Insomnia drives changes in suicide ideation: A latent difference score model of community adults over a brief interval

by

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Abstract

Insomnia is robustly associated with suicidal behavior (Bernert, Kim, Iwata, & Perlis, 2015), but limitations in existing studies hinder nuanced understanding of this relationship. The current study aimed to address limitations by utilizing a longitudinal design and advanced statistical modeling. Participants who endorsed lifetime experience of suicidal behavior were recruited through Amazon's Mechanical Turk ($N = 589$) and completed self-report online surveys at six time points over a 15-day period. Latent difference score modeling was utilized to investigate whether levels and/or changes in insomnia symptoms drive subsequent changes in suicide ideation, or vice versa. Results revealed that previous level of insomnia was predictive of positive changes in suicide ideation (e.g., level of insomnia at Wave 2 predicted lagged increases in suicide ideation at Wave 3). This relationship was not bidirectional (i.e., suicide ideation exerted no effects on insomnia). Additionally, only previous level, and not previous changes, in insomnia were predictive of changes in suicide ideation. Our results help clarify the nature of the relationship between insomnia and suicide ideation as one that is unidirectional, thereby offering evidence of insomnia as a variable risk factor for suicide ideation. These findings yield clinical implications, including the importance of screening for insomnia symptoms, and provide support for exploring the potential effectiveness of insomnia treatments to target suicide ideation. Moreover, our study design and methodology establish a foundation for more rigorous and nuanced investigations of imminent suicide risk in future studies, which ultimately can promote better clinical practice in the reduction in suicidal behavior.

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Over the past decade, the United States suicide rate has increased significantly, and it continues to rise (Drapeau & McIntosh, 2014). Even more prevalent than fatal suicidal behavior are its precursors, including suicide ideation, with recent estimates suggesting that over 8.2 million people in the United States experience suicide ideation annually (Crosby, Gfroerer, Han, Ortega, & Parks, 2011). The concerning increase in the suicide rate highlights the continued need to identify and investigate risk factors for suicide. A multitude of these risk factors have already been identified (e.g., age, sex, psychiatric diagnoses, and past suicide attempts; Joiner, 2005; O'Connor & Nock, 2014; Rudd & Joiner, 2004; Rudd et al., 2006). However, consideration of longstanding or stable risk factors, like those described above, are not sufficient to assess and predict suicide risk (Joiner, Walker, Rudd, & Jobes, 1999). In addition, consideration of risk factors conferring proximal risk for suicidal behavior (also called *imminent* or *acute* risk factors) is also crucial (Rudd et al., 2006). Proximal risk factors refer to the current state or conditions of an individual, which are likely to be more episodic and variable than the static, enduring risk factors described above. One proximal risk factor that has gained traction over the past decade as related to suicidal behavior is the presence of sleep disturbances, like insomnia (Bernert & Joiner, 2007). However, this literature basis is limited by the use of cross-sectional studies, and where longitudinal studies do exist, the measurement periods employed often exceed the bounds of what would be useful in investigating imminent risk. As such, the current project seeks to expand

and improve upon extant literature and better understand the temporal relationship between insomnia and suicidal behavior.

Proximal Risk Factors

Though factors conferring distal and proximal risk can actually be identical (e.g., hopelessness), studying proximal risk factors requires narrower temporal focus on the current state of an individual. However, findings from a recent meta-analysis have revealed a major limitation of existing longitudinal suicide research—its use of long follow-up time periods (i.e., most studies using a follow-up period of 61-121 months; Franklin et al., 2014), which renders investigation of proximal risk impossible. Given the field's reliance on long follow-up periods, or failure to conduct longitudinal research at all (in which case only *correlates* of suicide are being investigated, rather than potential risk factors; Kraemer et al., 1997), there remains a major gap in the suicide literature related to proximal risk factors in predicting all forms of suicidal behavior (i.e., ideation, attempts, death by suicide; Franklin et al., 2014). To remedy this limitation, Franklin and colleagues (2014) have suggested that prediction of suicidal behavior could be enhanced by shortening the follow-up time period to months, weeks, or even days. This focus would also better align with the primary goal of clinical interventions for suicide, which is to accurately identify individuals likely to attempt suicide within the very near future (i.e., hours, days, weeks). This goal is nearly impossible when depending on suicide risk research findings using cross-sectional designs or long follow-up periods, given the transient nature of proximal risk. For example, certain variable risk factors might only be related to suicide risk during a short temporal window (e.g., relationship break-up), whereas the predictive power of this risk factor over a longer period of time would be weak (i.e., stress associated with a relationship break-up would likely be weakly related to suicidal behavior a year later).

In addition, where longitudinal research does exist, it has largely focused on a handful of risk factors (e.g., hopelessness, depression, impulsivity) that are, in general, weakly predictive of eventual suicide ideation, attempt, and death (Franklin et al., 2014). Continued focus on these risk factors has severely limited any improvement in predicting suicide risk over the last 50 years, though other promising factors have received less attention (Franklin et al., 2014). For example, from a given list of risk factors, such as those described above, it is likely that few mental health professionals would hone in on sleep disturbances as a particularly important predictor of suicide over others (e.g., hopelessness; Ribeiro et al., 2012). However, to the contrary, sleep disturbances have been gaining attention over the last decade as a robust correlate for a range of suicidal behavior (Bernert et al., 2015; Pigeon, Pinquart, & Conner, 2012). Crucially, sleep disturbances also may be more easily amenable to psychological treatment, unlike other factors (Bernert & Joiner, 2007), which may assist in the development of clinical and behavioral health interventions for sleep disturbances that simultaneously lower risk for suicidal behavior (Pigeon & Caine, 2010). In addition, because people may be less hesitant to disclose sleep disturbances than other mental health symptoms (Pigeon, Britton, Ilgen, Chapman, & Conner, 2012), focus on this potential risk factor may provide an alternative route to identifying high-risk individuals, which has the potential to facilitate better screening of suicide risk in a variety of settings (e.g., primary care, military).

Though a link between sleep disturbances and suicidal behavior has been established (Bernert et al., 2015), the manner in which these variables relate over time is not clearly understood, especially as related to proximal risk. The current study aims to better understand the dynamic interplay between sleep disturbances and suicide over a brief interval, with the ultimate goal of informing and enhancing assessment and intervention efforts for proximal suicide risk.

Specifically, given the lack of research on proximal risk for all forms of suicidal behavior (Franklin et al., 2014), we will use suicide ideation as the outcome variable of interest, as it is typically a precursor to other forms of suicidal behavior, and is more prevalent in the general population.

Sleep Disturbances as a Proximal Risk Factor

The aforementioned association between sleep disturbances and suicidal behavior suggests that these constructs may share a number of underlying mechanisms, and indeed, several have been proposed in an effort to understand why these constructs would be related (for review, see Bernert & Joiner, 2007). One explored mechanism that is particularly relevant to consider in a proximal risk context is the physiological overarousal (i.e., overactivation of the sympathetic nervous system; heightened stress response) that accompanies sleep disturbances, and in particular, insomnia (Roth, 2007). For the purposes of the current study, insomnia refers to difficulty maintaining or initiating sleep, resulting in daytime consequences (e.g., fatigue; Roth, 2007).

Suicide decedents are often described as *agitated* or *keyed up* on the days and nights preceding their deaths (Hall, Platt, & Hall, 1999). Ribeiro and colleagues (2012) argue that this state of overarousal enables individuals to overcome the daunting and fear-inducing act of lethal self-injury, which, outside of this agitated state, might otherwise have prevented these individuals from attempting suicide. Because sleeplessness, or specifically, insomnia, is thought to be a key indicator of overarousal (Ribeiro et al., 2012), insomnia may be one manifestation of broader underlying physiological changes that lend to increased risk for suicide.

Beyond risk for suicide, insomnia is associated more broadly with experience of psychopathology, including depressive disorders and anxiety disorders (Ohayon, Caulet, &

Lemoine, 1998). In fact, insomnia symptoms are estimated to be found in as many as 80% of individuals experiencing a current major depressive episode, and up to 90% with a co-morbid anxiety disorder (Ohayon, Shapiro, & Kennedy, 2000). The high prevalence of insomnia among those with other forms of psychopathology, and also individuals without co-occurring mental disorders (Ohayon, 2002), suggests that insomnia may be considered a transdiagnostic risk factor for suicidal behavior (Harvey, 2008). Indeed, because of evidence that insomnia contributes to onset, relapse, and maintenance of a variety of psychiatric disorders, it is likely that insomnia functions as a transdiagnostic process, indicating that treatments for insomnia may be effective in treating a range of psychopathology (Harvey, 2008), which, as previously mentioned, may include suicidal behavior.

The frequency with which insomnia presents in the context of depressive disorders is reflected by the inclusion of insomnia as a symptom of depression in the *Diagnostic and Statistical Manual*. Because insomnia itself is considered a symptom of depression, there may be concern about whether insomnia is simply one manifestation of underlying depression, and that the relationship between insomnia and suicidal behavior may be spurious. However, as will be elaborated upon more in the following section (*Review of insomnia and suicidal behavior literature*), the link between insomnia and suicidal behavior appears to extend beyond co-morbid psychopathology (Bernert et al., 2015). In fact, research has also demonstrated that insomnia symptoms often precede depressive symptoms (Perlis et al., 2006), suggesting that insomnia symptoms may be a critical factor in the development of depression and suicidal behavior.

