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Linking Insomnia and Suicide Ideation: The Role of Socio-Cognitive Mechanisms in Suicide Risk

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The Role of Socio-Cognitive Mechanisms in Suicide Risk

by

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Dedication

This thesis is dedicated to the mentors, friends, and family who have supported me in my scientific endeavors. I would like to thank Dr. Edelyn Verona (my faculty advisor), and Dr. Marc Karver, for their support throughout my graduate training and for their thoughtful contributions to my professional and personal development. I am also truly thankful for the tireless support of my good friends, Kim O'Leary, Alexandra Cowden Hindash, and Alex De Nadai, who have motivated me to continue with and complete this work. This work is also dedicated to my parents, who instilled in me a love of learning that eventually lead me to begin this project, and to my grandparents, Jack and Amber King, who have been so supportive throughout this process. Lastly and most importantly, I am beyond grateful to my wonderful husband Jordan Hall – my best friend and strongest supporter. Without him, none of this work would have been possible.

Table of Contents

List of Tables

List of Figures

Abstract

Despite what is known about predictors of suicide risk and consequences of insomnia, research has yet to delineate mechanisms that may explain the known relationship between insomnia symptoms and suicide risk. There is some disagreement in the literature regarding whether this relationship could be primarily explained by recent depressive symptoms, or whether there may be other explanatory factors related to sleep deficits. The present study addressed this contention in the literature by examining 1) whether socio-cognitive variables (e.g. fatigue, appraised social problem-solving ability, and hopelessness) explained this insomnia-ideation relationship, and 2) whether these variables contributed some explanatory variance in suicide ideation above and beyond that explained by depressive symptoms. Approximately 483 female participants completed an online study survey. Cross-sectional path analyses were conducted in order to examine the initial hypothesized path, as well as whether the path persisted when depression was integrated in the model. Results suggest that the hypothesized socio-cognitive factors related to sleep deficits partially mediate the established insomnia-suicide ideation relationship. And, further, that the socio-cognitive pathway from sleep loss to suicide ideation persists even when accounting for recent depressive symptoms, such that both pathways separately explain some degree of this relationship. These findings have meaningful implications for understanding mechanisms by which insomnia symptoms may confer heightened risk for considering suicide.

Overview and Scope of the Problem

Suicide is the third leading cause of death among college-aged individuals in the United States (Centers for Disease Control and Prevention, 2012), resulting in approximately 3,500 deaths each year (Centers for Disease Control and Prevention, 2010). Individuals experiencing suicide ideation, essentially thoughts of engaging in behavior related to suicide (O'Carroll et al., 1996), are at heightened risk for attempting to end their lives since suicide ideation functions as a precursor to suicidal behavior (Reynolds, 1991). Indeed, among individuals who have thoughts of suicide, those who conceptualize a plan for carrying out suicide or who prepare for a suicide attempt are at greater risk of attempting or dying by suicide (Bertolote and Fleishmann, 2002; Kessler, Borges & Walters, 1999; Mann, 2002; Schmidtke et al., 2004). Further indicative of the importance of studying suicide ideation, the risk of an individual making a plan to engage in suicidal behavior and to subsequently make an attempt is elevated in the first year of the onset of ideation (Nock et al., 2008).

Given that suicide ideation tends to precede suicidal behavior, the heightened prevalence of suicide ideation among college students is particularly concerning. In a national survey, 55% of undergraduates endorsed having experienced some form of suicidal thinking during their lifetimes, and 6% of students endorsed having seriously considered suicide in the year prior to the survey (Drum et al., 2009). Among the students who endorsed recent ideation, 38% had made a plan for attempting suicide and 14% had made a suicide attempt (Drum et al., 2009). Given the importance of suicide ideation as a precipitant of suicidal behavior in general, and its

high prevalence among undergraduates, college students who experience suicide ideation comprise an important risk group. Thus, identifying potentially modifiable risk factors that contribute to college students' propensity to experience the onset of suicide ideation may enable more effective intervention and prevention of these deaths. The purpose of the present study is to examine insomnia symptoms as one such risk factor, and to identify putative mechanisms by which it contributes to suicide ideation.

Insomnia Symptoms

Insomnia symptoms may be one potentially modifiable risk factor that increases the risk of experiencing suicide ideation. Insomnia symptoms are characterized by difficulty falling asleep, staying asleep, or experiencing non-restorative sleep, accompanied by reduced daytime functioning (e.g. fatigue, attention deficits, memory impairment) as a result of the sleep disturbance (American Psychiatric Association, 2000) Among college-aged youth, approximately 4% meet DSM-IV criteria for clinical levels of insomnia, and a more expansive 11%-28% experience chronic insomnia symptoms at levels below the threshold needed for an insomnia diagnosis (Ford & Kamerow, 1989; Hicks, Conti, & Pellegrini, 1992; Johnson, 2006; Taylor & McFatter, 2003). These prevalence rates are particularly relevant to the present study since individuals who experience clinical and subclinical insomnia symptoms are more likely to have thoughts of suicide (Pigeon, Pinquart, & Connor, 2012). Indeed, individuals who experience persistent insomnia symptoms are more likely to develop thoughts of suicide in the future (Li et al., 2012; Suh et al., 2013). And, individuals who experience insomnia symptoms and suicide ideation during the same period experience more severe levels of suicide ideation than individuals who experience ideation without sleep deficits (Fawcett et al., 1990; Krakow et al., 2000; Pigeon, Pinquart, & Connor, 2012).

Depressive Symptoms as a Potential Explanation

While the temporal relationship between insomnia and suicide ideation has been established by research, the means by which insomnia symptoms contribute to suicide ideation have not yet been delineated by research. It may be that depressive symptoms explain this relationship. Indeed, research suggests that both subclinical (Breslau, Roth, Rosenthal, & Andreski, 1996; Buysse et al., 2008) and clinical (Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Neckelmann, Mykletun, & Dahl, 2007; Weissman, Greenwald, Nin˜o-Murcia & Dement, 1997) levels of insomnia symptoms increase the risk of future onset of depression, which is the mental disorder that is most commonly associated with suicide ideation (Janson-Fro¨jmark & Lindblom, 2008; Mann et al., 2005; Wulsin, Vaillant, & Wells, 1999). As such, insomnia may distally contribute to the onset of ideation via its relationship with depression (Breslau et al., 1996; Buysse et al., 2008; Morphy et al., 2007; Necklemann et al., 2007; Weissman et al., 1997). It is also known that insomnia symptoms often co-occur with depression (Ford & Kamerow, 1989; Janson-Fro¨jmark & Lindblom, 2008), and individuals who experience this concurrence of symptoms are more likely to become suicidal (Ağargün, Mehmed, Kara, & Solmaz, 1997). Thus, it may be that insomnia symptoms contribute indirectly to ideation through association with depression (Ford & Kamerow, 1989; Janson-Fro¨jmark & Lindblom, 2008).

Although there is some research to suggest that depressive symptoms may function as a mechanism explaining the insomnia-suicide ideation relationship, several studies suggest that insomnia symptoms contribute risk to suicide ideation independently of depression as well (Bernert et al., 2005; Keshavan et al., 1994; Smith, Perlis, & Haythornthwaite, 2004). Indeed, a meta-analysis examining sleep and mental health found that a significant relationship between insomnia symptoms and suicide ideation remains after controlling for the presence or absence of

depression (Pigeon, Pinquart, & Connor, 2012). This relationship holds even after controlling for symptoms specific to depression that are strongly associated with ideation, such as depressed mood. Thus, while research suggests that insomnia symptoms contribute unique variance to suicide ideation, studies have yet to identify mechanisms unique to sleep deficits that may explain this relationship. Theoretically, we propose that this independent relationship may be partially accounted for by social-cognitive consequences (Baumeister et al., 1998) of insomnia symptoms that may influence problem-solving.

Alternative Social-Cognitive Model

It may be that deficits in socio-cognitive functioning associated with insomnia symptoms function as a mechanism by which sleep disturbances contribute to suicide risk. Indeed, research suggests that experiencing chronic insomnia symptoms may reduce the energy available to address real-world problems or stressors (Van Dongen & Maislin, 2003). An individual experiencing such limited cognitive resources may develop low self-confidence in his or her ability to address problems (Maydeu-Olivares & D'Zurilla, 1996). As a result, the individual may begin to perceive that problems may not improve or be resolved in the future (Bonner & Rich, 1988). Such a hopeless thought process may eventually lead the individual to consider suicide (O'Connor, Armitage, & Gray, 2006). Exploration of such a process is of particular utility with regards to suicide prevention efforts, since delineating processes by which insomnia symptoms may contribute to suicide risk may aid understanding of the etiology of suicidal thinking among individuals with sleep deficits. And, furthering understanding of how such thoughts develop may indicate opportunities for early intervention with individuals most at risk of developing these thought processes. The subsequent sections review literature that lends theoretical support for the proposed model.

