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Reciprocal Relations between Parental Problem Drinking and Children's Sleep: The Role of Socioeconomic Adversity

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Abstract

Reciprocal relations between parental problem drinking and children's sleep were examined longitudinally and socioeconomic status was considered as a moderating variable. At wave 1, 280 children (*M* age = 10.33) and their parent(s) participated and 275 families returned one year later. At both waves, parent(s) reported on problem drinking and children wore actigraphs that measured established sleep parameters. After controlling for autoregressive effects, fathers' problem drinking predicted reduced sleep duration and efficiency in children over time. Supportive of reciprocal effects, more frequent long wake episodes predicted greater parental problem drinking. Fathers' problem drinking was a more robust risk factor for lower than higher income children. Results build on a growing literature that has considered children's sleep in a family context.

Keywords

Parental problem drinking; children's sleep; actigraphy; health disparities; longitudinal study

A growing literature has considered children's sleep in a family context (see El-Sheikh & Sadeh, 2015). Accumulating evidence indicates that many forms of family risk including marital (Insana, Foley, Montgomery-Downs, Kolko, & McNeil, 2014) and parent-child (Kelly, Marks, & El-Sheikh, 2014) conflict, attachment insecurity to parents (Belanger, Bernier, Simard, Bordeleau, & Carrier, 2015), reduced maternal emotional availability (Teti, Kim, Mayer, & Countermine, 2010) and parental psychopathology (Bernier, Belanger, Bordeleau, & Carrier, 2013) are robust correlates of sleep problems among children. Collectively, existing evidence has provided increased understanding of family factors that influence children's sleep and has underscored the importance of considering family processes when assessing sleep in youth.

Despite these advances, critical questions remain in the growing literature linking family functioning with children's sleep. Although the number of longitudinal studies is encouragingly on the rise (Belanger et al., 2015; Staples, Bates, & Petersen, 2015), the vast

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majority of pertinent studies have relied on cross-sectional designs (see El-Sheikh & Sadeh, 2015). Further, very few studies have considered directionality of effects between family processes and children's sleep. Explorations of transactional dynamics are imperative for clarifying pathways of effects and research calls have been placed to develop and test models that examine reciprocal relations between family functioning and sleep (El-Sheikh & Buckhalt, 2015). In this study, we investigated bidirectional associations between children's sleep and parental problem drinking (PPD) longitudinally.

Parental problem drinking refers broadly to several alcohol-related problems including dependence, abuse and other maladaptive drinking behaviors (Windle, 1997). The prevalence of PPD is of major public health concern with estimates indicating that over seven million U.S. youth under the age of 18 live with a parent who has an alcohol-related problem (Substance Abuse and Mental Health Services Administration, 2012). Other estimates indicate that over 40% of individuals are exposed to PPD at some point during their youth (Grant, 2000). PPD has been associated with many negative outcomes in children and adolescents including poor cognitive functioning (Diaz et al., 2008), socio-emotional problems (Keller, Cummings, Davies, & Mitchell, 2008) and externalizing behaviors (Finan, Schulz, Gordon, & Ohannessian, 2015). PPD has rarely been investigated in relation to children's sleep.

Sleep is a multi-faceted construct (Sadeh, 2015) and assessment of various parameters is imperative for explicating the sleep domains most strongly associated with PPD. Using actigraphs, we derived a commonly used measure of sleep duration, namely *sleep minutes* (refers to the number of minutes spent asleep between falling asleep and wake time). Three established indicators of sleep quality were also examined (Ohayon et al., 2017): *Sleep efficiency* (percentage of minutes spent asleep between falling asleep and wake time), *long wake episodes* (number of wake episodes greater than 5 minutes) and *sleep onset latency* (duration between bedtime and sleep onset time). Deficiencies in such parameters of sleep occur often in childhood (Sadeh, Gruber, & Raviv, 2002; Sadeh, Raviv, & Gruber, 2000) and are known risk factors for many developmental domains including adjustment (Sadeh et al., 2002) and academic performance (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010). Assessments of relations between PPD and sleep problems could thus have important implications for children's development. Although sleep efficiency and long wake episodes often correlate, they represent independent and distinct facets of sleep (Ohayon et al., 2017) and both were independently examined. Our assessment of sleep problems was along a continuum.

Exposure to family risk, including PPD, may compromise children's sleep. At the neurobehavioral level, vigilance is the antithesis of sleep, and ceasing awareness of the surrounding environment for an extended period is needed to achieve sufficient and high quality sleep (Dahl, 1996). Consistent with an evolutionary perspective, the ability to maintain arousal and detect threat in an unsafe environment could be highly adaptive and the broader social network including the family context are highly influential in creating sleep conditions that facilitate a sense of safety and reduced awareness (Worthman & Melby, 2002). Exposure to familial stress including PPD may interfere with the down-regulation needed to achieve optimal sleep (Dahl & El-Sheikh, 2007).

In one of the few studies linking PPD and children's sleep, school-aged children who had a parent with a history of an alcohol use disorder reported sleeping less and also had shorter actigraphy-derived sleep duration and more night time activity than their counterparts who resided in homes with less parental alcohol use (Conroy, Hairston, Zucker, & Heitzeg, 2015). Similarly, another study found that children of parents who had a dependency on alcohol had shorter self-reported and actigraphy-derived sleep duration (Hairston et al., 2016). In a recent investigation that used a community sample, PPD was associated cross-sectionally with shorter actigraphy-derived sleep duration and worse sleep quality particularly for African American children and those from lower income homes (Kelly & El-Sheikh, 2016). In that study, fathers' problem drinking (PD) was more influential than mothers' PD for children's sleep (Kelly & El-Sheikh, 2016). Although reasons for these differences are not clear, men often become more hostile than women after heavy alcohol consumption (Ogle & Miller, 2004), increasing the probability of vigilance and disrupted sleep in children. Overall, these studies have provided initial insight into relations between PPD and children's sleep. However, the cross-sectional nature of the findings limits conclusions and raises questions about directionality of effects.

