitivity (AS) in terms of preventing panic specifically and anxiety psychopathology generally. Risk factor research suggests AS modification among youth has implications for panic as well as generalized anxiety disorder (GAD). However, very little work has evaluated the impact of AS reduction among youth, which is unfortunate given adolescence is a period of “core risk” in terms of anxiety disorder onset. Further, no work has considered the effect of such a program on GAD-relevant outcomes, nor has any work included family-level intervention factors, despite evidence suggesting parents likely play a critical role in promoting prevention programming. To address these notable gaps in the literature, the primary aim of this project was to experimentally test the effects of an Adolescent Anxiety Sensitivity Amelioration Program (AASAP) among a sample of 69 adolescents (10 to 14 years) with elevated levels of AS. High AS youth and a parent were randomly assigned to either the AASAP, which consists of a single 50min session of psychoeducation as well as experimenter- and parent-led interoceptive exposures, or a general health information control condition. As expected, adolescents in the intervention condition evidenced decreased levels of AS and generalized anxiety symptoms compared to adolescents in the control condition at follow-up. Contrary to hypotheses, however, no differences were detected in panic- and GAD-relevant vulnerability indexed using sophisticated challenge procedures. Findings are discussed in terms of the development of the specified psychosocial intervention program for adolescents targeting empirically supported processes and its short-term effects on anxiety-relevant outcomes.

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Test of an Adolescent Anxiety Sensitivity Amelioration Program for At-Risk Youth

Anxiety disorders are among the most common classes of psychopathology among youth, with nearly a quarter of adolescents meeting 12-month diagnostic criteria (Kessler et al., 2012). They negatively impact functioning (e.g., school performance) and physical health, as well as increase risk for other psychopathology (Mychailyszyn, Mendez, & Kendall, 2010). Indeed, numerous studies suggest that anxiety disorders have a lasting impact, literally taking a toll, in human and financial terms, for many youth into their adult lives (Last, Hansen, & Franco, 1997; Messer & Beidel, 1994). These data underscore the importance of developing evidence-based strategies for preventing anxiety development among youth. The current study will therefore test the merit of a brief psychosocial intervention aimed at reducing vulnerability among at-risk adolescents. As described below, anxiety sensitivity was selected as the intervention target because it is a well-established, malleable vulnerability factor (Schmidt et al., 2007). Further, while AS is most robustly linked to panic, it can also be conceptualized as a “broad-based” risk factor with relevance to other types of anxiety, particularly generalized anxiety disorder (GAD; Naragon-Gainey, 2010). Thus, consistent with recommendations to target factors that cut across disorders (O’Connell, Boat, & Warner, 2009), the current study will evaluate the impact of changes in AS on vulnerability for panic and generalized anxiety.

Prevention of panic disorder (PD) and GAD represent important public health priorities. Both syndromes are characterized by significant functional impairment and substantial economic costs (Greenberg et al., 1999). In addition, panic attacks, particularly those that occur prior to the age of 20 years, constitute a risk factor for a variety of mental health problems (Baillie & Rapee, 2005; Craig, Hwang, & Bromet, 2002; Goodwin, Brook, & Cohen, 2005). Panic attacks are also necessary for PD, a debilitating and chronic condition linked to a number of serious mental

(Goldstein & Levitt, 2007) and physical (Chen, Hu, Lee, & Lin, 2010; Chen, Tsai, Lee, & Lin, 2009) health problems. Furthermore, individuals with PD are often not appropriately diagnosed (Weissman, 1990), resulting in frequent utilization of expensive and inefficient means of symptom management (e.g., emergency departments; Lynch & Galbraith, 2003). Similarly, GAD is linked to a range of negative sequelae among youth, including lowered academic functioning, impaired social relationships, sleep disturbances, and school absenteeism (Albano & Hack, 2004), as well as negative physical health outcomes (e.g., dampened immune functioning; Brosschot, Gerin, & Thayer, 2006).

Adolescence marks an important period in terms of psychological vulnerability (Dahl, 2004; Paus, Keshavan, & Giedd, 2008). As with other anxiety disorders, the prevalence of PD and GAD increase across the course of adolescence (Costello, Copeland, & Angold, 2011; Wittchen & Hoyer, 2001). Indeed, consistent with notions of adolescence as a period of “core risk” for anxiety psychopathology (Beesdo, Knappe, & Pine, 2009), it is typically during this phase that panic attacks are first experienced (Goodwin & Gotlib, 2004; Hayward et al., 1992; Ollendick, Mattis, & King, 1994) and the nature of worry, the hallmark feature of GAD, becomes more “adult like” (Szabó, 2009) as relevant cognitive competencies emerge (e.g., elaboration; Vasey, Crnic, & Carter, 1994). These data align with an emphasis in prevention research on early intervention (Beardslee, Chien, & Bell, 2011).

Reports from the National Research Council and Institute of Medicine suggest that prevention efforts should: a) occur prior to the diagnosis of disorder (cf., treatment), b) be tailored to unique aspects of the target population (e.g., universal, selective, and indicated strategies), c) be delivered during the appropriate “prevention window” with integration of relevant socializing agents (e.g., parent involvement during adolescence; Beardslee et al., 2011),

and d) target empirically-established, malleable risk factors for a given disorder (Mrazek & Haggerty, 1994; O'Connell et al., 2009). Notably, there is some evidence for the effectiveness of child and adolescent anxiety prevention programs (Barrett, Farrell, Ollendick, & Dadds, 2006; Ginsburg, 2009; Pina, Zerr, Villalta, & Gonzales, 2012; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005; Simon, Bogels, & Voncken, 2011), although extant programs are resource intensive (e.g., typically requiring 8-12 sessions; Neil & Christensen, 2009) and long-term effects appear to be modest (Barrett et al., 2006). In particular, absent from the literature is a brief, selective intervention program targeting a promising (malleable) risk factor for the development of panic and related psychopathology.

Anxiety Sensitivity: A Well-Established Panic-Relevant Risk Factor

One of the most well established panic-relevant risk factors is anxiety sensitivity (AS). AS is a cognitive vulnerability factor that reflects beliefs pertaining to the harmful consequences of anxiety (Reiss & McNally, 1985). Several lines of evidence support the conceptualization of AS as a specific panic-relevant risk factor (Kutz, Marshall, Bernstein, & Zvolensky, 2010; McNally, 2002). First, AS is elevated among adolescents with panic attack histories compared to those without (Ginsburg & Drake, 2002; Lau, Calamari, & Waraczynski, 1996). Second, laboratory studies indicate that AS predicts anxious responding to panic-relevant bodily arousal above and beyond variance accounted for by negative affect in both clinical and nonclinical populations of adolescents (Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky, 2005; Rabian, Embry, & MacIntyre, 1999; Unnewehr, Schneider, Margraf, Jenkins, & Florin, 1996). Finally, prospective studies with adolescents and adults indicate that AS predicts the future development of panic attacks and anxiety symptoms (Hayward, Killen, Kraemer, & Taylor, 2000; Schmidt, Lerew, & Jackson, 1997, 1999; Weems, Hayward, Killen, & Taylor, 2002).

Collectively, research suggests AS is a cognitive risk factor with relevance to panic psychopathology development and thus a promising target for intervention efforts.

Anxiety Sensitivity: A Broad-Based Risk Factor

While AS is a robust and specific risk factor for panic, it may also enhance risk for other problems. Therefore, AS modification may not only decrease panic vulnerability, but also the risk for developing other psychopathology (e.g., Schmidt et al., 2007). Indeed, a large body of work links AS to non-panic problems such as depression (e.g., Weems, Hammond-Laurence, Silverman, & Ferguson, 1997). Recent meta-analytic work, however, converges on the idea that AS is most strongly and consistently linked with PD, post-traumatic stress disorder (PTSD), and GAD (Naragon-Gainey, 2010; Olatunji & Wolitzky-Taylor, 2009). While almost no work has examined AS in relation to PTSD among youth (cf., Hensley & Varela, 2008), several studies link AS to self-reported frequency of worry (Leen-Feldner, Feldner, Tull, Roemer, & Zvolensky, 2006; Silverman, La Greca, & Wasserstein, 1995) as well as concurrently (e.g., Knapp, Blumenthal, Mischel, Badour, & Leen-Feldner, 2016; McLaughlin, Stewart, & Taylor, 2007; Muris, Schmidt, Merckelbach, & Schouten, 2001) and prospectively (Schmidt et al., 2010; Waszczuk, Zavos, & Eley, 2013) assessed GAD symptomatology and diagnosis. This linkage makes sense, given both AS (Pickett, Lodis, Parkhill, & Orcutt, 2012) and worry, the hallmark feature of GAD, are associated with avoidance of anxious states (Barlow, 2002). Further, consistent with contemporary GAD models highlighting avoidance of affective contrasts (Newman & Llera, 2011), high AS individuals are differentially sensitive to (perceived) changes in bodily states (Ehlers & Breuer, 1996; Pauli et al., 1991). Collectively, AS holds promise as a general risk factor relevant to panic and other types of anxiety psychopathology; the best available evidence emphasizes linkages with features of generalized anxiety (e.g., worry).

Importantly, while particular AS facets (e.g., mental incapacitations; Olatunji & Wolitzsky-Taylor, 2009) are theorized to map onto GAD, adult AS reduction programs that emphasize interoceptive exposure (IE) reduce global as well as AS facet scores, supporting the utility of AS reduction programs in impacting dimensions of AS pertinent to GAD-relevant vulnerability (Keough & Schmidt, 2012).

