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Parental Problem Drinking and Children's Sleep: The Role of Ethnicity and Socioeconomic Status

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Abstract

We examined relations between mothers' and fathers' problem drinking and school-aged children's sleep. Consistent with a health disparities perspective, children's ethnicity and socioeconomic status were examined as moderators of relations between parental problem drinking and children's sleep. Participants were 282 children (M age = 9.44 years) and their parents. Children were from diverse ethnic (65% European American, 35% African American) and socioeconomic backgrounds. Using a multi-informant design, parents reported on their own problem drinking and children's sleep was assessed with actigraphs over 7 nights. After controlling for several influential covariates, moderation findings indicated that associations between heightened levels of parental problem drinking (predominately fathers') and children's shorter sleep duration, reduced sleep efficiency, and greater long wake episodes were most evident for African American children and those from lower socioeconomic backgrounds. Findings are among the first to establish relations between parental problem drinking and children's sleep and indicate that not all children are at equal risk for sleep disturbances in such home environments. Results add to a growing literature that has examined children's sleep within the family context and highlight the importance of considering the broader sociocultural milieu.

Keywords

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

Insufficient and poor quality sleep among school-aged children are related to deficits in key developmental processes including academic (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010), socio-emotional (Kelly & El-Sheikh, 2014), brain maturation (Feinberg & Campbell, 2011), and physical health functioning (Knutson, 2012). Consequently, identifying factors that forecast children's sleep problems are warranted. A growing literature has considered sleep within the family context and accumulating evidence indicates that family risk is associated with children's sleep problems. For instance, parental inter-partner aggression (Kelly & El-Sheikh, 2014), parent-child conflict (Kelly, Marks, & El-Sheikh, 2014), and

parental psychopathology (e.g., symptoms of depression; Seifer, 2011) are not conducive to children's sleep. Despite these advances, relations between children's sleep and many family variables have been unexplored and there is a call in the literature for conducting such assessments (El-Sheikh & Buckhalt, 2015). Few studies have considered facets of parental substance use, including parental problem drinking (PPD) as they relate to children's sleep. PPD is a commonly occurring family stressor in the U.S.A. that may have ramifications for sleep disturbances in children. Towards addressing open scientific questions, we examined relations between mothers' and fathers' problem drinking and children's sleep. Consistent with a health disparities perspective (Buckhalt, 2011), we also investigated whether children's ethnicity and socioeconomic status (SES) moderated the effects of PPD on the sleep of children.

PPD is common, with over 40% of children estimated to be exposed at some point prior to adulthood (Grant, 2000). PPD can include a wide range of alcohol-related problems including dependence, abuse, and other maladaptive drinking problems (Windle, 1997). Many studies have demonstrated that PPD in both community and clinical samples can jeopardize children's development and relations have been well-established with social and emotional problems (Keller, Cummings, Davies, & Mitchell, 2008), reduced cognitive functioning (Diaz et al., 2008), and poor academic performance (Casas-Gil & Navarro-Guzman, 2002). In addition to these outcomes, it is also possible that PPD may affect children in ways that may be more subtle but not less important including disrupting their sleep. Along this line, PPD may disrupt bioregulatory systems including cortisol (Keller, Granger, Tyler, Gilbert, Haak, & Bi, 2015) and parasympathetic nervous system functioning (El-Sheikh, 2001). However, relations between PPD and children's sleep are scarce and are a focus of this investigation.

Sleep is a complex and multi-faceted construct and assessment of various sleep parameters is imperative (Sadeh, 2015). We investigated actigraphy-based *sleep duration* (number of minutes spent asleep) as well as two primary indicators of sleep quality, namely *sleep efficiency* (percentage of the night spent asleep) and *long wake episodes* (number of wake episodes > five minutes). Our reference to *sleep problems* includes those in the non-clinical range, which are common in community samples and are indicated by short sleep duration and poor sleep quality examined on a continuum.

