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The impact of daily and trait loneliness on diurnal cortisol and sleep among children affected by parental HIV/AIDS

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ABSTRACT

Dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis and disruptions of restorative processes (e.g., sleep) have been proposed as two key mechanisms through which loneliness leads to medical morbidity in adults and late adolescents. Whether loneliness acts through these biological and behavioral intermediaries in children as well remains unexplored. In a sample of 645 children aged 8–15 affected by parental HIV/AIDS in rural China, trait and state (i.e., daily) loneliness were measured in a 3-day diary study, wherein participants also provided cortisol samples and sleep measures. Whereas high levels of trait loneliness were found to predict lower morning cortisol levels, longer time in bed, lower sleep quality, and a higher number of night awakenings, daily loneliness was associated with a flatter diurnal cortisol slope and shorter time in bed. Although the association between trait loneliness and daily loneliness with HPA activity remained significant after controlling for psychological constructs that overlap with loneliness (e.g., depression and daily negative affect), some of the associations between loneliness and sleep measures became non-significant after including these additional covariates. These findings provide the first empirical evidence to our knowledge of associations between trait and state loneliness and health-related outcomes among school-aged children and young adolescents.

1. Introduction

The "Pit of Despair" and "Well of Loneliness" were among the most common names given by psychologist Harry F. Harlow to the small vertical apparatus he used for his experiments on social isolation in rhesus macaques. The consequences of placing animals in these inescapable metal cages were lethal; in Harlow's words: "Placed in a free-living situation, most of these animals would be driven off or eliminated before they could have an opportunity to learn to adapt to the group" (Harlow et al., 1965).

In humans, social isolation, the measurable condition of having a withered social network (i.e., few and infrequent social contacts), is associated with broad adult medical morbidity and mortality. For example, receiving low social support has been associated with higher risk of heart disease (Barth et al., 2010), susceptibility to common respiratory illness (Cohen et al., 1997), and mortality (Eng et al., 2002). Similar findings have emerged in people reporting high levels of loneliness (Caspi et al., 2006; Patterson and Veenstra, 2010), the emotional discomfort associated with the perceived discrepancy between desired and available quality and quantity of social contacts. Despite being obviously related, social isolation and loneliness can have non-overlapping effects on health (Steptoe et al., 2013), especially when social isolation does not predict loneliness (e.g., Fees et al., 1999).

Although loneliness is a common experience, stigmatized populations, such those living with HIV, are at greater risk of feeling socially isolated. HIV-infected individuals are often socially rejected and must face recurrent discrimination across a variety of social realms, including work and health care settings (Nyblade et al., 2009). This chronic psychological stressor can have daunting mental and physical consequences not only for HIV-positive individuals, but also for their children, who predominantly depend on them, regardless of their HIV status (Chi et al., 2014). Children

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affected by parental HIV, in fact, are more often socially excluded by peers and family members, and report recurrent experiences of humiliation and reduced social support (Cluver et al., 2008). Understanding how the multifaceted construct of loneliness influences health outcomes among children affected by parental HIV has important implications for tailoring multilevel (individual, family, community) intervention programs in this population.

The overarching aim of the current study was to investigate the contribution of different aspects of loneliness to two biobehavioral intermediaries—sleep and cortisol secretion—associated with physical health in a group of children affected by parental HIV. From this perspective, a distinction can be made between trait loneliness, the chronic perception—accumulated over time—of inadequate quantity and quality of social relationships, and state (i.e., daily) loneliness, a transient and more circumstantial feeling (De Jong-Gierveld and Raadschelders, 1982). This distinction is particularly meaningful in diary studies, where data from participants are collected as they live through their quotidian life, as it allows the researcher to disentangle the effect of sustained loneliness (i.e., trait loneliness) as well as between people variability in transient experiences of loneliness (i.e., daily loneliness), which provide a more proximal picture of the individual feelings of isolation during the testing days (Doane and Adam, 2010). This approach is also important in light of the fact that daily loneliness may not strongly correlate with trait loneliness, especially during childhood, wherein the degrees of stability and fluidity of one's social network are more malleable (Cairns et al., 1995).

Alteration in the activity of the Hypothalamic-Pituitary-Adrenal (HPA) axis has emerged as one of the key mechanisms underlying the association between loneliness and health. For example, several cross sectional studies have found small—but consistent—positive associations between subjective feelings of social isolation and salivary cortisol (Cacioppo et al., 2000; Steptoe et al., 2004; Pressman et al., 2005; Edwards et al., 2010) and urinary cortisol (Kiecolt-Glaser et al., 1984; Hawkley et al., 2006). These findings have been confirmed with daily dairy data, wherein daily assessments of loneliness were prospectively associated with larger cortisol responses to awakening in adults and late adolescents (i.e. cortisol awakening response, or CAR, Adam et al., 2006; Doane and Adam, 2010; but see Sladek and Doane, 2015), while trait loneliness was associated with a flatter decline in diurnal cortisol in late adolescence (Doane and Adam, 2010; but see, Sladek and Doane, 2015, for a null association between trait loneliness and cortisol slope in the same age group). Despite the insights provided by these data, critical gaps remain unaddressed in this literature. To date, no work has investigated the link between loneliness and health-related biology in children. Extending the link between the HPA axis and loneliness in children is important for at least two reasons. Recent empirical evidence showed that loneliness at a young age is predictive of health risk factors in young adulthood (Caspi et al., 2006) and dysregulation in cortisol secretion might be a key biological intermediary through which these effects take place.

