Violence and Vigilance: The Acute Effects of Community Violent Crime on Sleep and Cortisol

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The data combine objectively measured sleep and thrice-daily salivary cortisol collected from a 4-day diary study in a large Midwestern city with location data on all violent crimes recorded during the same time period for \( N = 82 \) children (\( M_{\text{age}} = 14.90, \text{ range} = 11.27–18.11 \)). The primary empirical strategy uses a within-person design to measure the change in sleep and cortisol from the person’s typical pattern on the night/day immediately following a local violent crime. On the night following a violent crime, children have later bedtimes. Children also have disrupted cortisol patterns the following morning. Supplementary analyses using varying distances of the crime to the child’s home address confirm more proximate crimes correspond to later bedtimes.

Children exposed to recent incidents of local violence perform worse on assessments of academic skills, executive function, and effortful control relative to children from the same or similar neighborhoods who were not recently exposed to violence (Gershenson & Tekin, 2015; Sharkey, 2010; Sharkey, Tirado-Strayer, Papachristos, & Raver, 2012). Despite evidence that violence has an acute, causal impact on academic performance, the mechanisms explaining how environmental stressors get “under the skin” or “into the minds” of children have received little attention. This study examines two potential causal pathways that might explain the effects of acute violent crime: sleep and the hypothalamic–pituitary–adrenal (HPA) axis.

Sleep is a stress-sensitive system (Hicken, Lee, Ailshire, Burgard, & Williams, 2013; Sadeh, 1996). Under the opponent processes model of sleep, individuals must suspend arousal to transition from wakefulness to sleep (Edgar, Dement, & Fuller, 1993). Crime may affect sleep through heightened vigilance arising from fear or stress, or through noise and disruption from gunfire, shouting, and expanded police presence (Dahl, 1996; Hale et al., 2013; Lacoe & Sharkey, 2016). Insufficient sleep is associated with greater fatigue, more difficulty concentrating, and reduced executive functioning (Alapin et al., 2000; Sadeh, Gruber, & Raviv, 2003; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010).

Stress exposure can also affect the HPA axis, a key biological stress response system, and its primary hormonal product, cortisol. Cortisol levels follow a predictable daily pattern. A sharp increase in cortisol about 30 min after waking is called the cortisol awakening response, or CAR (Frueßner et al., 1997). Normally, the CAR assists in the transition from sleep to waking and may provide a boost to help meet the expected demands of the upcoming day (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Clow, Hucklebridge, Stalder, Evans, & Thorn, 2010; Fries, Dettenborn, & Kirschbaum, 2009; Vrshek-Schalhorn et al., 2012). Following the CAR, basal activity of the HPA axis typically decreases during waking hours, reaching the lowest levels shortly after sleep begins (Adam et al., 2006).

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Stress exposure can change typical diurnal cortisol patterns (Adam, 2012; Sapolsky, Romero, & Munck, 2000). Acute stress is associated with an elevated CAR, with more chronic stress and burn-out associated with a blunted CAR (Chida & Steptoe, 2009). High daily stress is associated with low waking cortisol, a low CAR, slower memory speed, and reduced attention continuity (Maldonado et al., 2008).

Although exposure to traumatic events such as hurricanes is associated with long-term changes to sleep and the HPA axis (Lavie, 2001; Vigil, Geary, Granger, & Flinn, 2010), it is unclear whether somewhat more common stressors such as violent crime have similar effects. To our knowledge this is the first study examining the short-term effects of proximity to nearby violent crime on sleep and cortisol.

Our primary empirical strategy uses a within-person design. It measures the change in sleep or cortisol on the night and morning immediately following a local violent crime, relative to that same person’s typical pattern. Because the comparison occurs within individuals, any changes to the outcomes cannot be caused by characteristics that are constant for a person (e.g., neighborhood quality). The important assumption in the design is that, within a given person, the timing of violent crime is random with respect to the timing of the diary study. Exogenous variation in the timing of local crime allows for causal inference.

