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# Suicide risk among male substance users in residential treatment: Evaluation of the depression–distress amplification model



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## ABSTRACT

Suicide is a leading cause of death and is significantly elevated among those with substance use disorders (SUDs). However, specific mechanisms of suicide in this population have been relatively understudied. The depression–distress amplification model posits that one pathway to increased suicide risk is through the intensification of depressive symptoms by anxiety sensitivity cognitive concerns. However, this model has not been tested in populations with SUDs. The current study tested the depression–distress amplification model of suicide risk and examined the relation of anxiety sensitivity to suicide risk in a sample of men in residential SUD treatment. Consistent with prior work, anxiety sensitivity cognitive concerns were significantly associated with suicide risk. Moreover, and consistent with the depression–distress amplification model, anxiety sensitivity cognitive concerns related to elevated suicide risk among those with a current major depressive episode specifically, above and beyond insomnia (another risk factor for suicide) and relevant covariates. The results of this study corroborate the relevance of anxiety sensitivity cognitive concerns and the depression–distress amplification model to suicide risk in an at-risk clinical sample of SUD patients. Findings suggest the importance of assessing anxiety sensitivity cognitive concerns and targeting this vulnerability through brief interventions to reduce suicide risk.

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## 1. Introduction

Suicide is a major public health concern and one of the leading causes of death in the United States (Heron, 2015). One population that has been found to exhibit significantly elevated rates of death by suicide is individuals with substance use disorders (SUDs; Pompili et al., 2009). For example, heroin users are 14 times more likely to die from suicide than their non-heroin using peers (Darke and Ross, 2002) and alcohol dependent individuals are estimated to have 60–120 times greater suicide risk than non-psychiatric populations (Sher, 2006). Thus, there is a need to identify factors that may increase risk for suicidal behavior within this population.

One factor that warrants investigation in this regard is anxiety sensitivity (AS). AS is a cognitive–emotional vulnerability factor reflecting the fear of anxious arousal. It is composed of three facets

relating to fears stemming from the perceived negative physical, social, and cognitive consequences of anxiety (Zinbarg et al., 1997). Although AS was originally conceptualized as a risk factor for anxiety disorders (Cox et al., 1999), AS has also been found to be elevated among individuals with substance use disorders (Stewart et al., 1997; Lejuez et al., 2006).

The cognitive facet of AS, AS cognitive concerns (ASCC), has specifically been identified as a risk factor for suicidal ideation (Schmidt et al., 2001; Capron et al., 2012a, 2012b). ASCC refer to exaggerated fears of going crazy or losing control of mental processes in the context of anxiety and stress. Research has shown that this particular facet of AS is independently associated with mood and trauma-related disorders (Olthuis et al., 2014), in contrast to the pattern of findings for overall AS or AS physical concerns, which generally demonstrate the strongest associations with panic disorder (Olatunji and Wolitzky-Taylor, 2009). ASCC are theorized to increase suicide risk by increasing distress related to depression symptoms that are commonly found in these mood and trauma-related disorders – a process referred to as the

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depression–distress amplification model (Capron et al., 2013b).

There is emerging support for the depression–distress amplification model in adults and adolescents. In a sample of clinical outpatients, findings revealed a significant relationship between ASCC and suicidal ideation above and beyond distress tolerance, gender, and depressive symptoms. Moreover, consistent with the depression–distress amplification model, depressive symptoms significantly moderated the relationship between ASCC and suicidal ideation (Capron et al., 2013b), with ASCC relating to suicidal ideation only among those with elevated depression symptoms. A recent study of a large sample of young adults with elevated risk of suicidal ideation (Beck Scale for Suicidal Ideation > 0) corroborated these findings. Specifically, not only were ASCC the only factor of AS to evidence a significant association with suicidal ideation in this sample, they were related to elevated suicidal ideation only among individuals high (vs. low) in depression (Capron et al., 2014). Finally, results of a recent longitudinal study of 524 eighth graders revealed that ASCC significantly predicted suicidal ideation two years later among youth with high (but not low) levels of current depression (Capron et al., 2015).