Fatigue. Fatigue may be one symptom of insomnia that leads to detrimental cognitive consequences. It is a "weariness, weakness, and depleted energy" (Pigeon, Sateia, & Ferguson, 2003) that is associated with reduced duration of sleep (Bonnet & Arand, 1998) and requires rest for recovery purposes. Individuals who experience clinical and subclinical insomnia symptoms report higher levels of fatigue symptoms relative to individuals without sleep disturbances (Orff, Drummond, Nowakowski, & Perlis, 2007; Van Dongen et al., 2003). And, the experience of fatigue among such individuals is often chronic (Pigeon et al., 2003; Shen et al., 2006; Neu, Linkowski, & Le Bon, 2010), with its daytime consequences becoming increasingly pronounced in the presence of multiple nights of disturbed sleep (Bonnet & Arrand, 1998).

Dual-process theories (e.g. Epstein, 1994; Evans & Over, 1996; Metcalfe & Mischel, 1999; Schiffrin & Schneider, 1977; Sloman, 1996; Smith & DeCoster, 2000; Stanovich, 2009; Strack & Deutsch, 2004) suggest that the increased reasoning errors associated with fatigue (Rosekind et al., 1994) result from energy deficits that negatively impact cognitive functioning. Such theories suggest that an individual's ability to reason is reliant on two cognitive systems that process information (Bargh, 1994; Evans, 2008). System 1, the default system (Stanovich, 1999), automatically interprets environmental stimuli that is captured by attention (Egeth & Yantis, 1997), and initiates the individual's sequences of thoughts, feelings, or behaviors (Egeth & Yantis, 1997) to address multiple tasks simultaneously (Siemann & Delius, 1996). Conversely, System 2 processes may override System 1 processes in order to intentionally and consciously engage a problem, one task at a time (Egeth & Yantis, 1997; Evans, 2009; Siemann & Delius, 1996).

Of particular relevance to the proposed model, since System 2 requires focused attention and effort, it operates at a limited energy capacity, while System 1 is thought to operate with

little effort and thus requires little energy to function (Schiffrin & Schneider, 1977). Fatigue may impact how frequently and effectively System 2 reasoning processes may be engaged by limiting the amount of cognitive resources available (van der Linden, 2011; Lorist & Tops, 2003). These decrements may translate to a limited ability to access and retain information relevant to problem solving tasks (Helmreich, et al., 2004), decreasing the number of data points the individual is able to consider and logically connect (Oberauer, Suess, Wilhem, & Sander, 2007) during the reasoning process. In response to this depleted cognitive energy and reduced cognitive effectiveness, the fatigued individual may be motivated to conserve resources (Baumeister & Vohs, 2007) and redirect remaining energy to address only the most *essential* mental functions (Dawson & Reid, 1997; Rosekind et al., 1994). This alteration may consequently impact the *effectiveness* of the individual in managing daily tasks and stressors (Evans, 2009).

Perceived Social Problem-Solving Ability. Taken together, these consequences of sleep-related fatigue may result in perceived deficits in social problem-solving ability. Social problem-solving is the general coping strategy an individual employs to address or cope with problems encountered in the real world (e.g. regarding survival and social relationships), and is determined by an individual's appraisal of his or her own abilities (D'Zurilla & Nezu, 1999). It is defined by two components: problem orientation, and problem-solving style (D'Zurilla & Godfried, 1971; D'Zurilla & Nezu, 1982; Nezu & D'Zurilla, 1989; Maydeu-Olivares & D'Zurilla, 1996). Problem orientation refers to an individual's motivation to engage a problem, and is theorized to be determined by one's perception of both the problem solvability, and one's self-efficacy to resolve the problem (Maydeu-Olivares & D'Zurilla, 1996). It is thought to heavily contribute to the manner in which an individual conceptualizes and addresses a problem, termed problem-solving style (Maydeu-Olivares & D'Zurilla, 1996).

Of relevance to the present study, individuals who have a positive problem orientation are more motivated and confident in engaging problems, and tend to utilize a rational problem-solving style characterized by constructive problem-solving strategies (Maydeu-Olivares & D'Zurilla, 1996). In contrast, individuals who negatively orient to problems have low self-confidence, and perceive problems as threats to well-being (Maydeu-Olivares & D'Zurilla, 1996). These individuals impulsively address problems, implementing minimal solutions in an incomplete, careless, and hurried manner, or tend to avoid addressing them (Maydeu-Olivares & D'Zurilla, 1996). Fatigued individuals may be more likely to negatively orient to problems as a consequence of the increased cumbersome nature of tasks (Oberauer, Suess, Wilhem, & Sander, 2007), which may increase negative perceptions of solvability, as well as reasoning errors implicated in fatigue (Rosekind et al., 1994), which may increase perceptions of poor problem-solving self-efficacy. And, a fatigued individual may minimally address or avoid problems due to increasing motivation to conserve remaining resources (Baumeister & Vohs, 2007). Taken together, fatigued individuals may negatively orient to problems and maladaptively address problems, culminating in appraisals of poor social problem-solving ability (D'Zurilla & Nezu, 1999).

Hopelessness. Of particular relevance to the proposed model, self-appraised poor problem-solvers may be more likely to develop perceptions of hopelessness. Hopelessness is a series of negative or pessimistic expectations of the future (Beck, Steer, Kovacs, & Gamion, 1985), in which the individual expects both that negative outcomes will occur in important situations, and that he or she is helpless to change the negatively expected nature of these outcomes into positive results (Hopelessness Theory; Abramson, Metalsky, & Alloy, 1989). Expecting that they will be unable to effectively address problems, self-appraised poor problem-

solvers may put forth less effort in problem engagement (Self-efficacy Theory; Bandura, 1986), increasing the likelihood that unfavorable outcomes will occur. Experiencing negative outcomes may then reinforce beliefs of *helplessness* to resolve problematic situations in the future, and increase the likelihood of hopelessness when confronted with stressors (Beck, Steer, Kovacs, & Gamion, 1985). Indeed, self-appraised deficits in social problem-solving ability predict hopelessness (Bonner & Rich, 1988; Dixon, Heppner, & Anderson, 1991; Haaga, Fine, Terrill, Stewart, & Beck, 1995).

Hopelessness may then function as a mechanism by which appraised social problem-solving deficits contribute to suicide ideation. When an individual perceives him or herself as helpless to resolve problems, it may appear that the future will not improve, or will worsen (Abramson et al., 1989). As a result, the individual may become increasingly hopeless, and seek means of decreasing these negative thoughts. In the perceived absence of other options, suicide may be perceived as a means by which problematic feelings of hopelessness and hopeless life circumstances may be resolved (Beck, Steer, Beck, & Newman, 1993). Suggestive of this possibility, hopelessness has been associated with suicide ideation and increased suicide risk in general (American Psychiatric Association, 2003; Beck et al. 1985; Brown et al., 2000, 2005; Haney et al., 2012; Lamis & Lester, 2012). In particular, research suggests that hopelessness is a more important factor than depression in predicting and explaining suicide ideation (Beck et al., 1993), and further, that hopelessness is the construct that is most closely related to suicidality (O'Connor, Armitage, & Gray, 2006). Thus, hopelessness may function as a mediator by which more distal factors, such as fatigue and perceived poor social problem-solving ability, contribute to the occurrence of suicide ideation.

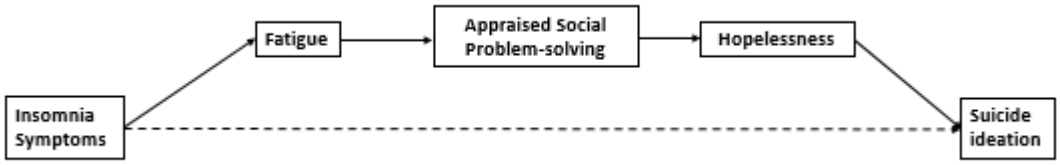

Figure 1. Conceptual model describing the relationship between insomnia symptoms, fatigue, appraised social problem-solving ability, hopelessness, and suicide ideation.