Review of Insomnia and Suicidal Behavior Literature

Given the aforementioned mechanism linking insomnia and proximal suicide risk (Ribeiro et al., 2012), the current study is focused on further exploring this relationship over

other forms of sleep disturbance (e.g., nightmares). Moreover, insomnia has received the most attention empirically, compared to other sleep disturbances (Ribeiro et al., 2012), and has been linked to several forms of suicidal behavior (i.e., ideation, non-fatal attempts, and death by suicide) within both the general population (Agargun, Kara, & Solmaz, 1997; Fujino, Mizoue, Tokui, & Yoshimura, 2005; Wojnar et al., 2009) and military samples (Ribeiro et al., 2012). However, existing literature is limited by its largely cross-sectional nature (e.g., Agargun et al., 1997; Chellappa & Araújo, 2007; Sjöström, Waern, & Hetta, 2007), which limits ability to speak to causal mechanisms or gain a more nuanced understanding of the directionality of this effect. Though fewer longitudinal studies exist, some longitudinal research also provides evidence of the relationship between insomnia and suicide ideation (McCall et al., 2010; Ribeiro et al., 2012), non-fatal attempts (Ribeiro et al., 2012) and death by suicide (Fujino et al., 2005); however, as will be discussed in more detail below, these studies are limited for a number of reasons.

In addition to reliance on cross-sectional data, it is also unclear whether the relationship between insomnia and suicidal behavior is spurious and better accounted for by the presence of other symptomatology. Several cross-sectional studies (Bernert, Joiner, Cukrowicz, Schmidt, & Krakow, 2005; Sjöström et al., 2007) have found that the relationship between insomnia and suicidal behavior (i.e., ideation and attempts, respectively) becomes non-significant after controlling for other symptomatology (i.e., depression, presence of other sleep disturbances like nightmares), while one longitudinal study using a military sample (Bryan et al., 2015) found that the relationship became non-significant upon inclusion of depressive symptoms as a mediator. However, two longitudinal studies have found that the association between insomnia and suicidal ideation remains significant after controlling for other symptoms, including depression (McCall et al., 2010) and depression, anxiety, hopelessness, and posttraumatic stress disorder (Ribeiro et

al., 2012). In addition, Ribeiro et al. (2012) found that insomnia outperformed all other symptoms as a predictor of suicidal behavior in a longitudinal investigation of young adults in the military.

Ribeiro et al. (2012), McCall et al. (2010), and Bryan et al. (2015) have conducted the most comprehensive investigations yet, using longitudinal designs; however, these studies are limited. Though Ribeiro et al. (2012) used a relatively short follow-up period (i.e., one month), which theoretically allows for investigation of proximal risk, only two time points were used, and the statistical methodology used (i.e., multiple regression) did not sufficiently allow for exploration of the temporal relation between these variables (i.e., investigation of lead-lag relationships; Ferrer & McArdle, 2010). Similarly, though McCall and Blocker (2010) utilized additional time points, their statistical methodology (i.e., mixed modeling) also does not establish temporal precedence of insomnia symptoms in the prediction of suicide ideation. Lastly, while Bryan et al. (2015) collected six time points over the course of a year and used a more advanced statistical model (i.e., latent difference score mediation model), this study was inadequately powered to test a complex model utilizing all time points (N = 168). Instead, Bryan and colleagues (2015) were only able to test this mediation effect in two models, each using two time points (i.e., one examining the mediation effect on a subset of data starting with baseline to a 12-month follow-up, and the other from 3-month to 18-month follow-up), which does not allow for study of temporal precedence.

Although the study by Bryan and colleagues (2015) represents a critical starting point in exploring more complex dynamics between these variables, inclusion of more time points drawn from a larger sample would provide more insight about sequencing of variables over time (Ferrer & McArdle, 2010). In addition, though Bryan et al. (2015) utilized several follow-ups, the

intervals between follow-up points were long, which does not allow for study of proximal risk, given that studies with two follow-up points are not truly longitudinal studies (Ployhart & MacKenzie, 2014). Though prevalent, especially in the field of psychology, studies with two time points are not adequately suited to speak to change over time, as they have a high potential for misestimating effect sizes and reliability and do not allow for examination of more nuanced dynamics related to onset, duration, or offset (Ployhart & MacKenzie, 2014). Moreover, other specifications of the latent difference score framework utilized by Bryan et al. (2015) could also allow for exploration of how previous levels and/or changes in one variable relate to subsequent changes in another variable (Grimm et al., 2012), allowing for exploration of leading and lagging processes and joint trajectories of insomnia and suicide ideation (i.e., increases in insomnia preceding increases in ideation; decreases in insomnia preceding decreases in ideation). Importantly, investigating these processes allows for establishing temporal precedence among variables, which is one of the primary conditions of establishing causality. Moreover, to establish insomnia as a *risk factor* for suicide, as opposed to simply a correlate, temporal precedence must be observed (Kraemer et al., 1997). Compared to more traditional longitudinal modeling techniques, latent difference score models are more complex (Grimm et al., 2012) and allow temporal precedence to be explored bi-directionally. In this way, in the current study, latent difference score modeling will allow examination of whether insomnia can be termed a variable risk factor for suicide ideation—that is, a factor preceding suicide ideation that can also can change, given that insomnia is not a permanent condition. As such, results from this study can help lay the foundation for examining insomnia as a potential *causal* risk factor for suicide (Kraemer et al., 1997), in which actual manipulation of insomnia can be observed to effect

suicide ideation. This would imply that an intervention targeting insomnia symptoms should result in a reduction in suicide ideation.

Present Study

The present study expanded and improved upon the existing insomnia and suicidal behavior literature, following a recent call for more methodologically rigorous studies of this relationship (Bernert et al., 2015). To account for prior limitations, the current study employed an intensive longitudinal design, assessing for suicide ideation, insomnia and other relevant symptoms at six time points, and utilizing a community sample with sufficient power to employ advanced statistical modeling; namely, latent difference score modeling (Ferrer & McArdle, 2010; Grimm et al., 2012). In addition, the use of several follow-up points over a brief interval (15 days) allowed for exploration of insomnia symptoms and proximal suicide risk, in line with recommendations from Franklin et al. (2014). Given that insomnia itself has been implicated in the classification of depression, it is particularly relevant to examine the relationship between insomnia and suicide ideation while parsing out the effects of depression; as such, we also sought to control for concurrent depressive symptoms to conduct the most stringent test of this relationship. Based on previous research on insomnia and suicidal behavior, we hypothesized that:

1) Level of insomnia will predict lagged changes in suicide ideation (e.g., level of insomnia at Wave 1 will predict worsening ideation at Wave 2), and not vice versa, controlling for depressive symptoms.
2) Changes in insomnia over time will predict lagged changes in suicide ideation (e.g., worsening insomnia between Waves 1 and 2 will predict worsening ideation at Wave 3), and not vice versa, controlling for depressive symptoms.

Methods

Participants and Procedure

Participants were English-speaking US residents recruited through Amazon's Mechanical Turk (MTurk). MTurk allows data collectors to recruit participants (workers) online to complete various tasks in exchange for wages and has been increasingly used in behavioral research to study community and clinical populations (Shapiro, Chandler, & Mueller, 2013). There are several advantages to using MTurk over a typical undergraduate participant pool, including increased diversity (e.g., age, race, socioeconomic status), expeditious data collection, and ability to attain more variability in key variable of interest (i.e., suicidal behavior) by screening large numbers of potential participants. All procedures were approved by our university's institutional review board prior to implementation of the protocol.

A screener questionnaire was available first on MTurk with questions about lifetime suicide behavior (i.e., ideation, plan, attempt), in order to recruit oversample for participants likely at elevated risk for suicidal behavior. However, to distract potential participants from the aim of the study, additional questions were also included in this screener questionnaire (e.g., items from the Patient Health Questionnaire-4; Löwe et al., 2010). We screened 1,940 workers and invited 1,029 (53.04%) of those screened to participate in the study based on endorsing a history of lifetime suicide ideation, plan or attempt (i.e., answering *yes* to any of the following questions: *Have you ever had thoughts of killing yourself?*, *Have you ever made a plan to kill yourself?*, or *Have you ever made an actual attempt to kill yourself in which you had at least*

some intent to die?). Of those who were sent an invitation, 589 (57.24%) ultimately participated in the study (i.e., completed at least Wave 1 of the study). Within the final sample, 257 (43.63%) of participants endorsed experiencing some degree of current suicide ideation (i.e., higher than a 0 on the Depressive Symptom Inventory – Suicide Subscale; DSI-SS; Joiner, Pfaff, & Acres, 2002), and 166 (28.18%) reported a past suicide attempt (i.e., answering *yes* on the question: *Have you ever made an actual attempt to kill yourself in which you had at least some intent to die?*). Of those who endorsed history of a suicide attempt, the modal number of attempts was one (n = 82; 49.70% of past attempters), and the range of suicide attempts observed in the sample was 1-100.

Participants were, on average, 34 years old (SD = 11.07) and mostly women (72.33%; n = 426). The majority of participants were non-Hispanic (92.36%; n = 544) and White (85.74%; n = 505); 9.68% Black (n = 57); 6.62% Asian (n = 39); 3.06% American Indian/Alaskan Native (n = 18); .68% Native Hawaiian or Other Pacific Islander (n = 4), with participants selecting all races that applied. Most participants had completed college or beyond (e.g., master's or doctoral degrees; 50.25%, n = 296), reported having full-time jobs (i.e., working over 30 hours per week; 47.03%, n = 277), and endorsed making under $39,999 a year (49.92%, n = 294).

Participants completed six waves of data collection over a 15-day time period, completing a battery of surveys every three days, receiving reminder emails to complete each wave of the study. Surveys administered included those assessing for symptoms of suicidality, insomnia, and depressive symptoms (see *Measures* section). Surveys were administered via Qualtrics, with links available through MTurk. Surveys were linked across time using participant's MTurk worker IDs, which are not linked to any identifiable information to which the researchers have access. Regarding attrition, 469 participants (79.63% of those who

participated in Wave 1) completed Wave 2; 461 (78.27%) completed Wave 3; 424 (71.99%) completed Wave 4; 455 (77.25%) completed Wave 5; 429 (72.84%) completed Wave 6.

