Longitudinal Relationships between Self-efficacy, Post-traumatic Distress and Salivary Cortisol among Motor Vehicle Accident Survivors

Roman Cieslak1,2*, Charles C. Benight1,3*, Aleksandra Luszczynska1,2 & Mark L. Laudenslager4

1Trauma, Health, and Hazards Center, University of Colorado at Colorado Springs, Colorado Springs, CO, USA
2Warsaw School of Social Sciences and Humanities, Warsaw, Poland
3Department of Psychology, University of Colorado at Colorado Springs, Colorado Springs, CO, USA
4University of Colorado Denver School of Medicine, Denver, CO, USA

Abstract

The present study tested if post-traumatic distress following a motor vehicle accident (MVA) and MVA-related self-efficacy beliefs were associated with diurnal salivary cortisol in the early post-traumatic period. Cortisol was collected upon awakening and at 1, 4 and 12 h after waking. Collection days were 1 week, 1 month and 3 months after MVA. A total of 30 participants provided their cortisol samples across all measurement points. Two methods for computing the area under the cortisol curve were used. Higher post-traumatic distress at 1 month predicted lower cortisol area under the curve (AUC) with respect to increase (AUCI, reflecting changes in cortisol secretions during daytime) at 3 months. AUC with respect to ground (AUCG, reflecting total cortisol release during daytime), measured at 1 month after trauma, predicted higher post-traumatic distress at 3 months. The results showed that self-efficacy at 1 week indirectly predicted 3-month AUCI through 1-month post-traumatic distress. These findings highlight the importance of self-efficacy and post-traumatic distress in explaining longitudinal diurnal patterns of cortisol secretion after trauma. Copyright © 2011 John Wiley & Sons, Ltd.

Keywords
diurnal cortisol; post-traumatic stress disorder; self-efficacy; trauma

*Correspondence
Roman Cieslak, Trauma, Health, and Hazards Center, University of Colorado at Colorado Springs, Colorado Springs, CO, 80933-7150, USA; Charles C. Benight, Department of Psychology, University of Colorado at Colorado Springs, Colorado Springs, CO, 80933-7150, USA.

Recovery from trauma challenges individual coping skills and capacities. Social cognitive theory (SCT) suggests that the processes of self-regulation allow humans to evaluate their coping successes and failures in order to strategically modify them (Bandura, 1997). It is through this self-regulation process that self-efficacy beliefs are generated. In the context of post-traumatic adaptation, self-efficacy perceptions refer to optimistic beliefs about the ability to deal with problems arising after trauma (Benight & Bandura, 2004). According to the SCT, self-efficacy plays a vital role in predicting emotional states, physiological responses and symptoms of mental disorder (Bandura, 1997).

Social cognitive theory indicates that self-efficacy beliefs are the most proximal predictor of mental health outcomes during post-traumatic recovery (Benight & Bandura, 2004). Longitudinal studies confirmed that self-efficacy is predictive of the development and maintenance of post-traumatic stress disorder (PTSD) symptoms (Benight, Cieslak, Molton, & Johnson, 2008; Heinrichs et al., 2005; for meta-analysis, see Luszczynska, Benight, & Cieslak, 2009). Further, the SCT suggests that self-efficacy constitutes an active agent in adaptation processes influencing distress states and producing biological and behavioural outcomes (Bandura, 1997). Therefore, the effects of self-efficacy on health outcomes or
biological indices (e.g. cortisol) may be mediated by distress.

Cortisol, a key hormone involved in the stress response, may be affected by efficacy beliefs (Bandura, 1997). Self-efficacy may be seen as a determinant of physiological stress response and thus predictive of basal cortisol levels (Nierop, Wirtz, Bratsikas, Zimmermann, & Ehlert, 2008; Schwerdtfeger, Konermann, & Schönhofen, 2008). Beliefs about one's own competence (a concept similar to self-efficacy, see Schwarzer, 2008) were negatively related to cortisol diurnal variation among individuals exposed to experimentally induced stress (Gaab, Rohleder, Nater, & Ehlert, 2005).