The Present Study

While prior research suggests that insomnia symptoms are predictive of suicide ideation (e.g. Pigeon, Pinquart, & Connor, 2012), research has yet to identify mechanisms other than depression that may explain this relationship. In particular, the relationships between insomnia-related fatigue and established suicide ideation risk factors, such as appraised problem-solving ability and hopelessness, have been relatively neglected in the literature. It is also unclear whether this social-cognitive pathway from sleep loss to ideation is distinct from one that highlights the role of depression in explaining the link between sleep loss and ideation. Thus, there were two main goals of the proposed study. First, we examined whether social-cognitive correlates of sleep loss (fatigue, perceived social problem solving, and hopelessness) will relate to levels of suicide ideation. Second, we assessed whether this "pathway" from sleep loss to ideation persists even when recent depressive symptoms were included as distinct mediator. As such, two models were tested. The first model tested the hypothesis that the relationship between insomnia symptoms and suicide ideation would be at least partially explained by social-cognitive correlates that may stem from insomnia. Specifically, we expected that more severe insomnia symptoms would be associated with worse fatigue, which would be associated with more negatively-appraised social problem-solving ability, which would be associated with increased levels of hopelessness, and, in turn, more severe suicide ideation (see Figure 1). The second model tested whether recent depressive symptoms at least partially mediated the relationship

between sleep loss and ideation, and whether the inclusion of this pathway explained overlapping or different variance from the social-cognitive pathway. In this model, we hypothesized that the relationship between insomnia symptoms and suicide ideation would be at least partially explained by *both* recent depressive symptoms and cognitive disruptions (see Figure 2).

Expanding knowledge in this area may increase understanding of the nature of suicide risk among college students who experience sleep deficits. It may also provide insight into different risk factors associated with sleep loss that are potentially modifiable, and thus, potentially informative, for tailoring suicide intervention and prevention strategies.

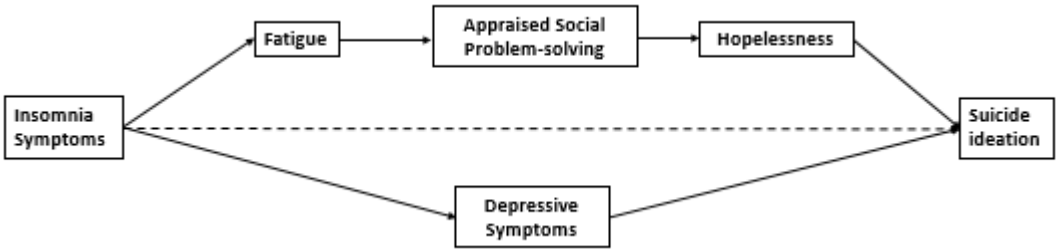

Figure 2. Conceptual model incorporating depressive symptoms as a separate mediation path explaining the relationship between insomnia symptoms and suicide ideation.

Method

Participants

Study participants were 438 female students recruited from the University of South Florida's undergraduate psychology participant pool using the SONA participant management system. This sample size was chosen based on guidelines suggesting a ratio of 20 participants per parameter for path analytic models (Kline, 2011). In order to account for potential participant response error and over-sampling of non-distressed individuals, we recruited 120 participants in addition to the 360 specified by our power analysis, bringing the total participants recruited to 480. In addition, only female participants were included in the study in order to over represent individuals with suicide ideation, since females are more likely to consider suicide than males (Centers for Disease Control and Prevention, 2010). Data were screened for validity using three criteria. A total of 42 participants who a) endorsed male gender, b) completed surveys in less than 10 minutes, and/or c) answered all 'True' or all 'False' to the Beck Hopelessness Scale were removed from inclusion in analyses.

The remaining participants ranged in age from 18 to 50 (M=20.4, SD=3.87), with the majority of participants being between 18 and 24 years old. Approximately 67.4% were Caucasian (most non-Hispanic), 15.9% were African American, 5% were Asian, 0.2% were American Indian or Alaskan Native, and 11.2% self-identified as other/multiple races. Participants were required to be above the age of 18 and fluent in English. No other specific exclusion criteria were employed above and beyond meeting inclusion criteria.

Procedure

All participants provided informed consent prior to completing the study survey online. Participants were compensated with extra credit points in their psychology class for their involvement in the study. At the end of the survey, participants were directed to a debriefing form, which provided information about the study purpose, as well as information about community-based and campus mental health resources. This study was approved by the institutional review board at the University of South Florida (See Appendix B).

Measures

Demographics. A brief demographics form (See Appendix A) was administered to participants in order to collect information on age, gender, race/ethnicity, and English fluency.

Insomnia Symptoms. The Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001) was used to assess severity of insomnia symptoms in the past month. The ISI is a self-report questionnaire comprised of 7 items that assess the nature, severity, and impact of specific symptoms of insomnia (e.g. "Difficulty falling asleep"). Responses are rated on a 5-point Likert Scale ranging from 0 to 4, with higher responses indicating greater distress. The ISI yields a total score ranging from 0 to 28, and has clinical cutoffs indicating the absence of insomnia, and sub-clinical, moderately severe, and severe levels of insomnia symptoms. The ISI demonstrates adequate concurrent validity with polysomnography and sleep diaries (Bastien et al., 2001; Savard, Savard, Simard, & Ivers, 2005), methods commonly utilized in establishing sleep diagnoses, and has been used to determine the presence or absence of clinically significant insomnia symptoms (Bernert, Turvey, Conwell, & Joiner, 2007; Tang, Wright, & Salkovskis, 2007). The ISI has demonstrated good internal consistency in both college student samples (Cronbach's alpha=0.84 -0.87; Nadorff, Nazem, & Fiske, 2011; Wilkerson, Boals, & Taylor,

2012) and in clinical samples (α =0.74; Bastien et al., 2001). In the present study, the ISI demonstrated acceptable internal consistency (α = .88).

Fatigue. The Multidimensional Fatigue Inventory (MFI; Smets, Garssen, Bonke, & De Haes, 1995) was used to assess the presence of fatigue symptoms. In the present study, the timeframe of the MFI was extended from assessing fatigue symptoms in the past two weeks to the past month, consistent with the time frame of the other measures (e.g., the Beck Hopelessness Scale). The MFI is a 20-item self-report measure, with 7 reverse-keyed items. It is comprised of scales spanning four dimensions of fatigue: general fatigue (e.g. "I feel tired"), reduced activity (e.g, "I think I do a lot in a day"), reduced motivation (e.g. "I dread having to do things"), and mental fatigue (e.g. "When I am doing something, I can keep my thoughts on it"). Participants are asked to indicate how true each statement has been of them in the past month, and rate responses on a 5-point Likert Scale ranging from 0 (yes that is true) to 4 (no that is not true). Ratings are summed to create a total score ranging from 20 to 100, with higher scores indicating greater levels of subjective fatigue. The MFI demonstrates good internal consistency among college students, and has been found to correlate with other measures of fatigue (α for scales = 0.76-0.93; Smets et al., 1995). In the present study, the MFI demonstrated acceptable internal consistency (α = .90).

Appraised Social Problem-Solving Ability. The Social Problem Solving Inventory – Revised: Short Form (SPSI-R-SF; D'Zurilla, Nezu, & Maydeu-Olivares, 2002) was used to assess appraised social problem-solving ability. The SPSI-R: SF is a 25-item self-report measure based on the social problem-solving model (D'Zurilla & Godfried, 1971). It is comprised of five scales. Two of the scales assess positive (PPO; e.g. "Whenever I have a problem, I believe it can be solved") and negative (NPO; e.g. When I am faced with a difficult problem, I doubt that I will

be able to solve it on my own no matter how hard I try") problem orientation. The remaining three scales assess different problem-solving styles, including the rational problem-solving style (RPS; e.g. "When I have a problem to solve, one of the first things I do is get as many facts about the problem as possible"), the impulsivity/carelessness style (ICS; e.g. "When making decisions, I go with my 'gut feeling' without thinking too much about the consequences of each option"), and the avoidance style (AS; e.g. "I spend more time avoiding my problems than solving them"). Each scale is comprised of five items. Items are rated on a 5-point Likert Scale ranging from 0 (not at all true of me) to 4 (extremely true of me). Scores are summed to create a global score of perceived problem-solving ability. Good appraised problem-solving ability is indicated by higher scores on the PPO and RPO scales, and lower scores on the NPO, ICS, and AS scales. In the present study, scores were reverse coded so that higher global scores reflected worse perceived problem-solving ability. The SPSI-R-SF has been found to have excellent internal consistency and good concurrent validity with other measures of problem-solving (e.g. the Problem Solving Inventory; Heppner & Petersen, 1982) in college students and in clinical samples (D'Zurilla et al., 2002).The SPSI-R-SF demonstrated acceptable internal consistency in the study sample ($\alpha = .87$).