Participants received monetary compensation for their participation. MTurk workers who completed the screener received $.10. Then, invited workers who participated in the study (N = 601) received payments for each wave of data collection, with increasing compensation over subsequent waves to incentivize participation: Wave 1: $2.50 (about 30 minutes), Waves 2-3: $0.50 (15 minutes each), Waves 4-6: $0.75 (15 minutes each), for a total of $5.75 per participant. A bonus ($0.50) was also sent with invitation to Wave 2 to incentivize continued participation, and participants who completed all six waves received another bonus ($0.50). As such, each participant had the opportunity to earn up to $6.75 for participating in the study, in addition to $.10 for completing the screener.

Measures

Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001). The ISI was used to assess for symptoms of insomnia at all time points. This measure assesses for the severity of insomnia (e.g., *Please rate the current severity of your difficulty falling asleep*) and interference with daily functioning (e.g., *To what extent do you consider your sleep to interfere with your daily functioning [daytime fatigue, mood, ability to function at work/daily chores, etc.]*) over the past three days using seven items on a 5-point scale. Prior research has reported the good psychometric properties of this measure (Bastien et al., 2001; Morin, Belleville, Bélanger, & Ivers, 2011). In the current study, a total score from this measure was derived for all time points. Internal consistency for the ISI was acceptable across waves (α's = .88-.93).

Depression Symptom Inventory - Suicide Subscale (DSI-SS; Joiner, Pfaff, & Acres, 2002). The DSI-SS is a brief, 4-item self-report questionnaire measuring suicide ideation and

impulses on a 4-point scale. The total score from this measure was used as a measure of suicide ideation at all time points, and participants were asked to consider their thoughts and behavior over past three days. Prior studies have demonstrated the good psychometric properties of this measure (Joiner et al., 2002; Metalsky & Joiner, 1997). Participants who endorsed a three or higher on this measure, indicating greater than low risk (Joiner et al., 2002) were immediately presented with instructions on how to make a coping card (Joiner, Van Orden, Witte, & Rudd, 2009; Rudd et al., 2004). Internal consistency was acceptable for the DSI-SS for all waves of the study (α's = .90-.95).

Patient Health Questionnaire - 4 (PHQ-4; Löwe et al., 2010). This brief, self-report questionnaire was used to assess for symptoms of depression. Specifically, we used the two items from the PHQ-4 that assess for depressive symptoms (i.e., *little interest or pleasure in doing things*; *feeling down, depressed, or hopeless*). These items are rated on a 4-point scale, and were summed to create a total depression score at each wave. These total scores were included as covariates in analyses (see *Statistical Procedure* section). This measure has been shown to have good psychometric properties (Löwe et al., 2010), and internal consistency in the current sample for the depression subscale was adequate across all time points (α's = .86-.88).

Statistical Procedure

Missing data and non-normality. Missing data were handled using full information maximum likelihood. Minimum covariance coverage in this investigation ranged from .64 to .97. All analyses were conducted using a robust maximum likelihood estimator (MLR) in Mplus version 7.2 (Muthén & Muthén, 2012), which has been shown to improve Type I error rates over traditional estimators in the presence of non-normal data (Enders, 2001).

Analytic plan. A latent difference score modeling (LDS) framework was used to analyze data and test hypotheses of the study that combines components of latent growth modeling and autoregressive cross-lagged models that are often used to analyze longitudinal data. LDS modeling is generally considered a framework (Grimm et al., 2012) because many longitudinal models typically applied to panel data (e.g., autoregressive cross-lag, latent curve models, time-varying covariate model; Grimm et al., 2012) can actually be re-specified to incorporate latent differences; however, common specifications of models included under this umbrella do not include such parameters. Similar to their statistical predecessors (e.g., classic autoregressive cross-lag models (Jöreskog, 1974; Jöreskog, 1970), LDS models allow for the examination of dynamic change relationships. These models also allow for directionality to be tested, and for changes across time to be examined within-person (Grimm et al., 2012).

Specification of LDS parameters. Following from classical test theory, observed scores on a given variable at time *t* are modeled as the latent 'true' score plus unique score (residual error). In LDS models, latent difference scores are then created such that the true score at a given time point is a function of the true score at the preceding time point plus change in the true score between measurement periods. Notably, difference scores are not created as they frequently are in other longitudinal models by taking the difference between observed scores. Rather, because change is estimated as a latent score, LDS models allow for change itself to be the focus of prediction. In the bivariate case, LDS models enable examination of whether changes in one process (e.g., suicide ideation) are determined by the previous state of the second process (e.g., insomnia), and vice versa. As such, the change in an outcome *y* at any measurement point is depicted below in Equation 1 (from Grimm et al., 2012 equation 7):

$$\Delta y[t]_n = \alpha_y \cdot s_{yn} + \beta_y \cdot y[t-1]_n + \gamma_{yx} \cdot x\,[t-1]_n + \phi_y \cdot \Delta y[t-1]_n + \xi_{yx} \cdot \Delta x[t-1]_n \quad (1)$$

where α_y is a fixed parameter and s_{yn} is the constant change component. In this equation, β_y represents proportional change in *y* between time points, which is a time invariant parameter that is not allowed to vary between subjects, implying that the dynamics of the system are constant over time. Similarly, γ_{yx} is the proportional change in *y* between time points, adding in the effect of proportional change in *x* on subsequent change in *y,* termed a coupling parameter in LDS. These two parameters (β_y *and* γ_{yx}) effectively model changes in the outcome over time as a function of the true scores on the same or different process at the previous time point. Applied to the current study, these parameters model lagged changes in suicide ideation (*y*) at a given assessment due to the *level* of both suicide ideation and insomnia reported at the previous assessment (Hypothesis 1). Specifically, a statistically significant, positive γ_{yx} parameter would indicate that level of insomnia at Wave 1 predicts positive change in suicide ideation at Wave 2. This would imply both that lower initial levels of insomnia are associated with smaller, but still positive, changes in ideation and that higher initial levels of insomnia are associated with larger, positive changes in ideation. Note, although we use patterns in insomnia and suicide ideation at Waves 1 and 2 as an example here, these patterns carry on throughout all time points.

Considering remaining parameters, Equation 1 also models how lagged *changes* (rather than *levels*) predict subsequent changes in the outcome. That is, ϕ_y describes how changes in *y* from *t-1* (i.e., *t* minus 1) to time *t* are determined by the changes from *t*—2 to time *t—1* in *y*, while ξ_{yx} describes the same process for changes in variable *x* predicting changes in *y*. In the current study, these dynamic change parameters model lagged changes in suicide ideation (*y*) from *t-1* to time *t* due to *changes* in suicide ideation and insomnia between *t*-2 to time *t-1*

(Hypothesis 2). In this case, a statistically significant, positive ξ_{yx} parameter would imply that, for example, increases positive in insomnia between Waves 1 and 2 predict positive change in suicide ideation at Wave 3. This would imply that smaller increases in insomnia between Waves 1 and 2 are predictive of smaller, but still positive, changes in ideation at Wave 3, and that a greater degree of positive change in insomnia between Waves 1 and 2 is associated with larger, positive changes in ideation at Wave 3. This dynamic change parameter in particular is distinct from other longitudinal models, where only the absolute or overall level of a variable often predicts change; in the case of the dynamic change parameter, independent of the absolute level of either variable modeled, previous states of a given variable play an instrumental role in predicting future change. While this dynamic change parameter is similar to the proportional change parameter (levels predicting changes), Grimm and colleagues (2012) describe it as unique in that it allows the model to take more information into account. For example, changes in suicide ideation may be affected by a certain level of insomnia, but these changes in ideation may be further accelerated when insomnia has recently increased. In this way, the inclusion of this parameter reflects "not just where you are, but where you have recently been" (Grimm et al., 2012; p. 12). The inclusion of this parameter is similar to the practice of adding additional lags reflecting not only the recent past ($t - 1$), but also earlier points in time ($t - 2$, $t - 3$, etc.), which is common practice in time series models (Grimm et al., 2012). We again note that although we use patterns in insomnia and suicide ideation between Waves 1 and 3 as an example here, these patterns carry on throughout all time points.

The introduction of these unique LDS parameters allows for testing whether previous levels and changes are leading indicators of subsequent changes both within and across variables. Importantly, though the examples provided above describe changes in the outcome y due to

levels and changes in *y* and *x,* LDS models allow for this model to be tested bi-directionally, modeling both processes simultaneously (a full coupling model; Grimm et al., 2012). Such a full coupling model is depicted graphically in Figure 1, portraying the dynamic interplay between two processes *y* and *x*.

Specification and comparison of models. In the current study, the use of an LDS framework was well suited to examine the bi-directional effects of suicide ideation and insomnia. Following procedures outlined by Grimm and colleagues (2012), we constructed a set of bivariate LDS models, with each model sequentially incorporating the parameters described above. Prior to testing these bivariate models, we first tested three nested univariate models for each outcome of interest (i.e., suicide ideation and insomnia). These models sequentially added univariate parameters shown in Equation 1. The first model (constant change) modeled variable *y* over time by a constant change component (s_y). This model was followed by a second dual change model, which included constant change and added the proportional change component (β_y). Finally, the third dynamic change model incorporated both aforementioned parameters, as well as the dynamic change component (ϕ_y). Univariate models were compared using Satorra-Bentler chi-square difference testing (Satorra & Bentler, 2001), and the best fitting model for each variable was selected.