Malleability of Anxiety Sensitivity

A large body of work supports the malleability of AS. For instance, Ollendick (1995) demonstrated, among adolescents, that brief cognitive behavioral therapy (CBT) that included IE reduced AS in a clinically significant manner. Further, a number of brief psychosocial interventions have been shown to reduce AS (e.g., Broman-Fulks & Storey, 2008; Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008). For example, Schmidt and colleagues (2007) found that a brief AS reduction training produced a 30% reduction in AS (compared to 17% in the control condition) as well as decreased likelihood of Axis I disorders at a two-year follow-up. In a follow-up study that included IE homework, Keough and Schmidt (2012) reported even larger reductions in AS at one and six months post-intervention. In the only published prevention test that evaluated effects on AS among youth, Balle and Tortella-Feliu (2010) adapted the Australian FRIENDS anxiety prevention program to a six-session school-based program for high AS youth (11 to 17 years) from the Balearic Islands. Compared to controls, participants in the prevention group evidenced decreased AS and, to a lesser extent, anxiety and depressive symptoms, at a 6-month follow-up. While promising, this intervention was resource intensive and relatively non-specific. Nonetheless, evidence indicates AS is malleable via brief psychosocial interventions and that such reductions impact panic and other Axis I psychopathology.

Importance of Intervening at the Family Level

Consistent with theoretical perspectives on the importance of including parents in adolescent prevention protocols (Beardslee et al., 2011), converging lines of evidence suggest parents are likely critical in promoting learning experiences designed to reduce anxious responding to panic-relevant cues (e.g., IE exercises) in contexts outside the laboratory. First, parents are key agents in efforts to generalize exposure-related learning (Hirshfeld-Becker & Biederman, 2002). Second, universal and indicated nonspecific anxiety prevention programs that integrate parent involvement evidence statistically and/or clinically significant anxiety reduction (Dadds, Spence, Holland, Barrett, & Laurens, 1997; Dadds et al., 1999; Feldner, Zvolensky, & Schmidt, 2004), and “child plus parent” conditions outperform “child only” conditions (Pina et al., 2012). Finally, evidence from other prevention literatures (e.g., substance use) suggests parent training can be an effective preventive intervention among youth (e.g., Flay, 2000; Mahabee-Gittens, Xiao, Gordon, & Khoury, 2013; McMahon & Forehand, 2004).

Relevance of Experimental Psychopathology Methods

Also absent from the child and adolescent literature is the use of sophisticated experimental psychopathology (EP) methods. This is unfortunate, as laboratory-based study of affective processes has the benefit of tightly controlled, multimodal measurement as well as attenuating self-reporting biases via “real-time” induction of states with hallmark characteristics of diagnostic syndromes (e.g., bodily arousal elicits panic-relevant responding; Zvolensky, Lejuez, Stuart, & Curtin, 2001). Further, EP methods index emotional reactivity (cf., symptom levels), thereby allowing for an evaluation of prevention programming on dimensions of psychopathology that are distinct from symptom presentation and central to understanding/ameliorating psychological disorders (Olatunji, Leen-Feldner, Feldner, & Forsyth,

2007). Further, such methods allow for the evaluation of vulnerability, and thus fit well with efforts to identify and intervene with high-risk populations (e.g., Beardslee et al., 2011). For instance, healthy individuals who respond fearfully to somatic arousal are more likely than those who do not to develop panic attacks (Schmidt & Zvolensky, 2007).

Despite recognition of AS as a malleable variable that is associated with increased risk for panic and other anxiety-related problems, there exists no test of the effects of directly modifying this risk factor among adolescents using a brief intervention format. The current study is designed to begin to fill this notable gap in the literature. It is significant and novel in a number of respects, as it will a) address the relative absence of brief interventions targeting specific risk factors among at-risk youth, b) use sophisticated EP methodologies, and c) incorporate parents. Several issues were carefully considered during the design of the proposed study, with an aim of optimally balancing theoretical and methodological concerns. Due to both financial (e.g., sample size to address incidence; Cuijpers, 2003) and temporal (e.g., following participants through disorder onset) constraints, the current investigation focuses on indexing panic and GAD-relevant vulnerability (cf., disorder onset) as primary outcomes.

Rationale for Studying Adolescents

As adolescence is a particularly important time to implement prevention programs (Cicchetti & Rogosch, 2002), this developmental period was the targeted age range for the current investigation. Adolescence is construed as the transition from childhood to adult roles characterized by tasks such as identity formation (Dahl, 2004). Further, puberty is underway by age 8 years for many American girls; outward signs appear around age 10 years and boys lag behind about 18 months (Susman & Rogol, 2004). Thus, while it is difficult to parameterize adolescence in terms of chronological age, it was reasoned that the span of 10 to 14 years best

captures key events of this period while 1) retaining reasonable homogeneity in participant developmental competency (e.g., most are moving into the period of formal operational thought; Kuhn, 2008) and 2) allowing us to secure the sample in a timely fashion (limiting the age range further may impact feasibility).

Primary Aim and Hypotheses

With this backdrop, the current study addressed both individual and family levels of intervention to target AS among at-risk adolescents. The primary aim of this research project was to evaluate a targeted psychosocial protocol that integrates experimenter and family directed IE exercises with the aim of reducing AS among high AS adolescents. Specifically, the project was designed to experimentally test the effects of an Adolescent Anxiety Sensitivity Amelioration Program (AASAP). Proximal and short-term distal effects of the AASAP were evaluated by comparing it to a general health information (GHI) control condition. It was hypothesized that, compared to the GHI, the AASAP would:

Hypothesis 1.a. result in decreased post-intervention: i) self-reported AS, ii) panic relevant responding indexed by intensity of panic symptoms elicited by a voluntary hyperventilation procedure, and iii) general anxious responding indexed using a worry induction procedure.

Hypothesis 1.b. reduce AS across a one-month post-intervention prospective assessment period.

Hypothesis 1.c. reduce self-reported symptoms of panic, as well as intensity of panic symptoms elicited by a voluntary hyperventilation procedure, assessed at one month post-intervention.

Hypothesis 1.d. reduce self-reported symptoms of generalized anxiety, as well as anxious reactivity elicited by a worry induction procedure at one month post-intervention.

Method

Design Overview

Sixty-nine high AS adolescents and a parent were randomly assigned to either the GHI or the AASAP. The initial protocol (phase I) lasted approximately three and a half to four hours, and included structured clinical interviews, the intervention, self-report assessments, and two laboratory-based procedures (counterbalanced). Computerized weekly homework assignments, as well as a two-week assessment (phase II), were completed off-site, and participants returned to the laboratory one month after the initial assessment for a one and half hour protocol (phase III) including self-report assessments, laboratory-based procedures, and debriefing. As has been done in prior AS reduction work (Keough & Schmidt, 2012), all self-report instruments were computer administered. Dyads were paid \$20 each for the initial visit and adolescents were compensated \$25 and the parent \$5 for the follow-up visit. In addition, adolescents were compensated \$5 for completing the two-week online assessment.

Participants

Sixty-nine nonclinical adolescents (58% male) between the ages of 10 and 14 years ($M_{age} = 12.52$ years, $SD = 1.37$) as well as one of their parents ($M_{age} = 41.03$ years, $SD = 6.95$) were recruited from the local community. Adolescent participants had a total CASI score ≥ 1 SD above the mean for males (i.e., ≥ 28) or females (i.e., ≥ 30), derived from prior community-based samples recruited from the locale, and the PI stratified by gender. This cutoff point was chosen because individuals with AS scores in this range are at increased risk of anxiety problems (Schmidt & Joiner, 2002). Please see Table 1 for adolescent and parent characteristics as a

function of condition. Exclusionary criteria for youth included: (a) current or past cardiopulmonary or respiratory illness; (b) possibility of being pregnant; (c) current enrollment in mental health treatment and (d) current or past PD, panic attacks, or GAD. Both parents and youth were excluded in the context of (a) current uncontrolled psychotic disorder; (b) current suicidal intent; or (c) limited mental competency/inability to give informed, written consent. Collectively, these exclusionary criteria ensured participant safety (Leen-Feldner et al., 2005). Criteria were assessed using the Medical Screening Interview (e.g., “Has a doctor ever said you have respiratory (breathing) problems, like asthma?”), Anxiety Disorders Interview Schedule-IV: Child version (ADIS-C; Silverman & Albano, 1996), Panic Attack Questionnaire (PAQ; Norton, Dorward, & Cox, 1986) and Mini-International Neuropsychiatric Interview (MINI; Sheehan et al. 1998). This exclusionary criterion has been successfully utilized in multiple laboratory studies involving emotion elicitation (Hawks, Blumenthal, Feldner, Leen-Feldner, & Jones, 2011). Finally, all participants with complete data (i.e., fully completed the measure of interest) were included in relevant analyses.

Measures

In addition to the primary study outcomes, measures were administered to index baseline characteristics and inclusionary/exclusionary criteria. The parent and adolescent self-report measures administered throughout the three phases were completed using Qualtrics, an on-line data acquisition system (please see Table 2 for the assessment timeline). The PI conducted baseline interviews prior to assigning participants to condition. Psychometric data for adaptations of instruments pertinent to DSM-5 were not available at the commencement of data collection, and therefore measures with established psychometrics, albeit for DSM-IV, were utilized.

Adolescent assessment. The *Anxiety Disorders Interview Schedule-IV: Child version* (ADIS-C; Silverman & Albano, 1996), which evaluates anxiety and other common childhood disorders according to the DSM-IV (American Psychiatric Association, 1994), was used to screen adolescents with histories of PD and GAD, as well as assess current suicidality and current psychotic psychopathology. This structured clinical interview has been validated with children and adolescents and evidences good reliability and validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002).