Hopelessness. The Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974; Beck & Steer, 1988) was used to assess negative expectations for the future, based on the person's thoughts in the past month. The BHS is a self-report measure intended to be used with individuals over the age of 17. It is comprised of 20 true/false items, 9 of which are reverse keyed. An example item is, "Even if I try, I will not be able to accomplish the things that are really important to me." Scores are summed to create a total hopelessness score ranging from 0 to 20. Higher scores indicate higher levels of hopelessness, with cutoff scores ranging from 0-3

indicating minimal hopelessness, 4-8 indicating mild hopelessness, 9-14 indicating moderate hopelessness, and scores greater than 14 indicating severe levels of hopelessness (Beck & Steer, 1988). The BHS has demonstrated concurrent validity with clinical ratings of hopelessness (Beck & Steer, 1988; Bisconer & Gross, 2007; Brown, Henriques, Sosdjan, & Beck, 2004), and divergent validity with measures of hope (Herth, 1991; Steed, 2001). Studies have reported good internal consistency for the total BHS score (α =0.82-0.93) in college student samples and clinical samples (Beck & Steer, 1988; Bisconer & Gross, 2007; Brown, Henriques, Sosdjan, & Beck, 2004; Steed, 2001). The BHS demonstrated acceptable internal consistency in the present study (α = .89).

Depressive Symptoms. The Depression, Anxiety, Stress, Scales -21 (DASS-21; Lovibond & Lovibond, 1995) were used to assess the dimensional presence of depressive symptoms in the past week. Although differing from the timeframe assessed in the other measures in the present study, this one-week timeframe was maintained for the depression measures given that norms for measuring depression are often assessed based on report of symptoms from the past week (e.g. the Center for Epidemiologic Studies Depression Scale, Radloff, 1977; Beck Depression Inventory – Second Edition, Beck, Steer, & Brown, 1996). The DASS-21 is a self-report measure comprised of 21 items that assess depression, anxiety, and stress symptoms. In the present study, only the 7 items pertaining to recent depression were included in analyses. Respondents indicate how frequently they experienced the symptoms in the past week using a 4-point scale ranging from 0 (“Did not apply to me at all”) to 3 (“Applied to me very much, or most of the time”). An example item is, “I felt down-hearted and blue.” Higher scores indicate higher severity of depressive symptoms, with scores of 7-10 indicating moderate levels, and scores greater than 11 indicating severe to extremely severe levels of symptoms

(Lovibond & Lovibond, 1995). The DASS-21 demonstrates convergent validity with other measures of depression and divergent validity from measures of anxiety and stress (Antony et al., 1998; Henry & Crawford, 2005). It also has shown good internal consistency in clinical and nonclinical samples (α = .94; Antony et al., 1998). The DASS-21 demonstrated good internal consistency in the present study (α = .92).

Suicide Ideation. The Adult Suicide Ideation Questionnaire (ASIQ; Reynolds, 1991) was used to assess presence of suicidal thoughts in the past month. It is comprised of 25 items that assess a range of mild-to-severe suicidal thoughts. Respondents rate how frequently they experienced each thought on a 7-point scale ranging from 0 ("I never had this thought") to 6 ("Almost every day"). As one of the options on the scale references a timeframe outside of one month (1; "I had this thought before but not in the past month"), this option was recoded to indicate an absence of the thought in the past month. An example item is, "I thought about how easy it would be to end it all." Scores on the ASIQ have been found to correlate with measures of depression, hopelessness, and history of prior suicide attempts (Osman et al., 1999), and to discriminate between suicide attempters and psychiatric controls (Osman et al., 1999). The ASIQ has demonstrated good internal consistency among college students (α = .97; Reynolds, 1991) and clinical samples (α = .98; Osman et al., 1999). The ASIQ demonstrated excellent internal consistency in the present study (α = .99).

Analytic Strategy

Preliminary Analyses. Prior to conducting hypothesis testing, all variables were screened for violations of assumptions of normality. The variable with a particularly non-normal distribution, as a result of many participants not endorsing any suicide ideation in the last month, was the suicide ideation variable (ASIQ; Skew = 4.09, SE = .12, Kurtosis = 18.1, Kolmogorov-

Smirnov D = .41, $p<.001$). Given that regression-based methods in particular assume that the dependent variable is normally distributed (Cohen, Cohen, West, & Aiken, 2002), we employed a log transformation, which is typically recommended to correct such a distribution[1], in order to better meet the underlying assumptions of these tests (Keene, 1995). However, since the transformation did not affect the distribution (Skew = 2.17, SE = .12, Kurtosis = 3.48, Kolmogorov-Smirnov D = .45, $p<.001$) or interpretation of the study models, we report analyses with the non-transformed values in order to better aid in interpretation of results (Howell, 2007; B. Small, personal communication, March 30, 2015).

Hypothesis Testing. In order to test the study hypotheses, we conducted path analytic models using Mplus 7.2 (Muthén & Muthén, 1998-2012). Due to concerns with normality regarding the dependent variable in the model, all analyses were initially conducted using a dichotomized dependent variable. Then, analyses were conducted using the robust estimator (MLM) in Mplus, which is robust to violations of normality in study variables[2] (Muthén & Muthén, 1998-2012). Results were then compared with models conducted using maximum likelihood parameter estimates (ML; Muthén & Muthén, 1998-2012). As results (e.g. fit indices, regression weights, model interpretation) did not change significantly across the dichotomized and continuous outcomes and estimators, and as the ML estimator allows for the inclusion of more participants (via full information maximum likelihood) as well as the usage of bootstrapping, results using the ML estimator are reported.

[1] These transformative calculations may be considered "reexpressions" of the data in other terms that enable the analyst to better meet the statistical assumptions of a test, and hence have more confidence in the results found. However, given that such a procedure expresses the distribution of a variable in different mathematical terms or units, such a procedure theoretically should not alter the results that would be found with the untransformed variable, and indeed such differing results are the exception and not the rule in clinical data.

[2] In particular, this estimator generates standard errors and a corrected Satorra-Bentler chi-square fit statistic that are robust to violations of normality.

With regards to bootstrapping, the collected data were resampled 10,000 times in order to provide a percentile-based and bias-corrected confidence interval for the indirect effects, which is the recommended approach to testing mediational pathways (Preacher & Hayes, 2004; Shrout & Bolger, 2002; Zhao, Lynch, & Chen, 2010). In addition to evaluating the significance and effect sizes of the hypothesized paths, we also evaluated overall model fit using the model-based chi-square value (Joreskog, 1969), the Comparative Fit Index (CFI; Bentler, 1990), the Tucker Lewis Index (TLI; Tucker & Lewis, 1973) the Standardized Root Mean Residual (SRMR; Hu & Bentler, 1999), and the Root Mean Square Error of Approximation (RMSEA; Steiger, 1990). Given that the chi-square test is more likely to erroneously reject well-fitting models in analyses involving large sample sizes (Reise, Widaman, & Pugh, 1993), we evaluated good model fit emphasizing a CFI $\geq$ 0.95, TLI $\geq$ 0.95, SRMR $\leq$ 0.08 (Hu & Bentler, 1999), and an RMSEA $\leq$ 0.08 (Browne & Cudeck, 1993).[3]

In order to test the first hypothesis, that the relationship between insomnia symptoms and suicide ideation would be explained by social-cognitive factors, we employed a path analytic model. In doing so, we described a direct path from insomnia symptoms (measured by the ISI) to suicide ideation (measured by the ASIQ), and an indirect path from insomnia symptoms to suicide ideation through fatigue (measured by the MFI), appraised social problem-solving ability (measured by the SPSI-R), and hopelessness (measured by the BHS). Given that our measures for appraised social problem-solving ability and hopelessness both assessed future orientation to some degree (e.g. SPSI: "When I am faced with a difficult problem, I doubt that I will be able to solve it on my own no matter how hard I try;" BHS: "In the future I expect to fail in what

[3] Of note, the robust MLM estimator adjusts fit indices that rely on the chi-square statistic (RMSEA, CFI, and TLI) based on an estimated scaling factor (Muthén & Muthén, 1998-2012). These fit indices did not differ across models using either the ML or the MLM estimators; this suggests that the model estimates are fairly robust across estimators employed, despite normality concerns.

concerns me most"), we also correlated the residuals of these variables to account for irrelevant shared variance. Then, we computed an alternative path model in which the order of model variables were reversed, in order to assess whether the hypothesized model better explained the relationship between insomnia symptoms and suicide ideation.