The current study represents the first examination of the depression–distress amplification model in a sample of male SUD patients. We chose to focus exclusively on men for several reasons, including (a) evidence that male SUD patients are at greater risk for completed suicide than female SUD patients (Conner et al., 2003); (b) the relative lack of research on suicidality in male versus female SUD populations (Ilgen et al., 2007); (c) differences in the symptom presentation of depression among men and women (Angst et al., 2002; Marcus et al., 2005); and (d) the well-documented gender differences in a variety of suicide-related risk factors in SUD patients, including psychiatric comorbidity, impulsivity, traumatic exposure, and substance use severity (Brady and Randall, 1999; Sonne et al., 2003; Zilberman et al., 2003; Lejuez et al., 2007). To begin to establish the unique relevance of the depression–distress amplification model of suicide risk within this population, we also explored whether this model relates to suicide risk above and beyond another well-established risk factor for suicide: insomnia symptoms (McCall et al., 2010; Nadorff et al., 2011). Indeed, one consideration that has not been addressed in the extant ASCC and suicide literature is the extent to which this relation may be accounted for by their shared associations with insomnia. As noted previously, ASCC have been found to evidence the strongest associations with mood and trauma-related disorders – disorders for which insomnia-type sleep disturbances are also key symptoms. Therefore, it is possible that the depression–distress amplification model is only a proxy for the more well-established relationship between insomnia and suicide.

The primary aim of the current study was to test the depression–distress amplification model of suicide risk within an at-risk clinical sample (i.e., patients in residential SUD treatment) by examining the interaction of ASCC and depression in relation to suicide risk, above and beyond another risk factor for suicide (i.e., insomnia) and relevant covariates. Based on the depression–distress amplification model, it was hypothesized that ASCC would interact with current major depressive episode (MDE) status to predict suicide risk (Capron et al., 2013b, 2014, 2015), above and beyond insomnia symptoms and other relevant covariates. Given the symptom overlap between insomnia and ASCC (e.g., racing thoughts, struggling to control thoughts), a secondary aim was to test an alternative model wherein MDE interacts with insomnia symptoms to predict suicide risk above and beyond ASCC and other relevant covariates. Given previously demonstrated support for the depression–distress amplification model (Capron et al., 2013b, 2014, 2015), we did not expect to find support for this alternative model.

## 2. Method

### 2.1. Participants

A sample of 111 adult male patients ( $M_{age}=34$ ,  $SD=10.47$ ) in a residential SUD treatment program participated in this study. Approximately half the sample self-identified as White (54%), with the other half identifying as Black/African-American (45%). One participant self-identified as Asian. In order to be included in the larger study, participants were required to be dependent on alcohol and/or cocaine, obtain a score of > 24 on a Mini-Mental Status Examination (Folstein et al., 1975), and report no current psychotic symptoms.

### 2.2. Measures

#### 2.2.1. Structured clinical interview for the DSM-IV for axis I disorders (SCID-I)

The research version of the SCID-I (SCID-I/NP; First et al., 2002) was used to assess current SUDs. The SCID-I has demonstrated good validity in SUD populations (Kranzler et al., 1996).

#### 2.2.2. MINI-international neuropsychiatric interview 6.0 (MINI)

The MINI (Sheehan et al., 1998) is a structured clinical interview that was used to assess current (non-SUD) psychiatric disorders (including current MDE) and suicide risk. To determine suicide risk, participants responded to 11 questions assessing suicidal ideation, plans, preparation, and attempt history. Suicide risk scores range from 0 to 52 and are used to classify individuals as having low (1–8), moderate (9–16), and high current suicidality ( $\geq 17$ ). The MINI has demonstrated high specificity for each evaluated disorder and excellent inter-rater reliability (Sheehan et al., 1998). The MINI suicidality total score has been previously used to measure suicide risk in clinical populations (Hearon et al., 2015). Interviews were conducted by bachelors- or masters-level clinical assessors trained to reliability with the principal investigator (MTT) and co-investigator (KLG). All interviews were reviewed by the principal investigator, with diagnoses and outcomes confirmed in consensus meetings.

#### 2.2.3. Anxiety sensitivity index-3 (ASI-3)

The ASI-3 (Taylor et al., 2007) is an 18-item questionnaire used to measure fears of anxiety symptoms due to perceived negative cognitive, physical, and/or social consequences. The ASI-3 has been found to have adequate convergent, discriminant, structural, and criterion-related validity (Taylor et al., 2007). Internal consistency of the cognitive subscale was excellent in the current study (Cronbach's  $\alpha=0.89$ ).

#### 2.2.4. Insomnia severity index (ISI)

The ISI (Bastien et al., 2001) is a 7-item self-report questionnaire that evaluates the nature, severity, and impact of insomnia. The ISI has demonstrated excellent internal consistency and good convergent validity with corresponding variables from sleep diaries (Bastien et al., 2001). Internal consistency was excellent in the present sample (Cronbach's  $\alpha=0.92$ ).