After selecting the best univariate models, we proceeded in a similar fashion to test seven bivariate models. The equations for the full coupling model are shown below for each outcome, with *SUI* representing suicide ideation (Equation 2) and *INS* representing insomnia (Equation 3). We first tested a constant change model (Model 1) with no coupling, then tested two dual change models (one including only $\gamma_{INS,SUI}$ and the other including only $\gamma_{SUI,INS}$; Models 2 and 3). The fourth model included both of these proportional coupling parameters (bi-directional coupling

model). Lastly, we tested two additional models that included either dynamic change parameters ($\xi_{INS \cdot SUI}$ or $\xi_{SUI \cdot INS}$; Models 5 and 6), and then included all of these coupling parameters in the final, full coupling model (Model 7). The equations for the full coupling model are shown below for each outcome, with *SUI* representing suicide ideation (Equation 2) and *INS* representing insomnia (Equation 3):

$$\Delta SUI[t]_n = \alpha_{SUI} \cdot s_{SUI} + \beta_{SUI} \cdot SUI[t-1]_n + \gamma_{SUI \cdot INS} \cdot INS[t-1]_n + \phi_{SUI} \cdot \Delta SUI[t-1]_n + \xi_{SUI \cdot INS} \cdot \Delta INS[t-1]_n \quad (2)$$

$$\Delta INS[t]_n = \alpha_{INS} \cdot s_{INS} + \beta_{INS} \cdot INS[t-1]_n + \gamma_{INS \cdot SUI} \cdot SUI[t-1]_n + \phi_{INS} \cdot \Delta INS[t-1]_n + \xi_{INS \cdot SUI} \cdot \Delta SUI[t-1]_n \quad (3)$$

Each progressive bivariate model was compared against the previous using information criteria including Akaike's Information Criterion (AIC) and Bayesian Information Criterion (BIC; Grimm et al., 2012), with lower values indicating relatively better fit. Because the bivariate models are not truly nested models, chi-square difference testing cannot be used to compare models. In addition, general fit of all models (both univariate and bivariate) was evaluated using conventional criteria for standard structural equation model fit indices: Comparative Fit Index (CFI) > .95, Tucker-Lewis Index (TLI) > .95, Standardized Root Mean Square Residual (SRMR) < .06, Root-Mean-Square Errors of Approximation (RMSEA) < .06 (Hu & Bentler, 1999).

Controlling for depression. In all bivariate models, we residualized observed indicators for insomnia and ideation from each time point on depression scores at each time point. This solution allowed us to examine the variance in both outcomes (i.e., suicide ideation and

insomnia) after depression had been accounted for, which is statistically equivalent to including depression as a covariate within an LDS framework.

Power analysis. Though traditional power analysis methods cannot be used with the outlined analytic strategy, given several features of this study (e.g., large number of parameters to estimate; non-normality of data; low base rate of suicidal behavior; Kline, 2010), a large sample size was indicated. As such, we aimed to recruit a large sample of around 500 for this study, and achieved a final *N* of 601.

Results

Univariate Models

Descriptive statistics and intercorrelations between all study variables are in Tables 1 and 2. Fit statistics for the univariate LDS models for both insomnia and suicide ideation are presented in Table 3. Changes in insomnia were best represented by a dynamic change model, and fit for this model was acceptable based on a priori cut-scores. The selection of a dynamic change model indicates that changes in insomnia over time were dependent on a constant change component, previous levels of insomnia (i.e., proportional change component), and how insomnia had previously changed (i.e., dynamic change component), such that previous higher levels of insomnia and prior upward changes in insomnia both predicted downward changes in insomnia. Parameter estimates for this selected univariate model are shown in Table 4. Note, mean group slope was constrained to zero in this model and all others, given we had no reason to expect group-level changes in our outcomes over our selected measurement period.

Changes in suicide ideation over time were best represented by the dual change model (Table 2), and fit for this model was adequate using predetermined cut-scores, aside from SRMR which was elevated above suggested cut-scores. According to this model, changes in suicide ideation over time were dependent on the previous levels (i.e., proportional change) of suicide ideation and constant change, but not on how suicide ideation had recently changed (i.e., dynamic change). Thus, higher levels of previous suicide ideation were predictive of downward changes in suicide ideation. Parameter estimates for this selected model are also presented in

Table 3. Given the univariate dynamic change parameter was not significant for suicide ideation, we did not retain this parameter in the bivariate models moving forward (i.e., parameter ϕ_{SUI} from Equation 2 was not included).

Bivariate Models

After establishing best fit for univariate models, we proceeded to test bivariate models, which build on the univariate models by testing whether the longitudinal trajectory for either outcome is influenced by lagged levels or changes in the other outcome. These models all controlled for depression symptoms as described in the *Methods* section.

Fit statistics for all bivariate models are contained in Table 5. Based on model fit, Model 3 demonstrated best fit. This is a dual change model that includes the proportional change coupling parameter for suicide ideation (i.e., $\gamma_{SUI \cdot INS}$) but not for insomnia. This model also retains the univariate parameters for each outcome (i.e., for suicide ideation, including the proportional change component β_{SUI}, and for insomnia, including both the proportional change component β_{INS} and dynamic change ϕ_{INS}). Though chi-square was slightly lower in Model 4 (i.e., the bi-directional coupling model), no other fit indices improved, and BIC slightly worsened (i.e., was larger than for Model 3). In addition, we examined parameter estimates in Model 4 and found that the coupling parameter $\gamma_{INS \cdot SUI}$ (i.e., level of suicide ideation influencing changes in insomnia) was not significant, which is the only parameter that is added from Model 3 to Model 4. As such, Model 3 was retained for further consideration. Parameter estimates for the selected Model 3 can be found in Table 6, and the equations for this selected model can be found below for each outcome:

$$\Delta SUI[t]_n = \alpha_{SUI} \cdot s_{SUI} + \beta_{SUI} \cdot SUI[t-1]_n + \gamma_{SUI \cdot INS} \cdot INS[t-1]_n \quad (4)$$

$$\Delta INS[t]_n = \alpha_{INS} \cdot s_{INS} + \beta_{INS} \cdot INS[t-1]_n + \phi_{INS} \cdot \Delta INS[t-1]_n \quad (5)$$

This model demonstrates that suicide ideation was significantly predicted by previous levels of insomnia ($\gamma_{SUI \cdot INS}$). Parameter estimates indicate that changes in suicide ideation were positively impacted by the previous level of insomnia; thus, changes in suicide ideation increased at a faster rate if the individual had higher previous levels of insomnia. Conversely, insomnia was not influenced by previous levels or changes in suicide ideation and was only predicted by previous levels and changes in insomnia (i.e., β_{INS} and ϕ_{INS}), as previously described in the univariate results.

Graphical Depiction of Results

Visual representations of these results are shown in Figures 2-3 based on parameter estimates for the selected model. In Figure 2, we depict a hypothetical participant with average levels of both suicide ideation and insomnia at Wave 1 (i.e., starting at the intercepts). In this figure, we depict what is predicted to happen in this dynamic system with only inputs from Wave 1 to show what the model predicts with no perturbations in the system. As is shown in Figure 2, this constant system consists of two downward sloping lines for both suicide ideation and insomnia. If we only had the scores for a participant at Wave 1, and no changes in suicide ideation or insomnia scores occurred, this is the projected trajectory of symptoms over a 15-day period.

In Figure 3, we also depict a hypothetical participant with average levels of both suicide ideation and insomnia at Wave 1 (i.e., starting at the intercepts). However, in this figure, we portray more realistic and expected fluctuations in scores over time, as occurred with the participants in our sample, who completed measures of suicide ideation and insomnia at all time

points. To depict the influence of suicide ideation from levels of insomnia (i.e., the proportional coupling parameter that was significant in our final bivariate model), we force changes in insomnia symptoms at Waves 3 and 5. At Wave 3, the hypothetical participant has an increase in insomnia symptoms to 15 (suggesting moderate to severe insomnia; Morin et al., 2011). As shown in the figure, after this forced increase in insomnia symptoms, representative of an individual who starts at average levels of insomnia and experiences increases to a clinical level after two time points, suicide ideation then increases at Wave 4. This model also depicts concurrent univariate effects, wherein suicide ideation tends to decrease over time (i.e., regression to the mean); however, with a large enough disruption in the system (i.e., spike in insomnia from 9.755 to 15), suicide ideation is affected and increases temporarily at Wave 4.

We then force the participant insomnia score back to average (i.e., the intercept) at Wave 5. The lower level of insomnia symptoms at Wave 5 contributes less influence on suicide ideation at Wave 6, such that an increase is no longer observable in the figure after the effect of univariate parameters (i.e., level of suicide ideation predicting decreases subsequent ideation) is also taken into account. This change represents a participant who returns to his or her baseline level of insomnia after an increase, and subsequently, with no disruptions to the system, levels of suicide ideation return to previous lower levels that were observed before the increase in insomnia.

Discussion

Examining the lead-leg relationships between suicide ideation and insomnia over a brief interval, our findings indicate that prior levels of insomnia positively influence changes in suicide ideation, in that higher levels of insomnia lead to greater increases in suicide ideation at the subsequent measurement point. Consistent with hypotheses, this relationship was not bidirectional. Though we hypothesized that a similar relationship would emerge when considering the effect of previous changes in insomnia on subsequent changes in suicide ideation, these results were non-significant. Our results suggest that when an individual experiences a high level of insomnia, this will result in subsequent increases in suicide ideation, whereas lower levels of insomnia will result in smaller increases, which is also graphically depicted in Figure 3. Given that our model allowed establishment of temporal precedence, our results indicate that insomnia can be considered a variable risk factor for suicide ideation (Kraemer et al., 1997), because it precedes changes in suicide ideation, and because insomnia itself can change and fluctuate over time, which is also captured by our model.

Though not the focus of the current hypotheses, considering univariate results, we found that previous levels of suicide ideation negatively influenced changes in suicide ideation, while prior levels of and changes in insomnia negatively impacted changes in insomnia. These negative effects are consistent with regression to the mean, in that if participants' scores on either of these variables increased, they were then likely to decrease, at least slightly, at the next measurement point. In the case of suicide ideation, past research also suggests that asking participants about suicide may actually decrease distress, particularly for high-risk individuals (e.g., Gould et al.,

2005; Reynolds, Lindenboim, Comtois, Murray, & Linehan, 2006); thus, it is likely we are capturing this phenomenon with this univariate parameter. However, these results cannot be interpreted solely in isolation, given significant bivariate results. As such, for suicide ideation, the negative univariate effects dampen the effect of the proportional coupling between previous levels of insomnia and changes in suicide ideation: as increased levels of suicide ideation drive subsequent decreases, higher levels of insomnia drive this subsequent suicide ideation score back up. Put metaphorically, if we think of suicide ideation as a rocket traveling up, insomnia is the jet fuel propelling it to do so. With a full tank, the rocket can accelerate faster and keep moving ahead; as fuel runs out or decreases, the rocket may slow down. In this way, level of insomnia drives suicide ideation up, but it is important to note that our model does not demonstrate that *decreases* in insomnia compel corresponding decreases in suicide ideation. Using the rocket metaphor, decreases in jet fuel do not turn the rocket back around, but rather, the slowing of the rocket allows other factors (e.g., wind) to more easily influence it, which could then send it in another direction. As the influence of insomnia decreases (e.g., lowering of insomnia scores), the effects of the univariate parameters described here have a more potent effect in driving suicide ideation scores down. Our model allows the complex dynamics of this system to be captured, which are nuanced and undetectable through traditional statistical methodology.