Consistent with family and developmental systems perspectives, reciprocal dynamics between family risk and children's sleep are likely operative (Kelly & El-Sheikh, 2011; Peltz, Rogge, Sturge-Apple, O'Connor, & Pigeon, 2016). As many parents have experienced, a tired child often struggles to function adequately during the day. Empirical investigations have repeatedly demonstrated that insufficient and poor quality sleep relates to lower self-regulation (Owens, Dearth-Wesley, Lewin, Gioia, & Whitaker, 2016) and greater adjustment problems (Sadeh et al., 2002). Disruption in these key developmental domains could increase parental distress and psychopathology symptoms (Meltzer & Westin, 2011).

Supportive of reciprocal relations, Kelly and El-Sheikh (2011) found that parental marital conflict predicted worsening of child subjective and actigraphy-based sleep problems over time and vice versa. Other reciprocal effects have been reported including those between child sleep problems and parents' negative emotionality (Bell & Belsky, 2008), lower maternal relationship satisfaction (Peltz et al., 2016) and lower levels of mothers' emotional availability (Philbrook & Teti, 2016). It is plausible that children's sleep problems may precede other forms of family risk including PPD and the examination of such reciprocal effects was our primary study objective.

A second objective was to examine whether relations between PPD and children's sleep varied across socioeconomic status (SES) lines. Adults from lower SES backgrounds may be at greater risk for drinking-related problems (Bonevski, Regan, Paul, Baker, & Bisquera, 2014) and children from lower SES backgrounds are more likely to have sleep problems (Spilsbury et al., 2006). In addition to such mean-comparison differences, SES effects may operate in other ways that have been less examined. For example, family risk (e.g., PPD) may interact with SES to affect children's sleep. The *health disparities view* advances that individuals who face socioeconomic adversity may have increased burden of adverse circumstances (Carter-Pokras & Baquet, 2002). This perspective rests on the premise that such individuals are exposed to increased levels of environmental stress including reduced access to community resources and suboptimal living conditions (Evans, 2003). Further,

children from lower SES backgrounds are more susceptible to the negative consequences of family risk (El-Sheikh & Sadeh, 2015; Repetti, Taylor, & Seeman, 2002). Specifically, the cumulative effect of high stress exposure may compromise cognitive and emotion regulation processes needed to effectively cope with family risk (Evans, 2003; Repetti et al., 2002), and increase vulnerability for poor health outcomes.

Available evidence supports the aforementioned perspective. Parental marital conflict was a more robust risk factor for increases in sleep problems over two years among children from lower in comparison to higher SES homes (Kelly & El-Sheikh, 2011). Similarly, longitudinal relations between several parent functioning variables (e.g., parenting stress, marital satisfaction, perceived social support) and children's sleep problems were moderated by SES and in most instances, the expected effects were more robust for lower SES children (Bernier et al., 2013). In addition, greater PD among fathers, and to a lesser extent among mothers, was related to shorter sleep duration and poorer sleep quality concurrently for children from lower but not higher SES homes (Kelly & El-Sheikh, 2016). These findings indicate that consideration of the broader socioeconomic context as a moderator of effects holds promise for gaining a better understanding of relations between family risk and children's sleep.

Current Study

We examined the reciprocal relations between PPD and children's sleep (actigraphy-derived sleep duration, sleep efficiency, long wake episodes, sleep latency) over one year, and assessed the role of SES as a moderator of effects. We expected that greater PPD would predict worsening of sleep problems over time and that the opposite direction of effects would also be evident. Given the novelty of our investigation, we did not hypothesize which variable (PPD or sleep) would be a more robust predictor of the other variable. For a fuller understanding of relations between PPD and children's sleep, we examined both mothers' and fathers' PD. Because this literature is in a newly developing stage, we had no expectations regarding whether mothers' or fathers' PD would confer greater risk and approached these questions as exploratory. The sample included a high representation of economic adversity allowing for adequate testing of SES as a moderator of the association between PD and children's sleep. Consistent with a health disparities perspective, we expected relations between PPD and sleep problems to be more robust for children from lower SES homes.

Method

Participants

Families participated in the Auburn Sleep Study, a multi-wave investigation focused on examining biopsychosocial influences on child development. Data for the current study come from waves 2 (W2) and 3 (W3; data collected in the 2010–2011 and 2011–2012 school years). Families were recruited through letters distributed at local public schools in Alabama and Georgia. Interested families called our on-campus laboratory. Eligibility criteria were based on parents' (mostly mothers) reports and included not having a learning disability nor a diagnosed sleep disorder; these criteria were implemented to reduce potential

confounds. At the initial wave of data collection (2009–2010 school year), 282 families participated. At W2, 79% of the original sample returned and an additional 56 families were recruited. For the remainder of the paper, W2 of the longitudinal study will be referred to as W1 and W3 will be referred to as W2.