In order to assess whether the indirect effect was sufficient in explaining the relationship between insomnia symptoms and suicide ideation, we compared the original unconstrained model to a constrained model where the direct path was fixed to zero. In such a comparison, the lack of significant differences in model fit would indicate that estimating the direct path in the unconstrained model does not increase model fit above the more parsimonious model that constrained the direct path to zero. Then, the intervening mediation paths (e.g., from insomnia to problem solving via fatigue) were examined in order to assess whether these relationships were present as specified. In this case, mediation analyses in which zero was not contained in a confidence interval for the effect were considered significant (MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008).

In order to test the second hypothesis, that the relationship between insomnia symptoms and suicide ideation would be at least partially explained by *both* depressive symptoms and social-cognitive factors, several analyses were employed. First, in order to examine whether there was preliminary evidence to suggest that the socio-cognitive model may contribute variance to suicide ideation above and beyond depressive symptoms, we conducted a multiple hierarchical regression. In doing so, we entered depressive symptoms on the first step, and then entered the mediators of the socio-cognitive model (fatigue, appraised social problem-solving ability, and hopelessness) on the second step, with suicide ideation as the outcome variable. Then, we conducted a second hierarchical multiple regression to examine the variance depressive

symptoms may contribute above and beyond the hypothesized mediators in the model. As an item from the depression measure (Item 10: “I felt that I had nothing left to look forward to”) overlapped with an item from the hopelessness measure (e.g. Item 3: “All I can see ahead of me is unpleasantness rather than pleasantness), we removed this item in order to better distinguish the variance explained by these constructs. Then, we proceeded to test the hypothesized expanded path analytic model. In doing so, we added a second indirect path with depressive symptoms (measured by the DASS) as a mediator of the relationship between insomnia symptoms and suicide ideation (see Figure 2).

Results

Descriptive Statistics

Table 1 presents the means, standard deviations, and intercorrelations of the study variables. Among study participants, approximately 34.1% (*n*=145) met cutoffs for subclinical levels of insomnia symptoms (scores ranging from 8-14), and 14.8% (*n*=63) met cutoffs for clinical levels of insomnia symptoms (scores of 15 or greater). With regards to depressive symptoms, 10.5% (*n*=46) of participants endorsed moderate levels, and 9% (*n*=39) endorsed severe to extremely severe levels of symptoms. Approximately 22% (*n*=98) of study participants endorsed some degree of suicide ideation in the past month, and 11% of study participants (*n*=48) endorsed more active ideation (e.g. preparatory behaviors such as suicidal planning).

Table 1. *Means, Standard Deviations, and Intercorrelations between Study Variables*

	1	2	3	4	5	6
1. Insomnia symptoms (ISI)	--					
2. Fatigue symptoms (MFI)	.47*	--				
3. Appraised social problem-solving ability (SPSI-R)	.20*	.40*	--			
4. Hopelessness (BHS)	.36*	.48*	.49*	--		
5. Suicide ideation (ASIQ)	.30*	.22*	.27*	.46*	--	
6. Depression (DASS)	.45*	.51*	.43*	.66*	.57*	--
Mean	8.10	52.28	35.22	4.25	5.45	9.16
Standard Deviation	5.82	13.19	13.28	4.17	17.64	3.82

Note. ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, SPSI-R = Social Problem Solving Inventory – Revised, BHS = Beck Hopelessness Scale, ASIQ = Adult Suicide Ideation Questionnaire, DASS = Depression, Anxiety, Stress Scales. *p<.001

Table 2 presents the intercorrelations, and descriptive statistics of the study variables with regard to suicide ideation endorsement. Individuals who endorsed suicide ideation endorsed significantly higher levels of insomnia symptoms ($t(403)=7.08$, $p<.001$, $d=.70$), fatigue ($t(414)=6.76$, $p<.001$, $d=.79$), negatively-appraised social problem-solving ability ($t(413)=6.26$, $p<.001$, $d=.73$), hopelessness ($t(112.76)=8.29$, $p<.001$, $d=1.10$), and depressive symptoms ($t(107.64)=9.95$, $p<.001$, $d=1.34$) compared to individuals who did not endorse ideation.

Table 2. *Descriptive Statistics and Intercorrelations by Suicide Ideation Endorsement*

	1	2	3	4	5	*M*	*SD*
1. Insomnia symptoms (ISI)	--	.42**	.14	.44**	.28**	11.50	6.14
2. Fatigue symptoms (MFI)	.41**	--	.36**	.57**	.51**	59.71	12.64
3. Appraised social problem-solving ability (SPSI-R)	.15**	.34**	--	.50**	.45**	42.17	12.49
4. Hopelessness (BHS)	.16*	.32**	.40**	--	.63**	7.76	5.38
5. Depression (DASS)	.36**	.43**	.30**	.33**	--	13.13	5.03
M	7.51	49.78	32.86	3.04	7.90		
SD	5.19	12.61	12.88	2.84	2.22		

Note. Correlations above the diagonal and descriptives in the columns to the right of the table are for individuals who reported suicide ideation (n=97); correlations below the diagonal and descriptives in rows at the bottom of the table are for individuals who did not report suicide ideation (n = 320). ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, SPSI-R = Social Problem Solving Inventory – Revised, BHS = Beck Hopelessness Scale, ASIQ = Adult Suicide Ideation Questionnaire, DASS = Depression, Anxiety, Stress Scales. *$p<.01$, **$p<.001$

Hypothesis 1

The first hypothesis was tested using a path analytic model describing fatigue, appraised social problem-solving ability, and hopelessness as an indirect effect to explain the relationship between insomnia symptoms and suicide ideation. First, we examined this model using a dichotomized outcome, in order to better manage violations of assumptions of normality. The model demonstrated acceptable fit to the data, $x^2_{[df=4]} = 17.19$, $p < .01$, CFI = 0.97, TLI = 0.93,

RMSEA = 0.09, SRMR = 0.04. Results indicated that the indirect effect was significant (*B* = .01, SE = .00, β = .10, 95% CI [0.06, 0.13], *p*<.001), and constituted a medium effect (Preacher & Kelley, 2011). Since this dichotomized outcome supported our hypothesized model, we proceeded to examine the model using a continuous dependent variable, which allowed for increased power in our analyses. The model demonstrated acceptable fit to the data, x^2 [df=4] = 17.93, $p < .01$, CFI = 0.97, TLI = 0.93, RMSEA = 0.09, SRMR = 0.04. Model results are described in Figure 3. Results indicated that the indirect effect was significant (*B* = .27, SE = .06, β = .09, 95% CI [0.05, 0.13], *p*<.001), and constituted a medium effect (Preacher & Kelley, 2011), supporting our hypothesized model. The relationship of insomnia and ideation prior to including the socio-cognitive pathway in the model (the C path) was a large effect. After the hypothesized pathway was included in the model, the remaining effect of insomnia symptoms on suicide ideation (the C' path) was significant (*B* = .47, SE = .12, β = .16, *p*<.01), and reduced to a medium effect (Preacher & Kelley, 2011), suggesting a partially mediated model. The indirect effect explained 20.5%, and the direct effect just 0.5% of the variance in suicide ideation. The overall model explained 21% of the variance in suicide ideation, and is described more fully in Figure 3.[4]

A series of analyses were then computed in order to assess the significance of the intermediate mediated paths. Results indicated that fatigue significantly explained the association between insomnia symptoms and appraised social problem-solving ability (*B* = .44, SE = .07, β = .19, 95% CI [0.13, 0.24], *p*<.001), and constituted a medium effect (Preacher & Kelley, 2011).

[4] Notably, a slightly improper solution is apparent for the standardized beta weight predicting hopelessness from appraised social problem-solving ability (i.e., standardized coefficient greater than 1). Following guidelines from Joreskog (1999), we inspected the variance/covariance matrices for additional improper solutions (e.g. variance). In the absence of such additional improper solutions, standardized coefficients above 1 may indicate a high degree of multicollinearity in the data. Given that hopelessness and appraised social problem-solving ability to some extent measure negative future expectations associated with ideation, such a result is unsurprising.

Additionally, appraised social problem-solving ability significantly explained the relationship between fatigue and hopelessness (B = .15, SE = .02, β = .48, 95% CI [0.41, 0.55], p<.001), and constituted a large effect (Preacher & Kelley, 2011). Finally, hopelessness significantly explained the association between appraised social problem-solving ability and suicide ideation (B = .50, SE = .12, β = .48, 95% CI [0.31, 0.66], p<.001), and constituted a large effect (Preacher & Kelley, 2011). Taken together, these findings support the hypothesis that these factors may be part of a larger chain explaining links between sleep loss and suicide ideation.