A second mechanism that has been recognized as crucial in mediating the effect of loneliness on physical health is the dysregulation of restorative processes (e.g., sleep) (Hawkley and Cacioppo, 2010). The intuitive association between sleep and various biological risk factors (for a reviews, see Irwin, 2015; Mullington et al., 2009) and, consequently, medical morbidity during adulthood (e.g., King et al., 2008) and mortality (Kripke et al., 2002) is well established. Within this framework, loneliness in adulthood has emerged as a reliable antecedent of poor sleep quality (Cacioppo et al., 2002b; Steptoe et al., 2004; Pressman et al., 2005) and disruptions in sleep continuity (Cacioppo et al., 2002a), but not sleep duration (Cacioppo et al., 2002b; Hawkley et al., 2010; Kurina et al., 2011). Studies on adolescents replicated and extended these findings by showing that not only was loneliness positively associated with sleep disturbances (e.g., number of night awakenings) (Mahon, 1994; Harris et al., 2013), but also with difficulty of falling asleep (i.e. sleep onset latency or SOL) (Harris et al., 2013). Overall, this pattern of results demonstrates the importance of measuring different sleep outcomes across subsequent days rather than single assessments at one point in time.

The aim of the current study was to investigate the impact of trait loneliness and daily loneliness on diurnal cortisol and daily reports of sleep in a large sample of children aged 8–15 years affected by parental HIV/AIDS in rural China. Loneliness among children in rural China is widespread (Chen et al., 2014) and, as mentioned above, this feeling can be further exacerbated among children whose parents belong to a stigmatized group, such as HIV infected individuals (Chi and Li, 2013). Working with this high-risk sample offers a unique opportunity to investigate the link between loneliness and biobehavioral health mechanisms among youth living in adversity.

2. Method

2.1. Participants

Seven hundred and ninety children aged 6–17 affected by parental HIV participated in a randomized controlled trial of a psychological intervention currently under way; the current investigation used baseline data (i.e., prior to intervention) drawn from this larger study. Of the larger sample of 790 children, 746 fit the inclusion criterion of 8 to 15 years of age, based on the age range for which the self-report measures used in the present analyses were normed. Further, of those 746 children, 645 (86.4%) provided valid saliva samples for cortisol analyses (48.1% female, age, $M = 10.67$ years, $SD = 1.79$ years) and were therefore used in the analyses. At the end of the study, each child received either toys or school supplies depending on their age as tokens of appreciation. Appropriate informed consent/assent was obtained before participation and all procedures received approval by the Institutional Review Boards at Wayne State University in the United States and Henan University in China.

2.2. Procedure

Children and their caregivers completed confidential survey questionnaires in Chinese. The survey included detailed measures of demographic information and several psychosocial scales. The majority of the child surveys were self-administrated in a small group in which two interviewers were present. The daily diary data collection occurred during the same time as daily saliva collection. From Thursday to Saturday, children were instructed in detail to fill the daily sleep diary immediately after the first saliva sample was collected, and to fill the daily mood diary (including momentary loneliness and other daily emotions). The primary caregiver was allowed to provide assistance during the collection of daily diary measures.

2.3. Measures

2.3.1. Trait loneliness

The Children's Loneliness Scale (CLS, Asher et al., 1984) was used to assess trait loneliness. An overall index ($M = 2.10$, $SD = 0.47$) is calculated by averaging children's self-reported scores on 16 items rated on a 4-point Likert scale, ranging from "Always True" to "Not True at All". Sample items included, "It's easy for me to make new friends at school"; "I feel alone" (reversed); "I have nobody to talk to" (reversed). The scale also contained 10 (8 in the original scale) additional filler items that are not included in the scoring. The high

reliability obtained in our sample ($\alpha = 0.78$) matches the strong reliability found in other non-clinical samples (Parkhurst and Asher, 1992), including Chinese children (Chen et al., 2004; Li et al., 2009; Du et al., 2014; Qiao et al., 2014).

2.3.2. Daily Sleep Outcomes

At the beginning of each day, participants were asked to report the time they woke up, the time they went to bed the night before, how many minutes it took them to fall asleep (i.e., SOL) ($M = 11.88$, $SD = 11.56$) and the number of night awakenings ($M = 1.20$, $SD = 0.98$). Total time in bed ($M = 10.11$ $SD = 1.25$) was calculated in hours by subtracting the time they woke up from the time they went to bed. Participants were also asked to rate, on a scale from 1 (very bad) to 4 (very good), their sleep quality ($M = 3.41$, $SD = 0.66$).

