Sleep and Sadness: Exploring the Relation Among Sleep, Cognitive Control and Depressive Symptoms in Young Adults

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Abstract

Background: Sleep disturbance is a common feature of depression. However, recent work has found that depression-vulnerable people, compared to their low-risk counterparts, report poorer sleep quality, suggesting that sleep disturbance may precede depression. In addition, both sleep disturbance and depression are related to deficits in cognitive control processes. Thus, we examined whether poor sleep quality predicts subsequent increases in depressive symptoms and whether levels of cognitive control mediated this relation. Methods: Thirty-five undergraduates participated in two experimental sessions separated by three weeks. Participants wore an actigraph watch between sessions, which provided an objective measure of sleep patterns. We assessed self-reported sleep quality and depressive symptoms at both sessions. Last, individuals completed an exogenous cuing task, which measured ability to disengage attention from neutral and negative stimuli, during the second session. Results: Using path analyses, we found that both greater self-reported sleep difficulty and more objective sleep stability measures significantly predicted greater difficulty disengaging attention (i.e., less cognitive control) from negative stimuli. Less cognitive control over negative stimuli, in turn, predicted increased depression symptoms at the second session. Exploratory associations among a CLOCK
polymorphism, rs11932595, sleep assessments, and depressive symptoms are also presented. **Conclusions:** These preliminary results suggest that sleep disruptions may contribute to increases in depressive symptoms via their impact on cognitive control. Further, variation in the CLOCK gene may be associated with sleep quality.

**Sleep and Sadness: Exploring the Relation Among Sleep, Cognitive Control and Depressive Symptoms in Young Adults**

Sleep disturbance is a common feature of depression. Some estimates suggest that greater than 90% of individuals with major depression also report insomnia or sleep disturbance. Further, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) includes sleep impairment (insomnia or hypersomnia) as one of nine symptoms of a Major Depressive Episode (MDE). Non-clinical sleep disturbances in depression have also been documented empirically. In a meta-analysis on sleep and psychiatric disorders, individuals with an affective disorder show reduced total sleep time, reduced sleep efficiency and increased sleep latency compared to healthy controls.

In addition to its presence during a MDE, recent research has found that sleep disturbance may precede the disorder. For example, Chen, Burley and Gotlib compared girls at high and low risk for depression on subjective and objective measures of sleep. Girls’ risk status was defined by their mothers’ depression history (either no history of depression or recurrent depression). While the two groups did not differ on any objective measures (sleep duration, onset latency, snooze duration), high-risk girls reported poorer sleep quality than their low-risk counterparts. Similarly, Baglioni and colleagues found that non-depressed individuals with insomnia are two times more likely to develop depression than non-depressed individuals without insomnia. Finally, a recent examination of a gene commonly associated with risk for depression (5HTTLPR) demonstrated that 1st year college students with the variant of 5HTTLPR associated with lower expression of serotonin transporter had greater depression symptoms and shorter sleep duration. Taken together, sleep difficulties may serve as a phenotype of risk for depression.

It is important to note that there is considerable variation in the types of sleep disturbances present before and during depression. As reviewed, differences in subjective (e.g., sleep quality) and objective measures (e.g., hours of sleep) of sleep between depressed and non-depressed individuals have been documented. Further, there is support that clinical levels of sleep disturbance are predictive of depression onset and that non-clinical sleep disturbance characterizes at-risk populations. Altogether, depression and risk for depression seems to include disturbances in sleep quality and real-world sleep patterns. It is possible, however, that negative reporting biases that precede depression onset may account for differences in self-reported sleep quality between depression risk groups. Therefore, it is important that any study of sleep and depressive symptoms use multiple methods to assess both subjective perceived sleep quality and objective sleep patterns.

**Measurement of Sleep**

Recent research has utilized naturalistic methods, such as actigraphy, to measure real-world sleep patterns. Actigraph devices use an accelerometer to measure movement across time and researchers can use movement data to infer periods of sleep and wakefulness. Actigraphy eliminates inevitable biases associated with subjective measures (e.g., reporting and memory biases). Further, as opposed to sleep deprivation methodologies, this method is intended to not disrupt sleep but instead provide an objective measure of naturalistic sleep patterns. The current
study is among few that have utilized this novel tool to explore the relation between sleep and depressive symptoms.