Method

Participants

The present study involves a sample of 82 adolescents (49% female) who participated in a take-home diary study in a large Midwestern city in fall 2012. Diary subjects were drawn from participants of a larger study of adolescent stress (N = 379) submitted through the DePaul University and Northwestern University Institutional Review Boards. Adolescents were recruited through three public schools selected for having racially, ethnically, and socioeconomically diverse student bodies drawn from across the city. Information about the study was presented orally and in writing to potential participants during homeroom or lunch periods at their school. Parents were informed about the study at parent meetings, at report card pick-up days, and through parent consent forms. Parents were invited to contact the final author and/or school administrators if they had questions and/or wished to see copies of the measures. Participants with parental consent completed assent forms before data collection.

A subsample of 138 adolescents participated in the take-home diary study, including 94 adolescents who also received activity-tracking watches. One adolescent did not return a watch and 11 watches contained no usable data, leaving 82 adolescents with at least one full night of sleep data. We obtained 87% of four expected nights of sleep data from the 82 adolescents. The mean age was 14.90 (SD = 1.87, range = 11.27–18.11), with 17% of participants identifying as Black (n = 14), 20% identifying as Hispanic White (n = 16), 18% identifying as non-Hispanic White (n = 15), 27% identifying as multiethnic (n = 22), and the remainder identifying as another category or missing race information. Table 1 provides descriptive statistics. Table 2 provides a correlation matrix.

Measures

Sleep

Adolescents participated in one of six collection periods in fall 2012. Participants wore an Activitywatch-64 (Mini-Mitter Respironics, Inc., Bend, OR, USA) for four consecutive nights to objectively measure sleep. Each period began on a Saturday. Participants were instructed to register bedtime and wake time by pressing a button on the Activitywatch immediately before they went to bed and immediately after they woke up, respectively. These timestamps were cross-checked using adolescent self-report diaries. The validated Actiware Sleep software (version 3.4, Mini-Mitter/Philips Respironics) calculated various sleep parameters using 1-min epochs, based on significant movement after at least 10 min of inactivity (Oakley, 1997).

The software calculated sleep measures including bedtime (time when adolescent got into bed; M = 11:07 p.m.), sleep latency (lag from bedtime to sleep onset; M = 26 min), wake time (time at which the adolescent woke up for the last time in the morning; M = 7:00 a.m.), and sleep duration (time actually spent asleep from sleep onset to final waking; M = 6.54 hr). Adolescents completed a make-up day on Wednesday night if they missed a day of data collection. The adolescents had a mean of 3.4 nights of data out of an expected four nights.

Cortisol

Participants collected three salivary cortisol samples (immediately after waking, 30 min after
waking, and at bedtime) each day for three consecutive days (typically Sunday, Monday, and Tuesday) during the same period as the sleep study. They also collected an evening sample on Saturday, and wake and 30 min postawakening samples on the final day (typically Wednesday). Participants used a passive drool technique by which they expressed unstimulated saliva through a small straw into a vial. Participants watched a saliva sample demonstration prior to the study and received reminder calls from the research team during the data collection to ensure they followed the protocol. Participants were instructed to avoid eating, drinking, and brushing their teeth 30 min prior to each sample collection. A kitchen timer preset to 30 min was provided to aid in the timing of the second daily sample. Participants were instructed to refrigerate their samples as soon as possible after collection. Most samples were taken at home and thus immediately refrigerated. Adolescents returned their samples to school at the end of the study week, at which point the research team paid the participants. Six of the 82 sleep participants returned no usable saliva samples, leaving $N = 76$ cortisol participants. Cortisol participants completed 91% of expected samples. Samples were stored at $-20^\circ \text{C}$ before shipment to Trier, Germany, where they were assayed in duplicate using time-resolved