### 2.3. Procedure

All procedures were reviewed and approved by the relevant Institutional Review Boards. Eligible participants were recruited for this study no sooner than 72 h after entry in the facility (to limit the possible interference of withdrawal symptoms on study engagement). Prospective participants were provided with information about study procedures and associated risks, following which written informed consent was obtained. After providing informed consent, bachelors- or masters-level study personnel administered the diagnostic interviews and a questionnaire packet including the measures previously described. Participants were reimbursed \$25 for this assessment session.

## 3. Results

### 3.1. Preliminary analyses

Consistent with past research, the suicide risk variable was positively skewed and kurtotic (skewness=3.17; kurtosis=10.18). Following square-root transformation (as recommended for samples of this size; (Pallant, 2007; Tabachnick and Fidell, 2007), the suicide risk variable approximated a normal distribution (skewness and kurtosis < 2). However, it should be noted that the pattern of findings was the same when using the non-transformed MINI suicide risk variable.

**Table 1**  
Means, standard deviations, and intercorrelations for included measures.

Measure	M (or %)	SD	1	2	3	4	5	6
1. AS physical concerns	7.24	6.05	–					
2. AS cognitive concerns	6.23	5.97	.66*	–				
3. AS social concerns	8.61	5.64	.58*	.72*	–			
4. Current MDE (%)	20.90	.41	.06	.18	.17	–		
5. Insomnia severity	10.04	7.95	.21*	.26*	.28*	.23*	–	
6. MINI – suicide risk	5.51	9.17	.05	.16	.04	.22*	.17	–
7. Number SUD diagnoses	6.56	3.20	–.05	–.05	.01	.12	.14	.36*

Note. AS=anxiety sensitivity. MDE=major depressive episode. MINI=mini international neuropsychiatric interview. SUD=substance use disorder (DSM-IV abuse and dependence).

\*  $p < .05$ .

Means, standard deviations, and intercorrelations for the variables included in analyses are provided in Table 1.

### 3.2. Primary analyses

A multiple regression analysis was performed to examine the relation of the AS facets to suicide risk on the MINI, above and beyond other well-established risk factors for suicidality, including insomnia symptom severity (McCall et al., 2010; Nadorff et al., 2011), SUD pathology (as indexed by the number of SUD diagnoses; see Wilcox et al. (2004)), and current MDE (Beck et al., 1985). The full model was statistically significant and accounted for 17% of the variance in MINI suicide risk scores ( $F(6,93)=4.38$ ,  $p=.001$ ). Among covariates, number of SUD diagnoses ( $t=3.05$ ,  $\beta=.29$ ,  $p=.003$ ,  $sr^2=.09$ ) and current MDE ( $t=2.60$ ,  $\beta=0.25$ ,  $p=0.01$ ,  $sr^2=0.07$ ) were significantly associated with suicide risk, but insomnia severity ( $t=0.63$ ,  $\beta=0.06$ ,  $p=0.53$ ,  $sr^2 < 0.01$ ) was not. In addition, AS physical ( $t=-0.35$ ,  $\beta=-0.04$ ,  $p=0.73$ ,  $sr^2 < 0.01$ ) and social ( $t=-1.75$ ,  $\beta=-0.24$ ,  $p=0.08$ ,  $sr^2=0.03$ ) concerns were not significantly associated with suicide risk. Accounting for these variables, ASCC ( $t=2.24$ ,  $\beta=0.34$ ,  $p=0.03$ ,  $sr^2=0.04$ ) were significantly associated with suicide risk on the MINI.

Next, a multiple regression analysis was performed to test the depression–distress amplification model in this population, above and beyond insomnia symptom severity and number of SUD diagnoses. In addition to the covariates of number of SUD diagnoses and insomnia symptom severity, this model examined the main effects of current MDE and ASCC, as well as the interaction of MDE and ASCC. All variables were mean centered prior to inclusion in the model. Results of the model are listed in Table 2. The full model was statistically significant. Consistent with the

**Table 2**  
Results of multiple regression testing depression distress amplification model and alternate model.

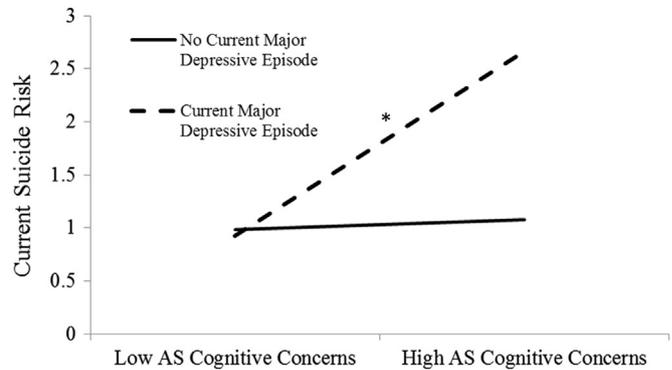
Variable	t	$\beta$	$sr^2$	$R^2$	$\Delta R^2$
Step 1:				.19***	
SUD diagnosis #	2.94	.28**	.08		
ISI	.41	.04	< .01		
MDE current	2.56	.25*	.06		
ASCC	1.38	.13	.02		
Step 2:				.23***	.04*
ASCC X MDE	2.24	.21*	.05		
Alternate Step 2:				.19***	.00
ISI X MDE	–.01	–.01	< .01		