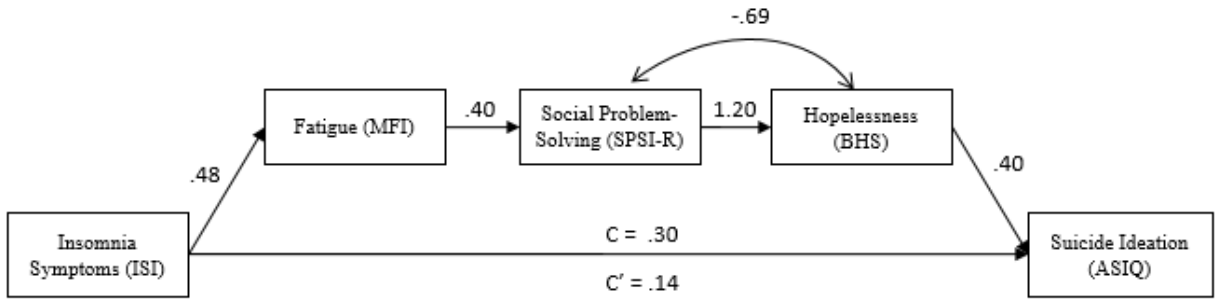

Figure 3. Standardized estimates of the first path model testing whether social-cognitive factors explain the relationship between insomnia symptoms and suicide ideation.

Note. ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, SPSI-R = Social Problem Solving Inventory – Revised, BHS = Beck Hopelessness Scale, ASIQ = Adult Suicide Ideation Questionnaire. All solid paths significant at p<.01.

Next, in order to address the limitation of our cross-sectional data and examine other possible ordering of effects, we then tested an alternative model in which the order of all of the variables were reversed, such that suicide ideation predicted hopelessness, which predicted appraised social problem-solving ability, which predicted fatigue, which in turn predicted insomnia symptoms.[5] The overall model fit was poor, x^2 [df=4] = 60.73, p < .001, CFI = 0.88, TLI

[5] We computed several additional path models in which the order of the mediators of the insomnia-suicide ideation relationship was reversed. Each model displayed poor fit to the data; only one of the indirect effects was significant, constituting a small effect in comparison to the medium effect obtained by the hypothesized model. Model 1 reversed the hopelessness and appraised social problem-solving ability variables. The model demonstrated poor fit to the data, x^2 [df=4] = 60.50, p < .0001, CFI = 0.87, TLI = 0.69, RMSEA = 0.18, SRMR = 0.07. The indirect effect

= 0.70, RMSEA = 0.18, SRMR = 0.08. The indirect effect was significant (*B* = .02, SE = .01, β = .05, 95% CI [0.01, 0.03], *p*<.001), constituted a small effect (Preacher & Kelley, 2011), and explained 25% of the variance in insomnia symptoms. As this model was not comparable to the hypothesized model with regards to model fit or strength of the indirect effect, we retained the hypothesized model in our analyses.

Finally, in order to assess whether the hypothesized indirect path was sufficient to explain links between insomnia and suicide ideation, the direct path from insomnia to suicide ideation was fixed to zero. The model demonstrated acceptable fit to the data, $x^2_{[df=5]} = 28.53$, $p < .0001$, CFI = 0.95, TLI = 0.90, RMSEA = 0.11, SRMR = 0.06. Then, a chi-square difference test was used to test whether there was a significant difference in fit across the nested models (unconstrained and direct-path-constrained models). As expected, the direct path constrained model demonstrated significantly worse fit to the data $x^2_{[df=1]} = 10.61$, $p < .005$. However, the chi-square statistic is sensitive to large sample sizes (Reise, Widaman, & Pugh, 1993), which may increase the likelihood of identifying even minimal differences as significant. Inspection of the remaining fit indices, including the RMSEA statistic in particular, which takes into account model parsimony (Browne & Cudeck, 1993), revealed that there was minimal change in fit between the two models. Additionally, our results suggest that the direct path contributes minimal variance above the indirect path. Taken together, these results suggest that the model without the direct path may demonstrate a more parsimonious means of describing the data.

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was significant (*B* = .12, SE = .03, β = .04, 95% CI [0.02, 0.06], *p*<.001), constituted a small effect (Preacher & Kelley, 2011), and explained 13% of the variance in suicide ideation. Model 2 reversed fatigue and appraised social problem-solving ability. The model also demonstrated poor fit to the data, x^2 [df=4] = 117.01, $p < .0001$, CFI = 0.75, TLI = 0.38, RMSEA = 0.26, SRMR = 0.13. The indirect effect was not significant (*B* = .02, SE = .01, β = .01, 95% CI [0.00, 0.02], *p*>.05). Model 3 switched fatigue and hopelessness, such that insomnia predicted social problem-solving ability, which predicted hopelessness, which predicted fatigue, which predicted ideation. The model demonstrated poor fit to the data, x^2 [df=4] = 144.59, $p < .0001$, CFI = 0.69, TLI = 0.23, RMSEA = 0.29, SRMR = 0.11. The indirect effect was not significant (*B* = .06, SE = .04, β = .02, 95% CI [-0.01, 0.14], *p*>.05).

Table 3. *Summary of the Hierarchical Multiple Regression Analysis Predicting Suicide Ideation from Mediation Variables Controlling for Depressive Symptoms*

Predictor	Beta	*SE B*	*β*	*t*	R^2	ΔR^2
Step 1					.33	
Depression	2.61	1.81	.57	14.24**		
Step 2					.36	.03**
Depression	2.403	.247	.53	9.73**		
Fatigue	-.19	.06	-.15	-3.10*		
Appraised Social Problem-Solving Ability	.01	.06	.01	.22		
Hopelessness	.78	.23	.19	3.39*		

Note. *p<.01, **p<.001.

Hypothesis 2

We then proceeded to test Hypothesis 2, that the relationship between insomnia symptoms and suicide ideation would be at least partially explained by *both* recent depressive symptoms and socio-cognitive risk factors. In order to preliminarily support that the socio-cognitive risk factors may contribute variance in explaining suicide ideation above and beyond depressive symptoms alone, we conducted a multiple hierarchical regression model. Results of the analysis are displayed in Table 3. Entry of the model mediators in the second step accounted for an additional 3% of the variance in suicide ideation, F change (3, 409) = 6.08, p <.001, or a small effect size (Cohen, 1988). In order to examine whether depressive symptoms explained variance in suicide ideation above and beyond our study mediators, we computed a second multiple hierarchical regression model. Results of the analysis are displayed in Table 4. Entry of the depressive symptoms in the second step accounted for an additional 15% of the variance in suicide ideation, F change (3, 409) = 98.56, p <.001, or a medium effect size (Cohen, 1988). Since the results of these analyses suggested that the hypothesized pathway contributed to suicide ideation to some degree above and beyond depression, we proceeded to test the path analytic model.

Table 4. *Summary of the Hierarchical Multiple Regression Analysis Predicting Suicide Ideation from Depression while Controlling for Mediation Variables*[6]

Predictor	Beta	*SE B*	*β*	*t*	R^2	ΔR^2
Step 1					.21	
Fatigue	-0.2	.07	-.01	-.24		
Appraised Social Problem-Solving Ability	.08	.07	.06	1.21		
Hopelessness	1.82	.23	.43	.43**		
Step 2					.36	.15**
Depression	2.15	.22	.55	9.93**		
Fatigue	-0.2	.06	-.15	-3.15*		
Appraised Social Problem-Solving Ability	.01	.06	.01	.22		
Hopelessness	.70	.23	.17	3.00*		

Note. *p<.01, **p<.001.

In testing the integrative model for Hypothesis 2, we expanded the path analytic model tested in Hypothesis 1 by adding a separate indirect path in which recent depression solely mediated the relationship between sleep loss and suicide ideation (see Figure 2). The overall model fit was poor, x^2 [df=7] = 213.40, p < .001, CFI = 0.74, TLI = 0.44, RMSEA = 0.27, SRMR = 0.15, partly because relationships between depression and the variables in the first path were not estimated. Since depression is naturally correlated with the other variables, but we did not estimate those in our model to stick to our apriori hypotheses about two distinct paths, this model would not adequately replicate the covariance matrix. Examining only the pattern of correlations, results indicated that both the indirect path through social-cognitive factors (B = .09, SE = 04, β = .04, p<.05) and the indirect path through depression (B = .53, SE = .13, β = .22, 95% CI [0.14, 0.28], p<.001) were significant, supporting that these two paths can operate separately.[7] In this

[6] Notably, fatigue and appraised social problem-solving abilities both relate to suicide ideation prior to the addition of hopelessness in the model.