Table 1
Descriptive Statistics

<table>
<thead>
<tr>
<th></th>
<th>$M$</th>
<th>$SD$</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Weekday Cronbach's $\alpha$</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black</td>
<td>0.17</td>
<td>0.38</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Hispanic White</td>
<td>0.20</td>
<td>0.40</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Non-Hispanic White</td>
<td>0.18</td>
<td>0.39</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Multiethnic</td>
<td>0.27</td>
<td>0.45</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Other</td>
<td>0.18</td>
<td>0.39</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Age</td>
<td>14.90</td>
<td>1.86</td>
<td>11.27</td>
<td>18.11</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Male</td>
<td>0.51</td>
<td>0.50</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Violent crime ever = 1</td>
<td>0.51</td>
<td>0.50</td>
<td>0.00</td>
<td>1.00</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Mean bedtime</td>
<td>23.12</td>
<td>1.07</td>
<td>20.17</td>
<td>26.12</td>
<td>.72</td>
<td>82</td>
</tr>
<tr>
<td>Mean latency</td>
<td>0.43</td>
<td>0.47</td>
<td>0.04</td>
<td>3.03</td>
<td>.44</td>
<td>82</td>
</tr>
<tr>
<td>Mean sleep duration</td>
<td>6.54</td>
<td>0.87</td>
<td>3.98</td>
<td>9.13</td>
<td>.61</td>
<td>82</td>
</tr>
<tr>
<td>Mean wake time</td>
<td>7.00</td>
<td>0.98</td>
<td>4.89</td>
<td>10.33</td>
<td>.76</td>
<td>82</td>
</tr>
<tr>
<td>Mean waking cortisol</td>
<td>0.27</td>
<td>0.19</td>
<td>0.01</td>
<td>1.48</td>
<td>.88</td>
<td>76</td>
</tr>
<tr>
<td>Mean CAR</td>
<td>0.12</td>
<td>0.17</td>
<td>-0.23</td>
<td>0.65</td>
<td>.53</td>
<td>74</td>
</tr>
<tr>
<td>Mean bedtime cortisol</td>
<td>0.07</td>
<td>0.07</td>
<td>0.00</td>
<td>0.37</td>
<td>.66</td>
<td>76</td>
</tr>
</tbody>
</table>

Note. Sleep outcomes measured in hours. Cortisol outcomes measured in $\mu$g/dl. Includes Cronbach’s alpha of outcome measures during the week. CAR = cortisol awakening response.

Table 2
Correlation Matrix for Outcome Measures

<table>
<thead>
<tr>
<th></th>
<th>Bedtime</th>
<th>Latency</th>
<th>Sleep duration</th>
<th>Wake time</th>
<th>Bedtime cortisol</th>
<th>Waking cortisol</th>
<th>CAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bedtime</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Latency</td>
<td>−.261</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep duration</td>
<td>−.672</td>
<td>−.106</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wake time</td>
<td>.303</td>
<td>.073</td>
<td>.328</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedtime cortisol</td>
<td>−.073</td>
<td>.066</td>
<td>.002</td>
<td>.074</td>
<td>1.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waking cortisol (next day)</td>
<td>−.112</td>
<td>.056</td>
<td>.206</td>
<td>.227</td>
<td>.222</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>CAR</td>
<td>.145</td>
<td>−.230</td>
<td>−.204</td>
<td>−.235</td>
<td>−.154</td>
<td>−.457</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Note. Correlations between outcomes measured in the main analysis. Note that some measures are mathematically related to each other. CAR = cortisol awakening response.
fluorescent detection immunoassay (Dressendörfer, Kirschbaum, Rohde, Stahl, & Strasburger, 1992).

Measures included waking cortisol (cortisol level at wake; $M = .27 \mu g/dl$; mean samples per person = 3.7 out of an expected 4), CAR (increase from waking measurement to the measure occurring about 30 min later; $M = .12 \mu g/dl$; 3.2 of 4), and bedtime cortisol (cortisol level at bedtime; $M = .07 \mu g/dl$; 3.7 of 4). Analyses dropped any CAR samples taken more than 60 or less than 15 min after the waking sample. All 76 cortisol participants had at least one morning and bedtime sample, and 74 participants had data to calculate at least one CAR.

**Violent Crime**

Crime data were obtained from a geocoded file identifying the date and location of every violent crime reported to the city police department. We created an indicator variable equal to one if a violent crime occurred in a participant’s home police beat in a given day, from midnight to midnight. Police beats are subdivisions of police districts that represent areas that officers can patrol by foot. Our city is divided into over 250 police beats. We had 80 instances of violent crime (43 assaults, 31 robberies, 4 criminal sexual assaults, and 2 homicides) in a participant’s beat from Saturday through Wednesday of the study, with 51% of adolescents ($N = 42$) having at least one crime in their beat during their participation period. For sensitivity analyses, exact distances between participants’ addresses and crime locations were calculated.