2.3.3. Daily loneliness and negative affect

At the end of each day, participants were asked to report, on a scale from 1 (not at all) to 3 (most of the day), how much they felt each of the following affective states: lonely, sad, upset, fear, angry, and worried. Responses on the "lonely" item were used for our measure of daily loneliness (across days, $M = 1.25$, $SD = 0.42$), while an average of the other moods were used for our measure of general negative affect (across days, $M = 1.37$, $SD = 0.37$) (for a similar approach, see Doane and Adam, 2010).

Depression. A short version of the Center for Epidemiologic Studies Depression Scale for children (Fendrich et al., 1990) was used to assess depression. Children were asked to report on a 4-point Likert scale (1 = not at all, 4 = a lot) how they felt or acted in the previous week. Examples of the 10 items include, "I was bothered by things that usually don't bother me," and "I felt like I was too tired to do things this past week" ($\alpha = 0.62$). Scores on this scale were obtained by summing the responses and were computed only for participants who answered all 10 items. Higher scores indicated higher depression symptoms ($M = 20.17$, $SD = 4.29$).

2.3.4. Perceived health status

We also controlled for perceived health status, which was self-reported by each youth and by their caregiver on a 5-point Likert-type scale ranging from 1 (very poor) to 5 (very good). This scale was constructed by calculating the mean of the two items and was computed for cases that had valid values on both items. Higher scores indicate higher perceived health status ($M = 4.1$, $SD = 0.75$).

2.3.5. Parental death

One-hundred and twelve children lost at least one of their parents, while 495 had both parents still alive. Data for this variable were not available for 38 children.

2.3.6. Other stressful life events

Fifteen items were used to assess children's experience of a number of stressful life events. Children reported whether these events occurred during the past six months ($M = 2.47$, $SD = 2.12$). Sample items included being in a traffic accident, being a witness to involuntary violence, hospitalization, natural disaster, severe sickness or death of friends, relocation of the family, and death of family members.

2.3.7. Additional covariates

Other covariates included age ($M = 10.67$, $SD = 1.79$), gender (48.1% female), family socioeconomic status (SES), which was derived by adding together caregiver education and family income after having *z* scored them ($M = 0.01$, $SD = 1.53$), daily wake up time, and day of the week (weekday = 0, weekend = 1). At the momentary level (i.e., at collection time of each saliva sample), youths reported whether they smoked or practiced any sport. Missing cases for these two variables were replaced by the mode. Variables listed in this section are standard covariates in diurnal cortisol studies (Adam et al., 2006).

2.3.8. Cortisol

Participants self-collected saliva samples at four time points each day for three days: immediately upon waking (prior to any eating, drinking, or exercise), thirty minutes later to assess cortisol awakening response (CAR), one hour before dinnertime, and then at bedtime. Prior to saliva collection, the investigators showed children the correct procedure to collect saliva samples using Salivettes (Sarstedt, Rommelsdorft, Germany) and emphasized the importance of compliance with the time of collection. Salivettes were stored at room temperature before being returned to the researchers, who refrigerated them until the day of the assay at Huaihe Hospital. Prior work has shown that Salivette storage at room temperature for as long as two weeks (much longer than in this study) does not adversely affect cortisol concentration (Garde and Hansen, 2005). Participants provided a total of 11.17 out of 12 samples on average ($SD = 1.60$), with 93% of all possible saliva samples collected. Altogether, 61.3% of participants did not miss any samples, with 90.4% providing between 10 and 12 samples, and 96% of participants providing at least 8 of the 12 possible saliva samples across the 3 days. Cortisol levels were determined via chemiluminescent immunoassay (Access Cortisol kit YZB/USA 2802, Beckman Coulter, Inc, Fullerton, CA). Cortisol values were natural log-transformed to correct for positive skew in the cortisol distribution (Adam and Kumari, 2009). In order to ensure that all transformed scores were positive, a constant of 1 was added before the transformation.

2.4. Statistical analyses

The incidence of missing data was 8.67% at the daily level (sleep analyses) and 5.4% at the person levels (sleep analyses and cortisol analyses). In order to curtail the bias associated with pairwise or listwise deletion of missing data (Collins et al., 2001), we used the expectation maximization (EM) algorithm to impute missing data. Estimates obtained with this approach are less biased than estimates obtained with ad hoc methods (e.g., listwise deletion of missing data) (Schafer and Graham, 2002; Peng et al., 2006; see also Harris et al.'s work on loneliness for a similar approach; Harris et al., 2013). Because this algorithm does not allow value replacement for categorical data, mode imputation was used to replace missing cases for parental death ($n = 38$).

2.5. Inferential statistics

2.5.1. Cortisol analyses

Hierarchical Linear Modeling (HLM) was employed in order to properly account for the strong diurnal rhythm of cortisol. HLM allows for the simultaneous estimation of more than one cortisol parameter (e.g., wakeup values, CAR, and slope) along with the prediction of individual differences in diurnal cortisol profiles. Based on previous diurnal cortisol research (Adam and Kumari, 2009), Time Since Waking, Time Since Waking-squared, and CAR (dummy coded 0 and 1) were modeled at Level 1 in order to estimate each participants' diurnal cortisol profile. In our sample CAR observations that deviated by 10 min or more from the requested 30-min interval were dropped from the analyses. At Level 2 (person-level), we tested the effect of trait and daily loneliness (i.e., average of loneliness across the three testing days). Analyses were conducted without controlling for covariates (Model 1), controlling only for demographic covariates (age, gender, family SES, average wakeup time, sport, smoke, parental death, stressful events, and health status) (Model 2), and then controlling for both demographics

and psychological covariates (depression and daily negative affect) (Model 3). Cortisol intercept, slope (effect of time), and CAR were all allowed to vary randomly at Level-2, while Time Since Waking-squared was considered as a fixed effect with no Level-2 predictors. Age was centered such that a score of zero would represent the youngest children in the sample (i.e., age 8), while stressful life events was left uncentered (0 = no stressful life events). All other continuous person-level variables were grand-mean centered. All significance tests were two-tailed ($\alpha = 0.05$) with robust standard errors.