At W1, 280 children and their parent(s) participated (55% boys; *M* age = 10.33 years, *SD* = 8.07 months; 66% European American and 33% African American). Mothers' reports on the Puberty Development Scale (1 = prepubertal, 2 = early pubertal, 3 = midpubertal, 4 = late pubertal, 5 = postpubertal; Petersen, Crockett, Richards, & Boxer, 1988) indicated that on average boys were prepubertal (*M* = 1.53, *SD* = .37) and girls were near the early pubertal stage (*M* = 1.96, *SD* = .62). Family SES was indicated by the income-to-needs ratio (U.S. Department of Commerce; www.commerce.gov). An income-to-needs ratio < 1 is considered below the poverty line (34% of sample),1–2 is considered living near the poverty line (32% of sample), 2–3 is lower middle class (24% of sample), and >3 is middle class standing (10% of sample). The mean income-to-needs ratio in our sample was 1.61 (*SD* = . 97).

In terms of children's living arrangements, 90% (*n* = 252) resided with their biological mothers; of these children 54% (*n* = 137) also lived with their biological father, 19% (*n* = 48) with their step-father or mother's boyfriend and in 27% of cases (*n* = 67) the mother was single. Further, 3% of children (*n* = 7) lived with their biological father and step-mother and 1% (*n* = 4) lived with a single father. The remaining 6% (*n* = 17) lived with their grandparent(s), adoptive parents or another family member. In analyses, we considered controlling for biological parent status and single-mother status; only single-mother status was related to some of the primary study variables and it was retained as a covariate. For simplicity, children's caregivers are referred to as parents.

About one year later (*M* time lag between W1 and W2 = 336 days, *SD* = 34 days), 275 of the families returned for W2 (95% of original sample; 53% boys; 66% European American, 33% African American). Average pubertal status at W2 was 1.80 (*SD* = .55) for boys and 2.35 (*SD* = .62) for girls, indicating prepubertal status for boys and early pubertal status for girls (Petersen et al., 1988). Independent-samples *t* tests and χ^2 analyses were used to assess whether retained and attrited families differed on control and primary study variables from W1 to W2; no differences emerged.

Procedure

At W1 and W2, actigraphs were delivered to participants' homes. Parents were instructed to have their children place the actigraph on their non-dominant wrist at bed time for seven consecutive nights and to remove upon waking each morning. Parents completed sleep diaries to cross validate actigraphy-generated sleep times (Acebo et al., 1999). To reduce confounds, the sleep assessments occurred during the regular school-year, excluding holidays and vacations. Shortly after the actigraphy assessment (typically the next day), families visited our on-campus lab. Parents completed questionnaires independently and children completed questionnaires with a trained interviewer. Approval from the institution's internal review board was obtained and participants gave informed consent and assent.

Measures

Parental problem drinking—At both waves, parents completed the Parental Alcohol Experiences Scale (Windle, 1997), which has established psychometric properties including retest reliability, internal consistency and convergent validity (Davies & Windle, 1997; Windle, 1996, 2000). The measure includes 15-items and assesses symptoms of alcohol dependence across legal, social, work and family contexts. Mothers and fathers reported on the occurrence of their own PD within the last year (e.g., "*I got into trouble with the law while drinking*," "*I got into a fight or heated argument with a stranger while drinking*," "*I drank before work or school*," "*My drinking resulted in an argument/fight with family members*"). For each item, a 5-point Likert response system was used that ranged from 1 (*never*) to 5 (*frequently/more than 10 times*). Endorsement of 5 or more items indicates potential clinical levels of an alcohol-related disorder; based on self-reports, 5% of women and 9% of men surpassed the cutoff at W1; percentages were similar at W2. For mothers' PD, α was .84 at W1 and .77 at W2. For fathers' PD, α was .85 at W1 and .89 at W2.

Actigraphy-measured sleep—At both waves, actigraphy was used to examine children's sleep. Actigraphy has demonstrated good reliability, especially when used over multiple consecutive nights (Acebo et al. 1999) and has shown a strong correspondence with polysomnography (Sadeh, Sharkey, & Carskadon, 1994). The actigraphs were Octagonal Basic Motionloggers (Ambulatory Monitoring, Inc., Ardsley, NY) and sleep variables were derived with the Actme software derived using Sadeh's scoring algorithm (Sadeh et al., 1994).

We assessed *Sleep Minutes* (number of minutes scored as sleep between sleep onset time and morning wake time). We also assessed three well-established sleep quality parameters: *Sleep Efficiency* – percentage of minutes between bedtime and wake time spent asleep; *Long Wake Episodes* – number of wake episodes ≥ 5 min; and *Sleep Latency* – total minutes between bed time and sleep onset time. We also examined *variability in sleep onset time* across the week of actigraphy, however this variable did not play a significant role in the fitted models and was not included in the final analyses.

Children had on average 5.94 nights (SD = 1.18) of valid actigraphy data at W1 and 5.71 nights at W2 (SD = 1.41); these rates are considered very good (Acebo et al., 1999). Reasons for missing data included forgetting to wear the watch and battery failure. In addition, nights with medication use for acute illnesses (e.g., Benadryl) were excluded. Fewer than 3 nights of valid actigraphy data may provide a poor estimate of sleep (Acebo et al., 1999). We fit the models before and after the removal of such cases (n = 5 at W1 and 6 at W2). A comparison of the findings yielded no major differences and final analyses were based on the full sample. Intraclass correlations indicated good night-to-night stability at W1 and W2 for sleep minutes (α = .78 and .79), sleep efficiency (α = .89 and .85), long wake episodes (α = .87 and .84) and sleep latency (α = .52 and .70). Lower coefficients for sleep latency are consistent with those reported in past studies (e.g., Knutson, Rathouz, Yan, Liu, & Lauderdale, 2007). Each sleep variable was derived by creating an average of all available nights.