Longitudinally, the relationship between cortisol and post-traumatic distress could be viewed as bidirectional. On the one hand, altered cortisol response (measured within days after trauma) may predict subsequent PTSD. For example, a study on the effects of cortisol secretion sampled during the 24 h after motor vehicle trauma showed that lower cortisol levels predicted the development of more severe PTSD symptoms at 1 month after a motor vehicle accident (MVA) (Delahanty, Raomonde, & Spoonster, 2000). However, in another study, cortisol levels (measured on admission to an emergency room, 1 month and 5 months later) did not predict PTSD diagnosis 5 months after trauma (Shalev et al., 2008). It has also been found that elevated post-traumatic distress may evoke changes in subsequently measured cortisol. For example, individuals having PTSD symptoms have disturbed sleep patterns (Spoormaker & Montgomery, 2008), which result in abnormal cortisol secretion on the days following such symptoms (cf. Vgontzas et al., 1999). Therefore, PTSD symptoms may predict subsequent diurnal cortisol secretion.

As indicated, the relationship between the indices of cortisol secretion and subsequent PTSD within the first few months after trauma is ambiguous (Delahanty et al., 2000; Shalev et al., 2008). Response and thus predictive distress within the first months after trauma rarely accounted for diurnal cortisol secretion patterns (Miller, Chen, & Zhou, 2007). Thus, studying diurnal cortisol secretion may help to explain these equivocal findings.

A recent meta-analysis on cortisol secretion among trauma survivors indicated that long-term effects of trauma include lower morning cortisol, higher afternoon/evening cortisol and higher total daily cortisol output (Miller et al., 2007). Further, meta-analyses suggest that changes in the diurnal cortisol secretion depend on the time elapsed since trauma exposure. Higher output is expected in the months directly after trauma, but a reverse pattern is expected over longer periods post-event (Miller et al., 2007). The dynamics of observed changes depend not only on time elapsed since the trauma but also on co-occurring post-traumatic distress (Miller et al., 2007). Because of the dearth of longitudinal research, key unanswered questions refer to patterns of diurnal cortisol secretion within shorter time intervals since trauma onset (Miller et al., 2007). Our study focuses on diurnal cortisol changes within the period of 3 months after traumatic exposure, in the context of post-traumatic distress and self-efficacy beliefs.

In line with the SCT (Bandura, 1997; Benight & Bandura, 2004), it was hypothesized that coping self-efficacy (CSE) beliefs would predict subsequent post-traumatic distress symptoms (Hypothesis 1). It was also hypothesized that PTSD symptoms would predict subsequent diurnal cortisol profile indices (Hypothesis 2). Alternatively, we also tested if diurnal cortisol indices would predict PTSD symptoms measured at later time points (Hypothesis 3). Although the evidence is insufficient to firmly hypothesize the directions of the altered diurnal cortisol response in the months after trauma, it may be expected that high post-traumatic distress would relate to the higher total diurnal output and lower sensitivity of the cortisol secretion system (Miller et al., 2007). Based on the SCT (Benight & Bandura, 2004), two mediation hypotheses were proposed. Hypothesis 4 assumes that self-efficacy at 1 week after trauma (the independent variable) would predict PTSD symptoms 1 month later (the mediator), which in turn would predict diurnal cortisol at 3 months (the dependent variable). Lower self-efficacy would result in greater levels of subsequent post-traumatic distress. Greater psychological distress would, in turn, predict altered diurnal cortisol. In the competing hypothesis (5), we assumed that diurnal cortisol at 1 month would mediate the relationship between CSE measured at 1 week after trauma and PTSD symptoms measured 3 months later: CSE would predict an altered diurnal pattern of cortisol secretion, which in turn would be predictive of higher post-traumatic distress.

Materials and method
Participants and procedures
Individuals admitted to the emergency room of a local hospital because of an MVA were initially identified by hospital trauma team personnel and were screened for exclusion criteria. The following exclusion criteria were applied: age below 18 or above 75 years, being inebriated or abusing illegal drugs at the time of the accident, being pregnant, having a mental illness diagnosis, taking any prescription drugs or reporting chronic diseases that could interfere with cortisol assessment and reporting post-traumatic amnesia longer than 10 min. Those who met the inclusion criteria were invited to participate in the study.

Respondents of the present study were a subsample of the respondents to a larger study on the recovery...
mechanisms after an MVA trauma (Benight et al., 2008), who agreed to provide saliva in collection tubes on days when they filled out the self-assessment instruments. They were supplied with the labelled tubes and were instructed on how to collect their saliva and store the tubes. Among 163 survivors invited to participate in the study, 67 provided saliva samples sufficient for cortisol analysis at Phase 1 (at awakening, 1, 4 and 12 h later).