**Empirical Strategy**

Our analysis uses individual fixed effects to measure the change in outcome from the person’s typical pattern on the day following a local violent crime, as follows:

$$Y_{it} = \alpha + \beta \text{Crime}_{it} + \nu_i + \gamma_t + \varepsilon_{it}$$

where $Y_{it}$ is the adolescent’s objectively measured sleep or cortisol on a given day, $\text{Crime}_{it}$ is an indicator for whether crime occurred the preceding day, $\nu_i$ is a fixed effect for the day of the week, $\gamma_t$ is an individual fixed effect, and $\varepsilon_{it}$ is an idiosyncratic error term. The parameter of interest is $\beta$; it captures the causal effect of exposure to crime on the outcome. The inclusion of individual fixed effects enables us to compare sleep and cortisol outcomes for the same adolescent in a day with proximity to crime relative to a day without proximity to crime. Only those adolescents who experience a nearby crime during the study period contribute to this estimate ($N = 42$). We control for day of the week to account for daily differences in sleep and cortisol patterns; participants with and without local violent crime contribute to this estimate. All standard errors are robust and clustered within individuals. We examine outcomes described in the Measures section in separate analyses.

Additional analyses add indicators for 2 and 3 days after the violent crime to measure the persistence of changes in the response to the acute crime event. We add an indicator for the day before a violent crime as a placebo test: Outcomes should not change in anticipation of a crime occurring the following day.

We also separate the analysis by type of violent crime (robbery, assault, criminal sexual assault, and homicide) to test whether responses differ by severity of crime. This analysis should be interpreted cautiously given the low $N$ for certain types of crime. We also examine whether using distance cutoffs rather than an indicator for crime within a police beat provides similar estimates.

**Results**

**Sleep**

Table 3 displays the sleep results. Because some measures are mechanically related to each other, these should be viewed as related outcomes rather than independent tests. From Panel A, adolescents had significantly later bedtime (by 26 min, $p = .043$) on evenings immediately following a violent crime in their police beat, relative to their typical bedtime, controlling for day of the week. A robustness check tested whether weekends (defined as Saturday night sleep) drove the results. When running the same estimate but excluding weekend sleep, the estimated bedtime effect was 38 min later ($p = .027$). The standard error was also larger, at least partly because the number of observations dropped from 281 to 211. This estimate did not statistically differ from the estimate including weekends.

The first four panels of Figure 1 display the effects of violent crime on sleep for days subsequent and prior to acute crime. Each chart was derived from a regression that included interactions between lagged indicators for whether a crime occurred within an individual’s police beat during a given day, as well as individual and day-of-the-
Participants’ bedtime increased on the night immediately following a crime ($p = .041$), but the effect dissipated by the second night. Day -1 tested a placebo: sleep should not have changed in anticipation of a crime occurring the following day. We found no evidence of an effect in the placebo days or for any of the other outcomes on any day.

Panel B in Table 3 displays the analysis by type of crime. Results should be interpreted with caution, given the small number of severe crimes (especially homicide and criminal sexual assault) during
the study. Homicide generated the strongest effect on sleep: bedtime increased by 1.81 hr \((p = .000)\) and total sleep decreased by 1.14 hr \((p = .000)\). Criminal sexual assault had a large effect on sleep latency (increased by 29 min, \(p = .026\)) and total sleep (decreased by 1.04 hr, \(p = .002\)). Coefficients for assault were generally large relative to Panel A, but the standard errors were large. Specifically, bedtime increased by 39 min \((p = .042)\). Effects for robbery were never statistically significant. As a check, we removed robbery from the Panel A estimate. This check resulted in larger estimated effects, but also larger standard errors for bedtime (estimated at 39 min, \(p = .030\)). The other estimates remained null.