Covariates—At W1, we controlled for other forms of family risk known to relate to PPD and children's sleep in the literature including interpartner conflict (Kelly & El-Sheikh, 2011). This approach was important for helping isolate the unique influence of PPD on children's sleep over time and vice versa, especially given that previous papers from our lab found relations between family conflict and sleep. Children from two-parent families reported on the frequency of parents' verbal and physical interpartner conflict tactics in the past year using the Revised Conflict Tactics Scale (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). These data were treated as missing for children from one-parent homes. Further, demographic variables known to relate to primary study variables in the literature were also controlled including child sex (Sadeh et al., 2000) and ethnicity (Kelly & El-Sheikh, 2016). Child age and pubertal status were initially considered in analyses but were not related to primary study variables and were excluded.

Data Analytic Plan

A path model was fit to examine the reciprocal relations between PPD and children's sleep (sleep minutes, sleep efficiency, long wake episodes, sleep latency) over one year. PPD at W1 was examined as a predictor of children's sleep at W2 and relations between children's sleep at W1 and PPD at W2 were also estimated. The mothers' and fathers' PD variables were examined in the same model; this approach allowed for controlling for fathers' PD while examining the role of mothers' PD and vice versa (in preliminary analyses we fit models separately for mothers' and fathers' PD and no differences were observed in comparison to the full model). All four sleep variables were included simultaneously in the same model. To account for autoregressive effects, PPD at W1 was allowed to predict PPD at W2 and the paths for each sleep parameter between W1 and W2 were also estimated. Controlling for autoregressive effects helps reduce bias in parameter estimates, allows for conclusions about predicted change over time and provides information about the direction of effects between variables (Selig & Little, 2012). Further, $\Delta\chi 2$ tests assessed whether estimated paths between PPD and children's sleep resulted in a significant change in model fit. A change in model fit provides additional support for the inclusion of the estimated path in the model. The fitted path model is depicted in Figure 1.

Interaction terms were added to examine whether SES at W1 moderated relations between PPD at W1 and children's sleep at W2 and vice versa. Interaction terms were created between mothers' and fathers' PD at W1 and SES at W1 (total of 2 interaction terms). Interaction terms were also created between each of the four sleep variables at W1 and SES at W1 (total of 4 interaction terms). To reduce multicollinearity (Babyak, 2004), only one interaction term was examined at a time rather than fitting a model that included all interaction terms simultaneously. SES was treated as a continuous variable. Significant interactions were plotted at high (+1 *SD*) and low (−1 *SD*) levels of PPD and SES using Preacher, Curran, and Bauer's (2006) online interaction tool. In the sample, 52 children were from "low SES" backgrounds and 53 children had "high SES." For each significant interaction the "regions of significance" was computed; this indicates the range of the moderator where the simple slopes are significantly different from zero (Preacher et al., 2006). Further, $\Delta\chi 2$ tests were used to determine whether the inclusion of each interaction term resulted in a significant change in model fit. In additional analyses not reported in this

paper for brevity, ethnicity (European American and African American comparisons) was examined as a moderator of relations between PPD and children's sleep while controlling for SES. However, no significant interaction effects involving ethnicity emerged.

Path models were fit using Amos 24. In terms of missing data, 89% of mothers (n = 245) and 72% of fathers (n = 147) reported on their own PD and 98% of children (n = 275) had valid actigraphy assessments at W1. At W2, 87% of mothers (n = 241) and 75% of fathers (n= 153) reported on their PD and 95% of children (n = 260) had valid actigraphy data. Missing data were handled using full-information maximum likelihood (Acock, 2005). Models were considered an acceptable fit if they satisfied at least two of the three following criteria: χ^2/df < 3, CFI > 0.90, and RMSEA ≤ 0.08 (Browne & Cudeck, 1993); each fitted model satisfied these criteria. Regarding the control variables, each was allowed to covary with PPD and the sleep variables at W1 and to predict PPD and sleep at W2. The control variables were allowed to covary with each other. The residual variances among PPD and the sleep variables at W2 were allowed to correlate. The PPD variables were skewed and were natural log transformed. To reduce outlier effects, we recoded values surpassing 4 *SD*s among primary study variables as the highest observed value below 4 *SD*s. In total, 5 values were recoded among the PPD variables and 9 were recoded among the sleep variables (Cousineau & Chartier, 2010).

Results

Descriptive Statistics and Preliminary Analyses

Means, standard deviations, and bivariate correlations among study variables are presented in Table 1. On average, children slept 7 hrs and 24 min per night (*SD* = 51 min) at W1 and 7 hrs and 17 min (*SD* = 55 min) at W2. Sex-related comparisons indicated that mothers and fathers did not differ in their PD at W1, however fathers had more PD than mothers at W2 (t[146] = −3.42, p < .001) (*M*s at W2 = 17.14 and 15.93 respectively).