The 18-item *Childhood Anxiety Sensitivity Index* (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991) was used to assess AS. The CASI was validated with youth, and demonstrates good psychometrics (e.g., Wright et al., 2010). The CASI is distinct from indices of panic symptoms (Ginsburg & Drake, 2002). Consistent with empirical precedent (e.g., Balle & Tortella-Feliu, 2010; Keough & Schmidt, 2012) and the underdeveloped state of adolescent AS reduction research, total CASI scores were used to index global fear regarding the consequences of anxiety-related sensations.

The *Panic Attack Questionnaire* (PAQ; Norton et al., 1986), which has sound psychometric properties and has been successfully employed to evaluate panic attack symptoms among adolescents (e.g., Ginsburg & Drake, 2002; Ginsburg, Lambert, & Drake, 2004), was administered to evaluate the exclusionary criterion of current or past panic attacks.

The *Revised Child Anxiety and Depressive Scales* (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000), a 47-item measure of DSM-IV anxiety and depressive disorder symptoms, was administered to assess generalized anxiety (RCADS-GA) and panic (RCADS-P) symptomatology. Adolescent participants indicated how often each of the statements occurred on

a 4-point Likert-type scale from “never” to “always.” The RCADS measure has good psychometric properties (Chorpita et al., 2000).

The negative affect subscale of the *Positive and Negative Affect Schedule for Children* (PANAS-C; Joiner, Catanzaro, & Laurent, 1996) assessed generalized negative affectivity by asking adolescents to endorse the degree to which they “have felt this way during the past few weeks” regarding 15 negatively valenced words (e.g., guilty, nervous, frightened) on a 5-point Likert-type scale from 1 (“very slightly or not at all”) to 5 (“extremely”). The PANAS-C evidences sound psychometric properties (Wilson, Gullone, & Moss, 1998).

Fearful reactivity to bodily arousal. Voluntary hyperventilation (VH) was utilized to index real-time panic-relevant reactivity because it can reliably produce bodily arousal that mimics panic attack symptoms and it has been shown to be safe when used with adolescents (Leen-Feldner et al., 2005; Unnewehr et al., 1996). The VH challenge involves a three-minute hyperventilation, with a breathing rate of 30 respiratory cycles/min (Fried & Grimaldi, 1993). A trained research assistant, blind to condition, delivered a standardized instructional set prior to a standard five-minute baseline period. Next, participants listened to audiotaped directions that guided them through the hyperventilation. Participants provided pre- and post-challenge ratings of panic symptom intensity using the *Acute Panic Inventory* (API; Dillon, Gorman, Liebowitz, Fyer, & Klein, 1987), a 23-item assessment of panic attack symptoms (e.g., “do you feel faint?”) that has sound psychometric properties and has been successfully employed in research with adolescents (Pine et al., 2000).

Worry induction procedure (WIP). GAD-relevant vulnerability was indexed using an ideographic worry induction procedure in which participants were given a standard definition of worry and asked to engage in this cognitive process for five-minutes. Recent experimental data

accord with a wealth of adult work (see Newman & Llera, 2011), suggesting the procedure effectively increases anxious arousal, as well as future-oriented mentation, relative to a control group (Frala, Mischel, Knapp, Autry, & Leen-Feldner, 2014). Participants reported current anxiety before and after the procedure using a 100-point *Subjective Units of Distress Scale* (SUDS-A; Wolpe, 1958), which has been successfully employed in many studies with adolescents (e.g., Bacow, May, Choate-Summers, Pincus, & Mattis, 2010).

Intervention Self-Monitoring and Manipulation Check Materials. An adapted version of the *Exposure Exercise Record* (EER; Craske & Barlow, 2000) was used by participants in the AASAP to monitor IE at home. Participants track IE practice (e.g., time/date) and rate fear/distress as well as intensity of sensations experienced during the exposure from 0 (“none”) to 8 (“extreme”). Meal planning forms (“food tracker”; USDA, 2013) was used in the control condition to monitor activities based on recommendations made in the intervention session. A manipulation check questionnaire was utilized asking participants to endorse the topics (i.e., healthy foods, AS, bodily sensations or feelings, sleep, and/or none of the above) “talked about” in the powerpoint presentation and related exercise.

Parent assessment. The *Mini-International Neuropsychiatric Interview* (MINI; Sheehan et al., 1998) was used to measure relevant exclusionary criteria (e.g., current suicidal intent, psychosis). The MINI was selected because it is a brief (~15min), psychometrically sound instrument. Parental affective vulnerability was evaluated via the *Anxiety Sensitivity Index* (ASI-3; Taylor et al., 2007). The well-established, 18-item ASI-3 was used to assess parent AS.

Procedure

Informed consent, screening, and baseline assessment. Interested adolescents and their parents were informed of the study protocol and administered a brief telephone screener to

evaluate eligibility [i.e., CASI; Medical Screening Interview (MSI)]. A laboratory visit was scheduled for eligible adolescents and their parents. Upon arrival, adolescent participants were provided written informed assent, and parents written informed consent (for personal and adolescent participation). Inclusion criteria were then evaluated by the PI (i.e., ADIS-C; PAQ; MINI; MSI). Due to the rigorous telephone screening prior to the first laboratory visit, no participants were deemed ineligible in the laboratory. Eligible participants and their parents then completed the pre-intervention self-report measures. Adolescents and their parents were next randomly assigned (stratified by gender) to intervention condition. No information regarding participants' "at risk" status was provided. To ensure standardization, the PI conducted all sessions and written manuals were followed at all times. Both programs lasted approximately 50min.

Adolescent anxiety sensitivity amelioration program. The AASAP consisted of two parts. Part I consisted of: (1) psychoeducation regarding panic and AS and (2) an individual experimenter-directed exposure session. The first component of the session consisted of psychoeducation regarding the nature of anxiety/fear adapted from the Anxiety Sensitivity Amelioration Training (Schmidt et al., 2007) to make it developmentally appropriate for adolescents. The second component involved an experimenter-directed exposure session. The exposure session consisted of 10 repeated trials of straw breathing given evidence that these exercises are among the most anxiety-provoking for individuals with PD (Schmidt & Trakowski, 2004). Thereafter, participants completed the post-intervention indices (i.e., CASI, VH, WIP). Part II consisted of (1) parent training in conducting at-home exposure exercises and (2) training adolescent/parent pairs to monitor exposure practice. Dyads were then instructed to practice the straw breathing exercise once per week during the entire follow-up period. The Therapist Guide

and Workbook for *Master of Anxiety and Panic for Adolescents: Riding the Wave* (Pincus, Ehrenreich, & Mattis, 2008) were referenced when adapting the ASAT manual (Schmidt et al., 2007) with regards to facilitation of the in-laboratory IE as well as training the parent on the at-home IE.

General health information control condition. The GHI control condition was designed to mirror the structure of the AASAP, except that no information relevant to anxiety or panic was presented. Part I included (1) psychoeducation about the general benefits of healthy dietary habits, and (2) practice using a “food tracker” to plan, record, and monitor nutritional information of meals. Information presented was based on the U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA; HHS; 2010) recommendations (e.g., understanding nutritional labels). Thereafter, participants completed the post-intervention indices (i.e., CASI, VH, WIP). Part II consisted of (1) parent training in using the food tracker and (2) training adolescent/parent pairs to monitor nutritional information of meals and meal planning.

At the end of the laboratory session, participants were compensated \$20/each (\$40/dyad), given instructions regarding homework completion and submission, and scheduled for a one-month follow-up.

Two-week assessment. As has been done in the past (e.g., Keough & Schmidt, 2012), adolescents completed the two-week assessment (i.e., CASI) using a secure on-line data acquisition program. A reminder was sent to participants 48 hours prior to the two-week mark and every 24 hours (up to three days) after the intended date of completion. Adolescents were paid \$5 for completing this assessment.

One-month assessment. Participants returned to the lab one month after the initial laboratory session to complete a follow-up (i.e., PSQ, RCADS, CASI, WIP and VH). At the end of this session, dyads were comprehensively debriefed and the adolescent was compensated \$25 and the parent \$5. Please see Figure 1 for a graphical representation of study procedures.

General Analytic Strategy

Sample size considerations. Drawing on effect sizes reported in the panic prevention (Gardenswartz & Craske, 2001), AS modification (Balle & Tortella-Feliu, 2010; Schmidt et al., 2007), and panic treatment (e.g. Pincus, May, Whitton, Mattis, & Barlow, 2010; Otto & Reilly-Harrington, 1999) literatures, data converge on a medium to large effect size (Cohen, 1992). An *a priori* power analyses suggested 68 subjects were needed to detect an effect size of $f = .35$ in an analysis of variance with two groups (power of .80).

Preliminary analyses. First, the equivalence of groups on key baseline and demographic characteristics were evaluated to assess the efficacy of random assignment. Specifically, t-tests were conducted to compare groups regarding the continuous variables of ASI, adolescent age, PANAS-CN, CASI, RCADS-GA, RCADS-P, pre-task SUDS-A, pre-task API at phase I as well as pre-task SUDS-A and pre-task API at phase III. Chi-square analyses were computed to examine gender differences as a function of intervention condition. Finally, zero-order correlations were conducted between continuous variables to evaluate relations between demographic, baseline, and outcome characteristics.

As a manipulation check of condition, a series of chi-square tests were utilized to compare groups in terms of topics (e.g., anxiety sensitivity, healthy food) addressed during the conditions' psychoeducation and related exercises. T-tests were conducted to confirm the efficacy of the hyperventilation procedure in increasing self-reported anxiety by comparing

groups (pre-hyperventilation; post-hyperventilation) on the continuous variable of SUDS-A scores across condition for phases I and III.

