Published in final edited form as:

J Anxiety Disord. 2010 June ; 24(5): 503–508. doi:10.1016/j.janxdis.2010.03.007.

Anxiety Sensitivity: Prospective Prediction of Anxiety among Early Adolescents

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Abstract

Emerging evidence suggests that anxiety sensitivity (AS) predicts subsequent development of anxiety symptoms and panic attacks as well as clinical syndromes in adult samples. The primary aim of the present study was to determine whether AS similarly acts as a vulnerability factor in the pathogenesis of anxiety symptoms among youth in early adolescence (ages 9–13). A large nonclinical community sample of youth ($N = 277$) was prospectively followed over one year. The Childhood Anxiety Sensitivity Index (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991) served as the primary predictor. After controlling for baseline anxiety symptoms as well as depression, AS significantly predicted the future development of anxiety symptoms. Consistent with the adult literature and expectancy theory, AS appears to act as a risk factor for anxiety symptoms in youth.

Keywords

anxiety sensitivity; anxiety; prospective; longitudinal

Expectancy theory (Reiss, 1991) and contemporary psychosocial accounts of anxiety psychopathology posit that fear of anxiety (i.e., anxiety sensitivity; AS) is important in the development and maintenance of these conditions (Barlow, 2002; Clark, 1986; McNally, 1990). AS is a relatively stable cognitive characteristic that predisposes individuals to the development of anxiety problems (Taylor, 1999); it encompasses fears of physical, mental, and publicly observable experiences (Zinbarg, Barlow, & Brown, 1997), all of which are believed to amplify preexisting anxiety (Reiss, 1991). Thus, individuals with high AS are theorized to perceive bodily sensations associated with autonomic arousal as a sign of imminent personal harm and, as a result, to experience elevated levels of anxiety and be at increased risk for a panic attack.

Individual differences in AS are hypothesized to emerge from the combined influences of genetic variation along with any number of learned experiences that ultimately lead to the

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acquisition of beliefs about the potentially aversive consequences of arousal and anxiety-related states (Reiss & Haverkamp, 1998). Research conducted across diverse populations has supported the AS model, providing strong evidence of cross-cultural and developmental specificity in terms of the latent structure and stability of the construct (Chorpita & Daleiden, 2000; Muris, Schmidt, Merckelbach, & Schouten, 2001; Zinbarg, Brown, & Barlow, 2001; Zvolensky, Kotov, Antipova, Leen-Feldner, & Schmidt, 2005; Zvolensky, McNeil, Porter, & Stewart, 2001). AS also is unique from, and demonstrates incremental validity to, trait anxiety (Rapee & Medoro, 1994) as well as negative affectivity (Zvolensky, Kotov, Antipova, & Schmidt, 2005).

Unlike many other cognitive conceptualizations of anxiety, AS is believed to be a dispositional characteristic that may precede the development of clinical anxiety symptoms or diagnoses. Empirical studies provide converging evidence that AS does indeed act as a risk factor for anxiety problems. First, laboratory studies indicate that baseline AS predicts fear responses to bodily sensations (Rabian, Embry, & MacIntyre, 1999; Unnewehr, Schneider, Margraf, & Jenkins, 1996; Zvolensky, Goodie, McNeil, Sperry, & Sorrell, 2001); these effects are observed above and beyond variance accounted for by trait anxiety (Zinbarg, et al., 2001). Second, AS levels are elevated among individuals with anxiety disorders compared to those without anxiety disorders (Kearney, Albano, Eisen, Allan, & Barlow, 1997; Rabian, Peterson, Richters, & Jensen, 1993; Taylor, Koch, & McNally, 1992). Third, prospective studies with healthy adults (Schmidt, Lerew, & Jackson, 1997, 1999) and adolescents (Hayward, Killen, Kraemer, & Taylor, 2000) indicate that AS predicts the future occurrence of anxiety symptoms and panic attacks, even after controlling for trait anxiety. Additionally, AS predicts the maintenance of panic disorder among untreated patients, the prospective emergence of panic attacks among infrequent (nonclinical) panickers, and the emergence of panic among individuals free from a history of panic attacks (Ehlers, 1995), thereby indicating that AS is a predictor of (future) panic-related problems over time. Finally, AS has been found to prospectively predict the development of clinically significant anxiety syndromes (Schmidt, Zvolensky, & Maner, 2006).