The MVA survivors completed self-report instruments at three time points: 1 week [Phase 1; mean (M) = 7.10 days after MVA, standard deviation (SD) = 2.56], 1 month (Phase 2; M = 36.56 days after MVA, SD = 8.81) and 3 months (Phase 3; M = 98.79 days after MVA SD = 17.28). Participants provided samples at spontaneous awakening (Hour 0; \(n_{\text{Phase1}} = 80\), \(n_{\text{Phase2}} = 54\), \(n_{\text{Phase3}} = 36\), 1 h (Hour 1; \(n_{\text{Phase1}} = 80\), \(n_{\text{Phase2}} = 50\), \(n_{\text{Phase3}} = 36\), 4 h (Hour 4; \(n_{\text{Phase1}} = 83\), \(n_{\text{Phase2}} = 54\), \(n_{\text{Phase3}} = 35\) and 12 h (Hour 12; \(n_{\text{Phase1}} = 80\), \(n_{\text{Phase2}} = 55\), \(n_{\text{Phase3}} = 34\)) after awakening. Waking was not triggered. On average, participants collected cortisol sample upon awakening at 7:24 AM (SD = 1:47) at Phase 1, 7:17 AM (SD = 1:49) at Phase 2 and 7:19 AM (SD = 1:31) at Phase 3.

Overall, 17 (57%) women and 13 (37%) men provided their cortisol samples across all 12 cortisol collection points. They were aged 18 to 69 years (M = 37.00, SD = 13.36). Thirty per cent declared an annual income below $30,000, 43% declared an income between $30,000 and $60,000 and 23% reported an income above $60,000. More than half of the participants had at least a bachelor’s degree (n = 18; 60%), the rest have a high school education (n = 12; 40%) and slightly more than half (n = 17; 57%) were married or were living with a partner. The participants self-reported as primarily Caucasian (n = 26; 87%), with the remainder reporting as African or Native American (n = 4; 12%). Almost half of the participants (43%) indicated that they caused the accident; 10% were involved in litigation related to the accident. The study sample demonstrated mild injuries (Injury Severity Index: M = 2.61, SD = 2.37). The study received ethical clearance from the Internal Review Board at the University of Colorado. All participants provided informed consent.

**Measures**

**Post-traumatic distress (post-traumatic stress disorder symptoms)**

The total scores of the Impact of Event Scale—Revised (Weiss & Marmar, 1997) were collected at 1 week, 1 month and 3 months after trauma. The scale assesses the presence of avoidance, hyperarousal and intrusive thoughts/images symptoms. In our study, the response scale was adapted from the older version of the scale (i.e. Impact of Event Scale; Horowitz, Wilner, & Alvarez, 1979), in line with previous studies on MVA survivors (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006). Cronbach’s alphas for three measurement points were 0.91, 0.92 and 0.96, respectively.

**Self-efficacy**

The MVA-CSE measure was applied to assess self-evaluative perceptions of the ability to cope with the aftermath of an MVA (Benight et al., 2008). This unidimensional scale consists of 41 items with the seven-point response scale ranging from 1 (not at all capable) to 7 (very capable) and has satisfactory validity (Benight et al., 2008). For example, the participants were asked to evaluate their perceived capability of successful dealing with all of the appointments (rehabilitation, car repair, insurance, etc.) or with fears of having another accident. At 7 days after trauma, the reliability of this scale was \(\alpha = 0.97\).

Participants were instructed to collect saliva immediately upon awakening and at 1, 4 and 12 h after awakening. They were asked to refrain from eating, smoking or drinking, excluding water, for at least 30 min before collecting each sample. Specifically, individuals were instructed to collect their saliva by chewing Salivette (Sarstedt, Inc., Newton, NC) cotton dental swab for 60 s. The cotton roll was placed in the holder. The Salivettes were spun in the laboratory at 3000 g. Saliva was stored in o-ring-sealed microcentrifuge tubes at \(-20^\circ\text{C}\).

Salivary cortisol concentration was analysed using a commercially available radioimmunoassay kit, following the producer’s instructions (Cortisol Coat-A-Count RIA from Diagnostic Products Company, Los Angeles, CA). In brief, the kit was modified to permit the determination of lower levels of cortisol found in saliva. A 100-μL (rather than the 25 μL recommended by the manufacturer) sample was transferred in duplicate to assay tubes coated with antibody. The standard curve was adjusted by serial dilutions of the standards supplied with the kit to include standards of 0.01, 0.05, 0.1, 0.25, 0.5, 1.0, 2.0 and 5.0 μg/dL. The standards were added in volumes of 100 μL as well. Finally, incubation time of the assay was increased from 30 min to 3 h. A calibrated low standard (range 4–5 μg/dL) was run in every assay both undiluted and diluted 1:10 in Dulbecco’s phosphate buffer to an approximate concentration of 0.4 μg/dL. These standards were included in the assay for the determination of intra-assay and inter-assay coefficients of variation. Intra-assay and inter-assay coefficients of variation for this assay were less than 7%. The minimal detectable level for this modified procedure is 0.05 μg/dL. Any duplicate determination whose percentage of coefficient of variation exceeded 10% were rerun in triplicate taking the median value as the assay result.
Diurnal cortisol activity index