2.5.2. Sleep outcomes analyses

Given the nested nature of our sleep data (i.e. days within people), hierarchical linear modeling (HLM) was used for data analyses. For each sleep outcome (subjective sleep quality, SOL, number of awakenings, total time in bed), analyses were conducted without controlling for covariates (Model 1), controlling only for demographics covariates (age, gender, family SES, weekend, parental death, stressful events, and health status) (Model 2), and then controlling for both demographics and psychological covariates (depression and daily negative affect) (Model 3). The intercept of each model was allowed to vary randomly at the day level (e.g., treated as random effects). All significance tests were two-tailed ($\alpha = 0.05$) with robust standard errors.

3. Results

3.1. Cortisol analyses

Interestingly, trait loneliness was only weakly associated with daily loneliness ($r = 0.119$, $p = 0.002$) and daily negative affect ($r = 0.113$, $p = 0.004$), but strongly associated with depression ($r = 0.497$, $p < 0.001$). Daily loneliness was associated strongly with daily negative affect ($r = 0.629$, $p < 0.001$) and moderately with depression ($r = 0.212$, $p < 0.001$). As reported in Table 1, Model 1, trait loneliness was associated with lower morning cortisol ($t = -2.678$, $p = 0.008$), while average daily loneliness was associated with a flatter cortisol slope ($t = 2.384$, $p = 0.017$), suggesting that those individual who experienced more daily loneliness across the three testing days had a less steep diurnal decline in cortisol (Fig. 1). No effects were found for CAR, which overall was not statistically significant in this sample. These effects remained significant once we controlled for demographic covariates (Model 2, Table 1) and demographics and psychosocial covariates (Model 3, Table 1).

3.2. Sleep outcomes analyses

As shown in Table 2, Model 1 and Model 2, high trait loneliness was associated with low sleep quality ($t = -2.612$, $p = 0.009$; $t = -2.451$, $p = 0.014$, after controlling for demographic covariates) and high self-reported number of night awakenings ($t = 3.897$, $p < 0.001$; $t = 3.113$, $p = 0.002$, after controlling for demographic covariates). Similarly, average daily loneliness was associated with a higher number of night awakenings ($t = 2.007$, $p = 0.045$; $t = 2.195$, $p = 0.028$, after controlling for demographic covariates). Average daily loneliness was also associated with lower sleep quality ($t = -2.042$, $p = 0.042$), but this relationship disappeared after controlling for demographic covariates ($t = -1.468$, $p = 0.143$). Trait loneliness was associated with more time in bed ($t = 3.747$, $p < 0.001$; $t = 2.524$, $p = 0.012$, after controlling for demographic covariates), while the opposite was true for average daily loneliness ($t = -2.596$, $p = 0.010$; $t = -2.084$, $p = 0.038$, after controlling for demographic covariates). None of the measures of loneliness was predictive of SOL.[1]

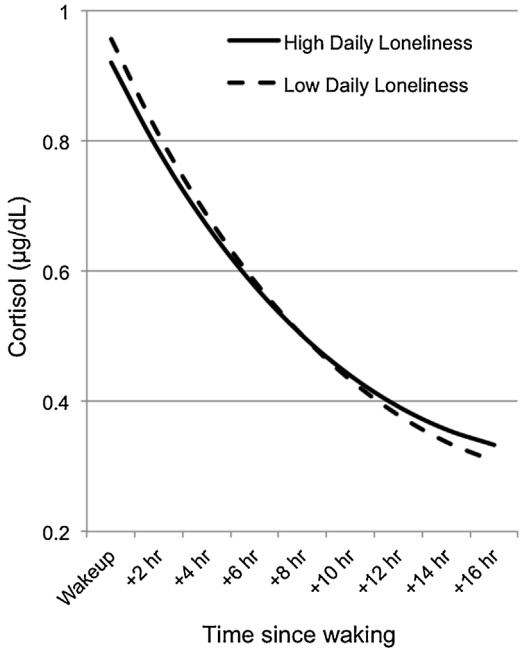

Fig. 1. Associations between daily loneliness and diurnal cortisol. Cortisol levels (µg/dL) is graphed as a function of time since waking, separately for children that reported low (1 SD below the mean) and high (1 SD above the mean) daily loneliness.

Notably, all these effects, except for the negative association between trait loneliness and number of awakenings and the positive association between trait loneliness and time in bed, disappeared when we controlled for demographic and psychosocial covariates (Table 2, Model 3), possibly because of the strong overlap between loneliness and depression and daily negative affect.