Our findings shed much needed light on a literature confounded by cross-sectional studies and weak statistical models. Though the relationship between insomnia and suicide ideation has been previously suggested (e.g., Ribeiro et al., 2012), to our knowledge, no prior studies have been able to establish temporal precedence between these variables due to statistical and methodological limitations, though some have approached this goal (e.g., Bryan et al., 2015; McCall et al., 2010; Ribeiro et al., 2012). In a large sample of community adults with a history of

suicidal behavior, this study establishes the directionality of this relationship, thereby providing evidence of insomnia as a variable risk factor for suicide ideation, while also accounting for the effects of depressive symptoms unrelated to sleep. Controlling for depression represents a stringent test of the relationship between insomnia and suicide ideation, casting doubt on suspicion that this relationship is spurious and better accounted for by other symptoms. Though inclusion of other covariates would provide even more rigorous tests, our decision to include depression was based on its robust relationship with suicidal behavior (Joiner, Brown, & Wingate, 2005); as such, the fact that a significant relationship between insomnia and suicide ideation emerged suggests the strength of this relationship. Unlike Bryan and colleagues (2015), who found that insomnia was related to suicidal behavior insofar as it is "embedded" within depression, our results provide evidence that it is insomnia itself, and not simply overarching symptoms of depression, that is driving changes in suicide ideation. These findings yield both critical clinical implications and a foundational agenda for future research in this area.

Clinical Implications

Prior studies have suggested the utility of clinical or behavioral interventions for sleep as indirect treatments for suicidal behavior (Pigeon & Caine, 2010). Given our results suggest that higher levels of insomnia precede worsening (i.e., increases) in suicide ideation, our findings lend support to this proposition. Based on the directionality of the effect we found, there are several clinical implications. First, our findings suggest that individuals who are experiencing symptoms of insomnia, but who are not experiencing suicide ideation, will likely develop these symptoms over time. Thus, interventions aimed at treating insomnia (e.g., Cognitive-Behavioral Therapy for Insomnia; CBT-I; Morin et al., 2006) should monitor changes in suicide ideation, expecting that if someone experiences an increase in insomnia symptoms, this will then lead to

increases in suicide ideation. Though treatment for insomnia will ideally indirectly reduce suicide ideation, it is still important to consider risk within treatment, when more intensive suicide risk management steps could be taken with the client. Moreover, because higher levels of insomnia precede worsening in suicide ideation, it also may be that targeting insomnia primarily through CBT-I or another empirically supported treatment may prevent high levels of suicide ideation from developing at all. Finally, outside the bounds of mental health treatment, our findings lend support to screening for insomnia as a means of indirectly assessing suicide risk, which is particularly relevant for the primary care setting, where people may be more likely to disclose sleep problems than other mental health symptoms, including suicidality (Pigeon, Britton, et al., 2012). As such, screening for insomnia may offer an indirect method of evaluating individuals' risk for suicidal behavior and allow these individuals to be referred to appropriate treatment providers.

In addition, our results also suggest that if a suicidal patient begins to develop sleep problems, this may further exacerbate his or her symptoms of suicidality. As such, our results also provide support for previous suggestions that screening for insomnia in the context of suicide management is advisable (Woznica, Carney, Kuo, & Moss, 2015). While we are unable to determine which set of symptoms ultimately appears first (i.e., insomnia or suicide ideation) based on the design of the current study, regardless of when insomnia appears symptomatically, our results provide evidence it will drive corresponding changes in suicide ideation.

Methodological Considerations for Future Research

Because we measured the relationship between insomnia and suicide ideation over a brief interval, our results are well suited to speak to clinical implications. We sought to address existing limitations in the literature by focusing on a narrow follow-up time period, in line with

recommendations by Franklin and colleagues (2014), and as such, our results also align with the primary goals of clinical work, which are focused on current symptom experience and functioning, and identifying individuals at risk in the very near future. Though the majority of longitudinal studies on suicidal behavior have utilized very long follow-up periods, assessing symptoms over a brief interval is more desirable to capture the complex dynamics between variables that may change rather quickly. The current study demonstrates a significant improvement upon the past literature by focusing on a narrow time period, and also demonstrates one method by which symptoms can be measured over such a brief interval.

Because our model allowed for the establishment of temporal precedence, and thus, suggests insomnia is a variable risk factor for suicide ideation, our study also sets the stage for future research on insomnia as a *causal* risk factor for suicide, which can yield even more valuable clinical information and guide development of treatments and preventative efforts. To establish insomnia as a causal risk factor (per criteria outlined by Kraemer and colleagues [1997]), manipulation of insomnia must be shown to change the outcome (i.e., suicide ideation). As such, one valuable line of future research may be investigating the course of suicide ideation after manipulating (i.e., treating) insomnia within a randomized control trial for an insomnia treatment (e.g., CBT-I). Our findings provide support for such research, in that other criteria for defining a risk factor have already been established. Thus, appropriate and necessary next steps should be taken to continue examining the plausibility of insomnia as a *cause* of suicide ideation, and potentially, other forms of suicidal behavior.

Beyond providing specific clinical implications for the insomnia and suicide ideation link, the present study more broadly represents an important contribution to the proximal suicide risk literature. Specifically, one hindrance to short-term follow-up may be the ability to identify

potentially at-risk participants, and then establish non-burdensome methodology to collect these data frequently. In the current study, we demonstrate that utilizing Amazon's Mechanical Turk (MTurk) may serve as a useful tool for investigating at-risk individuals with a history of suicidal behavior. Of the nearly 2,000 participants that were screened, about half endorsed some history of suicidal behavior (e.g., ideation, intent, attempt). This screening process adds another layer, and thus, more time, while conducting MTurk research, but our study suggests that such a screening process will yield the ability to test hypotheses on an at-risk sample. Moreover, given the high numbers of participants endorsing a history of suicidal behavior, our study highlights other recent findings that MTurk participants endorse clinical symptoms at a much greater degree than traditional nonclinical samples (Arditte, Çek, Shaw, & Timpano, 2016), indicating that the use of an MTurk sample, though not clinical, represents a more pathological sample than might otherwise be available. In addition, we have demonstrated the feasibility of conducting short-term, longitudinal research via this platform as well, which, to our knowledge, has not previously been attempted. Overall, the design of the current study could serve as a model for an expeditious, cost-effective, and feasible method for studying proximal risk for suicide outside the bounds of clinical samples.

Interpretive Caveats

Though the present study implemented many improvements upon existing research, we note several caveats. First, though we assert that MTurk represents an invaluable tool for studying pathological, non-clinical samples, we do note that the sample utilized in current study was comprised mostly of White, female participants. This is not entirely surprising, given our screening inclusion criterion was endorsing a history of suicidal behavior (i.e., ideation, intent, or attempt), and women have a higher lifetime prevalence rate of all forms of suicidal behavior

(Nock et al., 2008). Nevertheless, future studies should explore the hypotheses of the current study in samples with more men and with participants of varied racial backgrounds. Additional studies should also seek to replicate our findings with more severe, clinical samples, and in other samples outside of MTurk, given that MTurk worker pool does not necessarily represent the general population (Arditte et al., 2016).

In addition, though we sought to focus on suicide ideation, given its higher prevalence than other forms of suicidal behavior, and because it is typically a precursor to suicide attempt, we acknowledge that insomnia may differentially relate to suicide attempt as an outcome, rather than ideation. Future research should work on developing models using similar LDS modeling as in the current paper that can account for discrete variables such as suicide attempts; in principle, this should be possible for the same reason that other categorical generalization of linear models (e.g., logistic regression) are extendable to this type of framework, but whether or not this is a tractable goal for more complex models like LDS is worth investigating. Moreover, future studies might consider including different covariates in their models; though we opted only to include depression to reduce strain on the model, it is possible that other covariates might affect the relationship between these outcomes. However, we emphasize that our findings conformed to a priori hypotheses, and emerged as the result of statistically rigorous tests while controlling for a potent risk factor for suicide – depression. Thus, it is unlikely that other risk factors would completely negate the effects of insomnia on suicide ideation. We also point out that we observed relatively low variability in suicide ideation in our sample, indicating that if we were able to detect these effects in a sample with lower risk, these effects might even be amplified in samples with more variability in suicide ideation. Future studies should also seek to explore these relationships over different temporal epochs; though we opted to use a 15-day

measurement period, examining the dynamics between insomnia and suicide ideation over even smaller time periods (e.g., daily or perhaps even hourly assessment) may provide additional valuable information. Lastly, the current study used only self-report measures for feasibility reasons. Thus, it would be helpful for future studies to incorporate objective measures of insomnia, which has been a recurrent suggestion made in other recent longitudinal studies as well as this literature basis grows (e.g., Ribeiro et al., 2012).