The computation of the area under the curve (AUC) indices is often used to demonstrate the changes in the endocrinological factors over time. Recent research suggested that the AUC with respect to ground (AUCG) and the AUC with respect to increase (AUCI) may be particularly useful indices for cortisol secretion when studying the relationships between stress and health (Fekedulegn et al., 2007). These two indices are related to other profile indicators and patterns (including regression slopes) or rates of change over time (Fekedulegn et al., 2007). AUCG reflects the patterns of total release of cortisol over the time interval, whereas AUCI is more related to the sensitivity of the system and captures the pattern of change over the same interval (Fekedulegn et al., 2007). To compute the AUC indices, we applied formulas recommended by Pruessner, Kirschbaum, Meinlschmid and Hellhammer (2003). As saliva was collected four times a day at Phases 1, 2 and 3, we computed the AUC indices at each phase.

Research on diurnal cortisol among trauma survivors usually accounted for cortisol at awakening time (Miller et al., 2007), 1 h after awakening (Yehuda, Golier, & Kaufman, 2005) and evening time (e.g. 12–13 h after awakening; smil1379-bib-0017Miller et al., 2005; Yehuda et al., 2005). Altered cortisol levels have been documented at these times (Miller et al., 2007). Further, most of the studies on diurnal cortisol secretion also apply a midday measurement (Yehuda et al., 2005). Therefore, our diurnal cortisol indices were based on saliva samples collected at awakening time, 1, 4 and 12 h later.

Data analysis

Data were analysed with PASW Statistics (version 18; SPSS Inc., Chicago, IL, USA) and PASW macros for mediation effects with bootstrapping (Preacher & Hayes, 2004, 2008). Analysis of variance, correlation analysis and regression analysis were applied to test the hypotheses, with the significance level of 0.05. Mediation hypotheses were tested using regression analysis as outlined by Kenny, Kashy and Bolger (1998). To meet mediation conditions, the independent variable should predict the mediator (Path a), and the mediator should be significantly related to the dependent variable (Path b). To further confirm the significance of the mediation effect, Sobel test was calculated (MacKinnon & Dwyer, 1993).

Across the study variables, the distribution did not significantly differ from a normal distribution [D<1.18, not significant (ns)], except for the AUCI at Phase 3 [D(30)=1.51, p=0.02]. Bootstrapping was applied to aid non-normal distribution of this one-cortisol index and to reduce possible biases related to small sample size (Efron & Tibshirani, 1993; Preacher & Hayes, 2004).

Results

Preliminary analysis

Analyses comparing those who provided saliva samples at Phase 1 and those who dropped out at subsequent phases showed that completers and dropouts did not differ on self-efficacy [t(65)=−0.97, ns], PTSD symptoms [t(62)=0.38, ns], AUCG [t(65)=−0.28, ns], AUCI [t(65)=−0.72, ns], severity of injury [t(65)=−0.84, ns], gender [χ²(1)=0.22, ns], income [χ²(1)=0.63, ns], responsibility for the accident [χ²(1)=1.61, ns] or litigation [χ²(1)=0.52, ns]. Further, survivors who provided salivary cortisol at Phase 1 (n=65) and those who participated in the psychological assessment only (n=97) did not differ in any of the listed above variables, except severity of injury [t(160)=2.58, p<0.05], which was higher among those who provided cortisol samples at Phase 1.

The average profiles of cortisol secretion at 7 days, 1 month and 3 months after the MVA are presented in Figure 1. We compared cortisol secretion at each time of day, which was included in the analysis (awakening, 1, 4 and 12 h later) across the three phases of data collection (7 days, 1 month and 3 months after trauma). There was a significant main effect for the time of the day on cortisol level [F(1,15)=20.29, p<0.001, η²=0.58]. Contrast tests revealed that cortisol level upon awakening was significantly higher than 4-h cortisol [F(1,15)=27.28, p<0.001, η²=0.65] and 12-h cortisol [F(1,15)=27.25, p<0.001, η²=0.65], but it was approximatively at the same level as 1-h cortisol [F(1,15)=0.06, ns]. The main effect for the phases of data collection on cortisol was not significant [F(2,30)=0.09, ns]. The effect of interaction between the time of the day and the phases of the study was non-significant [F(6,90)=1.57, ns].