4. Discussion

Two main results emerged from our analyses of cortisol. First, high levels of trait loneliness—but not daily loneliness—were found to predict lower morning cortisol levels. Second, between-person variation in daily loneliness was associated with variation in diurnal cortisol profiles, such that those people reporting high loneliness across the study days experienced a flatter diurnal slope. These results remained significant after controlling for demographic covariates as well as psychological constructs that overlap with loneliness, namely depression and daily negative affect.

The paucity of studies that have looked at the relationship between loneliness and diurnal cortisol across days thwarts any attempt to derive a coherent picture of the most robust findings within this literature. For example, Doane and Adam (Doane and Adam, 2010) found that chronic loneliness was associated with a flatter diurnal cortisol slope in sample of 108 mostly female older adolescents, while the same association was not found by Sladek and Doane in a smaller, but demographically comparable, sample (Sladek and Doane, 2015). In our study, although trait loneliness was not associated with a flattened cortisol circadian rhythm, we found that children reporting high levels of daily loneliness—averaged across the three testing days—had a flatter cortisol slope. These results mirror work by Pressman et al. (Pressman et al., 2005), who found that high daily loneliness was

[1] SOL was strongly skewed, so analyses were re-run using log-transformed values instead of raw data. Notably, results did not change.

Table 1
HLM Models of Diurnal Cortisol Parameters.

	Model 1			Model 2			Model 3		
Fixed effect (independent variable)	Estimate	SE	*P*	Estimate	SE	*P*	Estimate	SE	*P*
Morning cortisol, π_0									
Average Morning Cortisol (Intercept), β_{00}	0.6620	0.0064	<0.001	0.6571	0.0164	<0.001	0.6569	0.0162	<0.001
Trait Loneliness, β_{01}	−0.0368	0.0137	0.008	−0.0329	0.0140	0.019	−0.0320	0.0158	0.043
Daily Loneliness, β_{02}	−0.0236	0.0152	0.120	−0.0234	0.0154	0.128	−0.0217	0.0197	0.272
Female, β_{03}	–	–	–	−0.0071	0.0131	0.590	−0.0072	0.0132	0.585
Parental death, β_{04}	–	–	–	−0.0137	0.0174	0.430	−0.0137	0.0174	0.431
Health status, β_{05}	–	–	–	−0.0017	0.0085	0.843	−0.0018	0.0086	0.837
Life events, β_{06}	–	–	–	−0.0032	0.0027	0.241	−0.0031	0.0027	0.254
Family SES β_{07}	–	–	–	0.0016	0.0041	0.693	0.0016	0.0041	0.698
Age, β_{08}	–	–	–	0.0057	0.0037	0.130	0.0057	0.0037	0.127
Wakeup time, β_{09}	–	–	–	−0.0003	0.0105	0.978	−0.0003	0.0105	0.975
Daily Negative Affect, β_{010}	–	–	–	–	–	–	−0.0029	0.0247	0.906
Depression, β_{011}	–	–	–	–	–	–	−0.0002	0.0017	0.907
Cortisol Awakening Response, π_1									
Average CAR, β_{10}	0.0023	0.0069	0.740	−0.0059	0.0155	0.705	−0.0056	0.0154	0.716
Trait Loneliness, β_{11}	−0.0057	0.0129	0.658	−0.0029	0.0132	0.826	−0.0037	0.0149	0.801
Daily Loneliness, β_{12}	−0.0029	0.0161	0.858	−0.0083	0.0162	0.609	−0.0093	0.0199	0.638
Female, β_{13}	–	–	–	−0.0311	0.0136	0.022	−0.0310	0.0135	0.022
Parental death, β_{14}	–	–	–	0.0178	0.0179	0.320	0.0178	0.0179	0.320
Health status, β_{15}	–	–	–	0.0077	0.0099	0.434	0.0078	0.0099	0.432
Life events, β_{16}	–	–	–	−0.0001	0.0034	0.979	−0.0002	0.0035	0.957
Family SES β_{17}	–	–	–	−0.0044	0.0042	0.296	−0.0044	0.0042	0.296
Age, β_{18}	–	–	–	0.0067	0.0037	0.071	0.0067	0.0037	0.070
Wakeup time, β_{19}	–	–	–	−0.0048	0.0129	0.710	−0.0047	0.0129	0.714
Daily Negative Affect, $\beta 1_{110}$	–	–	–	–	–	–	0.0017	0.0255	0.949
Depression, $\beta 1_{111}$	–	–	–	–	–	–	0.0002	0.0018	0.911
Time Since Waking, π_2									
Average Linear Slope, β_{20}	−0.0403	0.0020	<0.001	−0.0389	0.0022	<0.001	−0.0389	0.0022	<0.001
Trait Loneliness, β_{21}	0.0017	0.0012	0.162	0.0013	0.0012	0.288	0.0011	0.0013	0.402
Daily Loneliness, β_{22}	0.0034	0.0014	0.017	0.0033	0.0014	0.020	0.0038	0.0019	0.041
Female, β_{23}	–	–	–	−0.0008	0.0011	0.489	−0.0008	0.0011	0.506
Parental death, β_{24}	–	–	–	0.0020	0.0016	0.204	0.0020	0.0016	0.204
Health status, β_{25}	–	–	–	−0.0001	0.0007	0.890	−0.0001	0.0007	0.867
Life events, β_{26}	–	–	–	0.0002	0.0003	0.526	0.0002	0.0003	0.537
Family SES β_{27}	–	–	–	−0.0002	0.0004	0.702	−0.0002	0.0004	0.675
Age, β_{28}	–	–	–	−0.0006	0.0003	0.055	−0.0006	0.0003	0.054
Wakeup time, β_{29}	–	–	–	−0.0014	0.0009	0.145	−0.0014	0.0009	0.144
Daily Negative Affect, β_{210}	–	–	–	–	–	–	−0.0010	0.0023	0.658
Depression, β_{211}	–	–	–	–	–	–	0.0000	0.0002	0.742
Time Since Waking^2, π_3									
Average Curvature, β_{30}	0.0011	0.0001	<0.001	0.0011	0.0001	<0.001	0.0011	0.0001	<0.001
Smoke, π_4									
Intercept, β_{40}	–	–	–	0.1558	0.0442	<0.001	0.1566	0.0444	<0.001
Exercise, π_5									
Intercept, β_{50}	–	–	–	0.0227	0.0086	0.008	0.0229	0.0086	0.008