Primary analyses. Following preliminary analyses, a series of analyses of variance (ANOVAS) were conducted to examine the effect of group (AASAP vs. GHI) on hypothesized outcomes. To utilize the most powerful statistical approach for each analysis (Field, 2014; Maxwell, O'Callaghan, & Delaney, 1993), ANCOVAS were conducted for pre/post-intervention analyses (i.e., AASAP and GHI as the fixed factor; phase III RCADS-P and RCADS-GA as dependent variables; pre-intervention RCADS-P and RCADS-GA as covariates), whereas mixed ANOVAS were employed for post-intervention analyses (i.e., AASAP and GHI as the fixed factor; phase I and III post-task and pre-task SUDS-A and API as dependent variables) and AS levels across the intervention period (i.e., AASAP and GHI as the fixed factor; baseline CASI, phase I CASI, phase II CASI, and phase III CASI as dependent variables). Scores on the phase I and III post-task SUDS-A and CASI baseline evidenced a positive skew, whereas phase I API post-task scores and all CASI scores, RCADS-GA scores, post-task SUDS-A scores were kurtotic; accordingly, data from these measures were transformed using square root transformation prior to primary analyses (Field, 2014). Finally, effect size for ANOVAs were indexed via partial eta squared (η_p^2).

Results

Preliminary Analyses

Covariates. Preliminary analyses indicated that random assignment effectively equated groups across a number of conceptually relevant variables. Specifically, groups did not significantly differ as a function of ASI, adolescent age, PANAS-CN, CASI, RCADS-GA, RCADS-P, pre-task SUDS-A, pre-task API at phase I as well as pre-task SUDS-A and pre-task

API at phase III. Please see Table 3 for the reported means, standard deviations, and t-test scores as a function of condition. Additionally, the chi-square test evaluating group differences in terms of gender indicated no differences between adolescents in the control condition and those in the intervention condition [$\chi^2(1, n = 69) = .02, p = .888$]. Accordingly, only baseline scores were included as covariates for relevant primary analyses. Please see Table 4 for zero-order relations among the continuous demographic, baseline, and outcome variables.

Manipulation check of condition. Preliminary analyses suggested youth in the intervention condition were significantly more likely to report learning about AS [$\chi^2(1, n = 69) = 65.11, p < .001$] and bodily sensations [$\chi^2(1, n = 69) = 47.76, p < .001$] in the psychoeducation and related exercises compared to youth in the control condition, whereas youth in control condition were significantly more likely to report reviewing healthy food [$\chi^2(1, n = 69) = 57.95, p < .001$] and sleep [$\chi^2(1, n = 69) = 54.64, p < .001$] compared to youth in the intervention condition.

Manipulation check of the voluntary hyperventilation procedure. Preliminary analyses indicated that the VH procedure was successful in increasing self-reported anxiety across groups. Specifically, youth reported higher post-challenge SUDS-A scores ($M = 33.87, SD = 28.40$) compared to their baseline SUDS-A scores during phase I [$M = 17.49, SD = 20.62; t(134) = -3.85, p < .001$]. Similarly, youth evidenced significantly lower SUDS-A scores at baseline ($M = 13.59, SD = 20.11$) compared to their SUDS-A scores at the post-challenge assessment during phase III [$M = 25.80, SD = 27.42; t(109) = -2.68, p = .009$].

Primary Analyses

Analyses of covariance: Generalized anxiety and panic symptoms. After accounting for baseline RCADS-GA, youth in the intervention condition ($M = 9.72, SD = 2.46$) evidenced

significantly lower RCADS-GA at follow-up than youth in the control condition [$M = 11.41$, $SD = 3.82$; $F(1, 60) = 6.20$, $p = .016$, $\eta_p^2 = .09$]. However, youth in the control condition ($M = 12.94$, $SD = 4.74$) did not report significantly higher RCADS-P at the follow-up compared to youth in the intervention condition [$M = 10.93$, $SD = 1.94$; $F(1, 59) = 2.71$, $p = .105$, $\eta_p^2 = .04$], controlling for baseline RCADS-P.

Mixed analyses of variance: Anxiety sensitivity. Mauchly's test indicated that the assumption of sphericity had not been violated [$\chi^2(5) = 8.86$, $p = .115$], therefore Sphericity Assumed tests are reported (Field, 2014). There was not a significant main effect of condition regarding the different time points of AS [$F(1, 59) = 1.10$, $p = .298$, $\eta_p^2 = .02$]; however, there was a significant interaction between the different levels of AS and intervention condition [$F(3, 177) = 3.54$, $p = .016$, $\eta_p^2 = .06$], suggesting the different time points of AS differed by condition. To investigate this interaction, contrasts were performed comparing each AS time point to baseline AS across youth in the intervention and control conditions. The first contrast revealed a significant interaction when comparing youth AS scores at baseline to phase III [$F(1, 59) = 8.15$, $p = .006$, $\eta_p^2 = .12$]. The second and third contrasts comparing youth baseline AS scores to post-intervention [$F(1, 59) = 2.84$, $p = .097$, $\eta_p^2 = .05$] and phase II [$F(1, 59) = 1.49$, $p = .228$, $\eta_p^2 = .03$] were not significant. The interaction graph (please see figure 2) depicts that AS levels of youth in both conditions decreased from baseline to phase III, but this decrease was more pronounced at phase III for youth in the intervention condition, suggesting the AASAP intervention results in lower levels of AS compared to the control condition across the assessment period.

Mixed analyses of variance: Laboratory-based procedures. There was not a significant main effect of condition on WIP response at phase I [$F(1, 66) = .68, p = .414, \eta_p^2 = .01$], nor was there a significant interaction between SUDS-A ratings and condition during this phase [$F(1, 66) = 1.31, p = .256, \eta_p^2 = .02$], suggesting the pattern of SUDS-A responses were similar for adolescents in the intervention and control conditions (see Figure 3).

There was not a significant main effect of condition on WIP response at phase III [$F(1, 54) = .26, p = .610, \eta_p^2 = .01$], nor did a significant interaction emerge between condition and SUDS-A ratings [$F(1, 54) = 1.17, p = .284, \eta_p^2 = .02$], suggesting the SUDS-A ratings reported by adolescents in the intervention and control conditions were similar (please see Figure 4).

With regards to the VH task at phase I, there was not a significant effect of condition on response to the task [$F(1, 63) = .19, p = .663, \eta_p^2 = .00$], nor did a significant interaction emerge between the different levels of API ratings and condition [$F(1, 63) = .03, p = .856, \eta_p^2 = .00$], suggesting the adolescents in the control and intervention conditions had similar patterns of API ratings (please see Figure 5).

Finally, there was not a significant main effect of condition on response to the VH task at phase III [$F(1, 51) = 1.06, p = .307, \eta_p^2 = .02$], nor was there a significant interaction between condition and level of API ratings [$F(1, 51) = 1.86, p = .179, \eta_p^2 = .04$], suggesting the pattern of responses to the VH during this phase was similar for adolescents in the control and intervention conditions (please reference Figure 6).

Discussion

Adolescence is an important developmental period to investigate anxiety-relevant outcomes. Many adolescent youth evidence clinically-significant levels of anxiety, resulting in a

variety of debilitating outcomes (e.g., poor school performance, physical health; Kessler et al., 2012; Mychailyszyn et al., 2010). This highlights the need for effective, evidence-based preventive intervention programs targeting vulnerability factors that increase the risk for anxiety-related disorders among youth. The effects of a brief intervention program targeting the malleable vulnerability factor of AS have not yet been investigated among adolescent youth. The purpose of the current study was, therefore, to test the impact of a selective intervention program on anxiety-relevant outcomes among high-AS youth.

Anxiety Sensitivity

First, consistent with our predictions, youth in the intervention condition sustained low AS levels across the intervention period; however, significant differences in AS levels from baseline between the two conditions were only detected at the one-month follow-up period. Specifically, youth in the intervention condition evidenced significantly lower AS levels at the one-month follow-up period (i.e., phase III) from baseline compared to youth in the control condition. The trends in AS reductions at the follow-up period within both conditions (24% reduction in intervention condition, 12% in control condition at the one-month follow-up) are similar to the patterns observed in the adult (30% reduction in intervention condition, 17% in control condition at the one-year follow-up; Schmidt et al., 2007) and adolescent (25% reduction in intervention condition, 18% in control condition at the six-month follow-up; Balle & Tortella-Feliu, 2010) AS amelioration literatures. These data uniquely extend the extant literature base and suggest the AASAP intervention has sustained short-term effects on AS among youth.

The absence of group differences with regard to post-intervention AS line up with prior adolescent work (Balle & Tortella-Feliu, 2010), but are inconsistent with adult work (Keough & Schmidt, 2012; Schmidt et al., 2007). The discrepancy between the adult and adolescent findings

may be due to potential developmental differences. For example, as children transition into adolescence, they become better able to engage in abstract thought and propositional thinking (i.e., adolescents enter the formal operational stage; Inhelder & Piaget, 1958; Kuhn, 2008). Thus, older adolescents and adults may be better able to recall and apply concepts learned during psychoeducation (e.g., harmless nature of uncomfortable bodily sensations), resulting in relatively quick schematic accommodation (and immediate reductions in AS), whereas children and younger adolescents may need repeated IE experiences over time to accommodate new schemes. Future work is needed to elucidate mechanisms underlying the developmental differences in AS reduction trends observed here. For example, researchers could not only examine the effects of the AASAP on reductions in AS among different age groups, but also investigate whether putative cognitive mediating mechanism(s) differ across age groups.