In addition to measuring traditional sleep measures such as total sleep time, actigraphy can also provide measures of circadian rhythms. Especially in the cognition literature, there is evidence that circadian rhythm measures account for outcomes above and beyond those accounted for by average sleep duration or changes in sleep duration. And while some have conceptualized depression as a disorder of disrupted circadian rhythms, research on the relation between sleep-wake cycles and depression is lacking. Thus, in addition to subjective measures of sleep quality, the current study used actigraphy to obtain traditional and circadian sleep measures.

**Sleep Disruption and Cognitive Control**

Using the approaches described above, the current study sought to investigate the underlying mechanisms by which sleep difficulties may contribute to depression symptomatology. One possibility is that sleep disturbance influences depression symptoms via its impact on cognition. Indeed, research indicates that sleep disruption influences how individuals regulate emotions and interpret emotional stimuli, both of which have also been linked to depression. In addition, sleep difficulties and depression have been related to deficits in cognitive control separately. For example, sleep deficits negatively impact mental flexibility, working memory, and inhibition. Similarly, Nebes and colleagues found that, among healthy older adults, poor sleep quality was associated with worse performance on working memory and attentional shifting tasks.

Depression has also been linked to deficits in cognitive control, particularly control over emotional information. For example, depressed individuals exhibit difficulty inhibiting negative information from entering attention and working memory and, once engaged, they demonstrate difficulty disengaging attention from negative information. Further, negative attentional biases prospectively predict increases in depression. One goal of the present study is to examine whether the cognitive changes associated with sleep difficulties contribute to poor cognitive control over emotion stimuli, which in turn has been linked to the development and maintenance of depression.

**Current Study**

The current study recruited a sample of undergraduate students to explore the relation among sleep, cognitive control and depressive symptoms. First, we were interested in the relation between sleep and depression symptoms. In line with previous findings reporting that poor sleep quality and reduced sleep time precede depression, we hypothesized that poorer sleep quality and reduced sleep duration would predict increases in depressive symptoms. Second, as previous research has linked both sleep and depression to deficits in cognitive control, we hypothesized that cognitive control over negative stimuli would mediate the relation between sleep quality and change in depression symptoms. To test these hypotheses, we used path analyses—an analytic approach that allowed us to test these associations simultaneously within a single theoretically informed statistical model. As a final, exploratory objective of the study, we explored whether variation in a single nucleotide polymorphism (SNP) in the *CLOCK* gene relates to our primary variables of interest (sleep, cognitive control and depressive symptoms). *CLOCK* SNP rs11932595 was chosen because it has previously been linked to variation in daily sleep time.

**Methods**

**Participants**
Researchers posted flyers at The University of Texas at Austin advertising a study on sleep and cognition. Current undergraduate students at The University of Texas at Austin who were at least 18 years old were eligible for the study. Individuals who responded to the flyers via e-mail received a written description of the study. Individuals who remained interested in the study and who met inclusion criteria were scheduled for the first laboratory session.

Fifty-two individuals (22 women and 28 men) were originally enrolled into the study. One man and one woman failed to complete the second session and, consequentially, were not included in our sample. We removed 13 individuals who were missing greater than ten percent of their actigraph data, one individual who did not complete the emotional cuing task and one individual with missing actigraph and emotional cuing task data. Therefore, we used a sample of 35 individuals (14 women and 21 men) who had complete data across all measures in our analyses. Participants were on average 19.83 years old (SD = 1.25; range 18-23 years). Individuals received $80 for their participation in the study ($20 after completion of the first session; $60 after the second). The Institutional Review Board of the University of Texas at Austin approved all study procedures and participants provided both verbal and written informed consent.

Materials

Questionnaires. Participants completed three questionnaires: a demographics and health questionnaire, the Center for Epidemiologic Studies Depression Scale (CES-D) and the Pittsburgh Sleep Quality Index (PSQI).

We used the CES-D to assess participants’ depressive symptoms. The CES-D is a 20-item self-report measure of depressive symptoms across the past week. The CES-D has an internal consistency of 0.85 in the general population and 0.90 in patient samples. It has moderate test-retest reliabilities, ranging from 0.45 to 0.70. Higher reliability is obtained when tested across shorter time periods. Last, the CES-D has been found to correlate moderately with clinical interviewer ratings of depression.