Note: Intercepts indicate average cortisol values at wakeup; average slopes of time since waking indicate change in cortisol per 1-h change in time; average slopes of time since waking^2 indicate change in cortisol per 1-h change in time^2. CAR = Cortisol Awakening Response. SES: Socioeconomic Status.

associated with high evening cortisol, and complement work done among young adults by Stetler et al. (Stetler et al., 2004), who showed that days with more social activities were associated with a more normative (i.e., steeper) cortisol daily decline compared with days where social contact was less present.

Blunted cortisol levels at awakening were observed among children high in trait loneliness. Previous research in youth has shown that low morning cortisol—a result of the overnight down-regulation of an overactive HPA axis—is often observed in tandem with severe neglect (Dozier et al., 2006; Bruce et al., 2009) and maladaptive coping (Slatcher et al., 2015). Because these psychological manifestations are also recurrent among chronically lonely individuals (Cacioppo et al., 2000; Cornman et al., 2003), the association between low morning cortisol at awakening and loneliness found in our study can be read in light of this literature.

Lastly, inconsistent with the existing literature, we did not find any covariation between any measures of loneliness and CAR. A few reasons might lay behind this null effect. First, the directionality of the relationship between loneliness and CAR is not very clear, as evidence for both a positive association (i.e. greater loneliness/larger CAR) (Steptoe et al., 2004; Adam et al., 2006; Doane and Adam, 2010) and a negative association (i.e. greater loneliness/smaller CAR) have accumulated (Sladek and Doane, 2015). Second, previous work suggests that CAR might be less accentuated in youth at risk, especially during the early phase of puberty (Quevedo et al., 2012). This explanation, along with potential problems with compliance might explain why the effect of CAR in our sample was found to be in the opposite direction that expected. These aspects might have contributed to decrease the variability and range in CAR and/or inflate the error variance associated with it, and consequently obscured any modulatory effects of third variables (e.g., loneliness) on CAR.

Turning to the sleep results, we found that children with high levels of trait loneliness reported lower sleep quality and a higher number of night awakenings. Daily loneliness also predicted a greater number of night awakenings. These findings were inde-

Table 2
HLM Models of Sleep Outcomes.