Generalized Anxiety and Panic Symptoms

Second, as expected, adolescents in the intervention condition evidenced decreased generalized anxiety symptoms at the one-month follow-up compared to adolescents in the control condition. Importantly, the differences were detected even after controlling for baseline levels of generalized anxiety symptoms. These are the first data to empirically investigate the effects of a brief, AS amelioration program on specific symptoms (cf., total anxiety symptoms) among at-risk youth. Findings suggest that a relatively brief intervention decreases symptoms of generalized anxiety. These preliminary results have exciting implications for the youth anxiety prevention literature given the brief duration of the intervention as well as the plethora of future directions (e.g., increasing intervention accessibility and effectiveness) open to empirical investigation.

In contrast to prediction, there were no differences in panic symptoms at the one-month follow-up between adolescents in the intervention condition and control condition when controlling for baseline levels. While the AASAP may, in fact, have no effect on panic symptoms, it is also possible that reduction of these specific symptoms take longer to influence. For example, in a 10-week, school-based anxiety preventive intervention for at-risk youth, Dadds and colleagues (1997) reported a delayed intervention effect, in that they did not detect differences between the intervention and control conditions on diagnosable disorders until the six-month follow-up. An important next step in this line of work will be evaluation of longer follow-up periods (e.g., 1-2 years) to not only determine the distal impact of intervention programming more generally, but also to examine the particular outcomes that may take longer to impact specifically (e.g., panic symptoms).

Laboratory-Based Procedures

Third, in contrast to prediction, there were no differences in response to the somatic perturbation elicited by the VH task at phase I or phase III between adolescents in the intervention condition and control condition. Adolescents in both conditions reported increased anxiety levels from pre- to post-task at both phases and the frequency of panic symptoms endorsed post-task were comparable to levels typically reported in the adolescent VH literature (e.g., Leen-Feldner et al., 2005). This suggests the task was successful in increasing somatic arousal and subjective anxiety; however, the current findings indicate the intervention condition did not differentially impact adolescents' panic-relevant responding to the VH task. The absence of group differences is inconsistent with prior AS amelioration work among adults (Schmidt et al., 2007). This discrepancy may be due to a methodological difference between published work and the current study. Schmidt and colleagues utilized a more robust method to index real-time

panic-reactivity (i.e., the 20% CO₂ –enriched biological challenge). Despite the evidence indicating the VH was effective, adolescents' panic-relevant response was not extremely elevated (i.e., API moved from 28 to 36 and 26 to 32 overall, phases I and III respectively, on a scale of 23 to 92). These findings suggest the VH utilized in the current study may not have been a powerful enough induction to strongly influence somatic arousal given the voluntary nature of the procedure. To test the impact of this methodological discrepancy, future work could usefully employ a more robust induction procedure with adolescents (e.g., 5% CO₂ inhalation; Pine et al., 2005).

The effects of an AS amelioration program on GAD-relevant vulnerability have not been directly evaluated in the extant literature; however, the current findings are inconsistent with predictions based on our current understanding of ideographic worry induction procedures (Frala et al., 2014; Newman & Llera, 2011). Specifically, no differences were detected in reported anxiety in response to the worry induction at phases I and III between adolescents in the control and intervention conditions. It is interesting, in fact, that adolescents in the intervention condition reported decreased overall generalized anxiety symptoms at the one-month follow-up, but no differences were detected regarding GAD-vulnerability at this same time point. An important next step will be to further investigate the observed discordance between symptoms and emotion elicitation through induction procedures. For example, future work could examine the effects of the AS amelioration program on additional indices of reactivity to the ideographic worry induction, especially those that distinguish pathological worry from normative worry (e.g., physiological symptoms, uncontrollability of worry; APA, 2013; Borkovec, Shadick, & Hopkins, 1991).

A few additional limitations merit mention. First, as previously addressed, extended post-intervention assessment intervals are critical for determining the impact of intervention programming. An important next step in this line of work will be evaluation of the AASAP intervention over a longer follow-up period (e.g., 1-2 years). Relatedly, the current study only examined the short-term anxiety-relevant outcomes of panic and generalized anxiety symptomatology as well as anxiety vulnerability via inductions. While this is an important first step in program evaluation, future work will need to investigate the effects of the intervention on the incidence of psychopathology during a longer follow-up period. Second, to maintain standardization of the IE protocol, all adolescents engaged in and practiced the straw-breathing task over the intervention period. Future work could examine employment of tailored, and therefore potentially more robust, interoceptive exposure exercises (Keough & Schmidt, 2012). Employment of such exercises could increase the overall effects of the intervention and, accordingly, further enhance the contribution of such an intervention among youth. Third, while a novel aspect of the current study was incorporation of anxiety induction exercises to examine real-time responding, the majority of study measures were evaluated via self-report. A promising avenue for future work would be inclusion of additional modes of assessment to inform the design and evaluation of the AASAP intervention. For example, future research could meaningfully incorporate investigation of neurobiological indices to identify potential neurobiological mediators of change. Fourth, consistent with the anxiety treatment and preventive intervention literatures (e.g., Kendall & Sugarman, 1997; Dadds et al., 1997) a few parent-adolescent dyads were lost to attrition (87% completed phase III online survey; 81% completed phase III in-laboratory session). Interestingly, the majority of the dyads not retained were female and in the intervention condition. Future work would benefit from identifying

variables related to attrition that could be targeted in future interventions to increase participant retention, especially with family-level interventions where typically the child/adolescent is reliant on the parent for study completion (e.g., transportation to the clinic/laboratory; Harachi, Catalano, & Hawkins, 1997). Additionally, future work could investigate other effective intervention delivery modes (e.g., computer-delivered AS intervention; Capron & Schmidt, 2016) that may reduce participant burden and be more conducive to hectic family schedules. Fifth, employment of investigations empirically examining the intervention's mechanisms of change would increase our overall knowledge and help clarify the driving mechanisms causing decreases in AS and generalized anxiety symptoms. Similar to the youth anxiety treatment literature (e.g., Whiteside et al., 2015), future preventive intervention work would benefit from conducting dismantling studies to hone in on the specific mechanisms (e.g., psychoeducation regarding panic and AS, experimenter- and parent-directed IE) that have the most influence regarding the observed reductions in the current study. Relatedly, interventions designed to elucidate potential intervention moderators to further refine for whom the intervention is most effective and why would expand our research base in this area. For example, researchers could empirically investigate the relevance of parent factors (e.g., parental psychopathology) in administration and overall effectiveness of the parent-directed IE. Finally, the adolescents in the current sample primarily identified as Caucasian who took part in a laboratory-based investigation for financial compensation. Enrolling a more heterogeneous sample through more inclusive recruitment (e.g., school-based) techniques as well as utilizing more diverse compensation strategies would enhance generalizability of the observed findings.

Given the public health significance of decreasing risk for anxiety disorders among youth, the goal of current study was to investigate the effects of a brief intervention on anxiety-relevant

outcomes for at-risk youth. The current findings offer preliminary support for the AASAP intervention as a selective intervention for at-risk adolescents, with specific short-term effects on AS and generalized anxiety symptoms. Future work is now needed to replicate these effects as well as investigate the intervention effects over a longer period of time on an array of relevant outcomes (e.g., incidence of anxiety and related disorders; evaluation of neurobiological substrates).

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Table 1

Sample Characteristics and Dependent Variables as a Function of Condition (raw data)

	Condition	
	<i>GHI (Control)</i> <i>M or n</i> <i>(SD or %)</i>	<i>AASAP (Intervention)</i> <i>M or n</i> <i>(SD or %)</i>
<i>Adolescent</i>		
Age	12.36 (1.41)	12.68 (1.32)
Gender (Males)	20 (57.1%)	20 (58.8%)
Racial Composition		
White/Caucasian	25 (71.4%)	25 (73.5%)
Asian	1 (2.9%)	0 (0.0%)
Black/African American	1 (2.9%)	2 (5.9%)
American Indian/Alaska Native	2 (5.7%)	1 (2.9%)
Native Hawaiian/Other Pacific Islander	0 (0.0%)	1 (2.9%)
More than once race	5 (14.3%)	5 (14.7%)
Other	1 (2.9%)	0 (0.0%)
Ethnic Composition		
Hispanic/Latino	4 (11.4%)	1 (2.9%)
Non-Hispanic	31 (88.6%)	33 (97.1%)
Education		
Fourth grade	3 (8.6%)	2 (5.9%)
Fifth grade	6 (17.1%)	4 (11.8%)
Sixth grade	10 (28.6%)	9 (26.5%)
Seventh grade	7 (20.0%)	7 (20.6%)
Eighth grade	4 (11.4%)	6 (17.6%)
Ninth grade	5 (14.3%)	6 (17.6%)
Dependent Variables		
CASI-Post	28.33 (5.76)	27.14 (3.31)
CASI-PII ^a	27.52 (5.34)	26.68 (3.97)
CASI-PIII ^b	28.72 (6.20)	25.57 (4.09)
RCADS-P-PIII ^b	12.94 (4.74)	10.93 (1.94)
RCADS-GA-PIII ^b	11.41 (3.82)	9.72 (2.46)
SUDS-A-PI-Post	47.54 (33.85)	53.36 (31.53)
SUDS-A-PIII- Post ^c	32.48 (29.45)	45.76 (32.03)
API-PI-Post ^d	33.87 (11.08)	37.09 (14.43)
API-PIII-Post ^e	30.23 (6.60)	33.27 (10.24)

Table 1 (cont.)