In separate analyses, we tested whether adolescents who resided in high-crime areas were more sensitive to local violence. We defined high-crime areas as those that had an above-median number of violent crimes in their police beat in the months of the study. When we interacted acute exposure to crime \((\text{Crime}_s)\) with residence in high-crime areas, we found no difference in the estimated change in sleep across adolescents living in high- and low-crime areas. Given the small sample size, this test should be interpreted with caution.

**Cortisol**

Next, we reviewed the effects of violent crime on same-night bedtime, next-day waking, and next-day CAR cortisol measures (Table 4). We found a .13 \(\mu g/dl\) increase in next-day CAR in Panel A, representing a 111% increase over the mean CAR \((p = .025)\). We found no effects for other cortisol outcomes. Robustness checks that dropped two CAR outliers, controlled for prior night sleep and wake time, or excluded weekends (defined as Sunday morning) did not substantially change the results.

The final two panels of Figure 1 display cortisol effects over time. Each chart was derived from a regression that included interactions between lagged indicators for whether a crime occurred within an individual’s police beat during a given time period, as well as individual and day-of-the-week fixed effects. The effect was limited to the CAR on the day following violent crime \((p = .032)\), and effects dissipated by the next day. As in the sleep panels in Figure 1, prior-day placebo tests did not show anticipatory effects (see Day -1).

Panel B of Table 4 separates crime by type. Homicide was associated with the largest effects, with a .45 \(\mu g/dl\) increase in CAR the day following a homicide, relative to an individual’s typical CAR \((p = .000)\). Same-night cortisol also increased by a marginally significant .03 \(\mu g/dl\) \((p = .080)\). Assault was associated with a .22 \(\mu g/dl\) increase in CAR \((p = .011)\) and a marginally significant .09 \(\mu g/dl\) decrease in waking cortisol \((p = .059)\). Criminal sexual assault was associated with a .18 \(\mu g/dl\) increase in cortisol on the night of the crime \((p = .012)\). Effects for robbery were never statistically significant. Removing robbery from the Panel A estimate resulted in a larger coefficient for the effect on CAR (estimated at .22 \(\mu g/dl\), \(p = .003\)).

**Sensitivity to Distance of Crime From Home**

Our primary analyses used police beats as measures of local neighborhoods, as they account for local landmarks and roads that separate different localities. Here, we consider a violent crime as local if it occurred within various distance cutoffs from a participant’s home (see Figure 2; range = 0.33–1.00 miles). Each line represents the estimated coefficient from a separate regression. The figure includes five outcomes: bedtime, sleep latency, sleep duration, next-day waking cortisol, and next-day CAR.

More proximate crimes were necessarily rarer and generally had larger standard errors. Forty percent of adolescents experienced a violent crime within 0.33 miles of their home during their study participation, whereas the rate was 67% by 0.50 miles.

Coefficients were generally farther from zero at closer cutoffs. A violent crime within a half mile of

### Table 4

**Effect of Acute Violence on Cortisol Measures**

<table>
<thead>
<tr>
<th></th>
<th>Same-day cortisol</th>
<th>Next-day waking cortisol</th>
<th>Next-day CAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panel A: Overall violent crime</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crime</td>
<td>.005 (.019)</td>
<td>-.048 (.038)</td>
<td>.133 (.058)*</td>
</tr>
<tr>
<td>Panel B: Type of violent crime</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assault</td>
<td>-.019 (.028)</td>
<td>-.085 (.044)*</td>
<td>.221 (.085)*</td>
</tr>
<tr>
<td>Criminal sexual assault</td>
<td>.177 (.069)*</td>
<td>.056 (.119)</td>
<td>.190 (.119)</td>
</tr>
<tr>
<td>Homicide</td>
<td>.033 (.018)*</td>
<td>-.038 (.057)</td>
<td>.453 (.076)***</td>
</tr>
<tr>
<td>Robbery</td>
<td>.019 (.023)</td>
<td>-.020 (.041)</td>
<td>.019 (.051)</td>
</tr>
<tr>
<td>No. of adolescents</td>
<td>76</td>
<td>76</td>
<td>74</td>
</tr>
<tr>
<td>Observations</td>
<td>283</td>
<td>281</td>
<td>244</td>
</tr>
</tbody>
</table>