We used the PSQI as a subjective measure of sleep quality. The PSQI is a 19-item self-report measure of sleep quality and disturbances across the past month. Scores on individual items generate seven component scores that, together, yield a global score of sleep quality. In this study, we used the global measure of sleep quality. The PSQI has an internal reliability coefficient of 0.83 and a test-retest reliability of 0.85 across approximately one month.

Actigraphy. We used Motionlogger Actigraphs (Ambulatory Monitoring, Inc., Ardsley, NY) to objectively measure real-world sleep patterns. Motionlogger Actigraphs are watch-like actigraphs that are worn on the wrist. Participants were instructed to continuously wear an actigraph for the three weeks that separated the first and second laboratory session. They were instructed to only remove the watch during situations where it could get wet or damaged. Motionlogger Actigraphs contain an internal accelerometer that generates a signal when motion is detected across three planes. Depending on the time resolution set, the actigraph surveys the number of summated voltages generated per unit time. For the current study, we examined 60-second epochs of movement data continuously across the 3-week period. In addition, the Motionlogger Actigraph measures epidermal microvibrations to generate an index (LIFE) of whether someone is wearing the watch at any given time. We removed data where the LIFE measure indicated the actigraph was off for periods greater than 30 minutes (missing data). As previously stated, individuals missing more than ten percent of actigraph data were excluded from our sample.
Actigraph data generation. From the raw actigraph data, a number of measures were generated across the three week inter-session period: two measures of sleep (average sleep duration and sleep standard deviation) and two circadian rhythm variables (described below). It is important to note that an actigraph does not measure sleep directly; it measures motor activity from which sleep/wake periods may be inferred. We used the Cole-Kripke PCD ZCM algorithm with an epoch length of one minute via Action 4 software (Ambulatory Monitoring, Inc., Ardsley, NY) to estimate sleep periods from activity data. This algorithm is widely used among automatic actigraphical sleep scoring. Output from the algorithm defines each minute as either a period of sleep or wakefulness. From this, we calculated the mean and SD of 24-hour “sleep” periods across three weeks resulting in an estimate of average daily sleep and its standard deviation (SD) across the 3-week period.

To examine aspects of circadian rhythms, we applied the non-parametric circadian rhythm analysis provided as part of the Active 4 software. Two measures were generated. The first, interdaily stability (IS), reflects the degree to which individuals have a stable sleep-wake rhythm between 24 hour periods and is the ratio between the variance of the average 24-hour pattern around the mean and the total variance. This calculation will range from 0 to 1 where higher values indicate a more consistent sleep/wake schedule. We also calculated intradaily variability (IV). The IV is the period of rest and sleep activity that is fragmented. It is calculated as a ratio of the mean squares of the difference between successive hours (first derivative) and the mean squares around the grand mean (overall variance). The value will indicate how often and to what extent there are transitions between rest and activity. The calculation will range from 0 to 2 with higher numbers indicating more fragmented rhythms. These measures have previously been used to reveal differences in motor activity between schizophrenic and depressed individuals.

Emotional Cuing Task (ECT). Originally developed by Posner, the emotional cuing task has since been modified to incorporate emotional stimuli. As such, this task measures individuals’ ability to disengage from irrelevant but emotionally salient stimuli. Each trial began with a fixation cross presented at the center of the computer screen. Fixation cross presentation varied from 2000 to 3000 ms. Next, either a neutral or sad face (cue) appeared on the left or right side of the visual field. These cues were presented for 1000 ms. After the cue disappeared, either a single or double asterisk (target) appeared in the same location as the cue (valid cue trial) or on the opposite side of visual field (invalid cue trial) for 250 ms. Participants were instructed to identify target type as quickly and accurately as possible. Specifically, participants were told to press the “1” key if one asterisk appears and the “2” key if two asterisks appear, and participants had 2000 ms to respond. Figure 1 provides an example of a valid and invalid cue trial. Cue stimuli were faces from the Karolinska Directed Emotional Faces (KDEF) set. We converted images into black and white, cropped the images around the facial expression to remove any distracting and extraneous features (e.g., hair, ears), and fit into an oval with a height of 205 pixels and a width of 155 pixels (see Figure 1). There were 48 sad face cue and 48 neutral face cues.