	Condition	
	<i>GHI (Control)</i> <i>M or n</i> (<i>SD</i> or %)	<i>AASAP (Intervention)</i> <i>M or n</i> (<i>SD</i> or %)
<i>Parent</i>		
Age	41.71 (7.26)	40.32 (6.65)
Gender (Females)	31 (88.6%)	31 (91.2%)
Family Income ^f	\$79,333 (\$67,872)	\$54,241 (\$50,498)
Racial Background		
White/Caucasian	30 (85.7%)	27 (79.4%)
Asian	2 (5.7%)	0 (0.0%)
Black/African American	0 (0.0%)	2 (5.9%)
American Indian/Alaska Native	0 (0.0%)	2 (5.9%)
Native Hawaiian/Other Pacific Islander	1 (2.9%)	0 (0.0%)
More than once race	2 (5.7%)	3 (8.8%)
Ethnicity		
Hispanic	4 (11.4%)	3 (8.8%)
Non-Hispanic	31 (88.6%)	29 (85.3%)
Missing	0 (0.0%)	2 (5.9%)
Education		
Attended high school	0 (0.0%)	2 (5.9%)
Graduated high school	1 (2.9%)	1 (2.9%)
Attended college	13 (37.1%)	8 (23.5%)
Graduated 2-year College	0 (0.0%)	3 (8.8%)
Graduated 4-year College	9 (25.7%)	9 (26.5%)
Attended graduate school	2 (5.7%)	2 (5.9%)
Completed graduate school	9 (25.7%)	9 (26.5%)
Missing	1 (2.9%)	0 (0.0%)
Marital Status		
Never married	1 (2.9%)	2 (5.9%)
Divorced/Annulled	3 (8.6%)	6 (17.6%)
Separated	1 (2.9%)	1 (2.9%)
Married/Living with someone	30 (85.7%)	25 (73.5%)

Note: $N = 69$; $n = 35$ (GHI/Control); $n = 34$ (AASAP/Intervention). CASI-Post = Child Anxiety Sensitivity Index, post-intervention; CASI-PII = Child Anxiety Sensitivity Index, phase II; CASI-PIII = Child Anxiety Sensitivity Index, phase III; RCADS-P-PIII = Revised Child Anxiety and Depressive Scales- Panic Symptom Subscale, phase III; RCADS-GA-PIII = Revised Child Anxiety and Depressive Scales- Generalized Anxiety Symptom Subscale, phase III; SUDS-A-PI-Post = Subjective Units of Distress, Anxiety phase I post-task; SUDS-A-PIII-Post = Subjective Units of Distress, Anxiety phase III post-task; API-PI-Post = Acute Panic Inventory, phase I post-task; API-PIII-Post = Acute Panic Inventory, phase III post-task. Inventory, pre-task phase III.

^a $N = 64$. ^b $N = 63$. ^c $N = 56$. ^d $N = 66$. ^e $N = 54$. ^f $N = 18$.

Table 2

Timetable of Baseline/Initial, Two-week, and One-month Assessments

	Screening/ Baseline	Post- Intervention Lab Session	Two Week Assessment	One Month Lab Session
<u>Assessments</u>				
<u>Adolescent</u>				
Childhood Anxiety Sensitivity Index	X	X	X	X
Panic Attack Status- Panic Attack Questionnaire	X			
Medical Screening Interview	X			
Anxiety Disorders Interview Schedule-IV: Child version	X			
Revised Child Anxiety and Depressive Scales	X			X
Positive and Negative Affect Schedule for Children	X			
<u>Voluntary Hyperventilation Task</u>		X		X
Acute Panic Inventory (pre and post hyper task)		X		X
<u>Worry Induction Procedure</u>		X		X
Subjective Units of Distress (pre and post worry task)		X		X
<u>Parent</u>				
The Mini-International Neuropsychiatric Interview	X			
Anxiety Sensitivity Index	X			

Table 3

Evaluation of the Efficacy of Random Assignment among Demographic and Baseline Characteristics (raw data)

	Condition		<i>t</i>
	<i>GHI (Control)</i> <i>M (SD)</i>	<i>AASAP (Intervention)</i> <i>M (SD)</i>	
<i>Baseline Variables</i>			
ASI-Base ^a	10.65 (8.42)	9.10 (8.15)	.75
Adolescent Age	12.36 (1.41)	12.68 (1.33)	-.98
PANAS-CN-Base ^b	25.61 (8.03)	25.58 (8.42)	.02
CASI-Base	32.86 (3.90)	33.71 (4.47)	-.84
RCADS-GA-Base	11.23 (3.05)	11.15 (3.49)	.10
RCADS-P-Base ^c	12.71 (4.03)	11.53 (2.30)	1.48
SUDS-A-PI-Pre ^c	15.17 (23.89)	10.52 (12.97)	1.01
API-PI-Pre ^d	27.67 (5.74)	28.26 (5.37)	-.44
SUDS-A-PIII-Pre ^e	11.39 (19.55)	7.76 (13.33)	.79
API-PIII-Pre ^f	25.87 (5.30)	26.96 (5.77)	-.73

Note: $N = 69$. No statistically significant differences were observed. ASI-Base = Anxiety Sensitivity Index, baseline; PANAS-CN-Base = Positive and Negative Affect Schedule for Children, Negative Affect subscale, baseline; CASI-Base = Child Anxiety Sensitivity Index, baseline; RCADS-GA-Base = Revised Child Anxiety and Depressive Scales- Generalized Anxiety Symptom Subscale, baseline; RCADS-P-Base = Revised Child Anxiety and Depressive Scales- Panic Symptom Subscale, baseline; SUDS-A-PI-Pre = Subjective Units of Distress, Anxiety pre-task phase I; API-PI-Pre = Acute Panic Inventory, pre-task phase I; SUDS-A-PIII-Pre = Subjective Units of Distress, Anxiety pre-task phase III; API-PIII-Pre = Acute Panic Inventory, pre-task phase III.

^a $N = 63$. ^b $N = 66$. ^c $N = 68$. ^d $N = 67$. ^e $N = 56$. ^f $N = 55$.

Table 4

Zero-order Relations among Demographic, Baseline, and Outcome Variables (raw data)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age-A	--												
2. CASI-Base	-.02	--											
3. CASI-Post	-.06	.53**	--										
4. CASI-PII ^a	.08	.31*	.67**	--									
5. CASI-PIII ^b	-.13	.33**	.62**	.55**	--								
6. RCADS-GA-Base	.03	.43**	.51**	.42**	.31*	--							
7. RCADS-GA-PIII ^b	.01	.12	.43**	.42**	.62**	.50**	--						
8. RCADS-P-Base ^c	-.12	.23	.58**	.49**	.47**	.50**	.47**	--					
9. RCADS-P-PIII ^b	-.19	.20	.55**	.42**	.67**	.26*	.59**	.68**	--				
10. SUDS-A-PI-Post	.01	.06	.05	.24	.10	.22	.15	.34**	.23	--			
11. SUDS-A-PIII-Post ^d	.09	.01	-.04	.12	-.01	.22	.14	.26	.13	.71**	--		
12. API-PI-Post ^e	.07	.41**	.21	.19	.12	.33**	.19	.33**	.12	.48**	.41**	--	
13. API-PIII-Post ^f	.11	.31*	.20	.33*	.30*	.38**	.29*	.30*	.28*	.43**	.47**	.83**	--

Note: $N = 69$. Age-A = Adolescent Age; CASI-Base = Child Anxiety Sensitivity Index, baseline; CASI-Post = Child Anxiety Sensitivity Index, post-intervention; CASI-P-II = Child Anxiety Sensitivity Index, phase II; CASI-P-III = Child Anxiety Sensitivity Index, phase III; RCADS-GA-Base = Revised Child Anxiety and Depressive Scales- Generalized Anxiety Symptom Subscale, baseline; RCADS-GA-P-III = Revised Child Anxiety and Depressive Scales- Generalized Anxiety Symptom Subscale, phase III; RCADS-P-Base = Revised Child Anxiety and Depressive Scales- Panic Symptom Subscale, baseline; RCADS-P-P-III = Revised Child Anxiety and Depressive Scales- Panic Symptom Subscale, phase III; SUDS-A-P-I-Post = Subjective Units of Distress, Anxiety phase I post-task; SUDS-A-P-III-Pre = Subjective Units of Distress, Anxiety phase III post-task; API-P-I-Post = Acute Panic Inventory, phase I post-task; API-P-III-Pre = Acute Panic Inventory, phase III post-task.

^a $N = 64$. ^b $N = 63$. ^c $N = 68$. ^d $N = 56$. ^e $N = 66$. ^f $N = 54$.

* $p < .05$. ** $p < .01$.