Note. SUD=substance use disorder; ISI=insomnia severity index; MDE=major depressive episode; ASCC=anxiety sensitivity index-3 cognitive concerns.

\*\*\*  $p < .001$ .

\*\*  $p < .01$ .

\*  $p < .05$ .



**Fig. 1.** Interaction of anxiety sensitivity cognitive concerns by current major depressive episode predicting level of suicide risk as hypothesized by the depression distress amplification model. AS=Anxiety sensitivity. \* $p < 0.05$ .

depression–distress amplification model the interaction of MDE and ASCC ( $p=.03$ ) was significant (see Fig. 1). Probing this interaction revealed a significant relation between ASCC and suicide risk among participants with current MDE ( $t=2.64$ ,  $\beta=0.53$ ,  $p=0.01$ ,  $sr^2=0.07$ ); this relation was not significant among participants without current MDE ( $t=0.28$ ,  $\beta=0.03$ ,  $p=0.78$ ,  $sr^2 < 0.01$ ).

Results of a similar model examining the interaction of current MDE and insomnia symptom severity in suicide risk (above and beyond the main effects of number of SUD diagnoses, insomnia symptom severity, ASCC, and current MDE) provide further support for the particular relevance of the ASCC–MDE interaction in suicide risk. Specifically, although the full model was statistically significant, the interaction of current MDE and insomnia symptom severity ( $p=0.98$ ) was not significantly associated with the MINI suicide risk score (see Alternate Step 2 in Table 2).

## 4. Discussion

Consistent with previous studies (Capron et al., 2012c, 2013a), the present study found that ASCC were the only facet of AS to demonstrate significant associations with suicide risk. Moreover, this relationship was significant even after accounting for a number of other suicide-related risk factors, including number of SUD, current MDE, and current insomnia symptom severity. Results also provide further support for the depression–distress amplification model, with ASCC demonstrating a significant association with suicide risk only among patients with current depression. Notably, the interaction of current MDE with insomnia symptom severity (a construct that overlaps with ASCC) was not significantly related to suicide risk.

Findings suggesting the specificity of the depression–ASCC interaction in relation to suicide risk within this sample add to the literature on the utility of the depression–distress amplification model for understanding suicide risk (Capron et al., 2013b, 2014, 2015), extending this research to an at-risk clinical population of SUD patients and providing preliminary support for the relevance of ASCC above and beyond the related risk factor of insomnia. Although caution must be taken when interpreting null findings, findings that current MDE interacted with only ASCC and not insomnia in predicting suicide risk are consistent with the theoretical rationale of the depression–distress amplification model. Specifically, this model is based on the larger AS literature showing that AS constructs such as ASCC amplify the distress and experience of aversive internal sensations (Taylor, 2003; Schmidt et al., 2007). ASCC appear to predispose individuals to respond with increased distress to aversive physical and psychological mood

symptoms. In the depression–distress amplification model, suicidal ideation is considered not only a symptom of depression but a marker of the severity of the depression. Just as AS is thought to increase distress responses in the context of uncomfortable physical sensations (Schmidt et al., 2007), the depression–distress amplification model posits that ASCC amplify distress brought on by the uncomfortable sensations experienced in the context of emerging or existing dysphoria (e.g., lack of concentration, insomnia, anhedonia). Suicidal ideation is then expected to emerge when the distress caused by the amplified depression reaches severe levels.