Preliminary analysis indicated that age was unrelated to self-efficacy and post-traumatic distress (Table I). Gender was unrelated to cortisol indices, post-traumatic distress and self-efficacy (Table I). Several cross-sectional and longitudinal relationships among post-traumatic distress and the cortisol indices were found (Table I). Higher AUCG at Phase 2 was related to higher post-traumatic distress at 1 month and at 3 months after trauma. Reporting more post-traumatic distress at all three Phases (1 week, 3 month and 3 months after trauma) was related to lower AUCI at Phase 3. There were no associations between PTSD symptoms at Phase 1 and cortisol indices at Phase 1 (Table I).

Associations among self-efficacy, diurnal cortisol and post-traumatic distress symptoms

The first hypothesis suggested that stronger self-efficacy (Phase 1) would be associated with reporting less PTSD symptoms (at all three points of assess-
The correlation analysis (Table I) supports this hypothesis. The second hypothesis indicated that PTSD symptoms would be related to subsequent diurnal cortisol profile indices. Correlation analysis (Table I) supports this hypothesis: higher post-traumatic distress at Phase 1 and Phase 2 was related to lower AUCI at Phase 3. In line with the third hypothesis, higher cortisol index (AUCi at Phase 2) was related to higher level of PTSD symptoms at Phase 3. The strength of the relationship between cortisol profile index and subsequent PTSD symptoms (Hypothesis 3) did not differ (Z=0.24, ns) from the strength of the relationship between PTSD symptoms and subsequent cortisol profile index (Hypothesis 2). In line with our assumptions, higher post-traumatic distress was related to higher total output (AUCG) and lower sensitivity (AUCI) of the cortisol secretion system.

**Self-efficacy predicting post-traumatic distress and cortisol indices: the mediation analysis**

Hypothesis 4 referred to the longitudinal meditational relationships between self-efficacy (the independent variable), PTSD symptoms (the mediator) and cortisol indices (the dependent variables). Mediation analysis indicated that Path a was significant: stronger self-efficacy (Phase 1) predicted lower PTSD symptoms (Phase 2) (β=−0.77, p<0.001). Path b was also significant: lower level of PTSD symptoms (Phase 2) predicted higher AUCi index of cortisol at Phase 3 (β=−0.71, p<0.05). The direct effect of self-efficacy (Phase 1) on AUCi index of cortisol at Phase 3 was not significant, both with and without controlling for the effect of PTSD symptoms on cortisol. Sobel test