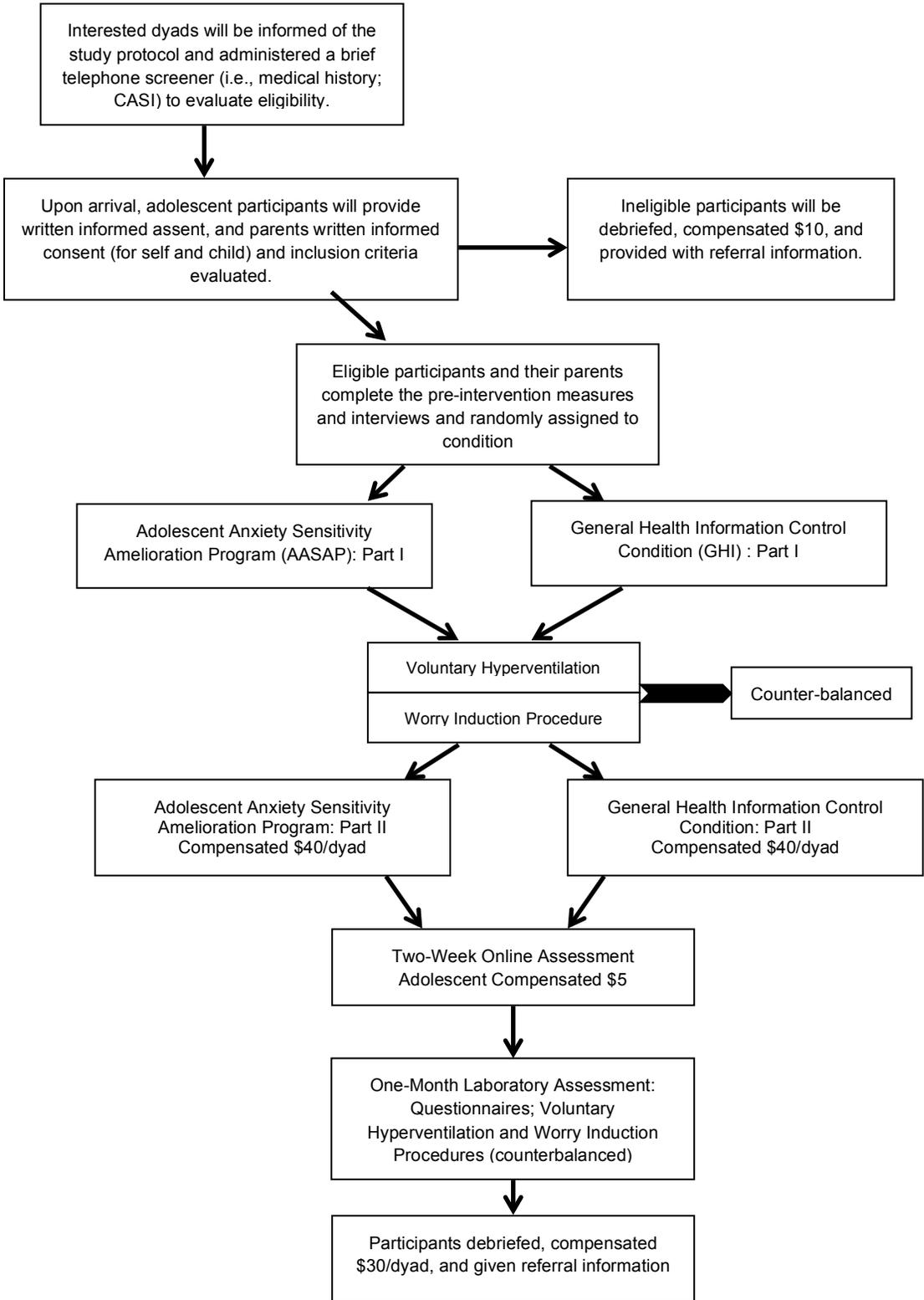


Figure 1. Graphical illustration of procedures to be utilized with participants.

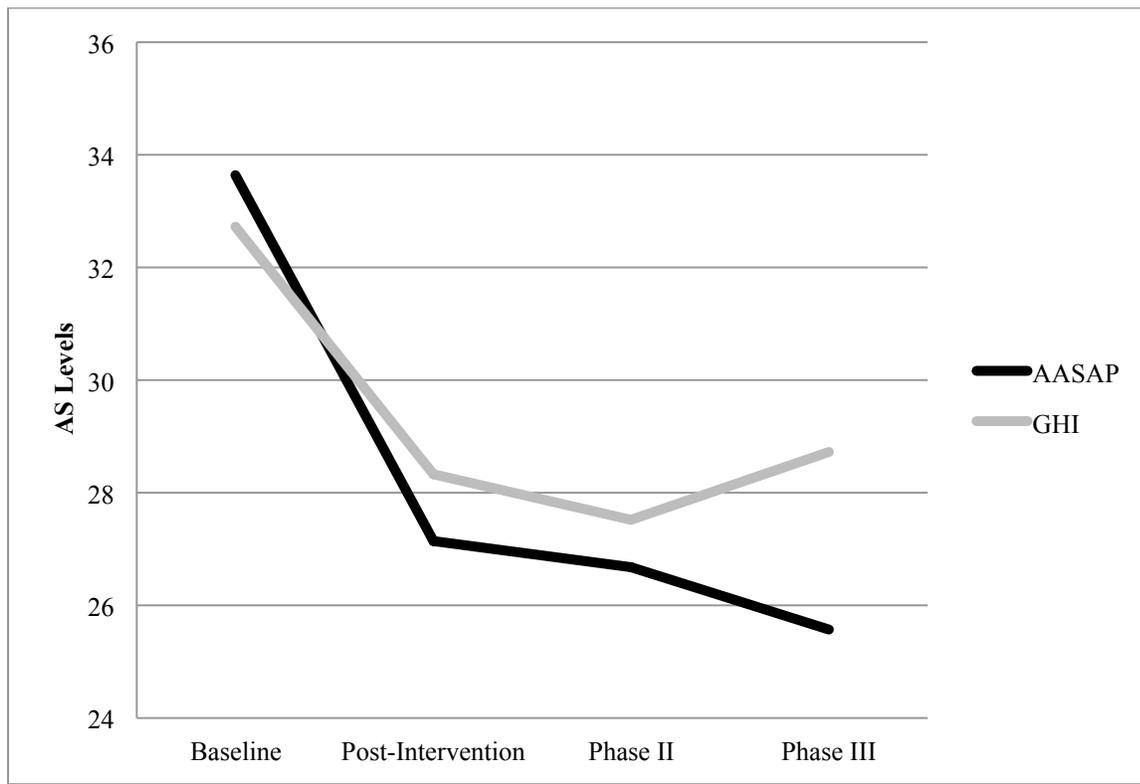


Figure 2. Trajectories of AS across the study phases as a function of condition.

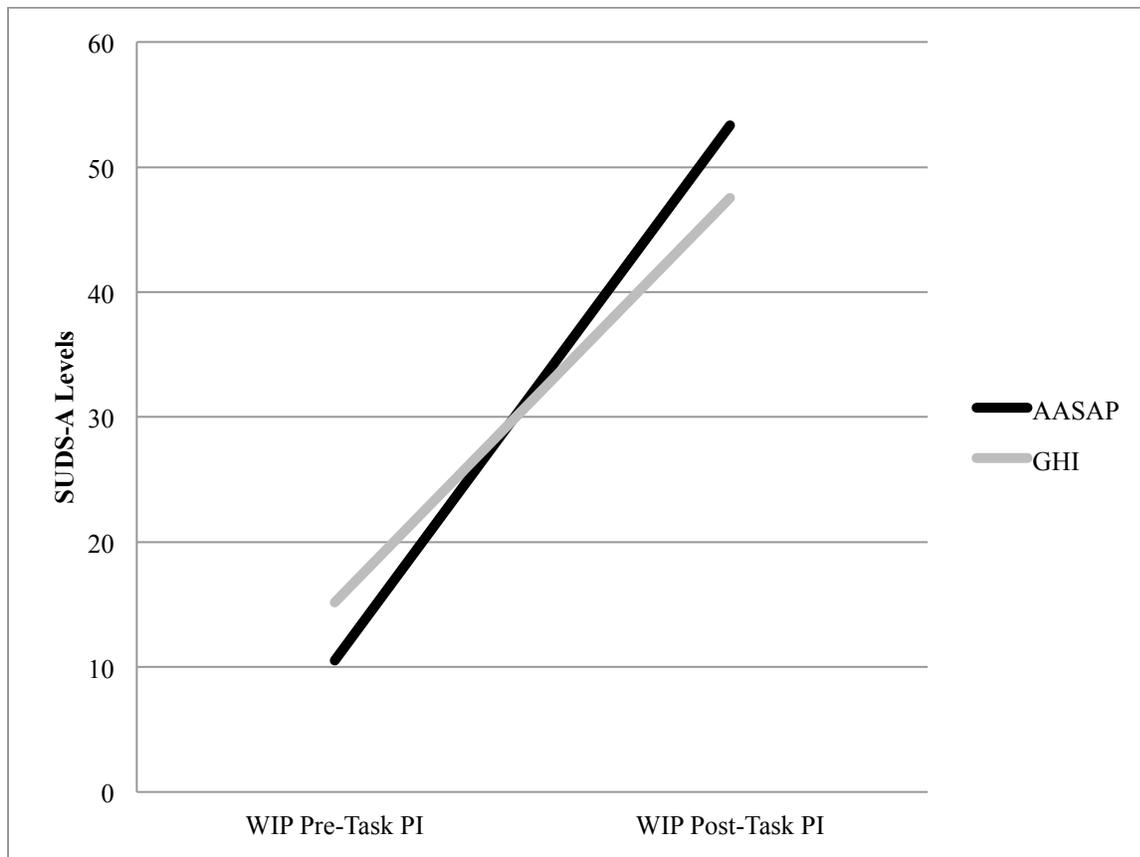


Figure 3. Graphical illustration of SUDS-A pre- and post-worry induction at phase I as a function of condition.