Conclusion

The findings of the current paper demonstrate the importance of insomnia in driving changes in suicide ideation, and importantly, revealed that this relationship is unidirectional. Our results converge with the few prior longitudinal studies that exist, highlighting the relationship between insomnia and suicide ideation (e.g., McCall et al., 2010; Ribeiro et al., 2012), but also expanded from these studies by utilizing a short-term longitudinal design, and applying a statistical approach that allowed for exploration of lead-lag (i.e., causal) relationships between these variables and establishment of insomnia as a variable risk factor for suicide ideation. Findings underscore the potential effectiveness of clinical interventions for insomnia in reducing risk for suicide, the utility of screening for insomnia to indirectly infer suicidality, and also suggest that additional research should be conducted to examine insomnia as a potential causal risk factor for suicidal behavior. Finally, this study represents a novel contribution to the proximal risk literature, demonstrating a feasible methodology for improving upon this limited literature and better examining risk factors for suicide over brief, clinically meaningful intervals.

References

Agargun, M. Y., Kara, H., & Solmaz, M. (1997). Sleep disturbances and suicidal behavior in patients with major depression. *Journal of Clinical Psychiatry*, *58*, 249–251.

Arditte, K. A., Çek, D., Shaw, A. M., & Timpano, K. R. (2016). The importance of assessing clinical phenomena in Mechanical Turk research. *Psychological Assessment*, *28*, 684–691.

Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, *2*(4), 297–307. http://doi.org/10.1080/15402002.2011.606766

Bernert, R. A., & Joiner, T. E. (2007). Sleep disturbances and suicide risk: A review of the literature. *Neuropsychiatric Disease and Treatment*, *3*(6), 735–743. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2656315/

Bernert, R. A., Joiner, T. E., Cukrowicz, K., Schmidt, N. B., & Krakow, B. (2005). Suicidality and sleep disturbances. *Sleep*, *28*(9), 1135–41.

Bernert, R. A., Kim, J. S., Iwata, N. G., & Perlis, M. L. (2015). Sleep disturbances as an evidence-based suicide risk factor. *Current Psychiatry Reports*, *17*(3), 1–9. http://doi.org/10.1016/j.jsmc.2014.11.004

Bryan, C. J., Gonzales, J., Rudd, M. D., Bryan, A. O., Clemans, T. A., Ray-Sannerud, B., … Etienne, N. (2015). Depression mediates the relation of insomnia severity with suicide risk in three clinical samples of US Military personnel. *Depression and Anxiety*.

Chellappa, S. L., & Araújo, J. F. (2007). Sleep disorders and suicidal ideation in patients with depressive disorder. *Psychiatry Research*, *153*, 131–136.

Crosby, A., Gfroerer, J., Han, B., Ortega, L., & Parks, S. E. (2011). Suicidal thoughts and

behaviors among adults aged >18 Years: United States, 2008-2009. Retrieved from http://www.cdc.gov

Drapeau, C. W., & McIntosh, J. L. (2014). U.S.A. suicide 2012: Official final data. Retrieved from http://www.suicidology.org

Enders, C. K. (2001). The impact of nonnormality on full information maximum-likelihood estimation for structural equation models with missing data. *Psychological Methods*, *6*, 352–370.

Ferrer, E., & McArdle, J. J. (2010). Longitudinal modeling of developmental changes in psychological research. *Current Directions in Psychological Science*, *19*, 149–154.

Franklin, J. C., Fox, K. R., Ribeiro, J. D., Kleiman, E. M., Harris, J., & Nock, M. K. (2014). Are we getting better at predicting suicidal behaviors? In *Association for Behavioral and Cognitive Therapies*. Philadelphia, PA.

Fujino, Y., Mizoue, T., Tokui, N., & Yoshimura, T. (2005). Prospective Cohort Study of Stress, Life Satisfaction, Self-Rated Health, Insomnia, and Suicide Death in Japan. *Suicide and Life-Threatening Behavior*, *35*(2), 227–237.

Gould, M., Marrocco, F. A., Kleinman, M., Thomas, J. G., Mostkoff, K., Cote, J., & Davies, M. (2005). Evaluating iatrogenic risk of youth suicide screening programs: A randomized controlled trial. *JAMA*, *293*, 1635–143.

Grimm, K. J., An, Y., McArdle, J. J., Zonderman, A. B., & Resnick, S. M. (2012). Recent changes leading to subsequent changes: Extensions of multivariate latent difference score models. *Structural Equation Modeling: A Multidisciplinary Journal*, *19*, 268–292.

Hall, R. C., Platt, D. E., & Hall, R. C. (1999). Suicide risk assessment: A review of risk factors

for suicide in 100 patients who made severe suicide attempts: evaluation of suicide risk in a time of managed care. *Psychosomatics*, *40*(1), 18–27.

Harvey, A. (2008). Insomnia, psychiatric disorders, and the transdiagnostic perspective. *Current Directions in Psychological Science*, *17*, 299–303.

Hu, L., & Bentler, P. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*, 1–55.

Joiner, T. E. (2005). *Why people die by suicide.* Cambridge, MA: Harvard University Press.

Joiner, T. E., Brown, J. S., & Wingate, L. R. (2005). The psychology and neurobiology of suicidal behavior. *Annual Review of Psychology*, *56*, 287–314.

Joiner, T. E., Pfaff, J. J., & Acres, J. G. (2002). A brief screening tool for suicidal symptoms in adolescents and young adults in general health settings: reliability and validity data from the Australian National General Practice Youth Suicide Prevention Project. *Behaviour Research and Therapy*, *40*(4), 471–481. http://doi.org/10.1016/s0005-7967(01)00017-1

Joiner, T. E., Van Orden, K. A., Witte, T. K., & Rudd, M. D. (2009). *The interpersonal theory of suicide: Guidance for working with suicidal client*. Washington, DC: American Psychological Association.

Joiner, T. E., Walker, R. L., Rudd, M. D., & Jobes, D. A. (1999). Scientizing and routinizing the assessment of suicidality in outpatient practice. *Professional Psychology: Research and Practice*, *30*(5), 447–453. http://doi.org/10.1037/0735-7028.30.5.447

Jöreskog, K. (1974). Analyzing psychological data by structural analysis of covariance matrices. In R. Atkinson, D. Krantz, R. Luce, & P. Suppas (Eds.), *Contemporary developments in*

mathematical psychology (pp. 1–56).

Jöreskog, K. (1970). Estimation and testing of simplex models. *British Journal of Mathematical and Statistical Psychology*, *23*, 121–145.

Kline, R. B. (2010). *Principles and practice of structural equation modeling* (3rd ed.). New York: Guilford Press.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, *54*, 337–343.

Löwe, B., Wahl, I., Rose, M., Spitzer, C., Glaesmer, H., Wingenfeld, K., … Brähler, E. (2010). A 4-item measure of depression and anxiety: Validation and standardization of the Patient Health Questionnaire-4 (PHQ-4) in the general population. *Journal of Affective Disorders*, *122*(1), 86–95.

McCall, W. V., Blocker, J. N., D'Agostino, R., Kimball, J., Boggs, N., Lasater, B., & Rosenquist, P. B. (2010). Insomnia severity is an indicator of suicidal ideation during a depression clinical trial. *Sleep Medicine*, *11*(9), 822–827.

Metalsky, G. I., & Joiner, T. E. (1997). The Hopelessness Depression Symptom Questionnaire. *Cognitive Therapy and Research*, *21*, 359–384.

Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, *34*(5), 601–608.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: update of the recent evidence

(1998-2004). *Sleep*, *29*, 1398–1414.

Muthén, L. K., & Muthén, B. O. (2012). *Mplus User's Guide* (7th ed.). Los Angeles, CA: Muthén & Muthén.

Nock, M., Borges, G., Bromet, E., Alonso, J., Angermeyer, M., Beautrais, A., … Williams, D. (2008). Cross-national prevalence and risk factors for suicidal ideation, plans and attempts. *British Journal of Psychiatry*, *192*, 98–105.

Nrugham, L., Larsson, B., & Sund, A. M. (2008). Specific depressive symptoms and disorders as associates and predictors of suicidal acts across adolescence. *Journal of Affective Disorders*, *111*(1), 83–93.

O'Connor, R. C., & Nock, M. K. (2014). The psychology of suicidal behaviour. *The Lancet*, *1*, 73–85.

Ohayon, M. M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews*, *6*, 97–111.

Ohayon, M. M., Caulet, M., & Lemoine, P. (1998). Comorbidity of mental and insomnia disorders in the general population. *Comprehensive Psychiatry*, *39*, 185–197.

Ohayon, M. M., Shapiro, C. M., & Kennedy, S. H. (2000). Differentiating DSM-IV anxiety and depressive disorders in the general population: Comorbidity and treatment consequences. *The Canadian Journal of Psychiatry*, *45*, 166–172.

Perlis, M. L., Smith, L. J., Lyness, J. M., Matteson, S. R., Pigeon, W. R., Jungquist, C. R., & Tu, X. (2006). Insomnia as a risk factor for onset of depression in the elderly. *Behavioral Sleep Medicine*, *4*, 104–113.

Pigeon, W. R., Britton, P. C., Ilgen, M. A., Chapman, B., & Conner, K. R. (2012). Sleep

disturbance preceding suicide among veterans. *American Journal of Public Health*, *102*(S1), S93–S97.

Pigeon, W. R., & Caine, E. D. (2010). Insomnia and the risk for suicide: Does sleep medicine have interventions that can make a difference? *Sleep Medicine*, *11*(9), 816–817.

Pigeon, W. R., Pinquart, M., & Conner, K. (2012). Meta-analysis of sleep disturbance and suicidal thoughts and behaviors. *The Journal of Clinical Psychiatry*, *73*(9), e1160–e1167. http://doi.org/10.4088/jcp.11r07586

Ployhart, R., & MacKenzie, W. (2014). Two waves of measurement do not a longitudinal study make. In C. Lance & R. Vandenberg (Eds.), *More statistical and methodological myths and urban legends* (New York). Routledge.

Reynolds, S. K., Lindenboim, N., Comtois, K. A., Murray, A., & Linehan, M. M. (2006). Risky assessments: Participant suicidality and distress associated with research assessments in a treatment study of suicidal behavior. *Suicide and Life-Threatening Behavior*, *36*, 19–34.