Work on AS has expanded dramatically as accumulating evidence supports its relationship to anxiety psychopathology. Although the vast majority of studies on AS have focused on college age or adult samples, AS research among children and adolescents has recently emerged and is consistent with the more extensive adult AS literature. For example, AS has been associated with general anxiety symptoms in both children and adolescents and has shown specificity to anxiety over depression (e.g., Joiner, et al., 2002; Lambert, Cooley, Campbell, Benoit, & Stansbury, 2004). AS also is associated with symptoms of various anxiety disorders in youth. Specifically, cross-sectional research with children and adolescents has indicated that AS is associated with panic symptomatology (Calamari, et al., 2001; Deacon, Valentiner, Gutierrez, & Blacker, 2002) and with panic disorder (Kearney, et al., 1997; Lau, Calamari, & Waraczynski, 1996). Further, research with trauma exposed children and adolescents indicates that AS is associated with PTSD symptoms above and beyond trauma exposure (Hensley & Varela, 2008; Leen-Feldner, Feldner, Reardon, Babson, & Dixon, 2008). Biological challenge studies have further validated this construct among nonclinical youth by demonstrating that AS is predictive of fearful reactivity to voluntary hyperventilation paradigm among adolescents (Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky, 2005) and to a stair stepping challenge among children (Rabian, et al., 1999).

Similar to the adult AS literature, the prospective evaluation of AS among community samples of youth is limited. There are two relevant studies with youth well into adolescence. In a 6-month longitudinal investigation among African-American adolescents (*M* age = 15.6), Ginsburg and colleagues (2002) reported that AS was associated with elevations in panic symptoms. However, baseline AS levels did not predict subsequent panic symptoms after

controlling for baseline panic symptoms. Hayward and colleagues (2000) conducted a four year, longitudinal study within a large diverse sample of high school students (M age = 15.4). Results indicated that elevated AS predicted the future occurrence of 4-symptom panic attacks (Hayward, et al., 2000). AS also predicted more severe panic attacks and anxious avoidance (Wilson & Hayward, 2006). Additionally, Hayward et al. (2000) reported that when controlling for relevant factors, AS showed specificity in predicting panic, but not depression. Further exploration of this sample, indicated that those with stable high or escalating AS over time were more likely to have experienced a panic attack; however, panic was not a strong predictor of future AS scores (Weems, Hayward, Killen, & Taylor, 2002)

To date, Weems and colleagues (2007) have conducted the only longitudinal investigation of AS including early adolescents ($n = 145$; $M = 11.36$ years). Among this ethnically-diverse sample, fifty-two participants completed a follow-up assessment one year later ($M = 11.15$ years), where baseline AS was predictive of anxiety disorder symptomatology as measured by the Revised Child Anxiety and Depression Scales (RCADS) at the one year follow-up. Although this investigation meaningfully contributes to the prospective AS literature among youth, the follow-up sample was a small subset of the original sample, and due to limited details, it is difficult to determine the extent to which this subset differs systematically from the overall sample. Perhaps most importantly, the analyses did not control for baseline anxiety, which renders the predictive validity of the CASI in this case largely uninterpretable (i.e., it is unclear if AS is a predictor of change in anxiety symptoms).

The extant literature examining the effects of AS on youth is consistent with the adult literature and suggests that AS may act as a premorbid risk factor for the genesis of anxiety problems among children and adolescents. However, this has not been clearly demonstrated due to the limited prospective investigations of AS among youth, particularly among youth in early adolescence. This is particularly notable given the age of onset for many anxiety disorders tends to peak in adolescence or early adulthood (American Psychiatric Association, 2000; Kessler, et al., 2005). Thus, it is crucial to prospectively investigate AS in this age group to truly evaluate AS as a risk factor in the development of these anxiety disorders.

Together, the current study provides a more definitive evaluation of whether AS acts as a premorbid risk factor for the pathogenesis of anxiety problems by including a large nonclinical sample of youth in early adolescence. The study prospectively followed 277 nonclinical participants ages 9-13 over a one-year time frame. It was hypothesized that AS would act as a cognitive diathesis, placing individuals at risk for the development of anxiety during the follow-up period, even after controlling for baseline levels of anxiety (see Lilienfeld, Turner, & Jacob, 1993). More specifically, it was hypothesized that, independent of baseline levels of anxiety, AS would predict the changes in anxiety symptoms during follow-up. We also predicted that AS would show specificity in terms of being relatively more strongly associated with anxiety versus mood symptoms over time.