Heavy parental alcohol consumption often results in erratic and unpredictable behavior. In addition, parents who drink heavily might perform poorly at work, experience legal trouble, and provide inadequate child care (Hussong et al., 2008). Collectively, PPD can create a home environment marked by increased stress and instability (Hussong et al., 2008), and such living conditions could affect the sleep of family members. According to Dahl's (1996) propositions, a sense of safety and stability is required to achieve the down-regulation and diminished attentiveness needed for sufficient and high quality sleep. In contrast, continued exposure to threatening and chaotic circumstances can create heightened vigilance, which is the antithesis of sleep. Paired with the emotional security theory (Cummings & Davies, 2010), which posits that a sense of emotional security derives from family relationships that children perceive as safe, exposure to family stress including PPD can produce feelings of

worry and interfere with a sense of security in the family (Keller, Gilbert, Koss, Cummings, & Davies, 2011).

A small empirical literature with clinical samples supports relations between PPD and children's sleep. School-aged children who had at least one parent with an alcohol use disorder had actigraphy-derived shorter sleep duration and more night time activity compared to their same-aged counterparts with a parent from a non-alcoholic comparison group (Conroy, Hairston, Zucker, & Heitzeg, 2015). However, in studies that utilized laboratory-based polysomnography, having a parent with a history of alcohol abuse and dependence was not related to children's sleep duration and quality (Tarokh & Carskadon, 2010; Tarokh et al., 2012). The discrepancy in findings across studies may indicate that the effects of PPD are not uniform across all children and that third variables may be operative. Building on findings based on clinical samples, we investigated relations between PPD and children's sleep in a community sample characterized by less severe drinking problems. Further, addressing potential individual differences and incorporating a biopsychosocial perspective (El-Sheikh, Hinnant, & Erath, 2015), we investigated whether the broader sociocultural milieu moderated relations between PPD and children's sleep.

In comparison to European Americans, African American children (AA) often sleep less and experience more night wakings (Adam, Snell, & Pendry, 2007; Buckhalt, El-Sheikh, & Keller, 2007). Similarly, those from lower socioeconomic (SES) homes tend to have insufficient and poor quality sleep in comparison to their counterparts from higher SES homes (Bagley, Kelly, Buckhalt & El-Sheikh, 2015). In addition to mean differences, it is also possible that ethnicity and SES may alter the influence of family risk on children's sleep (Kelly & El-Sheikh, 2011). Elevated exposure to a range of environmental stressors is likely among ethnic minorities (e.g., racism, unequal opportunity; El-Sheikh, Tu, Saini, Fuller-Rowell, & Buckhalt, 2016) and children from families with lower SES (e.g., financial challenge, inadequate living conditions; El-Sheikh et al., 2016). Consistent with a health disparities perspective (Buckhalt, 2011), individuals who face sociocultural disadvantage may be more greatly impacted by exposure to an additional stressor, such as family risk (Schonberg & Shaw, 2007), than their less disadvantaged counterparts (McEwen, 2009).

Marital aggression was a more robust predictor of children's sleep problems over one year, as assessed by self-reports and actigraphy, for AA children (compared to European American [EA] children) and those lower SES homes (Kelly & El-Sheikh, 2011). In another study, observed relations between a number of parental functioning variables and children's sleep, as measured by sleep diary methodology, were moderated by family SES and in most instances, the expected effects were stronger in lower SES families (Bernier, Belanger, Bordeleau, & Carrier, 2013). Across studies, an emerging pattern of moderation effects is consistent with cumulative risk (Evans, 2003; Rutter, 1993) and dual-risk (Sameroff, 1983) perspectives, which suggest that some children may have a vulnerability that increases risk, or a protective factor that reduces risk, in the context of an environmental stressor.

Current Study and Hypotheses

Relations between mothers' and fathers' problem drinking and children's sleep were examined. In addition, ethnicity and SES, as indexed by income-to-needs ratio, were assessed as moderators of relations between PPD and children's sleep. We controlled for ethnicity while examining SES as a moderator and vice versa to help disentangle the effects of these variables. We hypothesized that greater PPD, among both mothers and fathers, would relate to shorter sleep duration, reduced sleep efficiency, and more frequent long wake episodes among children. Further, we expected these associations to be pronounced for AA children and those from lower SES homes.