Reciprocal Relations between Parental Problem Drinking and Children's Sleep

The path model fit to examine the reciprocal relations between PPD and children's sleep over one year fit the data well, χ2(14) = 39.15, p < .001; χ2/*df* = 2.80; CFI = .99; RMSEA = .07*ns*, 95% CI [.05 to .10] (Figure 1). Several control variables were related to children's sleep at W2 as well as other primary study variables (not depicted in figure for clarity). African American status was related to fewer sleep minutes (B = −23.53, β = −.20, p = .002), and male status was related to greater long wake episodes (B = .48, β = .13, p = .05). Single mother status was related to fewer sleep minutes (B = −38.57, β = −.15, p = .01), reduced sleep efficiency (B = −6.10, β = −.17, p = .008), and greater long wake episodes (B = 2.09, β = .21, p = .01). More verbal interpartner conflict at W1 predicted an increase in mothers' PD at W2 (B = .04, β = .13, p = .05) and greater physical interpartner conflict at W1 predicted lower rates of mothers' PD at W2 (B = −.12, β = −.13, p = .01). As shown in the figure, the autoregressive effects were significant for mothers' and fathers' PD as well as for children's sleep minutes, sleep efficiency, long wake episodes, and sleep latency.

Examination of PPD as a predictor of change in children's sleep revealed that higher level of fathers' PD at W1 predicted reductions in sleep minutes and sleep efficiency over time (Figure 1). $\Delta\chi 2$ tests indicated that the estimation of each of these paths resulted in significant change in model fit. The assessment of reciprocal relations indicated that more frequent long wake episodes among children at W1 forecasted an increase in mothers' and fathers' PD at W2. The estimation of these two paths resulted in significant change in model fit, based on $\Delta\chi 2$ testing. In total, the model explained 25% of the variance in children's sleep minutes, 17% in sleep efficiency, 18% in long wake episodes and 9% in sleep latency at W2. In addition, 56% of the variance in mothers' PD and 31% in fathers' PD at W2 was accounted for in the model.

The Moderating Role of SES

Next, interactions between PPD and SES at W1 were examined in relation to children's sleep at W2 (path models fit to examine moderation effects are not depicted for brevity). The interaction between fathers' PD and SES at W1 was significantly related to all four child sleep parameters at W2. Based on $\Delta\chi 2$ testing, the estimation of these paths resulted in significant change in model fit. The interaction between fathers' PD and SES at W1 in the prediction of children' sleep minutes at W2 ($B = 2.42$, $\beta = .25$, $p = .001$) is depicted in Figure 2a. The examination of simple slopes yielded a significant negative association between fathers' PD at W1 and children's sleep minutes at W2 for children from lower SES homes ($n = 52$ children; predicted $M = 485$ min and 427 min at lower and higher levels of PD). The simple slope was not significant for children from higher SES homes (predicted $M = 453$ min and 449 min at low and high levels of PD, respectively). Further, the shortest sleep was observed for lower SES children who had fathers with greater PD. Calculation of the regions of significance indicated that relations between greater PD among fathers and fewer sleep minutes was significant for those with an SES < 2.08 ($n = 153$ families).

Family SES also moderated relations between fathers' PD at W1 and children's sleep efficiency at W2 ($B = .60$, $\beta = .31$, $p < .001$; Figure 2b). Higher levels of PD among fathers was associated with sleep efficiency over one year, but only among children from lower SES backgrounds (predicted $M = 94\%$ and 86% at low and high levels of PD, respectively). Children from higher SES homes tended to have relatively high sleep efficiency regardless of fathers' PD (predicted $M = 90\%$ at low and high levels of PD). Children with the lowest level of sleep efficiency were those from lower SES homes with fathers with higher levels of PD. Testing of the regions of significance indicated that relations between higher levels of fathers' PD and reduced sleep efficiency were significant for children with an SES < 1.99 ($n = 153$ families).

Similarly, family SES moderated relations between fathers' PD at W1 and children's long wake episodes at W2 ($B = -.10$, $\beta = -.20$, $p = .02$; Figure 2c). Greater PD among fathers at W1 predicted greater long wake episodes over time, but only for children from lower SES homes (predicted $M = 2.13$ and 3.53 at low and high levels of PD). Testing of the regions of significance indicated that these relations were significant for children with an SES < 1.73 ($n = 126$ families). Children from higher SES homes tended to have similar wake episodes regardless of fathers' PD (predicted $M = 2.91$ and 2.99 at low and high levels of PD).

Children at greatest risk for long wake episodes were those from lower SES homes who had fathers with greater PD.

Family SES moderated associations between fathers' PD at W1 and children's sleep latency at W2 ($B = -.38$, $\beta = -.22$, $p = .01$; Figure 2d). Higher levels of fathers' PD was associated with longer sleep latency only for children from lower SES backgrounds (predicted $M =$ 9.83 min and 13.29 min at low and high levels of PD). These relations were significant for children with an SES < 1.00 ($n = 79$ families). For children from higher SES backgrounds, relations between fathers' PD and children's sleep latency were not significant. Children from homes marked by high levels of fathers' PD in conjunction with low SES had the longest sleep latency.

Regarding the examination of reciprocal relations, SES did not moderate relations between children's sleep at W1 and PPD at W2.

Discussion

We investigated the reciprocal relations between PPD and school-aged children's sleep over one year and considered SES as a moderator of these associations. Fathers' PD was a risk factor for shorter sleep duration and worsening of sleep quality among children over time. Reciprocal relations were also observed and poor sleep quality predicted increases in mothers' and fathers' PD. Moderation analyses revealed that associations between fathers' PD and worsening of sleep problems was most evident for children from lower SES backgrounds. The results provide novel evidence of transactional dynamics between a prevalent familial stressor and children's sleep and illustrate the importance of considering the broader socioeconomic context.