1. Method

1.1. Participants

Data were collected from a community sample of 277 early adolescents and their primary caregiver (80.5% mothers) participating in a larger prospective study of behavioral, environmental, and genetic mechanisms of risk for HIV-related risk behaviors in youth. Of the original 277 subjects, 5 subjects (1%) were deleted due to incomplete data specific to hypotheses in this manuscript. These 5 subjects did not significantly differ from the retained subjects on any demographic variable. The final sample consisted of 272 subjects. At baseline, the average age of this sample was 11.00 years ($SD = .82$; range, 9-13 years) and 44.1% ($n = 120$) of the sample was female. In terms of racial/ethnic background, 49.4% ($n = 134$) of the

sample indicated European American, 35.1% ($n = 95$) indicated African American, 3.0% ($n = 8$) indicated Hispanic, and 12.5% ($n = 35$) indicated other. At follow-up, 31 subjects did not return for the study (11%). These 31 dropout subjects did not significantly differ from the retained subjects on any demographic variable or major study variable (Follow-up $n = 244$).

Permission to conduct research was obtained from the University of Maryland Institutional Review Board (IRB). Families were recruited through media outreach in the greater Washington, DC metropolitan area, as well as through contact with area schools, libraries, and Boys and Girls Clubs. The study was advertised as an investigation of youth health related behaviors. Interested families called to set up an appointment and were informed on the phone that we were conducting a study examining youth risk-taking behaviors. Families were eligible for participation if the child was in 5th or 6th grade and both primary caregiver and child were fluent in English. Interested families who met inclusion criteria were invited to come to the Center for Addiction, Personality, and Emotion Research (CAPER) located on the University of Maryland campus. Upon arrival at the assessment session, a more detailed description of the study procedures was provided and the primary caregivers and youth signed informed consent/assent.

1.2. Measures

1.2.1. Demographics—The parent/guardian completed a basic demographics form for personal information, as well as information about the child. The form included age, gender, race, status of biological father's presence in the home (yes/no), and annual family income. The total family income variable was positively skewed ($s = 4.81$, $SE = .15$); thus, a square root transformation was conducted (Tabachnick & Fidell, 1996). The resulting skew was less than 2 ($s = .94$, $SE = .15$).

1.2.2. The Revised Child Anxiety and Depression Scales—(RCADS; Chorpita & Daleiden, 2000). The RCADS was designed to assess symptoms of DSM-IV defined anxiety disorders and major depression including subscales targeting major depression (ten items; e.g., "I feel sad or empty"), social phobia (nine items; e.g., "I am afraid of looking foolish in front of people"), panic disorder (nine items; e.g., "My heart suddenly beats too quickly for no reason"), separation anxiety disorder (seven items; e.g., "I fear being away from my parents"), generalized anxiety disorder (six items; e.g., "I worry that bad things will happen to myself"), and obsessive-compulsive disorder (six items; e.g., "I have to do things just right to stop bad things"). Items ask about frequency of symptoms (never, sometime, often, always) and are scored on a 4-point Likert scale. RCADS total anxiety score was obtained by summing across all relevant anxiety items. Likewise RCADS depression score was obtained by summing the depression items. The RCADS has demonstrated convergent validity with existing measures of childhood anxiety and anxiety disorders (Chorpita & Daleiden, 2000). The internal consistency in the present sample was good with alpha coefficients of .82 for the depression subscale and .94 for all the anxiety subscales.

1.2.3. Childhood Anxiety Sensitivity Index—(CASI; Silverman, et al., 1991). The 18-item CASI, a modification of the adult 16-item Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986), was used to index anxiety sensitivity. Participants rated their perceptions of the aversive nature of anxiety symptoms by endorsing 1 (none), 2 (some), or 3 (a lot). An example item is, "it scares me when my heart beats fast." CASI scores range between 18 and 54, with higher scores indicating higher levels of anxiety sensitivity. This scale was adapted for, and validated with, a population of children and adolescents (Silverman, et al., 1991; Weems, Hammond-Laurence, Silverman, & Ginsburg, 1998). In the present report, a total CASI score was used because CASI subscales have not been consistently derived in the literature (e.g., Lambert, et al., 2004; Silverman, Ginsburg, & Goedhart, 1999; Silverman,

Goedhart, Barrett, & Turner, 2003). The scale has good psychometric properties and satisfactory convergent validity estimates (Silverman, et al., 1991).