Method

Participants

The current study is part of a larger investigation examining biopsychosocial influences on children's development (Blinded for Review; data were collected in 2009–2010). Children were recruited from semirural school districts in the Southeastern United States. Exclusion criteria included children having a diagnosed sleep disorder or learning disability based on mother report. More detail about recruitment is provided in Blinded for Review. In total, 282 families participated.

Of participants, 52% were boys and 48% were girls ($M_{\text{age}} = 9.44$ years, $SD = 8.55$ months; Range = 7.92 to 12.33 years). Regarding ethnicity, 65% were EA and 35% were AA.

Mothers reported on annual family income using the following categories (a) less than \$10,000 (18% of families); (b) \$10,000–\$20,000 (12% of families); (c) \$20,000–\$35,000 (19% of families); (d) \$35,000–\$50,000 (20% of families); (e) \$50,000–\$75,000 (18% of families); and (f) > \$75,000 (13% of families). For moderation analyses, our assessment of SES was indexed by income-to-needs ratio, which was determined by using family size, family income, and the federal cutoff for poverty (U.S. Department of Commerce; <http://www.commerce.gov>). Families who received an income-to-needs ratio < 1 were considered to be living in poverty (32% in the current study), 1–2 living near the poverty line (34% of families), 2–3 lower middle class (24% of families), and 3 middle class standing (10% of families). The mean income-to-needs ratio was 1.71 ($SD = 1.04$; Range = .34 to 4.37). For simplicity, we refer to income-to-needs ratio as SES. Attempts were made to recruit AA and EA families from a wide range of backgrounds, however ethnicity and SES were still correlated ($r = .40$), such that AAs had lower SES. Based on mothers' reports on the Puberty Development Scale (1 = prepubertal, 2 = early pubertal, 3 = midpubertal, 4 = late pubertal, and 5 = postpubertal; Petersen et al., 1988), boys ($M = 1.44$, $SD = 0.30$) and girls ($M = 1.71$, $SD = 0.44$) were prepubertal on average.

Regarding living arrangements, 89% of children lived with their biological mother ($n = 252$). Of these children, 21% ($n = 54$) lived with a single mother, 58% ($n = 146$) also lived with their biological father, and 21% ($n = 52$) lived with their mothers' partner (e.g., stepfather, boyfriend). Further, 6% of children ($n = 16$) lived with either a single father ($n = 1$), father and step-mother ($n = 11$), or adoptive parents ($n = 4$). Lastly, 5% of children ($n = 14$) lived with extended family members including grandparents and aunts/uncles. For simplicity, we

refer to the child's caregivers as parents. In analyses, single-mother status, biological parent status, and family composition were included as covariates.

Procedure

The university's internal review board approved the study. Actigraphs were delivered to families' homes and parents were instructed to have their children wear the watches for seven consecutive nights (bedtime to morning wake time) on their non-dominant wrist. Parents completed sleep diaries for children to corroborate actigraphy-derived bed and wake times. The sleep assessments occurred during the regular school year, excluding holidays and vacations. Shortly after the actigraphy assessment ($M = 3.47$ days, $SD = 8.73$ days), families visited our on-campus laboratory. Mothers and fathers completed questionnaires about demographics and drinking behavior, and children completed questionnaires with a trained interviewer. Monetary compensation was provided to families for their time and effort.

Measures

Parental problem drinking—Mothers and fathers reported on their own problem drinking using the Parental Alcohol Experiences scale (PAE; Windle, 1997), which is designed to assess a wide range of alcohol-related problems that are likely to occur in community samples. The PAE has demonstrated good psychometric properties (Davies & Windle, 1997; Windle, 1996, 2000). The measure consists of 15-items, which assess symptoms associated with alcohol dependence and abuse across a range of social, legal, family, and work/school contexts (e.g., “*got into a fight or heated argument with a stranger while drinking*,” “*got into trouble with the law while drinking*,” “*drinking resulted in an argument/fight with family members*,” “*regretted afterward the things that were done while drinking*”). For each item, participants report how often they experienced each symptom within the past year on a likert scale ranging from 1 (*never*) to 5 (*frequently/more than 10 times*). Endorsement of an item is indicated by a score greater than 1, and endorsement of 5 or more items suggests potential clinical levels of alcohol use disorders. In the current sample, 3% of women and 12% of men exceeded the cutoff. Cronbach's alpha = .70 for women and .86 for men.