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Age</td>
<td>0.19</td>
<td>0.05</td>
<td>0.07</td>
<td>0.08</td>
<td>0.02</td>
<td>0.03</td>
<td>0.04</td>
<td>0.05</td>
<td>0.06</td>
<td>0.07</td>
<td>0.08</td>
<td>0.09</td>
<td>0.1</td>
</tr>
<tr>
<td>3. CSE at P1</td>
<td>0.17</td>
<td>0.11</td>
<td>0.08</td>
<td>0.04</td>
<td>0.02</td>
<td>0.03</td>
<td>0.04</td>
<td>0.05</td>
<td>0.06</td>
<td>0.07</td>
<td>0.08</td>
<td>0.09</td>
<td>0.1</td>
</tr>
<tr>
<td>4. PTSD symptoms at P1</td>
<td>0.13</td>
<td>0.04</td>
<td>0.02</td>
<td>0.03</td>
<td>0.02</td>
<td>0.03</td>
<td>0.04</td>
<td>0.05</td>
<td>0.06</td>
<td>0.07</td>
<td>0.08</td>
<td>0.09</td>
<td>0.1</td>
</tr>
<tr>
<td>5. PTSD symptoms at P2</td>
<td>0.18</td>
<td>0.03</td>
<td>0.02</td>
<td>0.03</td>
<td>0.02</td>
<td>0.03</td>
<td>0.04</td>
<td>0.05</td>
<td>0.06</td>
<td>0.07</td>
<td>0.08</td>
<td>0.09</td>
<td>0.1</td>
</tr>
<tr>
<td>6. PTSD symptoms at P3</td>
<td>0.20</td>
<td>−0.05</td>
<td>−0.12</td>
<td>−0.08</td>
<td>−0.11</td>
<td>0.09</td>
<td>0.03</td>
<td>0.02</td>
<td>0.01</td>
<td>−0.08</td>
<td>−0.12</td>
<td>−0.16</td>
<td>−0.20</td>
</tr>
<tr>
<td>7. AUCi at P1</td>
<td>−0.07</td>
<td>−0.09</td>
<td>−0.37</td>
<td>0.35</td>
<td>0.52*</td>
<td>0.59**</td>
<td>0.16</td>
<td>−0.16</td>
<td>0.21</td>
<td>−0.16</td>
<td>0.21</td>
<td>−0.16</td>
<td>0.21</td>
</tr>
<tr>
<td>8. AUCi at P2</td>
<td>−0.12</td>
<td>0.08</td>
<td>−0.11</td>
<td>0.09</td>
<td>0.35</td>
<td>0.22</td>
<td>−0.12</td>
<td>0.44</td>
<td>−0.12</td>
<td>0.44</td>
<td>−0.12</td>
<td>0.44</td>
<td>−0.12</td>
</tr>
<tr>
<td>9. AUCi at P3</td>
<td>0.0</td>
<td>0.4</td>
<td>0.1</td>
<td>0.22</td>
<td>0.28</td>
<td>0.29</td>
<td>0.49</td>
<td>0.02</td>
<td>0.13</td>
<td>0.02</td>
<td>0.13</td>
<td>0.02</td>
<td>0.13</td>
</tr>
<tr>
<td>10. AUCi at P4</td>
<td>0.20</td>
<td>−0.53*</td>
<td>−0.21</td>
<td>0.40</td>
<td>0.26</td>
<td>0.30</td>
<td>0.11</td>
<td>0.19</td>
<td>0.24</td>
<td>0.45</td>
<td>0.65**</td>
<td>−3.33</td>
<td>4.03</td>
</tr>
<tr>
<td>11. AUCi at P5</td>
<td>−0.16</td>
<td>0.00</td>
<td>0.30</td>
<td>−0.50***</td>
<td>−0.54***</td>
<td>−0.65***</td>
<td>0.00</td>
<td>−0.56***</td>
<td>−0.37*</td>
<td>−0.16</td>
<td>−0.28</td>
<td>−1.93</td>
<td>3.61</td>
</tr>
</tbody>
</table>

PTSD, post-traumatic stress disorder; CSE, coping self-efficacy; P1, Phase 1 (1 week after motor vehicle accident); P2, Phase 2 (1 month after motor vehicle accident); P3, Phase 3 (3 months after motor vehicle accident); AUCG, the area under the curve with respect to ground, cortisol secretion index; AUCI, the area under the curve with respect to increase, cortisol secretion index.

*p<0.05, **p<0.01 and ***p<0.001.
confirmed that the mediation was significant ($Z=2.26, p<0.05$) (Figure 2). Moreover, the mediation remained significant after controlling for the effects of age ($\beta=-0.07, ns$) and gender ($\beta=-0.02, ns$) on Phase 2 PTSD symptoms and the effects of age ($\beta=0.02, ns$) and gender ($\beta=-0.17, ns$) on AUC at Phase 3.

To manage the non-normal distribution of AUC (Phase 3), the analyses were repeated using bootstrapping (Preacher & Hayes, 2008). Controlling for age and gender and assuming the normal distribution, the indirect effect of self-efficacy at Phase 1 on AUC at Phase 3 was 1.66. The bootstrap estimate (based on 1000 bootstrap samples) was 1.56, with 95% bias corrected and accelerated confidence intervals of 0.09–5.04, indicating a significant indirect effect.

The mediation analysis with bootstrapping was repeated with self-efficacy (Phase 1) playing additionally a moderating role in the relationship between PTSD (Phase 2) and AUC (Phase 3). Accounting for the moderating effect, the results showed that Paths a and b were significant (Path a: $\beta=-16.67, p<0.001$; Path b: $\beta=-0.29, p<0.001$; the moderator effect: $\beta=0.05, p<0.05$), but the indirect effect of self-efficacy on AUC was not significant. The participants with high self-efficacy (Phase 1) had similar values of AUC (Phase 3), regardless of the levels of post-traumatic distress (Phase 2). Means for AUC index were $-0.03$ (low PTSD symptoms, $-1SD$) and $1.32$ (high PTSD symptoms, $+1SD$). By contrast, among those with low self-efficacy (Phase 1), AUC index sharply declined with the increase of PTSD symptoms (from $M=4.35$ when post-traumatic distress was low, i.e. at $-1SD$, to $M=-0.64$ when post-traumatic distress was high, $+1SD$). Thus, the decline of the cortisol sensitivity system was observed when PTSD symptoms were increasing but only among the survivors with low self-efficacy.