1.3. Procedure

After providing informed consent, participants were escorted into a private room where they completed the self-report questionnaires as part of a larger assessment battery (see Daughters, et al., 2009). All measures were randomly ordered to limit systematic order effects. Sessions lasted approximately 1 hr at baseline and 1.5 hrs at follow-up.

After the baseline session, participants were contacted on a regular basis to minimize attrition. Participants were allowed to complete the follow-up assessment 12 months (+/- 2 months) following baseline. Participants and their guardians were both compensated \$25 at each visit.

2. Results

Table 1 provides means and standard deviations or %'s as appropriate, as well as a correlation matrix of variables assessed at both baseline and Year 1 follow-up. Data indicate a strong baseline to Year 1 correlation for CASI (.54, $p < .01$), RCADS Anxiety (.61, $p < .01$), and RCADS depression (.60, $p < .01$). Baseline CASI was related to RCADS Anxiety at baseline and Year 1 (.64 and .50 respectively; both p 's $< .01$), and to RCADS Depression somewhat more modestly at baseline and Year 1 (.48 and .39 respectively; both p 's $< .01$).

As for the demographic variables, age was negatively related to depressive symptoms at baseline ($r = -.15$, $p < .05$), European Americans had lower CASI scores than African American participants at baseline (10.46 (5.0) vs. 13.85 (7.3); $p < .01$), and income was negatively related to CASI scores at baseline ($-.19$, $p < .01$). Gender was not related to any other variable; however, given the theoretical and empirical links between gender and anxiety/depression, we used gender as a covariate in further analyses along with the other demographic variables despite the absence of correlations with any key predictors.

We conducted two primary regression analyses. The first regression predicted RCADS Anxiety at Year 1 controlling for the demographic variables in the first step, as well as baseline anxiety and depression. Step 2 included the CASI at baseline. In the first step, only RCADS Anxiety at baseline was related to RCADS Anxiety at Year 1, accounting for 11.2% of variance. When added in the second step, CASI was significant accounting for an additional 2.1 % of variance (see Table 2). In addition to including gender and racial/ethnic background as covariates, we examined whether these demographic factors served as moderators in the relationship between CASI at baseline and RCADS Anxiety at Year 1. These analyses indicated that neither gender nor racial/ethnic background were significant moderators.

As summarized in Table 3, the second regression was conducted to examine the specificity of CASI to change in RCADS Anxiety by predicting Year 1 RCADS Depression. All other aspects of the regression remained the same. In the first step, both RCADS Anxiety and Depression at baseline were related to RCADS Depression at Year 1, with the latter accounting for 9.3% of variance and the former 1.2%. When added in the second step, CASI was not significant ($p = .08$; $sr^2 = .008$).

We also conducted exploratory analyses to evaluate whether the CASI showed differential relations among the RCADS Anxiety subscales. Univariate analyses indicated that baseline CASI scores (uncontrolled) were moderately and fairly equivalently associated with follow-up RCADS subscales. As might be expected, significant associations were evident for each subscale including PD ($r = .45$, $p < .001$), OCD ($r = .42$), Separation Anxiety ($r = .42$), GAD ($r = .38$), and Social phobia ($r = .36$) subscales. Five separate regression analyses controlling

for each respective baseline subscale as well as RCADS depression indicated that the CASI added significant incremental contributions for OCD ($p < .001$; $sr^2 = .034$), PD ($p < .01$; $sr^2 = .02$), Separation Anxiety ($p < .001$; $sr^2 = .015$), and GAD ($p = .04$; $sr^2 = .034$); Social phobia did not evidence a significant incremental contribution in its regression ($p = .074$; $sr^2 = .008$).

3. Discussion

Expectancy theory proposes that AS may serve as a premorbid risk factor for the development of anxiety pathology (Reiss, 1991). Empirical evidence for AS as a risk factor for anxiety problems is well-developed (Hayward, et al., 2000; Maller & Reiss, 1992; Schmidt, et al., 1997, 1999; Schmidt, et al., 2006; Weems, et al., 2007). The present study provides a more definitive, prospective test of whether AS acts as a dispositional risk factor for anxiety in a sample of youth in early adolescence. Consistent with expectation, AS incrementally predicted changes in anxiety symptoms over a one-year interval, controlling for depressive symptoms. In addition, consistent with previous findings among adolescents (Weems, et al., 2007), AS showed specificity in predicting anxiety but not depressive symptoms over the follow-up interval. In conjunction with the Weems et al. (2007) prospective study, as well as other work, these data provide additional evidence for AS as a general risk factor for the development of anxiety problems in children and youth. The present report bolsters the status of AS as a general risk factor but additional work is needed to delineate its causal status. Controlled research designs are necessary to document causal effects because they can serve to rule out other competing alternative explanations (e.g., “third variables”).