Ribeiro, J. D., Pease, J. L., Gutierrez, P. M., Silva, C., Bernert, R. A., Rudd, M. D., & Joiner, T. E. (2012). Sleep problems outperform depression and hopelessness as cross-sectional and longitudinal predictors of suicidal ideation and behavior in young adults in the military. *Journal of Affective Disorders*, *136*(3), 743–750.

Roth, T. (2007). Insomnia: Definition, prevalence, etiology, and consequences. *Journal of Clinical Sleep Medicine*, *4*(S5), S7.

Rudd, M. D., Berman, A. L., Joiner, T. E., Nock, M. K., Silverman, M. M. Mandrusiak, M., Van Orden, K., & Witte, T. K. (2006). Warning signs for suicide: Theory, research, and clinical applications. *Suicide and Life-Threatening Behavior*, *36*, 255–262.

Rudd, M. D., Joiner, T. E., & Rajab, M. H. (2004). *Treating suicidal behavior: An effective, time-limited approach*. New York: Guilford Press.

Satorra, A., & Bentler, P. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika*, *66*, 507–514.

Shapiro, D. N., Chandler, J., & Mueller, P. A. (2013). Using Mechanical Turk to study clinical populations. *Clinical Psychological Science*, *1*, 213–220.

Sjöström, N., Waern, M., & Hetta, J. (2007). Nightmares and sleep disturbances in relation to suicidality in suicide attempters. *Sleep*, *30*(1), 91–95.

Wojnar, M., Ilgen, M. A., Wojnar, J., McCammon, R. J., Valenstein, M., & Brower, K. J. (2009). Sleep problems and suicidality in the National Comorbidity Survey Replication. *Journal of Psychiatric Research*, *43*(5), 526–531.

Woznica, A. A., Carney, C. E., Kuo, J. R., & Moss, T. G. (2015). The insomnia and suicide link: toward an enhanced understanding of this relationship. *Sleep Medicine Reviews*, *22*, 37–46.

Appendix 1: Tables

Table 1. Descriptive statistics for analysis variables

	N	Univariate Statistics					
		Mean	Std. Dev.	Min.	Max.	Skewness	Kurtosis
Suicide Ideation							
Wave 1	585	1.415	2.018	0.000	12.000	1.457	2.074
Wave 2	468	0.801	1.751	0.000	12.000	2.605	7.947
Wave 3	461	0.664	1.578	0.000	12.000	3.165	13.344
Wave 4	424	0.700	1.747	0.000	12.000	3.317	13.726
Wave 5	455	0.747	1.723	0.000	12.000	2.956	10.879
Wave 6	429	0.699	1.763	0.000	12.000	3.369	13.633
Insomnia							
Wave 1	581	11.296	6.287	0.000	28.000	0.293	-0.513
Wave 2	469	9.431	6.093	0.000	28.000	0.668	0.012
Wave 3	461	9.534	6.407	0.000	28.000	0.661	-0.045
Wave 4	423	9.099	6.450	0.000	28.000	0.669	-0.071
Wave 5	455	9.308	6.585	0.000	28.000	0.616	-0.130
Wave 6	429	9.179	6.748	0.000	28.000	0.697	0.032
Depression							
Wave 1	581	2.430	1.936	0.000	6.000	0.489	-0.819
Wave 2	468	1.893	1.922	0.000	6.000	0.783	-0.509
Wave 3	461	1.892	1.892	0.000	6.000	0.785	-0.460
Wave 4	423	1.920	2.016	0.000	6.000	0.785	-0.624
Wave 5	454	1.817	1.954	0.000	6.000	0.889	-0.349
Wave 6	429	1.918	2.012	0.000	6.000	0.811	-0.554

Note. All descriptive statistics computed in SPSS Version 21.

Table 2. Intercorrelations for analysis variables

		Correlations																	
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	Suicide Ideation W1	1																	
2	Suicide Ideation W2	.683	1																
3	Suicide Ideation W3	.595	.716	1															
4	Suicide Ideation W4	.596	.679	.765	1														
5	Suicide Ideation W5	.651	.712	.694	.791	1													
6	Suicide Ideation W6	.566	.689	.616	.712	.756	1												
7	Insomnia W1	.230	.231	.157	.167	.166	.211	1											
8	Insomnia W2	.234	.273	.190	.240	.229	.274	.837	1										
9	Insomnia W3	.237	.252	.263	.279	.225	.228	.758	.839	1									
10	Insomnia W4	.197	.237	.169	.223	.188	.199	.743	.810	.851	1								
11	Insomnia W5	.156	.228	.143	.196	.211	.237	.704	.809	.812	.848	1							
12	Insomnia W6	.172	.258	.166	.237	.239	.265	.709	.795	.791	.834	.857	1						
13	Depression W1	.498	.397	.353	.339	.360	.313	.392	.403	.396	.358	.304	.322	1					
14	Depression W2	.471	.489	.434	.381	.410	.404	.343	.422	.380	.377	.367	.377	.763	1				
15	Depression W3	.460	.430	.452	.437	.380	.373	.280	.365	.398	.370	.306	.336	.711	.770	1			
16	Depression W4	.427	.443	.407	.459	.377	.357	.267	.372	.424	.400	.356	.358	.684	.709	.770	1		
17	Depression W5	.393	.383	.376	.421	.446	.402	.286	.388	.402	.393	.431	.440	.658	.699	.765	.792	1	
18	Depression W6	.405	.446	.380	.429	.407	.441	.324	.412	.449	.423	.411	.500	.660	.707	.729	.779	.818	1

Note. All correlations computed in SPSS Version 21. All correlations are significant at $p < .01$. W1 = Wave 1; W2 = Wave 2; W3 = Wave 3; W4 = Wave 4; W5 = Wave 5; W6 = Wave 6.

Table 3. Fit statistics for univariate latent difference score models fit to (A) Insomnia and (B) Suicide ideation

(A) Insomnia

	Constant Change	Dual Change	Dynamic Change*
χ^2	155.128	96.728	78.057
df	22.000	21.000	20.000
RMSEA	.102	.079	.071
RMSEA 90% CI	.087-.117	.063-.095	.055-.087
CFI	.921	.955	.965
TLI	.946	.968	.974
SRMR	.064	.039	.035
χ^2 Difference[a]		63.254	38.530
χ^2 Diff p value		< .001	< .001

(B) Suicide ideation

	Constant Change	Dual Change*	Dynamic Change
χ^2	145.989	86.081	111.691
df	22.000	21.000	20.000
RMSEA	.098	.073	.088
RMSEA 90% CI	.083-.113	.057-.089	.073-.105
CFI	.882	.938	.913
TLI	.919	.956	.934
SRMR	.086	.195	.168
χ^2 Difference[a]		29.635	.260
χ^2 Diff p value		< .001	.610

Note. [a]χ^2 difference results compare model to preceding model using Sattora-Bentler Scaled Chi-Square Difference test. Value provided is Chi-square difference value from this test. *Denotes selected univariate model for each variable based on best fit.

Table 4. Parameter estimates for selected univariate models for (A) Insomnia and (B) Suicide Ideation

(A) Insomnia (Dynamic Change Model)

	Estimate	SE	p
μ_{y0}	10.906	.252	<.001
μ_s	--	--	--
β	-.066	.008	<.001
ϕ	-.610	.064	<.001

(B) Suicide Ideation (Dual Change Model)

	Estimate	SE	p
μ_{y0}	.867	.075	<.001
μ_s	--	--	--
β	-.728	.089	<.001

Note. μ_y0 = mean intercept; β = proportional change parameter; ϕ = dynamic change parameter. Mean slope (μ_s) was constrained to zero in both models and thus, this parameter was not estimated.

Table 5. Fit statistics for bivariate models

	Model 1	**Model 2**	**Model 3***	**Model 4**	**Model 5**	**Model 6**	**Model 7**
χ^2	220.670	220.026	212.952	212.361	216.833	211.279	213.175
df	108	107	107	106	105	105	104
RMSEA	.042	.042	.041	.041	.043	.041	.042
RMSEA 90% CI	.034-.050	.034-.050	.033-.049	.033-.049	.034-.051	.033-.050	.034-.050
CFI	.974	.974	.976	.976	.974	.976	.975
TLI	.967	.966	.969	.968	.966	.968	.967
SRMR	.031	.031	.026	.026	.027	.026	.026
AIC	33805.467	33807.001	33798.207	33799.821	33801.349	33800.676	33801.362
BIC	34160.119	34166.032	34157.238	34163.230	34169.137	34168.464	34173.529
BIC Difference[a]	--	5.913	-8.794	5.992	5.907	-.673	5.065

Note. Model 1: Constant change with no coupling; Model 2: Dual change model with unidirectional coupling (SUI --> ΔINS); Model 3: Dual change model with unidirectional coupling (INS --> ΔSUI); Model 4: Bidirectional coupling with both proportional coupling parameters included (SUI -->ΔINS; INS -->ΔSUI); Model 5: Model 4 plus unidirectional dynamic change parameter (ΔSUI --> ΔINS); Model 6: Model 4 plus unidirectional dynamic change parameter (ΔINS --> ΔSUI); Model 7: All parameters included in full coupling model. INS = Insomnia; SUI = Suicide ideation. [a]BIC Difference values reflect BIC value in current model minus BIC value from previous model. As such, negative values here reflect improvement in fit. *Denotes selected bivariate model.

Table 6. Parameter estimates for selected bivariate model

	Suicide Ideation (SUI)			Insomnia (INS)		
	Estimate	**SE**	**p**	**Estimate**	**SE**	**p**
μ_{y0}	.742	.377	.049	9.775	.402	<.001
μ_s	--	--	--	--	--	--
β	-.719	.104	<.001	-.073	.020	<.001
ϕ	--	--	--	-.521	.091	<.001
γ	.020	.008	.012	--	--	--

Note. Parameters correspond with model in Equations 4 and 5. μ_y0 = mean intercept; β = proportional change parameter; ϕ = dynamic change parameter; γ = proportional change coupling parameter. Mean slope (μ_s) for both outcomes (SUI and INS) was constrained to zero. As described in text, ϕ was not estimated for SUI, and γ was not estimated for INS.