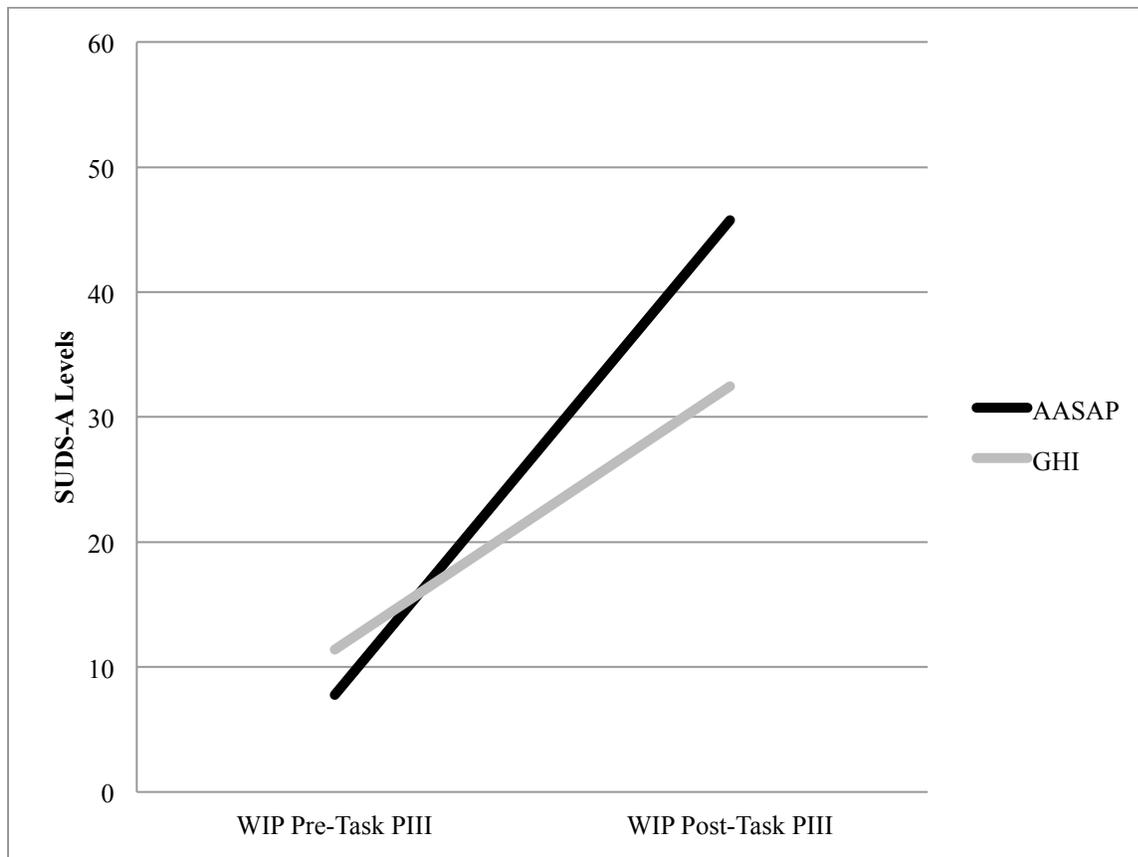


Figure 4. Graphical illustration of SUDS-A pre- and post-worry induction at phase III as a function of condition.

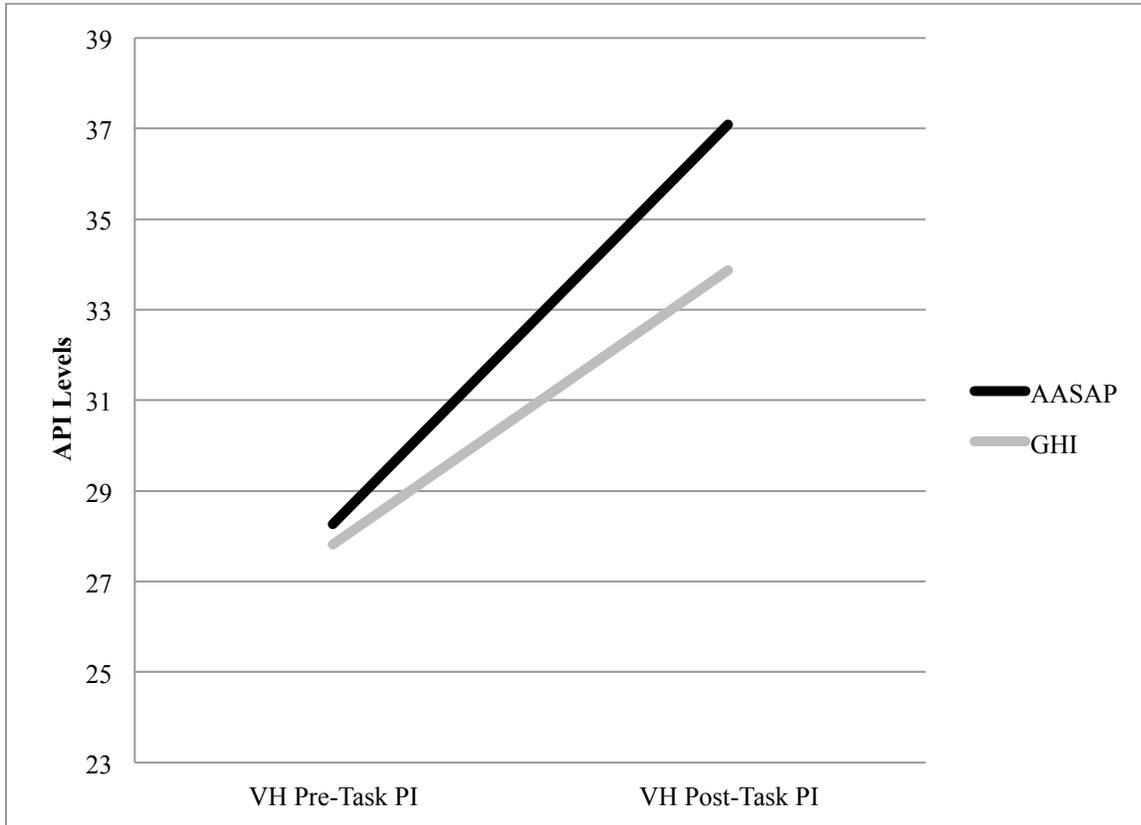


Figure 5. Graphical depiction of API pre- and post-VH task at phase I as a function of condition.

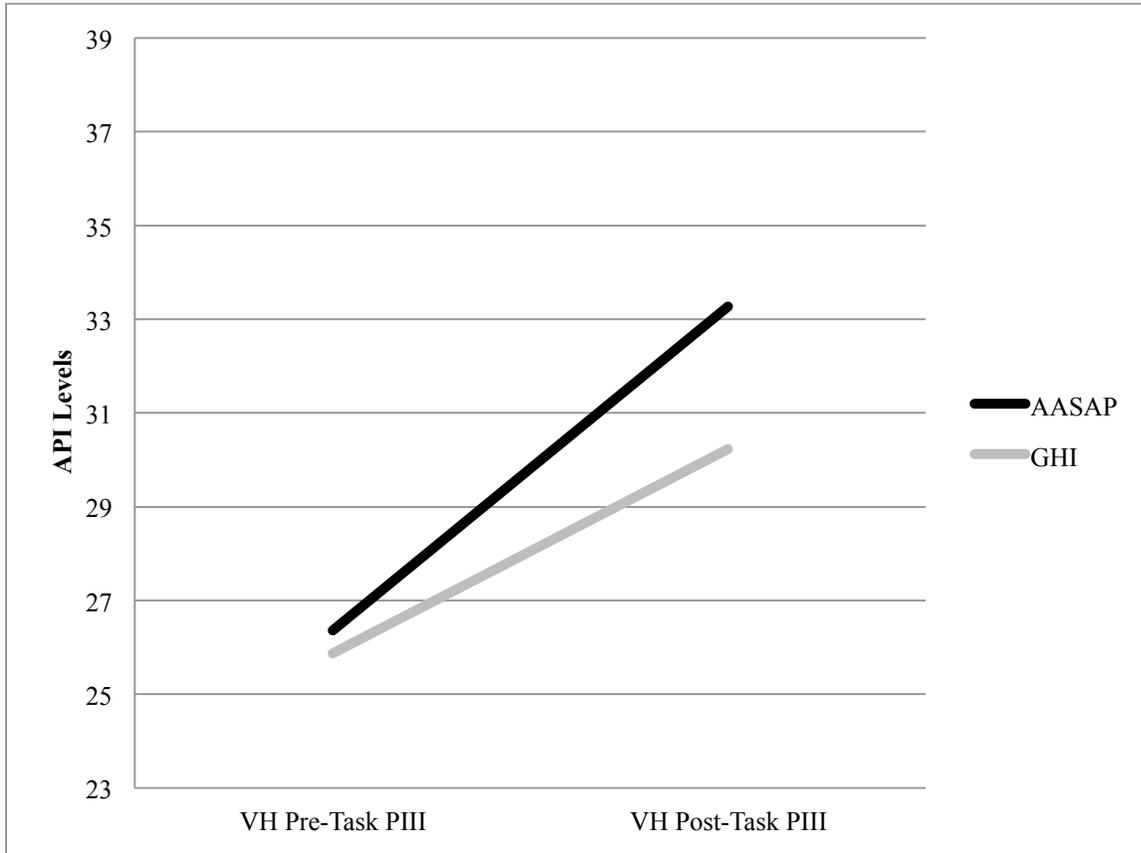


Figure 6. Graphical depiction of API pre- and post-VH task at phase III as a function of condition.

Appendix A



Office of Research Compliance
Institutional Review Board

August 6, 2013

MEMORANDUM

TO: Ashley Knapp
Ellen Leen-Feldner

FROM: Ro Windwalker
IRB Coordinator

RE: New Protocol Approval

IRB Protocol #: 13-07-028

Protocol Title: *A Test of an Adolescent Anxiety Sensitivity Amelioration Program for At-Risk Youth*

Review Type: EXEMPT EXPEDITED FULL IRB

Approved Project Period: Start Date: 08/05/2013 Expiration Date: 08/04/2014

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form *Continuing Review for IRB Approved Projects*, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (<http://vpred.uark.edu/210.php>). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 200 participants. If you wish to make any modifications in the approved protocol, including enrolling more than this number, you must seek approval *prior to* implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 210 Administration Building, 5-2208, or irb@uark.edu.