	Model 1			Model 2			Model 3		
Fixed effect (independent variable)	Estimate	SE	*P*	Estimate	SE	*P*	Estimate	SE	*P*
Time in bed, π_0									
Average Time in Bed (Intercept), β_{00}	10.1238	0.0360	<0.001	10.4030	0.0831	<0.001	10.3925	0.0835	<0.001
Trait Loneliness, β_{01}	0.3089	0.0824	<0.001	0.1938	0.0768	0.012	0.2229	0.0889	0.012
Daily Loneliness, β_{02}	−0.2385	0.0919	0.010	−0.1838	0.0882	0.038	−0.0955	0.1149	0.406
Female, β_{03}	–	–	–	−0.0404	0.0694	0.560	−0.0436	0.0698	0.532
Parental death, β_{04}	–	–	–	−0.0541	0.1021	0.596	−0.0520	0.1020	0.611
Health status, β_{05}	–	–	–	−0.0513	0.0446	0.251	−0.0541	0.0444	0.223
Life events, β_{06}	–	–	–	0.0087	0.0176	0.620	0.0125	0.0181	0.491
Family SES β_{07}	–	–	–	−0.0154	0.0260	0.554	−0.0170	0.0258	0.511
Age, β_{08}	–	–	–	−0.1571	0.0199	<0.001	−0.1563	0.0198	<0.001
Daily Negative Affect, β_{09}	–	–	–	–	–	–	−0.1494	0.1400	0.286
Depression, β_{010}	–	–	–	–	–	–	−0.0064	0.0099	0.519
Weekend, β_{10}	–	–	–	0.4448	0.0464	<0.001	0.4448	0.0464	<0.001
SOL, π_0									
Average SOL (Intercept), β_{00}	11.8929	0.3973	<0.001	10.9313	0.8736	<0.001	10.9747	0.9043	<0.001
Trait Loneliness, β_{01}	−0.1615	0.8299	0.846	0.0275	0.8449	0.974	0.3680	1.0800	0.733
Daily Loneliness, β_{02}	0.0311	0.8780	0.972	−0.2731	0.9086	0.764	−1.3752	1.5227	0.367
Female, β_{03}	–	–	–	−0.4118	0.7982	0.606	−0.4579	0.7923	0.564
Parental death, β_{04}	–	–	–	0.7329	1.3129	0.577	0.7337	1.3189	0.578
Health status, β_{05}	–	–	–	−0.3930	0.4780	0.411	−0.3516	0.4813	0.465
Life events, β_{06}	–	–	–	0.1828	0.2099	0.384	0.1785	0.2072	0.389
Family SES β_{07}	–	–	–	0.5107	0.4392	0.245	0.5434	0.4437	0.221
Age, β_{08}	–	–	–	0.2510	0.2266	0.269	0.2469	0.2283	0.280
Daily Negative Affect, β_{09}	–	–	–	–	–	–	2.3238	1.8952	0.221
Depression, β_{010}	–	–	–	–	–	–	−0.0916	0.1323	0.489
Weekend (Intercept), β_{10}	–	–	–	−0.2702	0.2356	0.252	−0.2702	0.2356	0.252
Sleep quality, π_0									
Average Sleep quality (Intercept), β_{00}	3.411	0.018	<0.001	3.5094	0.0458	<0.001	3.5038	0.0460	<0.001
Trait Loneliness, β_{01}	−0.102	0.039	0.009	−0.0981	0.0400	0.014	−0.0719	0.0461	0.119
Daily Loneliness, β_{02}	−0.096	0.047	0.042	−0.0681	0.0464	0.143	−0.0375	0.0597	0.530
Female, β_{03}	–	–	–	0.0137	0.0357	0.700	0.0106	0.0357	0.766
Parental death, β_{04}	–	–	–	0.0538	0.0514	0.295	0.0552	0.0512	0.281
Health status, β_{05}	–	–	–	0.0993	0.0245	<0.001	0.0984	0.0244	<0.001
Life events, β_{06}	–	–	–	−0.0211	0.0093	0.024	−0.0188	0.0094	0.047
Family SES β_{07}	–	–	–	0.0092	0.0121	0.447	0.0090	0.0120	0.457
Age, β_{08}	–	–	–	−0.0322	0.0102	0.002	−0.0317	0.0102	0.002
Daily Negative Affect, β_{09}	–	–	–	–	–	–	−0.0414	0.0691	0.549
Depression, β_{010}	–	–	–	–	–	–	−0.0061	0.0050	0.224
Weekend, β_{10}	–	–	–	0.0721	0.0255	0.005	0.0721	0.0255	0.005
Night awakenings, π_0									
Average Night awakenings (Intercept), β_{00}	1.194	0.028	<0.001	1.2894	0.0702	<0.001	1.2910	0.0703	<0.001
Trait Loneliness, β_{01}	0.234	0.060	<0.001	0.1900	0.0610	0.002	0.1904	0.0697	0.006
Daily Loneliness, β_{02}	0.153	0.076	0.045	0.1664	0.0758	0.028	0.1456	0.0877	0.097
Female, β_{03}	–	–	–	0.0237	0.0559	0.671	0.0236	0.0562	0.675
Parental death, β_{04}	–	–	–	0.0676	0.0798	0.397	0.0674	0.0797	0.398
Health status, β_{05}	–	–	–	−0.0286	0.0386	0.459	−0.0279	0.0385	0.469
Life events, β_{06}	–	–	–	0.0050	0.0138	0.715	0.0046	0.0142	0.746
Family SES β_{07}	–	–	–	−0.0307	0.0196	0.118	−0.0302	0.0196	0.124
Age, β_{08}	–	–	–	−0.0441	0.0165	0.008	−0.0442	0.0164	0.007
Daily Negative Affect, β_{09}	–	–	–	–	–	–	0.0400	0.1022	0.696
Depression, β_{010}	–	–	–	–	–	–	−0.0003	0.0079	0.975
Weekend (Intercept), β_{10}	–	–	–	−0.0380	0.0343	0.268	−0.0380	0.0343	0.268

Note: SES: Socioeconomic Status. SOL: Sleep Onset Latency.