The main task was composed of 192 trials (128 valid cue trials; 64 invalid cue trials). Cues and targets were with equal frequency on the left and right side of the screen. Each cue was presented twice, and cue stimuli were randomized across trials. Participants first completed ten practice trials of the task. Participants must have correctly identified the target on eight of the ten trials to advance to the main task. Individuals who failed to correctly identify the target on at
least eight trials had to repeat the set of ten practice trials before they could advance to the main
task.

To assess cognitive control, we computed a cue validity score for each individual for
each cue type using the following formula: Cue Validity (CV) = Mean RT (Invalid trials) – Mean
RT (Valid trials). Positive CV scores putatively reflect difficulty disengaging attention from
invalid cues to identify the target on opposite side of visual field. Lower CV scores reflect
relatively little difficulty with attention disengagement. We focused on difficulty with
disengagement from sad cues, as this is the primary cognitive control deficit typically observed
in depression.\footnote{39}

**Genotyping.** Ethanol precipitation was used to extract DNA from collected saliva
samples. Samples were genotyped using a MassEXTEND Sequenom assay based on the
annealing of an oligonucleotide primer adjacent to the SNP of interest. The addition of DNA
polymerase along with a mixture of terminator nucleotides allows extension of the primer
through the polymorphic site and generates allele-specific extension products, each having a
unique molecular mass. The resultant masses of the extension products are then analyzed by
matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS)
and a genotype is assigned. The assay was performed in multiplex with 27 reactions in a single
well. Primer sequences for rs11932595 are as follows: PCR Primer 1 (forward):
ACGTGGATGATCTCATCCATCTTATGTC, PCR Primer 2 (reverse):
ACGTGGATGAGACTGGTGAACACTGTAGC, Extension Primer: ttGTTTAGACCCCTGCC. The
genotyping fail rate was 2.80\% (n=1) for rs11932595 for all included participants. Genotypes
were determined by investigators blinded to phenotypic data.

**Procedure**

The current study consisted of two experimental sessions, which were separated by three
weeks. In addition to the measures discussed here, participants also completed a number
of cognitive and affective measures both at baseline and after the 3-week period. A list of these
additional measures can be made available by the authors upon request. During the first
experimental session (Time 1), the study was described to participants who then provided
informed consent, completed a set of questionnaires (demographics and health questionnaire,
CES-D, and PSQI) and were equipped with an actigraph watch. Between the two experimental
sessions, participants continuously wore the actigraph and completed a daily sleep log where
they reported the duration of each sleep period. Following this 3-week period, participants were
scheduled for a second experimental session (Time 2) where they completed the CES-D, the
PSQI, and the ECT. Participants also returned their actigraph watch and gave two 2-mL saliva
samples for genotyping. The first laboratory session lasted approximately 30 minutes and the
second session lasted approximately 90 minutes. Individuals were compensated, debriefed and
thanked for their participation.

**Results**

**Descriptive statistics.** Means, standard deviations and correlations among the relevant
variables are presented in Table 1. Depressive symptoms and self-reported sleep difficulties were
moderately correlated. Total sleep was moderately associated with depression symptoms at time
two but fell short of statistical significance (p = .08). Sleep stability (IS), fragmentation (IV) and
variability (SD) were weakly associated with depressive symptoms at both time points.
Cognitive control over negative stimuli was associated with depressive symptoms at Time 2,
self-reported sleep difficulties and sleep stability. Mean depressive symptoms were relatively
stable from Time 1 to Time 2, as there was only a point difference in CESD at Time 1 and Time 2.

Path analyses. Path analysis was used to test hypotheses about relations between sleep, cognitive control and depressive symptoms. Specifically, we examined whether self-reported sleep difficulties and sleep stability contribute to altered cognitive control over negative stimuli, which in turn predicts change in depressive symptoms. Several indices are often used to determine quality of path model fit. Among the most commonly used are: $\chi^2$, Comparative Fit Index (CFI), and Root Mean Squared Error of Approximation (RMSEA). Model fit that includes CFI ≥ .90 and RMSEA ≤ .10 is generally acceptable. These criteria were used in the present study.