Children's sleep—Actigraphs recorded children's activity at night. Actigraphy has been frequently used to assess children's sleep duration and quality and has demonstrated good psychometric properties based on comparisons with polysomnography (Sadeh, Acebo, Seifer, Aytur, & Carskadon, 1995; Sadeh, Sharkey, & Carskadon, 1994). The actigraph was an Octagonal Basic Motionlogger (Ambulatory Monitoring Inc., Ardsley, NY), a lightweight (45 g) device about the size of a wrist watch. Activity was recorded in 1-min epochs between bedtime and morning wake time. Data were downloaded and sleep variables were created using the Octagonal Motionlogger Interface with ACTme software and the analysis software package (Action W-User's Guide, 2002). Procedures for determining sleep onset time followed a lab protocol created at the E.P. Bradley Hospital Laboratory at Brown University (Acebo & Carskadon, 2001). Sadeh's algorithm was used to determine whether children were awake or asleep (Sadeh et al., 1994, 1995).

Three frequently used sleep parameters were assessed. Sleep duration was assessed by (a) Sleep Minutes – total number of minutes between sleep onset time and wake time that were scored as sleep. Sleep quality was assessed by (b) Sleep Efficiency – percentage of minutes scored as sleep between sleep onset and wake time; and (c) Long Wake Episodes – number of wake episodes > 5 min.

On average, children had 5.89 nights ($SD = 1.89$) of valid actigraphy data. More specifically, 57% of children had 7 nights of valid actigraphy data, 18% had 6 nights, 12% had 5 nights, and 13% had 4 nights or fewer. This rate of valid data is considered very good (Acebo et al., 1999). Reasons for missing data included the use of medication known to influence sleep (e.g., cold medicine) and exclusion of these nights from analyses, not wearing the watch, and technical failure. Researchers have recommended at least 5 nights are needed for a valid actigraphy assessment (Acebo et al., 1999; Sadeh, 2011). Consequently, sleep data points (not cases) for those with fewer than 5 nights were removed. Intraclass correlations indicated good night-to-night stability during the week for sleep minutes ($\alpha = .85$), sleep efficiency ($\alpha = .90$), and long wake episodes ($\alpha = .77$).

Control variables—To make a unique contribution to the literature beyond studies linking mental health, family conflict and children's sleep, available variables known to co-occur with PPD or children's sleep were controlled. These were children's symptoms of anxiety and depression (Sadeh, Tikotzky, & Kahn, 2014), parents' symptoms of depression (for a review Seifer, 2011), parent-child conflict (Kelly et al., 2014), and inter-partner aggression (Kelly & El-Sheikh, 2014). All measures used to assess the covariates have demonstrated good psychometric properties and have been used frequently in the extant literature. Children reported on their own symptoms of anxiety and depression using the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978), and the Children's Depression Inventory (Kovacs, 1985), respectively. Parents reported on their own symptoms of depression using the Center for Epidemiologic Studies Depression Scale (Radloff, 1977). Children reported on verbal and physical parent-child conflict (for both mothers and fathers) using the Parent-Child Conflict Tactics Scale (Straus, 1999). The scale assesses the extent to which parents inflict both verbal and physical conflict tactics toward the child. Further, children reported on parents' verbal and physical inter-partner aggression tactics (for both mothers and fathers) using the Revised Conflict Tactics Scale (Straus et al., 1996).

Many demographic variables were also controlled including child sex, pubertal status, age, ethnicity, family SES, asthma ($n = 33$), and chronic illness ($n = 10$).

Data Analytic Plan

A path model was fit to examine the direct effects between PPD and children's sleep. Mothers' and fathers' problem drinking often co-occur (Keller et al., 2008) and thus both were examined simultaneously in the same models. Such an approach allows for controlling for mothers' problem drinking while examining the influence of fathers' problem drinking and vice versa. In a similar fashion, each of the three actigraphy-measured sleep parameters (sleep duration, sleep efficiency, long wake episodes) were examined simultaneously in the same model. In total, one path model was fit for the examination of direct effects.