Appendix 2: Figures

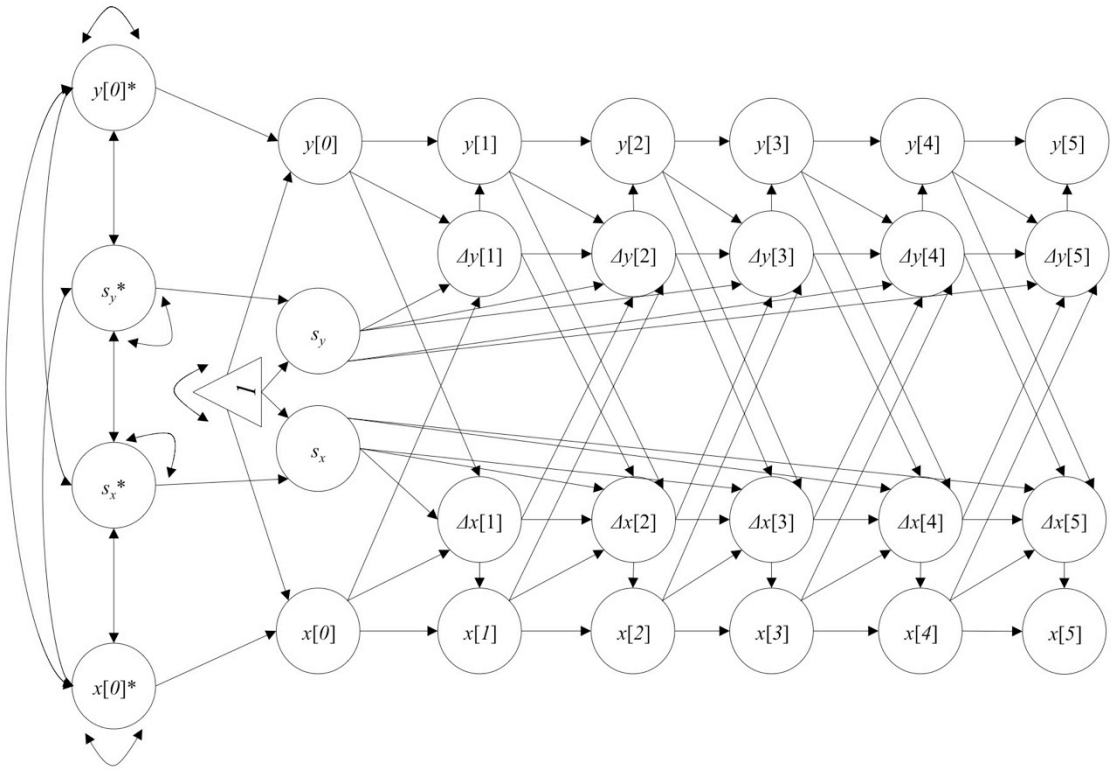

Figure 1. Path diagram of the full coupling model from Equation 1 with six measurement occasions and two processes. For process *y*, latent true scores after the initial true score ($y[1]$-$y[5]$) receive inputs from the prior true score and the corresponding latent difference score ($\Delta y[1]$-$\Delta y[5]$). Regression weights for the latent difference scores are equal to 1, which means that each true score is the sum of the prior true score and latent difference score. For the latent difference scores following $\Delta y[1]$, each of these scores have five predictors: a constant (s_y), the prior true score of the same process (corresponding to β_y in Equation 1), the prior true score of the second process (γ_{yx} from Equation 1), the prior latent different score of the same process (ϕ_y from Equation 1), and the prior latent difference score of the second process (ξ_{yx} from Equation 1). Note, observed variables and their residuals are not depicted in this diagram.

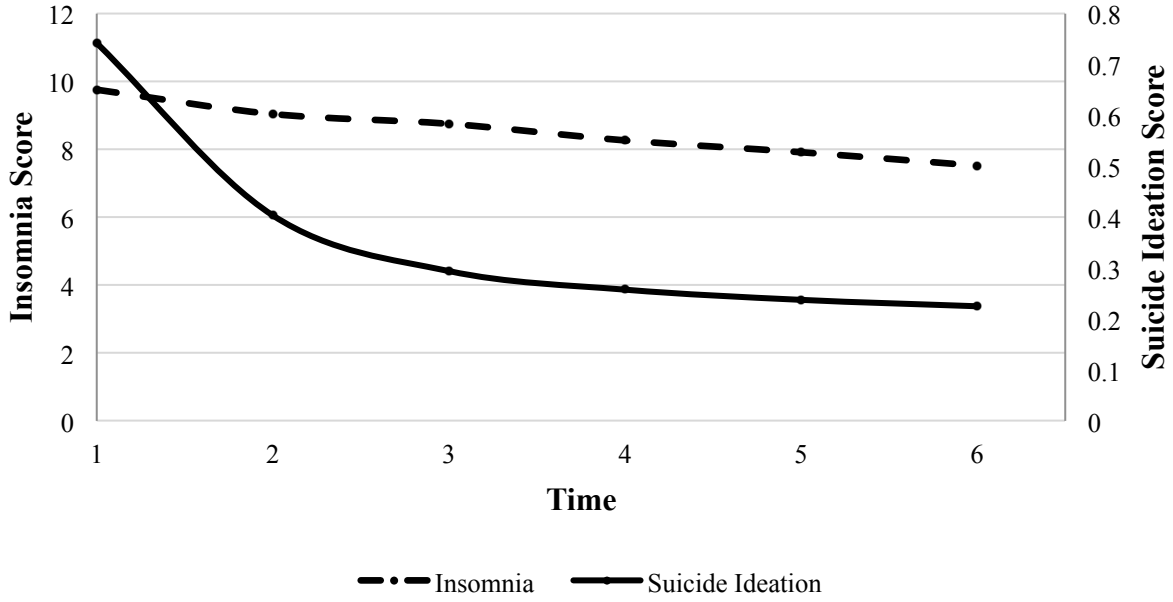

Figure 2. Visual depiction of bivariate model with no perturbations in system. Insomnia and suicide ideation symptoms over measurement period (15 days) for hypothetical participant who begins with average levels of insomnia and suicide ideation (i.e., the intercepts). This figure depicts the projected trajectory of an individual experiencing average levels of both insomnia and suicide ideation at Wave 1, with no additional input into the system (i.e., no additional measurement time points).

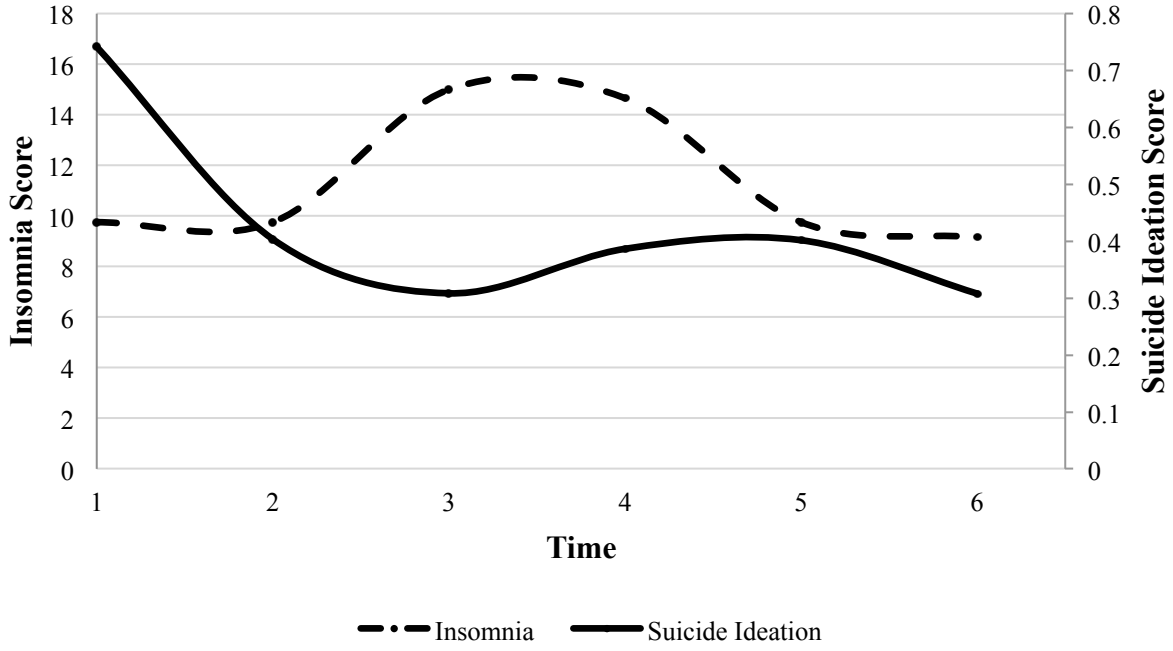

Figure 3. Visual depiction of bivariate model with perturbations in system. Insomnia and suicide ideation symptoms over measurement period (15 days) for hypothetical participant who begins with average levels of insomnia and suicide ideation (i.e., the intercepts). This figure depicts the projected trajectory of an individual experiencing average levels of both insomnia and suicide ideation at Wave 1, who then experiences an increase in insomnia symptoms at Wave 3. As can be seen in the figure, after this forced increased in insomnia, positive changes in suicide ideation at Wave 4 can be observed, as the model would predict. After level of insomnia returns to average at Wave 5, positive changes in suicide ideation are smaller, and no longer observable due to concurrent univariate effects of the previous level of suicide ideation (i.e., regression to the mean). This figure portrays the impact of a significant increase in insomnia symptoms on changes in suicide ideation, followed by the system returning to its previous state after levels of insomnia return to average.