The initial path model posited direct effects between self-reported sleep difficulties and sleep stability to cognitive control over negative stimuli. A direct effect between cognitive control over negative stimuli and depressive symptoms at Time 2, controlling for depression symptoms at Time 1, was also estimated. This model is presented in Figure 2 and provided good fit to the data $\chi^2 (df = 2) = 0.95, p = .62, \text{RMSEA} = .00, \text{CFI} = 1.00$. Greater sleep difficulty and more sleep stability both significantly predicted greater difficulty disengaging attention (i.e., less cognitive control) from negative stimuli. Less cognitive control over sad stimuli, in turn, predicted greater depressive symptoms at the 3-week time point controlling for baseline depressive symptoms. Depressive symptoms at Time 1 were not strongly correlated with cognitive control three weeks later, but they were strongly correlated with Time 2 depressive symptoms.

A separate nested model found that removing the path from cognitive control over negative stimuli to Time 2 depressive symptoms significantly degraded model fit $\Delta \chi^2 (df = 1) = 12.06, p < .001$. This suggests that the initial model should be retained and, more importantly, cognitive control over emotion stimuli provides significant prediction of Time 2 depressive symptoms beyond the contribution of baseline depressive symptoms.

Finally, we tested for indirect effects among associations in the initial model for which there was a possible intervening (or mediating) variable. For Time 2 depressive symptoms, there was a marginally significant indirect effect for self-reported sleep difficulty ($p = .07$) and a significant indirect effect for sleep stability ($p = .014$). Cognitive control over negative stimuli was the intervening variable for these indirect effects. Taken together, these data suggest that sleep quality and circadian rhythm measures (self-reported and objectively measured, respectively) over a three-week period can contribute to increases in depression symptoms via its impact on cognitive control (see Figure 2).

Exploratory SNP analyses

Individuals were genotyped for polymorphisms in CLOCK SNP rs11932595. Genotype data was missing for one participant so the following analyses reflect an N of 34. Given the association between the CLOCK gene and sleep, we were primarily interested in the association between CLOCK gene variation and sleep variables but we also included other variables for exploratory analyses. For correlation analyses, rs11932595 was coded to reflect number of G alleles (i.e., 0, 1, 2). rs11932595 was not associated with any objective measures of sleep, cognitive control or depressive symptoms at either time point. However, number of G alleles in rs11932595 was positively associated with self-reported sleep difficulty. Correlations between rs11932595, self-reported sleep quality, cognitive control and depressive symptoms are presented in Table 1.
Discussion

We explored the relation among sleep, cognitive control and depressive symptoms in a two-session study with a sample of undergraduate students. We had three aims in this study: (1) to examine the relation between sleep and depressive symptoms; (2) to test an integrative model that links sleep, cognitive control and change in depressive symptoms; and (3) to explore the relation among the CLOCK gene, sleep, cognitive control and depressive symptoms.

Our first aim was to examine the relation between sleep and depressive symptoms. Prior research has shown that poor sleep quality may precede depression. Therefore, we expected that sleep duration and quality would predict changes in depressive symptoms. In line with our prediction, self-reported sleep quality between the two laboratory sessions predicted depressive symptoms at Time 2 after controlling for depressive symptoms at Time 1. Specifically, poorer sleep quality was associated with greater increases in depressive symptoms. This finding supports those of Chen and colleagues, suggesting that sleep disturbance is not solely a by-product of depression but, instead, may precede the disorder.

While clinically reduced sleep (i.e., insomnia) has also been found to predict depression, we did not find a relation between sleep duration and depression symptoms. However, it is possible that our negative finding may be a result of our non-clinical sample. We did not recruit individuals based on defined sleep criteria and, as expected, there was a fair amount of variability in participants’ amount of daily sleep. However, few, if any, of our participants would meet objective criteria for insomnia. Previous research has found good correspondence between PSQI global scores greater than 8 and insomnia. Only one participant in our sample reported a PSQI score greater than 8. Thus, insomnia, as opposed to variation in sleep duration, may be a unique predictor of depression symptoms. Consistent with Chen et al., although depression risk groups differed in subjective sleep ratings, there were no differences in sleep duration.