Next, interaction terms were added to examine ethnicity and SES as moderators of relations between PPD and children's sleep. To reduce multicollinearity (Babyak, 2004), each interaction term was examined in a separate model. Four interaction terms were examined: Mothers' Problem Drinking $\times$ Ethnicity; Fathers' Problem Drinking $\times$ Ethnicity; Mothers' Problem Drinking $\times$ SES; and Fathers' Problem Drinking $\times$ SES. In total, four path models were fit to investigate moderation effects (see Figures 1 and 2 for examples). Significant interactions were plotted at high (+1 *SD*) and low (-1 *SD*) levels of mothers' and fathers' problem drinking and SES. Ethnicity (1 = AA, 0 = EA) was treated as a dichotomous variable. Using estimates obtained from the fitted models, interactions were plotted using Preacher, Curran, and Bauer's (2006) interaction utility. In addition, χ^2 tests were used to determine whether removing the interaction effect significantly weakened model fit; such an approach provides additional support for the inclusion of the interaction term in the model. Ethnicity was included as a control variable when SES was examined as a moderator and vice versa.

Path models were fit using Amos 22. Full information maximum-likelihood estimation was used to handle missing data. This approach is robust against violations of normality and is one of the best methods for handling missing data (Acock, 2005). All control variables were treated as exogenous variables and were included in all fitted models. Exogenous variables that were significantly related were allowed to covary. The residual variances among endogenous variables were allowed to correlate. Acceptable model fit included satisfying at least two of the following three criteria: $\chi^2/df < 3$, CFI > 0.90 , and RMSEA < 0.08 (Browne & Cudeck, 1993); each model satisfied these criteria. Based on skewness (+2) and kurtosis (-2) statistics and visual inspection, mothers' and fathers' problem drinking were skewed and natural logged transformed. To reduce outlier effects among primary study variables, data points that surpassed 4 *SDs* were recoded as the highest value below 4 *SDs* ($n = 9$ values; Cousineau & Chartier, 2010). Models were initially fit prior to recoding outliers and no major differences were detected before or after the outliers were recoded.

Results

Descriptive Statistics

Descriptive statistics and bivariate correlations among primary study variables are presented in Table 1. On average, children slept 7 hr and 37 min per night (*SD* = 59.23) and had a sleep efficiency of 88.22% (*SD* = 8.22); researchers have suggested that sleep efficiency below 90% is an indicator of poor sleep quality among school-aged children (Sadeh et al., 2000, 2002). Similarly, children averaged 3.57 long wake episodes (> 5 min each) per night (*SD* = 2.14); greater than 3 has been considered a characteristic of poor sleep (Sadeh et al., 2000, 2002). Lower SES was associated with more long wake episodes. Mothers' and fathers' problem drinking were marginally associated ($p = .08$). Testing of mean differences indicated that fathers ($M = 16.86$, *SD* = 4.26) reported more problem drinking than mothers ($M = 15.84$, *SD* = 3.06) ($t[128] = -3.23$, $p = .002$). In comparison to EA children ($M = 7$ hr 41 min, *SD* = 63 min), AA children had shorter sleep duration (7 hr 25 min, *SD* = 51 min) ($t[259] = 2.02$, $p = .045$).

Direct Relations between Parental Problem Drinking and Children's Sleep

The path model fit to examine direct relations between PPD and children's sleep fit the data well, $\chi^2(157) = 147.79$ $\chi^2/df = .94$; CFI = 1.00; RMSEA = .00*ns*, 95% CI [.00 to .02]; model not depicted in a Figure for brevity). Child sex and pubertal status were related to sleep duration, such that male status ($B = -14.84$, $\beta = -.14$, $p = .05$) and a higher pubertal status ($B = -22.97$, $\beta = -.16$, $p = .03$) were associated with shorter sleep duration. Older children tended to have shorter sleep duration ($B = -1.19$, $\beta = -.17$, $p = .006$). Asthma status was related to reduced sleep efficiency ($B = -4.60$, $\beta = -.18$, $p = .003$) and more long wake episodes ($B = .91$, $\beta = .14$, $p = .03$). No significant direct effects emerged for relations between PPD and children's sleep. In total, the model explained 13% of the variance in sleep minutes, 11% in sleep efficiency, and 10% in long wake episodes.