Although the debate regarding the differentiation between AS and trait anxiety (Lilienfeld, Turner, & Jacob, 1996; McNally, 1996) has been resolved to a great extent, it is still useful to demonstrate that AS is predictive of psychopathology beyond the variance it shares with measures of general anxiety symptoms (Lilienfeld, et al., 1993), particularly among youth in early adolescence, a group who have received modest attention in this debate. Consistent with prior work (Schmidt et al., 1999), findings indicated that AS acted as a significant risk factor even when the effects of baseline anxiety were partialled out. The size of the effect of AS tends to be small, which is not surprising due to the relatively shorter follow-up interval in the present study. Similar effect sizes have been obtained in other studies (e.g., Zvolensky et al., 2005a) that have examined the distinctiveness and incrementality of AS in predicting anxiety symptoms, controlling for potential confounds. These data further highlight the unique role AS may play in the genesis of psychopathology in youth. This finding is particularly notable because children and adolescents are at particularly high risk for the development of an anxiety disorder with the onset of many disorders peaking in adolescents to early adulthood (American Psychiatric Association, 2000; Kessler, et al., 2005). Future work may benefit from extending such types of tests to the maintenance of psychopathology. To the extent that AS increases the risk for maladaptive emotional problems in youth, it would not be surprising if it also facilitated the maintenance of psychopathology among youth with pre-existing clinical conditions, or increased the probability of relapse among youth receiving treatment.

There has been considerable interest in evaluating the three subscales of the original ASI to provide additional clarity regarding the components of AS (Lambert, et al., 2004; Zvolensky & Forsyth, 2002). Some studies suggest that an evaluation of specific sensitivities may yield better predictions depending on the outcomes of interest (Schmidt, et al., 1999; Zinbarg, et al., 2001). For example, Schmidt et al. (1999) found that the cognitive concerns subscale of the ASI was particularly predictive of panic attacks during acute stress. We did not evaluate the subscales in this sample because the youth AS literature is mixed regarding the number of coherent factors in the CASI (e.g., Lambert, et al., 2004; Silverman, et al., 1999; Silverman, et al., 2003). Four first-order factors (Disease concerns, Unsteady concerns, Mental Incapacitation concerns, and Social concerns) were found in a primarily Caucasian sample of

youth (ages 7-16) (Silverman, et al., 2003), whereas only two factors (Physical concerns and Mental Incapacitation concerns) were found in an African-American sample of youth (ages 8-12) (Lambert, et al., 2004). It is unclear if social concerns are a unique component of AS in African-American youth (Lambert, et al., 2004). Carter and colleagues (1999) speculated that African Americans may have different social concerns than European Americans. They noted that African Americans' social concerns about anxiety symptoms may encompass broader concerns about mental incapacitation. Because of this inconsistency with CASI factors across ethnic groups, we did not include analyses of the subscales predicting the development of anxiety symptoms as such analyses may not be prudent until a clearer sense of the most appropriate factor structure is available.

Conclusions must be considered in light of study limitations. First, the present study utilized a community sample that did not receive diagnostic interviews at baseline or follow-up. Therefore, it is difficult to clearly ascertain the clinical significance of the changes in anxiety symptoms that were predicted. Second, the follow-up interval was relatively short to examine the onset of anxiety psychopathology. Future studies, including those that will be available with this sample in future follow-up assessments, should examine increases in and development of anxiety symptoms over longer periods of time.

4. Conclusions

Overall, the present findings add to the existing knowledge base implicating AS as a risk factor for psychopathology. These findings provide strong evidence suggesting that AS serves as a risk factor for development of anxiety symptoms among youth in early adolescence. Because instruction in cognitive behavioral skills can significantly reduce AS (Schmidt, et al., 2007; Smits, Powers, Cho, & Telch, 2004), this offers an exciting possibility for the implementation of a primary prevention intervention that can effectively anticipate and prevent anxiety and panic reactions among high-risk youth (Zvolensky, Schmidt, Bernstein, & Keough, 2006).