Hypothesis 4 referred also to the mediational relationships between self-efficacy (the independent variable), PTSD symptoms (the mediator) and the other cortisol index, AUC$_G$ (the dependent variable). Regression analyses indicated that PTSD symptoms measured at Phase 2 remained unrelated to AUC$_G$ index (Phase 3) ($\beta=0.34, ns$). Because Path b of the mediation model was not significant, the mediating hypothesis was not supported for the AUC$_G$ index.

In sum, the mediation analysis (Figure 2) indicated that stronger self-efficacy at 1 week after trauma predicted lower post-traumatic distress at 1 month after the exposure. Low levels of PTSD symptoms were, in turn, related to larger change in cortisol level during the day (an index of the sensitivity of the cortisol secretion system).

Hypothesis 5 referred to a competing hypothesis that cortisol at 1 month would mediate the relationship between CSE at the time of trauma and PTSD symptoms 3 months later. Self-efficacy (Phase 1) remained unrelated to AUC at Phase 2 ($\beta=-0.37, ns$) or AUC$_G$ at Phase 2 ($\beta=0.21, ns$). Therefore, the first condition of the mediation analysis (i.e. the effect of the independent variable on the mediator) was not met, and Hypothesis 5 was not supported.

**Discussion**

The study provides support for the interplay between self-efficacy and psychological distress following MVA trauma and corroborate SCT in the context of post-traumatic distress (Benight & Bandura, 2004). Our findings indicate that among MVA survivors, strong self-efficacy generated within 7 days after trauma exposure were related to lower current and subsequent post-traumatic distress (PTSD symptoms). This relationship is consistent with the results of a recent meta-analysis, suggesting strong effects of self-efficacy on subsequent PTSD among trauma survivors (Luszczyńska et al., 2009).

The present study did not support a direct relationship between self-efficacy and long-term diurnal variation in cortisol. Importantly, it appears that self-efficacy may predict subsequent physiological stress reactivity (i.e. cortisol diurnal decline) through post-traumatic stress symptoms. Specifically, high self-efficacy indirectly predicted greater time-dependent change in cortisol (e.g. AUC$_G$). Thus, lower psychological distress was associated with more normal function.

![Figure 2](image-url) The effect of self-efficacy (at 1 week after trauma) on cortisol area under the curve (AUC) with respect to increase (AUC$_G$ at 3 months after trauma) mediated by post-traumatic distress at 1 month after trauma.
of hypothalamus–pituitary axis. Cross-sectional re-
search among trauma survivors suggested that diurnal
secretion of cortisol is a function of post-traumatic
distress (Miller et al., 2007). Our findings indicate that
accounting for self-efficacy, beliefs may further eluci-
date the changes in diurnal cortisol secretion across
3 months after trauma exposure.

Importantly, the SCT (Bandura, 1997) suggests that
self-efficacy should have a dampening effect on
physiological arousal, and thus, it may have a direct
effect on cortisol secretion. Although the present study
suggests that self-efficacy was not related to either of
diurnal cortisol indices, relationships that were iden-
tified were in a theoretically consistent direction, with
weaker self-efficacy related to higher total cortisol
output and lower sensitivity of cortisol secretion system
at subsequent measurements. The small sample size for
the present study should be taken into consideration
when interpreting these non-significant findings.

Hypotheses 2 and 3 for the present study dealt with
the bidirectional relationships between diurnal cortisol
indices and post-traumatic distress, taking into account
longitudinal relationships. We found some support for
both directions. Participants with higher 1-month total
output of the cortisol during daytime (AUCG) reported
higher post-traumatic distress at that same data
analysis phase and at 3 months after the MVA. These
results are in line with findings indicating higher diurnal
cortisol output among survivors who have been recently
exposed to trauma (Miller et al., 2007). Further, results
of the present study indicate that respondents with high
post-traumatic distress 1 month after the MVA had low
AUCl (the index of sensitivity of the cortisol secretion
system) at 3 months after MVA. Low AUCl reflects
smaller change in cortisol level during the day. Recent
meta-analysis of cross-sectional studies suggested that
flat diurnal profiles are typical in trauma survivors
(Miller et al., 2007). Our study adds to this evidence, as
it is based on longitudinal findings. We showed that
elevated PTSD symptoms may precede a flat diurnal
profile. These results, indicating that elevated post-
traumatic distress predicts lower sensitivity of cortisol
secretion system (measured 2 months later), are in line
with research on the effects of disturbed sleep (which
may reflect higher PTSD symptoms) on flat profiles of
cortisol secretion (cf. Vgontzas et al., 1999).