[7] In order to provide a more stringent test of this analyses, we also computed the path analytic model using mediators in the socio-cognitive pathway that had been residualized upon depressive symptoms. Such a technique enabled any variance explained in suicide ideation by this pathway to be independent of depressive symptoms. The results supported that the hypothesized socio-cognitive pathway contributed unique variance to suicide ideation that

case, the indirect path through socio-cognitive factors constituted a small effect, and the indirect path through depression constituted a medium effect (Preacher & Kelley, 2011). Additionally, the direct path from insomnia symptoms to suicide ideation was no longer significant in this model (B = .04, SE = .12, β = .02, p=.75), suggesting that depression fully mediated the relationship between insomnia and suicide ideation. The overall model explained 29% of the variance in suicide ideation, with the addition of the recent depressive symptoms path contributing an additional 8% of the variance above and beyond the first hypothesized model. The results of this model are described more fully in Figure 4.

was independent of depressive symptoms, (B = .02, SE = .01, β = .01, 95% CI [0.004, 0.048], p<.001). However, this pathway constituted a very small effect. Yet, this approach is highly conservative and does not reflect the real-life influence of these factors since aspects of depressive symptoms (e.g. depressed mood) are important in the experience of suicide ideation. Additionally, these are cross-sectional data and thus these results may not represent the importance of these variables over time.

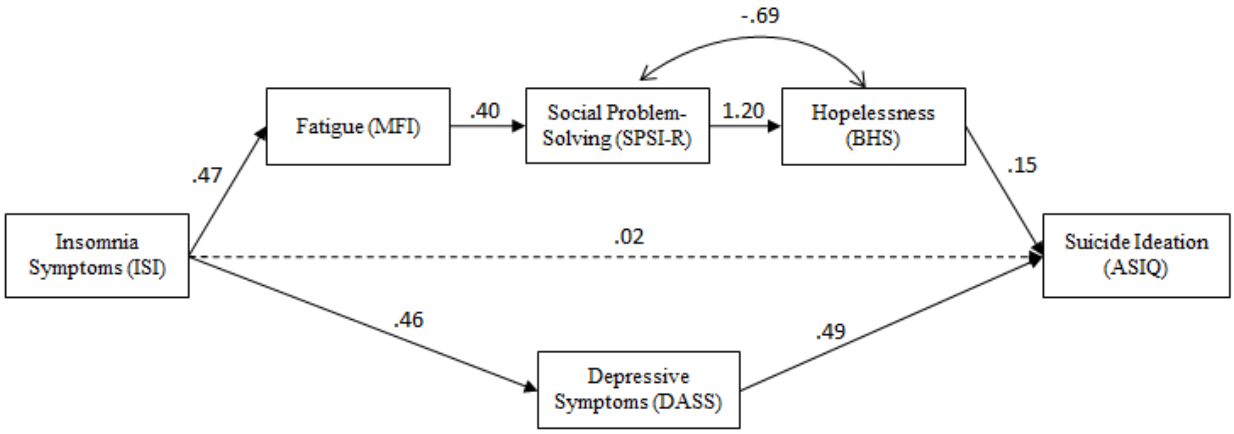

Figure 4. Standardized estimates of second path model incorporating depressive symptoms as a separate mediational path explaining the relationship between insomnia symptoms and suicide ideation.

Note. Standardized estimates displayed. ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, SPSI-R = Social Problem Solving Inventory – Revised, BHS = Beck Hopelessness Scale, ASIQ = Adult Suicide Ideation Questionnaire, DASS = Depression, Anxiety, Stress Scales. All solid paths significant at $p<.05$.

Discussion

To our knowledge, this study was the first to jointly investigate potential socio-cognitive factors and depression as distinct mediators of the established relationship between insomnia symptoms and suicide ideation. There are at least two major findings advanced by the present study. First, our results build on the extant literature by expanding our knowledge of putative mechanisms that may explain the relationship between insomnia symptoms and suicide ideation. Specifically, we found that socio-cognitive factors related to sleep deficits partially mediate this relationship. Second, it addressed the contention in the literature regarding whether this relationship could be primarily explained by recent depressive symptoms, or whether there may be other explanatory factors related to sleep deficits. Our findings suggest that the socio-cognitive pathway from sleep loss to suicide ideation persists even when accounting for recent depressive symptoms, albeit contributing a small unique effect, such that both pathways separately explain some degree of this relationship.

Hypothesis 1

A major contribution of the present study is the finding that fatigue, appraised social problem-solving ability, and hopelessness partially explain the relationship between insomnia symptoms and suicide ideation, supporting Hypothesis 1. This model expands on the existing literature by combining dual process theories (e.g. Schiffrin & Schneider, 1977) and the social problem-solving model (D'Zurilla & Godfried, 1971) with existing research suggesting hopelessness as the construct most closely related to suicidality (Beck et al., 1993; O'Connor et

al., 2006). In particular, we established that the insomnia-ideation relationship is most parsimoniously explained through a model retaining the hypothesized indirect effect of insomnia through the socio-cognitive pathway, which comprised a medium effect, with the remaining relationship between insomnia symptoms (residualized) and suicide ideation explaining minimal variance in the overall model.

Two possible explanations the mechanisms that drive our model include cognitive dysfunction and appraisal explanations. For the first, our findings that individuals with insomnia symptoms who experience fatigue perceive more deficits in addressing and resolving problems are in line with dual process theories (e.g. Stanovich, 2009), which suggest that reduced energy resources may contribute to cognitive functioning deficits. Under conditions of non-restorative sleep, fatigue may increase vulnerability to negative perceptions of one's ability to manage life stressors by increasing the difficulty of managing daily tasks. These results parallel findings of ego depletion studies that demonstrate increased cognitive deficits in the presence of fatigue (see Hagger, Wood, Stiff, & Chatzisarantis, 2010 for a review).

An appraisal explanation is also supported by evidence that fatigue compels individuals to increasingly prioritize resources for problems most central to well-being or to survival, such as threatening situations (Barclay & Ellis, 2013). As such, fatigue may increase tendencies to perceive circumstances more negatively. Our results suggesting that fatigue is associated with more negatively appraised problem-solving ability and future (assessed as hopelessness) may thus naturally follow from such negative appraisals. As suggested by the social problem-solving model (D'Zurilla & Godfried, 1971), actual or perceived reductions in problem-solving ability resulting from fatigue may contribute to negative self-efficacy more generally. Such perceptions may in turn contribute to reduced motivation to engage problems, due to preliminary

expectations of unsatisfactory outcomes (Bandura, 1986). And, in the presence of prolonged fatigue, such a thought process may become automatic (e.g. Beck et al., 1993), contributing to perceptions of hopelessness (Abramson et al., 1989), as suggested in our findings. And, our finding that suicide ideation results from this chain of variables may reflect the tendency of individuals in such circumstances to perceive suicide as a means of resolving otherwise hopeless life circumstances and emotions (Beck et al., 1975).

Taken together, our results provide quantitative support for the notion that the desire to die (e.g. suicide ideation) develops through the confluence of multiple factors over time (Maris, 2002). This work is consistent with other theories of suicide, including the stress-process model of suicide in particular (Sandin, Chorot, Santed, Valiente, & Joiner, 1998). This model posits that negative appraisals (e.g. appraised social problem-solving ability) mediate the relationship between negative life events and chronic stressors (e.g. insomnia symptoms) and suicide ideation. The fact that we cannot draw causal conclusions from these findings, due to the cross-sectional nature of the data, indicates that this study represents preliminary evidence for developing and testing longitudinal process models implicating the cognitive disruption resulting from sleep loss in risk for suicide ideation.