Studies investigating children's sleep in a family context are on the rise and relations between familial risk and children's sleep are being increasingly reported (El-Sheikh & Sadeh, 2015). Despite many advances, most investigations have been cross-sectional and longitudinal assessments are needed to explicate relations between family risk and children's sleep and to identify directionality of effects (El-Sheikh & Sadeh, 2015). Building on recent cross-sectional evidence of associations between PPD and children's sleep (Kelly & El-Sheikh, 2016), the current results demonstrate that children living in homes marked by higher levels of fathers' PD experienced shorter sleep duration and worsening of sleep efficiency over one year. We controlled for prevalent forms of family risk associated with children's sleep problems including verbal and physical marital conflict (Insana et al., 2014), which helped isolate the role of PPD. The longitudinal nature of the findings illustrates that the consequences of fathers' PD on disruptions in children's sleep may be persist over development. National estimates of the number of children exposed to PPD in U.S. homes is high (Substance Abuse and Mental Health Services Administration, 2012; Grant, 2000) and established longitudinal relations with such a fundamental component of health and development is of significance.

Plausible explanations exist regarding why fathers' PD may confer risk. Fathers in our sample experienced various drinking problems at W1 including regretting afterward the

things that occurred while drinking (10%), fighting with others (10%), drinking before work or school (9%) and drinking to forget troubles (9%). These occurrences, along with other behaviors that coexist with intoxication including slurred speech, unpredictability and confusion may affect children and interfere with the suspension of vigilance and relaxation needed for adequate sleep (Dahl, 1996).

Building on a small literature that has considered the influence of children's sleep on family functioning (e.g., Peltz et al., 2016), novel findings indicate that children's sleep forecasted increases in PPD over time. After controlling for highly stable autoregressive effects, more frequent long wake episodes forecasted an increase in mothers' and fathers' PD. The amount of support found for children's sleep as a predictor of PPD was similar in comparison to the opposite direction of effects (i.e., PPD as a predictor of children's sleep); 2 of the 6 tested pathways for both directions were significant. The continued consideration of transactional dynamics holds promise for moving the field forward and explicating ways in which children's sleep functions in various family contexts.

Night wakings interfere with sleep continuity and often lead to poor daytime functioning among children including adjustment problems (Sadeh et al., 2002) and poor regulation (Owens et al., 2016). Such child outcomes might contribute to a stressful home and promote risky coping methods among parents, including PD (Swendsen et al., 2000). In addition, children's long wake episodes often forecast mothers' night wakings (Kouros & El-Sheikh, 2017) and disrupted sleep is an established risk factor for PD in adulthood (Stein & Friedmann, 2005). Further, consistent with a systems perspective (Ford & Lerner, 1992), exposure to PPD may lead to disruption in children's sleep, which in turn might forecast greater PPD and a repetitive cycle may ensue. We offer our explanations as tentative pending empirical assessments. Testing of mechanisms will clarify why PPD and children's sleep are reciprocally related and advance this literature.

A secondary objective was to investigate SES as a moderator of associations between PPD and children's sleep. Family risk including interpartner aggression (Kelly & El-Sheikh, 2011), parenting stress, reduced social support among parents and less marital satisfaction (Bernier et al., 2013) compromise sleep to a greater degree for children from lower SES backgrounds. The longitudinal analyses build on these studies and on a previous investigation that demonstrated cross-sectional relations between fathers' PD and children's sleep problems to be more robust for lower SES children (Kelly & El-Sheikh, 2016). For children from lower but not higher SES backgrounds, fathers' PD predicted shortening of sleep duration and worsening of sleep quality including sleep efficiency, long wake episodes and sleep latency one year later. The pattern of effects for all the moderation effects illustrate health disparities in that children most at risk for disrupted sleep were those from families characterized by both higher levels of fathers' PD and lower SES. In the societal milieu of semirural Alabama, families from poor economic backgrounds commonly face stressful living conditions including substandard housing and living arrangements, reduced human capital and financial challenges (Bagley, Kelly, Buckhalt, & El-Sheikh, 2015). The wear and tear commonly experienced after prolonged stress exposure may deplete coping systems that aid in effectively responding to additional risk (Evans, 2003), including family adversity (Repetti et al., 2002). In contrast, children from higher SES homes may maintain the ability

to cope with family stress exposure in more effective ways and may be less vulnerable to its consequences, including sleep problems. To offer another explanation, the stronger association between fathers' PD and children's sleep among those from lower SES backgrounds may be attributed to physical environmental factors (Spilsbury, Frame, Magtanong, & Rork, 2016). For example, lower SES homes are often smaller and have poor insulation, and children may sleep closer in proximity to where fathers drink resulting in noise exposure and sleep disruption. Overall, the observed interaction effects illustrate that consideration of the broader socioeconomic context holds promise for identifying children most vulnerable to the negative consequences of familial risk.

The interaction effects predicted meaningful differences in children's sleep. For lower SES children, an average difference of 58 minutes in sleep duration was observed between those with fathers who had lower (M = 485 minutes per night) and higher levels of PD (M = 427 minutes per night). This difference may have important implications for development. Work conducted by others has illustrated that restriction of sleep duration by one hour compromised cognitive functioning (Sadeh et al., 2002) and emotion regulation capacities (Gruber, Cassoff, Frenette, Wiebe, & Carrier, 2012). Important differences were also observed for sleep efficiency. Children from lower SES backgrounds who had fathers with lower levels of PD had relatively good sleep efficiency (M = 94% per night). However, reduced sleep efficiency was observed for lower SES children who had fathers with higher levels of PD (M = 86% per night). Sleep efficiency below 90% has been considered an indicator of poor sleep quality (Sadeh et al., 2000, 2002) and thus the observed differences in our sample are likely meaningful.