pendent of individual differences in self-reported health, recent stressful events, and other demographic variables, and corroborate the well-established link between loneliness and diminished sleep quality (Cacioppo et al., 2002b; Steptoe et al., 2004; Pressman et al., 2005) and augmented night awakenings (Cacioppo et al., 2002a). The general consensus in the field is that trait loneliness does not impair sleep duration among older adolescents and adults (Mahon, 1994; Hawkley et al., 2010; Kurina et al., 2011; but see, Cacioppo et al., 2000, for a positive relationship between the two). In contrast with this literature, children in our study who scored higher on trait loneliness also reported more time in bed. One possible explanation for the positive association between trait loneliness and sleep duration is that the former causes distress and fatigue (Jaremka et al., 2013) and heighten sensitivity to pain (Oishi et al., 2013), and may lead the individual to sleep longer in the attempt to recover from chronic loneliness-related (e.g., ostracism) demands. Longer sleep duration might also be a way to offset the physiological (Pressman et al., 2005) and immune (Jaremka et al., 2013) costs associated with loneliness. Another possibility is that sleep duration is simply a byproduct of a coping strategy that relies on social withdraw as the main behavior to deal with the emotional adversities experienced during the day (Cacioppo et al., 2000). However, in our study we also found that high levels of daily loneliness were associated with less time in bed, suggesting that transient (i.e., daily) feelings of loneliness might lead to shorter sleep duration. One possibility is that daily loneliness ratings partially overlapped with the frequency and severity of daily stressors, which have been showed to

lead to shorter sleep duration in precarious family environments (Hanson and Chen, 2010).

An important caveat about our sleep results was that, except for the trait loneliness/number of awakenings link and the trait loneliness/time in bed link, the significance of the presented associations disappeared after controlling for psychological constructs overlapping with loneliness, namely depression and daily negative affect. Interestingly, however, neither negative affect nor depression was associated with sleep disturbances, suggesting a multicollinearity issue. Future research is needed to more comprehensively understand the specific contribution of each construct by collecting data across a wider span (e.g., weeks) than the current study (i.e., days) as a way to reduce multicollinearity.

Both dysregulation of the HPA axis, in the form of morning hypocortisolism and flattening of the diurnal cortisol rhythm, and disruptions of restorative processes lead to adverse health outcomes (Goodyer et al., 1996; Gunnar and Vazquez, 2001; Bower et al., 2005; King et al., 2008; Matthews et al., 2006), including mortality (Kripke et al., 2002; Kumari et al., 2011). Accumulating research among adults and older adolescents point at these two mechanisms as the main pathways through which loneliness leads to medical morbidity and mortality. However, prospective studies have showed that the cumulative detrimental effects of loneliness on physical health start at a young age (Caspi et al., 2006; Harris et al., 2013). Notably, our study provides the first empirical evidence that some of these negative effects of loneliness might be mediated by alterations in cortisol secretion and sleep.

Our work is not without limitations. First, the restricted number of sampling days (i.e., 3) precluded us from modeling within-person changes in loneliness and testing its relations to HPA axis functioning and sleep. Likewise, the decision to implement two-level multilevel models prevented us from modeling daily predictors (e.g., weekday) in cortisol analyses. Future studies should include loneliness, cortisol, and sleep assessments over a greater number of days to fully capture the complex relationships among our study variables. That said, the limited sampling regime was offset by a large sample, which lends increased confidence about the current findings. Second, because of the correlational nature of our study, some of the relationships between loneliness and sleep outcomes we observed could be explained in terms of sleeping habits influencing loneliness. For example, it is possible that spending more time in bed, either sleeping or engaging in ludic activities, may lead to more opportunities for children to socially isolate themselves. Thus, future studies involving multiple and longitudinal sleep and loneliness measures will be necessary before stronger causal claims could be made. Third, one trade-off in our study was the reliance on self-reported measures of sleep in favor of a large sample size. Despite being widely used, subjective reports of sleep disturbances might deviate from objective measures of sleep duration and quality (Bertocci et al., 2005; Tremaine et al., 2010). For example, in their study, Bertocci et al. (2005) found no difference in objective EEG-recorded number of awakenings between children with major depressive disorder and matched controls, despite the fact that the former group self-reported a greater number of awakenings than the latter group. Thus, it is important for future studies to replicate our findings by using more sophisticated sleep measures, such as actigraphy (Sadeh et al., 1995). Fourth, because the age range of our sample was broad (8–15 years old), children at presumably different stages of development (e.g., older youths undergoing puberty) were included. This weakness of our study should be considered in future research aiming at understanding the role played by developmental switch points on cortisol modulation. A last caveat to consider in our study concerns the uniqueness of our sample, which might limit the generalizability to other populations.

To summarize, in a sample of 645 children affected by parental HIV, we found that high levels of trait loneliness predicted lower morning cortisol levels, longer time in bed, lower sleep quality, and a higher number of night awakenings, while daily loneliness was associated with a flatter diurnal cortisol slope and shorter time in bed. Although the association between trait loneliness and daily loneliness with HPA activity remained significant after controlling for psychological constructs that overlap with loneliness (i.e., depression and daily negative affect), some of the associations between loneliness and sleep measures disappeared after including these additional covariates. We hope that the results of our study can help guide future intervention studies and stimulate further research among high-risk populations on the biobehavioral pathways through which loneliness exerts its effects on health across the lifespan.

Author contributions

S.Z., R.B.S., P.C., X.L., J.Z., G.Z.: study conceptualization. S.Z.: data analyses. S.Z. and R.B.S.: writing. S.Z., R.B.S., P.C., X.L.: editing.

Conflict of interest

None declared.

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