Our second aim was to investigate possible mechanisms through which sleep patterns might contribute to depressive symptoms. Disturbed sleep has been found to contribute to emotion dysregulation and interpretation biases, abnormalities that are often seen in depressed individuals. In addition, both sleep and depression have been independently linked to deficits in cognitive control. Therefore, we tested a comprehensive, theoretically informed model that incorporated sleep, cognitive control and depressive symptoms. Poor sleep quality was associated with greater difficulty disengaging attention from negative stimuli. Cognitive control, in turn, predicted changes in depressive symptoms. Specifically, worse cognitive control was associated with greater increases in depression symptoms. Thus, altered sleep quality may contribute to the development of depression by impairing cognitive control.

We also found an indirect relation between sleep-wake cycle stability (IS) and depressive symptoms. IS did not predict depressive symptoms at either time point. However, IS predicted cognitive control over emotional information, which predicted increases in depressive symptoms. Interestingly, stable sleepers exhibited poorer cognitive control than individuals with less consistent sleep patterns, putting them at greater risk for depression. Although not measured in the current study, we hypothesize that a third, possibly trait-like, variable (e.g., behavioral inhibition) may be associated with both sleep stability and cognitive control. However, while this possibility may account for the unexpected relation, it is merely speculation. Thus, future research should further explore this possibility.

Our third, exploratory aim was to explore the relation of the CLOCK gene to our variables of interest (sleep, cognitive control and depressive symptoms). Although CLOCK SNP rs11932595 has previously been linked to sleep duration, it did not correlate with any objective
measures of sleep in the current study. It was, however, associated with sleep quality. Specifically, greater number of G alleles was associated with higher levels of self-reported sleep difficulties. Last, rs11932595 was not related to cognitive control or depressive symptoms at either time point. In a recent commentary, Goel noted that the fields of sleep and mood disorders share the overlap of phenotypes and genotypes and that future research should “exploit these commonalities”. Therefore, we present these findings as preliminary evidence to suggest that polymorphisms within the CLOCK gene may influence sleep quality. Future research should continue to explore the relation among these variables within a single model in order to develop a more comprehensive understanding of depression than unidimensional models that focus on one particular domain.

There are several limitations to the current study. Perhaps the most important limitation is sample size. Indeed, effects in smaller samples are at greater risk for Type I error than those initially found in larger samples. As such, replication with a larger sample is necessary before further development of the proposed model. Further, several of the associations tested in the model were cross-sectional. While we did assess sleep across a 3-week period and depressive symptoms across two time points, cognitive control was only assessed at the end of the three-week period. Thus, causal inferences would have been stronger if we had temporally separated the assessments of cognitive control and depression. Nevertheless, future research that experimentally manipulates these variables (e.g., sleep deprivation, cognitive bias modification, etc.) is needed before one can conclude with confidence that we are observing causal relations among these variables.

Despite these limitations, we present a model in which perceived sleep quality and sleep-wake cycle stability predicts cognitive control over negative stimuli, which in turn predicts changes in depressive symptoms. To our knowledge, we are the first to report that sleep disturbance may contribute to changes in depressive symptoms via its impact on cognitive control over negative stimuli. Sleep stability and self-reported sleep difficulties predicted cognitive control over sad stimuli that, in turn, predicted changes in depression symptoms. In addition, a SNP within the CLOCK gene was associated with sleep quality. We believe our proposed model and exploratory SNP analyses are an excellent starting point for the development of interdisciplinary models of depression vulnerability that incorporate findings across levels of analysis.

Acknowledgements
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Table 1. Correlations, means, and standard deviations for depression severity, self-reported sleep difficulty, cognitive control of emotion stimuli, objective sleep measures, and CLOCK polymorphisms.

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Mean 13.34 12.43 5.37 17.04 0.55 0.50 7.16 1.94 1.42
SD 8.75 7.71 1.78 34.27 0.13 0.08 0.79 0.51 0.61

CES-D = Center for Epidemiological Studies – Depression scale; PSQI-G = Pittsburgh Sleep Quality Inventory – General Functioning; CV = Cue Validity; IS = Interdaily Stability; IV = Intradaily Variability; Sleep duration = mean sleep duration; Sleep variability = sleep duration SD; * indicates p < .05; N = 35 for all correlations except those with rs11932595 where N = 34
**Figure 1.** Trial sequence for valid and invalid trials on the Emotional Cuing Task. Faces and targets are not to scale for purposes of this figure.

**Figure 2.** Path analysis predicting change in depressive symptoms.