Few studies have considered relations between both mothers' and fathers' drinking on children's development and sex/gender-related comparisons are needed (Guttmannova et al., 2016). Of available findings, some have indicated that men's PD compromises children's development to a greater degree (Guttmannova et al., 2016; Keller et al., 2008), including in relation to sleep problems (Kelly & El-Sheikh, 2016). Consistent with these findings, the current results indicate that fathers' PD was directly related to change in two of the four examined sleep parameters (while controlling for mothers' PD) and all four tested interactions between fathers' PD and SES were significant. Conversely, mothers' PD did not predict children's sleep (while either controlling for fathers' PD or not controlling for such effects as indicated by preliminary analyses).

There are plausible reasons for why children's sleep was affected by fathers' but not mothers' PD. After drinking heavily, men may be particularly sensitive to social threat cues and interpret the intentions of provocateurs as more hostile (Ogle & Miller, 2004), which could lead to stressful family dynamics and evoke distress in children compromising their sleep. Further, compared to women, men who drink excessively are more likely to exhibit antisocial behaviors (Nolen-Hoeksema, 2004), which could interfere with children's abilities to relax at night. In addition, men are more likely to report drinking to cope with distress (Nolen-Hoeksema & Harrell, 2002), and the combined influence of distress and PD in the home may disrupt children's sleep.

There are study strengths, limitations and directions for future research. The use of actigraphy is a strength (Sadeh, 2015) and allowed for the assessment of important sleep parameters that cannot be accurately obtained with subjective reports. Our sample was composed of school-aged children and assessments of other developmental periods might yield a different pattern of effects, and thus it is critical for researchers to address this issue. In addition, the community sample was composed largely of adults with subclinical levels of PPD and children with normative sleep problems and findings should be interpreted and generalized within these boundaries. Also, families in the sample were predominately living near the poverty line or were of lower middle class status. Although the sample has good representation across the lower SES spectrum, it lacks adequate representation of upper middle or upper class families. Findings may be more or less robust with samples of different SES distributions. Moreover, for conservative model testing we controlled for many variables known to relate to children's sleep. Nevertheless, other untested variables could have been influential (e.g., temperament). Lastly, we note a recent critique of cross-lagged panel models used often to assess transactional dynamics in contemporary developmental research and similar to those fitted in the current study. The concern is that such models cannot adequately disaggregate between- and within-subject effects and parameter estimates may be difficult to interpret in a meaningful way (Berry & Willoughby, 2017).

Acknowledging these limitations, the findings provide new insight into the longitudinal influence of PPD on children's sleep while demonstrating reciprocal relations and socioeconomic effects. Related prevention and intervention efforts would benefit from recognition of these transactional dynamics when considering relations between PD and children's health and development. Moreover, family risk may have a particularly deleterious influence on those exposed to socioeconomic adversity, and clinical efforts should be tailored based on an individual's access to financial resources and associated challenges.

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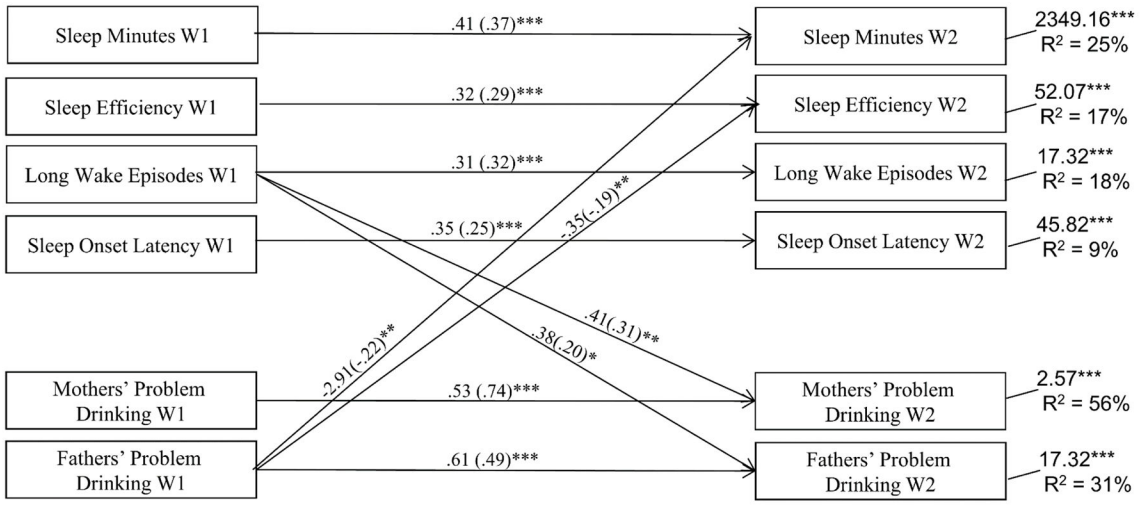

Figure 1.

Examination of reciprocal relations between mothers' and fathers' problem drinking and children's sleep over one year. Model fit: $\chi 2(14) = 39.15$, $p < .001$; $\chi 2/df = 2.80$; CFI = .99; RMSEA = .07*ns*, 95% CI [.05 to .10]. Unstandardized and standardized coefficients (in parentheses) are provided. Residual variances among the problem drinking and sleep variables at W2 were allowed to correlate. Significant relations among exogenous variables were allowed to covary. For clarity, only significant paths are depicted. Children's sex, ethnicity, socioeconomic status, single-mother status, and verbal and physical interpartner conflict were included as covariates. W1 = Wave 1; W2 = Wave 2.