*Note. Robust standard errors clustered at the individual level and included in parentheses. Models include individual and day of the week fixed effects. Outcomes measured in \(\mu g/dl\).\*\* CAR = cortisol awakening response. *\(p < .10\). *\(p < .05\). ***\(p < .001\).
Discussion

We explored the relation between an acute external stressor (prior-day local violent crime) and two stress-responsive systems: sleep and the HPA axis. There were nearly 1.2 million violent crimes in the United States in 2015 (U.S. Department of Justice, 2016). Crimes may affect victims, but our study indicates that they may also affect nearby residents. Local prior-day violence disrupted sleep and increased next-day CAR. We argue that our findings are causal because, within a person, whether local violent crime happens to occur on a given day in our study is random from the perspective of each participant.

The effects on sleep and the CAR were generally large for homicide, moderate for assault, and null for robbery. These patterns could occur either because adolescents reacted more strongly to more violent crime or because news of such crimes spread more prominently through social networks, media, or police presence. The analysis by type of crime should be interpreted cautiously, given the low number of participants who experienced local criminal sexual assault or homicide in this study.

Estimated effect sizes for sleep were generally larger for more proximate crimes; such crimes may be more likely to be communicated or even directly overheard. The effects for CAR were not statistically significant across distance measures, making these results less robust than the sleep analyses. This difference may be partially related to differences in reliability across measures; the Cronbach’s alpha for CAR was .53, compared to .72 for bedtime.

Many of our estimates were not statistically significant. Latency did not change following a crime, perhaps because the students went to bed later. Wake time increased by a statistically insignificant 11 min. Inflexible school start times may prevent adolescents from adjusting their wake time. We
predicted an increase in same-day bedtime cortisol and a decrease in next-day waking cortisol, though neither was statistically significant.

A key limitation of our study is the low N. We view our results as preliminary, and we call for replication to confirm the patterns in larger samples. Larger sample sizes would also allow analysis of subgroup differences by gender and race/ethnicity. Moreover, the 18% decrease in sleep duration and 18% decrease in waking cortisol in the main specification are meaningfully sized, but the large standard errors prevented us from differentiating them from zero (p = .260 and .202, respectively). Future research should reexamine these outcomes.

Because our data do not allow identification of adolescents who were direct witnesses of violent events, our estimates are averaged across exposed and nonexposed adolescents. If the effect of violence is heterogeneous across these two types of adolescents (with direct witnesses arguably experiencing more stress), our estimates should be considered a lower bound for the effect that directly exposed adolescents would have experienced. Similarly, if robberies and assaults are underreported and measured with error, the true estimates would be larger in absolute value.

One way to test why crime affects adolescents is to test if the timing of crime matters. Evening crime mattering more than earlier crime, for instance, may provide more support for the opponent processes model of sleep (Edgar et al., 1993). In preliminary tests, crimes that occurred later in the day (after 4 p.m., when the child was more likely to be home) had larger effects on bedtime and sleep duration than the baseline specification, but we do not have the power to formally test this difference. Future research should investigate whether it is near-bedtime crime or crime that occurs while the child is home that most affects sleep.

Changes in puberty may change the association between acute stressors, sleep, and cortisol. An initial review found no differences in the association between acute violent crime and the stress response by pubertal status. Given the low N, this analysis should be interpreted with caution. For example, the CAR estimate is .05 μg/dl for participants at the lowest end of the pubertal scale (meaning the pubertal transition has not started) and .14 μg/dl for those at the top end of the scale (meaning the pubertal transition has completed). Although the difference between these estimates is not statistically significant, the difference is intriguing for future research, especially given prior research indicating that adolescence is associated with increased cortisol reactivity relative to children (e.g., Stroud et al., 2009). Future researchers with larger sample sizes should explore differences by pubertal status in more detail.

Future research should add objective measures of cognitive functioning to examine relations between semirandom stressful events, sleep, cortisol, and daily cognitive functioning. Such research may help uncover the mechanisms explaining some of the prior findings on acute local violence and performance on standardized tests (Gershenson & Tekin, 2015; Sharkey, 2010; Sharkey et al., 2012).

References