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Table 1
Bivariate correlations and descriptive statistics for the main study variables

	<i>M (SD) / %</i>	1	2	3	4	5	6	7	8	9	10
1. Gender	56% Male	XXX	.07	.04	.07	-.10	-.10	-.09	.02	-.01	.03
2. Age	11.0 (0.8)		XXX	-.04	.02	-.12	-.11	-.15*	-.03	-.10	-.05
3. Ethnicity	49% Caucasian			XXX	.35**	-.19**	-.03	.02	-.04	-.02	.06
4. Income	\$93K (74K)				XXX	-.22**	-.11	-.08	-.02	-.03	.00
5. CASI BA	11.6 (6.2)					XXX	.64**	.48**	.54**	.50**	.39**
6. Anxiety BA	25.4 (15.4)						XXX	.79**	.50**	.61**	.54**
7. Depress BA	6.6 (4.5)							XXX	.34**	.50**	.60**
8. CASI Y1	10.1 (5.7)								XXX	.66**	.46**
9. Anxiety Y1	22.4 (13.4)									XXX	.76**
10. Depress Y1	6.1 (4.3)										XXX

Note: Gender coded as Female = 0 and Male = 1; Ethnicity coded as NonCaucasian = 0 and Caucasian = 1; CASI BA and Y1 indicates Total Score on the Child Anxiety Sensitivity Index at the baseline and year 1 assessments; Anxiety BA and Y1 indicates anxiety symptoms score on the Revised Child Anxiety and Depression Scale at the baseline and year 1 assessments; Depress BA and Y1 indicates depressive symptoms score on the Revised Child Anxiety and Depression Scale at the baseline and year 1 assessments. * indicates $p < .05$ and ** indicates $p < .01$.

Table 2
Predicting RCADS Anxiety at Year 1 using baseline CASI scores controlling for demographics as well as baseline RCADS Depression and Anxiety

	B	SE	F	P	Sr2
Step 1: $F(6, 234) = 23.35, p < .001, R^2\Delta = 0.375$					
Age	1.232	1.404	0.877	.381	0.002
Gender	-0.442	0.859	-0.514	.607	0.001
Ethnicity	0.910	1.534	0.593	.554	0.001
Total Family Income	0.003	0.008	0.427	.670	<0.001
Y1 RCADS Depression	0.297	0.247	1.202	.231	0.004
Y1 RCADS Anxiety	0.476	0.073	6.470	<.001	0.112
Step 2: $F(1, 234) = 8.07, p < .01, R^2\Delta = 0.021$					
Age	1.451	1.385	1.047	.296	0.003
Gender	-0.253	0.849	-0.298	.766	0.000
Ethnicity	0.350	1.524	0.230	.819	0.000
Total Family Income	0.006	0.008	0.793	.429	0.002
Y1 RCADS Depression	0.331	0.243	1.358	.176	0.005
Y1 RCADS Anxiety	0.356	0.084	4.252	<.001	0.047
Y1 CASI	0.432	0.152	2.841	.005	0.021

Table 3
Predicting RCADS Depression at Year 1 using baseline CASI scores controlling for demographics as well as baseline RCADS Depression and Anxiety

	B	SE	F	P	Sr ²
Step 1: F(6, 234) = 24.94, p < .001, R ² Δ = 0.390					
Age	0.728	0.441	1.652	.100	0.007
Gender	0.225	0.270	0.836	.404	0.002
Ethnicity	-0.378	0.481	-0.785	.434	0.002
Total Family Income	0.000	0.002	0.145	.885	0.000
Y1 Depression	0.462	0.077	5.965	<.001	0.093
Y1 Anxiety	0.049	0.023	2.117	.035	0.012
Step 2: F(1, 234) = 3.18, p .076, R ² Δ = 0.008					
Age	0.772	0.439	1.757	.080	0.008
Gender	0.263	0.269	0.976	.330	0.002
Ethnicity	-0.489	0.483	-1.012	.312	0.003
Total Family Income	0.001	0.002	0.372	.710	0.000
Y1 Depression	0.469	0.077	6.073	<.001	0.095
Y1 Anxiety	0.025	0.027	0.943	.346	0.002
Y1 CASI	0.086	0.048	1.783	.076	0.008