* $p < .05$. ** $p < .01$. *** $p < .001$.

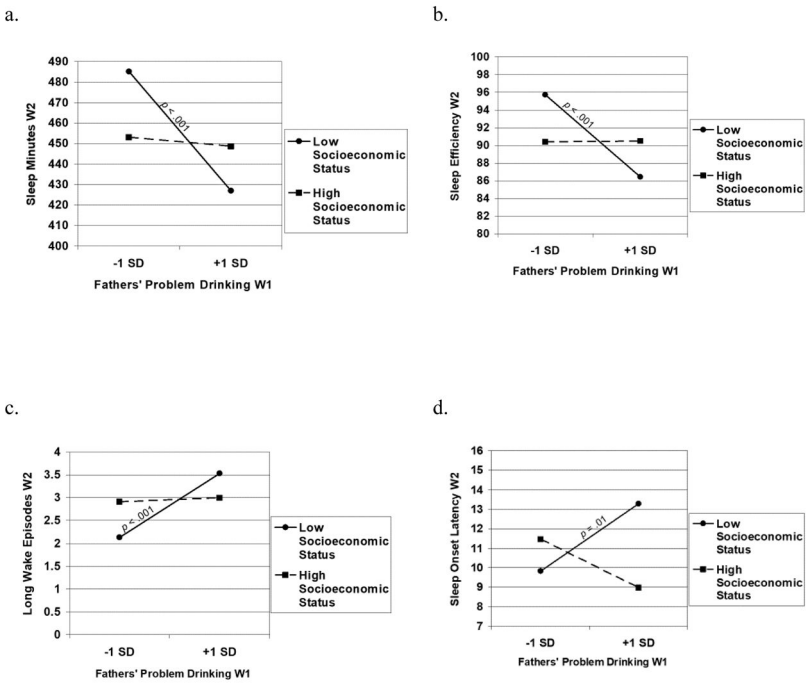

Figure 2.

Socioeconomic status (as assessed by income-to-needs ratio) as a moderator of relations between fathers' problem drinking at wave 1 (W1) and children's sleep at wave 2 (W2). For slopes that differ from zero, the *p* value is presented next to the slope. Low socioeconomic status included 52 children and high socioeconomic status included 53 children.

Table 1

Descriptive Statistics and Correlations among Study Variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
1. Child sex	-																	
2. Ethnicity	−.04	-																
3. Single mother status	−.01	−.03	-															
4. Socioeconomic status W1	.11	−.39*	−.08	-														
5. Verbal interpartner conflict W1	−.02	−.21*	−.06	.02	-													
6. Physical interpartner conflict W1	.01	.13*	−.05	.06	.32*	-												
7. Mothers' problem drinking W1	−.08	−.08	−.07	.08	.32*	.24*	-											
8. Fathers' problem drinking W1	.17	.02	.01	−.05	.13	.07	.23*	-										
9. Children's sleep minutes W1	−.08	−.04	.11	.14*	.01	.01	.01	−.01	-									
10. Children's sleep efficiency W1	−.09	.11	.03	.08	−.01	.05	.00	.02	.74*	-								
11. Children's long wake episodes W1	.09	−.11	−.02	−.03	.02	−.02	.02	−.03	−.63	−.94*	-							
12. Children's sleep latency W1	−.03	.03	−.07	−.04	.08	−.02	.01	−.11	−.31	−.38*	.34*	-						
13. Mothers' problem drinking W2	−.01	−.04	−.05	.05	.25*	.12	.75*	.30*	.03	.03	−.01	.06	-					
14. Fathers' problem drinking W2	−.01	.02	−.01	−.05	.07	−.01	.39*	.51*	−.13	−.06	.04	−.08	.44*	-				
15. Children's sleep minutes W2	−.10	−.20*	−.11	.11	.06	−.06	.03	−.05	.35*	.23*	−.25*	−.11	−.02	−.04	-			
16. Children's sleep efficiency W2	−.11	−.09	−.15*	.12	.08	−.01	.05	.01	.20*	.30*	−.31*	−.12	.07	.01	.75*	-		
17. Children's long wake episodes W2	.14*	.01	.21*	−.06	−.08	−.03	−.07	.00	−.22*	−.34*	.37*	.11	−.09	−.01	−.61*	−.88*	-	
18. Children's sleep latency W2	.02	.09	.04	−.12	−.06	.00	.05	−.06	−.17*	−.21*	.22*	.27*	−.02	−.04	−.27*	−.30*	.29*	-
M	-	-	-	1.62	6.02	.69	16.17	16.65	443.78	88.39	3.46	9.32	15.93	17.14	437.47	88.82	3.19	11.23
SD	-	-	-	.97	6.10	2.72	3.38	4.04	50.60	7.35	2.29	5.23	2.49	4.99	54.83	7.89	2.17	7.13

Note. W1 refers to Wave 1 and W2 refers to Wave 2. Child sex (1 = male, 0 = female), ethnicity (1 = African American, European American) and single mother status (1= single mother family, 0 = not a single mother family) were dichotomized and thus, descriptive statistics are not included for these variables. For children's sleep minutes, 443.78 min equates to 7 hrs and 24 min and 437.47 min equates to 7 hrs and 17 min.

*$p < .05$.