Hypothesis 2

A secondary aim of the study was to examine whether the socio-cognitive pathway from insomnia symptoms to ideation examined in Hypothesis 1 significantly contributed to the model after incorporating recent depressive symptoms as a separate mediator. Our results suggest that recent depressive symptoms and socio-cognitive disruptions together fully explain the relationship between insomnia symptoms and suicide ideation, supporting our second hypothesis. While the recent depression path accounted for more of the variance in suicide ideation than the

pathway involving social-cognitive factors (e.g. a medium effect versus a small effect), both pathways contributed significantly to the outcome. These findings are consistent with prior research indicating that depression functions as a mechanism explaining the insomnia-suicide ideation relationship (e.g. Buysse et al., 2008; Janson-Fro¨jmark & Lindblom, 2008; Morphy et al., 2007). However, they also support studies suggesting that insomnia symptoms confer at least a small proportion of risk to experiencing suicide ideation through processes other than depressive symptoms as well (Bernert et al., 2005; Pigeon, Pinquart, & Connor, 2012; Ribeiro et al., 2012; Smith, Perlis, & Haythornthwaite, 2004).

This is one of the first studies to provide preliminary evidence for a social-cognitive path from sleep deficits to suicide risk that may not involve depression. Such findings may reflect some degree of the uniqueness of hopelessness apart from depressive symptoms in explaining the occurrence of suicide ideation (Beck et al., 1993; O'Connor, Armitage, & Gray, 2006). Additionally, such results may reflect that the hypothesized pathway examined in this study is illustrative of a broader process by which individuals who experience sleep deficits begin to also experience reductions in socio-cognitive functioning that are not fully defined by the essence of depression. Indeed, research suggests that insomnia symptoms contribute risk to suicide ideation that is not fully explained by depressive symptoms that are strongly associated with ideation, including depressed mood in particular (Ribeiro et al., 2012).

Nonetheless, these findings must be interpreted with some reservation since the social-cognitive pathway explained such a small proportion of variance after depressive symptoms are considered in the model. Indeed, including depressive symptoms in the model reduces the contribution of the hypothesized pathway from a medium effect to a small effect. Such shared variance may reflect the interrelationships of depressive symptoms with each of the variables in

the hypothesized pathway. For example, insomnia symptoms have been found to precede the onset of depression (Morphy et al., 2007; Neckelmann et al., 2007). And, reasoning abilities and cognitive functioning more generally have been found to decline in the presence of depression, potentially as a consequence of depression (see Hammar & Ardal, 2009 for a review). Furthermore, the variables in the hypothesized pathway are diagnostic symptoms of a depressive episode (American Psychiatric Association, 2013), such that it is difficult to ascertain the unique contribution of each pathway in explaining ideation. Alternatively, our measure of depressive symptoms was primarily comprised of items indicating negative affect, and the measures for the remaining variables in the model were also saturated with negative affect. It may be that negative affect accounted for the majority of the variance explained in ideation. Such possibilities may provide some explanation for the reduced effect of the hypothesized pathway in explaining ideation in the presence of depressive symptoms. Research employing experimental methodologies to isolate and test functions (e.g. cognitive rigidity; future orientation) common to both depression and the hypothesized socio-cognitive pathway may be particularly informative in furthering understanding of putative factors that underlie the insomnia-ideation relationship.

Limitations and Future Research

There are several limitations of the present study that should be acknowledged. Our study is cross-sectional, and thus our findings indicate preliminary evidence in support of the proposed model, rather than causality. As such, future studies examining these socio-cognitive mechanisms over time with regards to insomnia symptoms and suicide risk, as well as experimentally manipulating these mediators, are warranted. Potential studies in this vein could examine the development of cognitive deficits over time among individuals with an insomnia diagnosis, and assess the degree to which manipulating fatigue severity and longevity may

deleteriously impact aspects of cognition implicated in reasoning (e.g. working memory deficits, cognitive rigidity) as well as mood regulation.

Additionally, while all of the instruments utilized demonstrated good reliability in the present sample and good psychometric properties with regard to college students, the measures utilized were self-report instruments, and thus potentially subject to biases in social desirability as well as individual differences in insight (Podsakoff & Organ, 1986; Hammen, 2006). Future studies assessing these constructs more objectively, and utilizing interview assessments in examining clinical symptoms, are needed. Furthermore, the sample utilized in our study was restricted to females, and were predominantly Caucasian. Future research with more diverse samples (e.g. males and racial and ethnic minorities) may be useful in ascertaining the generalizability of our results. Finally, although sampling methods were employed to increase the likelihood of recruiting individuals at greater risk for suicide (e.g. recruiting females), our sample was comprised of college students. Thus, further replication is needed in more diverse clinical samples with higher rates of suicide ideation.

Despite these limitations, the present study is one of the first to investigate mechanisms other than depression that may help explain the relationship between insomnia symptoms and suicide ideation. It expands upon existing research by increasing knowledge of such explanatory mechanisms, and addresses a point of contention in the literature by identifying putative mechanisms that stem from sleep deficits, even after accounting for the influence of recent depressive symptoms. These findings have meaningful implications for understanding mechanisms by which insomnia symptoms may confer heightened risk for considering suicide.

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Appendix A

Demographics Form

Please be aware that all responses are kept strictly confidential and any research data is kept separate from your identifying information.

1. What is your age in years? _____

2. What is your gender?
a) Male
b) Female
c) Trans-gender
d) Other (please specify) _____

3. Which of the following best describes your current academic level?
a) Freshman
b) Sophomore
c) Junior
d) Senior

4. What is your current living situation?
a) Off-campus apartment
b) Off-campus fraternity or sorority house
c) Off-campus, at home with family
d) On-campus residence hall
e) On-campus fraternity or sorority house
f) Other (please specify) _____

5. What is your sexual orientation?
a) Heterosexual
b) Homosexual
c) Bisexual
d) Unsure
e) Other (please specify) _____
f) Prefer not to answer

6. What is your ethnicity?
a) Hispanic or Latino/a
b) Non-Hispanic or Non-Latino/a

7. How would you describe your race? (Select all that apply)
a) America Indian or Alaskan Native
b) Asian
c) Black or African American
d) Native Hawaiian or Pacific Islander
e) White
f) Other, please specify _____

8. Which of the following best describes your current marital status?
a) Single
b) Married or domestic partnership
c) Widowed
d) Divorced
e) Separated

9. How well do you speak English?
a) Very well
b) Well
c) Average
d) Poorly
e) Very Poorly

Appendix B

Documentation of Institutional Review Board Approval

USF
UNIVERSITY OF
SOUTH FLORIDA

RESEARCH INTEGRITY AND COMPLIANCE
Institutional Review Boards, FWA No. 00001669
12901 Bruce B. Downs Blvd., MDC035 • Tampa, FL 33612-4799
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October 9, 2014

Melanie Bozzay
Psychology
Tampa, FL 33647

RE: **Exempt Certification**
IRB#: Pro00019500
Title: Sleep, Cognition, and Mental Health

Study Approval Period: 10/8/2014 to 10/8/2019

Dear Ms. Bozzay:

On 10/8/2014, the Institutional Review Board (IRB) determined that your research meets USF requirements and Federal Exemption criteria as outlined in the federal regulations at 45CFR46.101(b):

(2) Research involving the use of educational tests (cognitive, diagnostic, aptitude, achievement), survey procedures, interview procedures or observation of public behavior, unless:
(i) information obtained is recorded in such a manner that human subjects can be identified, directly or through identifiers linked to the subjects; and (ii) any disclosure of the human subjects' responses outside the research could reasonably place the subjects at risk of criminal or civil liability or be damaging to the subjects' financial standing, employability, or reputation.

Approved Items:

Sleep and Cognition Study Protocol

Informed Consent

Your study qualifies for a waiver of the requirements for the documentation of informed consent as outlined in the federal regulations at 45CFR46.117(c) which states that an IRB may waive the requirement for the investigator to obtain a signed consent form for some or all subjects if it finds either: (1) That the only record linking the subject and the research would be the consent document and the principal risk would be potential harm resulting from a breach of

confidentiality. Each subject will be asked whether the subject wants documentation linking the subject with the research, and the subject's wishes will govern; or (2) That the research presents no more than minimal risk of harm to subjects and involves no procedures for which written consent is normally required outside of the research context.

As the principal investigator for this study, it is your responsibility to ensure that this research is conducted as outlined in your application and consistent with the ethical principles outlined in the Belmont Report and with USF IRB policies and procedures. Please note that changes to this protocol may disqualify it from exempt status. Please note that you are responsible for notifying the IRB prior to implementing any changes to the currently approved protocol.

The Institutional Review Board will maintain your exemption application for a period of five years from the date of approval or for three years after a Final Progress Report is received, whichever is longer. If you wish to continue this protocol beyond five years, you will need to submit a new application at least 60 days prior to the end of your exemption approval period. Should you complete this study prior to the end of the five-year period, you must submit a request to close the study.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638.

Sincerely,

Kristen Salomon, Ph.D., Vice Chairperson
USF Institutional Review Board