The relationships between self-efficacy, diurnal
cortisol secretion and PTSD symptoms may be further
explained by other factors that may determine the
AUC indices but were not controlled for in the present
study (e.g. other physical illnesses, cf. Vedhara et al.,
2007; mood disorders, adiposity and lipoprotein
profiles, Veen et al., 2009). Further, other cognitive
predictors may better elucidate the complex relation-
ships among these self-beliefs and health outcomes
(Schwarzer, 2008). Therefore, future research in this
area will need to address other possible contributors to
the relationships among self-regulatory cognitions,
post-traumatic distress and cortisol. Finally, experi-
mental studies are needed to more firmly establish the
role of self-efficacy in the relationship between post-
traumatic distress and cortisol secretion.

The study has several limitations. The biggest concern
for the present study is the small sample size making it
difficult to find important associations and limiting the
confidence in the ones that were identified. Previous
studies enrolled samples with 20–34 participants
(Benight et al., 2004; Edwards, Hucklebridge, Clow, &
Evans, 2003; Stephon, Sapolsky, Kraemer, & Spiegel,
2004). Based on the effects found in previous studies, 29
participants should secure power of 0.70. In any case,
small sample and reduced power is the major limitation
of the present study. These issues may partially explain
why some relationships were not significant, although
the coefficients were substantial in size (cf. the
relationship between self-efficacy at 1 week after
exposure and cortisol at 3 months later, also the effects
of sex on cortisol secretion). Further, smaller samples
make it nearly impossible to control for the multitude of
confounding factors. Although, ideally, one should
control for relevant important confounding variables
(e.g. smoking, menstrual cycle, age, sleep), our sample
size made this impossible. In our analyses, we were
unable to control for potential confounding variables
of cortisol secretion, such as compliance with saliva
analysis protocol, menstrual cycle, oral contraceptive
use, exercise patterns and sleep patterns, because this
information was not collected. In addition, our PTSD
symptom measure is not a diagnostic assessment limiting
our ability to diagnose. In consequence, analyses
addressing the effect of PTSD diagnosis were not possible.
The present study shows preliminary support for the
importance of the moderating role of self-efficacy in the
relationships between PTSD and diurnal cortisol. Thus,
alternative hypotheses assuming moderating effects of
self-efficacy should be further tested. The mediating
effects of post-traumatic distress on the relationship
between self-efficacy and diurnal cortisol should be
replicated. Only one daily profile was taken at each phase,
and cortisol was collected only twice within 60 min from
awakening. The age range was large, which could affect the
obtained results (although age was controlled for in
mediation hypotheses). Results also should be interpreted
with caution as there was no control group. The
generalisability is restricted to a non-clinical sample of
MVA survivors. Further research should test for the
relationships between diurnal cortisol slopes, AUC indices
referring to the first hour after awakening and PTSD
symptoms. The design of the present study (two types of
outcomes and five hypotheses) does not allow for adding
more outcome indices (such as diurnal cortisol slopes)
without increasing the likelihood of chance findings.

In spite of these limitations, this theoretically based
longitudinal study provides important steps in helping
to understand the inter-relationships among self-
regulation, psychological distress and salivary cortisol
during the first few months of post-traumatic adaptation. This is the first study of which we are aware to follow trauma survivors across a 3-month period attempting to elucidate the adaptation process utilizing SCT. Post-traumatic distress was positively correlated with AUC<sub>C</sub> (reflecting total cortisol release during daytime) and inversely related to AUC<sub>T</sub> (representing the sensitivity of the cortisol secretion system). These findings may further help to explain the inconsistent findings in PTSD and diurnal cortisol relationships. Self-efficacy beliefs may facilitate post-traumatic adaptation, as they are directly related to co-occurring and subsequent symptoms of PTSD. Finally, post-traumatic distress may play a crucial role in diurnal cortisol secretion among non-clinical MVA survivors.

Acknowledgments

The study was supported by the National Institutes of Mental Health grant RO3 MH59621-02 to Charles C. Benight and from the National Institutes of Health grants MH37373, AA013973 and CA196971 to Mark L. Laudenslager. We thank Christy Warren and Mark Goldstein for their assistance in this project.

REFERENCES