Despite providing additional support for the depression–distress amplification model, findings must be considered in light of the study limitations. First, our sample consisted entirely of male SUD patients in residential treatment. Therefore, our findings may not generalize to broader clinical populations or female SUD patients. In addition, the cross-sectional nature of the study precludes causal inferences regarding the predictive value of the depression–distress amplification model in relation to increased suicide risk. Although previous work in this area has shown that both ASCC and the depression–distress amplification model are longitudinal predictors of increased suicide risk (Capron et al., 2012a, 2015), additional prospective studies are needed to better establish the temporal relationship between the variables under investigation in this study. Prospective studies would also allow us to better determine if the depression–distress amplification model is associated with future suicide attempts and not just suicide risk. Next, although this study examined several relevant suicide risk factors (including SUD pathology, current MDE, and insomnia symptom severity), suicide risk is multi-faceted and complex. Future studies are needed to demonstrate that the depression–distress amplification model predicts suicidality above and beyond other well-established suicide risk factors (e.g., severe mental illness, hopelessness). Likewise, even though results are consistent with previous literature, the current analyses do not establish that depression–distress amplification is the active mechanism related to increased suicide risk. Future work needs to examine more specifically the amplification aspect of this model and the extent to which ASCC operates to facilitate the transition from suicidal ideation to suicide attempts.

Moreover, given our exclusive reliance on participant self-report (through either the use of questionnaires or diagnostic interviews) to assess all constructs of interest, the relation between ASCC and depression may have been inflated due to common method variance (see Lindell and Whitney (2001)). Multi-method approaches to the investigation of the depression–distress amplification model are needed. Specifically, future studies may benefit from the assessment of ASCC using laboratory-based procedures. Finally, AS was conceptualized as a continuous variable in the present study, given evidence that the AS construct is best represented as a dimensional variable (Broman-Fulks et al., 2008; 2010). Although the vast majority of extant research on the AS construct is based on a dimensional model, it is important to note that alternative conceptualizations of AS taxa (Bernstein et al., 2011) and latent classes (Allan et al., 2014) have been proposed. Future research on the depression–distress amplification model that utilizes a categorical representation of the AS construct may help identify a group of individuals most susceptible to the amplifying effects of AS (i.e., those in higher AS classes), and thus, at greater risk for suicide.

Despite these limitations, there were several notable strengths of this study that deserve mention. First, our study utilized a severe sample of SUD patients. Indeed, participants in this study met criteria for an average of six DSM-IV SUD diagnoses. Despite evidence that substance users are at heightened risk for suicide (Darke and Ross, 2002), research on suicide risk among SUD

patients is limited, and studies on the role of ASCC in suicide risk within this population are particularly scarce. Another strength of the present study was that suicide risk was determined using a semi-structured interview that assessed the full range of the construct from ideation, to plans and preparations, to previous attempt history. Previous work on the depression–distress amplification model has focused exclusively on suicidal ideation outcomes. Recent work in the broader suicidology field has emphasized the importance of better understanding the transition from suicidal ideation to suicide attempt (Klonsky and May, 2014). The results of this study suggest that the depression–distress amplification model may have implications for understanding aspects of suicidality beyond just suicidal ideation. Expanding research on suicide risk beyond suicidal ideation is also important because ideation alone is not as strong a predictor of death by suicide as more proximal suicide-related behaviors, such as plans and preparation (Suominen et al., 2004).

Male patients in residential SUD treatment programs represent a clinically-relevant, at-risk sample for examining suicide-related outcomes. Not only are SUD patients in general at increased risk for death by suicide (Darke and Ross, 2002; Pompili et al., 2009; Sher, 2006), patients in residential SUD treatment programs are at elevated risk for more severe forms of psychopathology and maladaptive, self-destructive behaviors (Chen et al., 2011; Gratz and Tull, 2010). Findings from the present study highlight the importance of assessing ASCC and MDE in male SUD patients in order to identify which patients may be at greatest risk for suicide. Findings also suggest the relevance of incorporating suicide risk reduction interventions into standard residential SUD treatment and highlight potential targets (i.e., ASCC and/or depression symptoms) for these interventions. Given that the stays within residential SUD treatment programs are often short (i.e., approximately 30 days; SAMHSA, 2014), brief interventions are required in this context. One intervention that may hold promise is Cognitive Anxiety Sensitivity Treatment (CAST; Schmidt et al., 2014). CAST is a one-session fully computerized treatment that has been shown to significantly reduce ASCC for at least one month. By reducing ASCC, the amplification of depression symptoms may be prevented, thus reducing suicide risk. Given that CAST is brief and can be delivered in the absence of trained providers, it is a low-resource intervention that may be ideal for this population and context. Moreover, given that AS has been shown to be a unique predictor of residential SUD treatment dropout (Lejuez et al., 2008), CAST may also help reduce risk for other negative outcomes in addition to suicide. The amplification of depression symptoms (and, thus, suicide risk) may also be reduced by directly targeting depression through brief interventions, such as Brief Behavioral Activation (Lejuez et al., 2011). Such interventions may be used alone or in conjunction with each other to target both components of the depression–distress amplification model (ASCC and depression symptoms).

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### Conflicts of interest

All authors declare no conflicts of interest.

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