Ethnicity and SES as Moderators of Relations between Parental Problem Drinking and Children's Sleep

Ethnicity—Interaction terms were added to the path model to examine whether ethnicity served as a moderator. Only one interaction term was assessed at a time. We first fit a model to examine whether ethnicity moderated relations between mothers' problem drinking and children's sleep (while controlling for fathers' problem drinking), $\chi^2(168) = 161.83$ $\chi^2/df = .96$; CFI = 1.00; RMSEA = .00*ns*, 95% CI [.00 to .02] (model not depicted in figure). No significant interaction effects emerged.

Next, a path model was fit to examine ethnicity as a moderator of relations between fathers' problem drinking and children's sleep (while controlling for mothers' problem drinking), $\chi^2(168) = 155.79$ $\chi^2/df = .93$; CFI = 1.00; RMSEA = .00*ns*, 95% CI [.00 to .02] (Figure 1). Three cases of moderation were detected. Specifically, ethnicity moderated the association between fathers' problem drinking and children's sleep duration ($B = -295.01$, $\beta = -.15$, $p = .05$; $R^2 = .03$). Plotting of the interaction indicated that in the context of lower levels of problem drinking among fathers, children's sleep duration was rather similar for all children regardless of ethnicity (Figure 3a; predicted $M = 468$ min for EAs and 481 min for AAs). However, in the context of greater problem drinking, AAs ($M = 451$ min) had an average of 30 min less sleep than EAs ($M = 481$ min). Neither slope was statistically different from zero. As depicted in the plot, the shortest sleep duration was observed for AA children with fathers who had elevated levels of problem drinking.

Ethnicity also moderated relations between fathers' problem drinking and children's sleep efficiency ($B = -58.13$, $\beta = -.21$, $p = .005$; $R^2 = .05$) and the pattern of effects was similar to that observed for sleep duration. Based on the examination of simple slopes, a significant negative association between fathers' problem drinking and sleep efficiency was observed for AA children (Figure 3b). The simple slope was not significant for EAs who tended to have relatively high levels of sleep efficiency regardless of fathers' problem drinking (Predicted $M = 89\%$ at lower levels and 90% at higher levels of problem drinking). However, for AAs, sleep efficiency was much lower at higher (Predicted $M = 85\%$) than lower levels of fathers' problem drinking (Predicted $M = 93\%$). The children who had the lowest level of sleep efficiency were AA children with fathers who had greater problem drinking.

Similar to the pattern of moderation effects observed for sleep duration and efficiency, ethnicity moderated relations between fathers' problem drinking and children's long wake episodes ($B = 20.25$, $\beta = .28$, $p < .001$; $R^2 = .06$). Testing of the simple slopes indicated that problem drinking among fathers was not related to long wake episodes for EA children (Predicted M was similar at lower [3.46] and higher [3.22] levels of problem drinking; Figure 3c). However, the simple slope was significant for AA children and rather large differences in long wake episodes were observed in the context of lower ($M = 1.72$) and higher ($M = 4.68$) problem drinking. The children who experienced the most frequent long wake episodes were AAs with fathers who had greater problem drinking.

Lastly, χ^2 tests indicated that the removal of each of the aforementioned moderation effects pertaining to ethnicity significantly decreased model fit, providing support for their roles in the model.

SES—A model was fit to examine whether SES moderated relations between mothers' problem drinking and children's sleep (while controlling for fathers' problem drinking), $\chi^2(168) = 156.55$, $\chi^2/df = .93$; CFI = .99; RMSEA = .00 ns , 95% CI [.00 to .02] (model not depicted in a figure for brevity). One interaction effect was detected. SES moderated relations between mothers' problem drinking and children's long wake episodes ($B = -6.76$, $\beta = -.17$, $p = .02$; $R^2 = .04$). Plotting of the interaction indicated that within the context of lower problem drinking among mothers, the frequency of long wake episodes was rather similar for all children regardless of family SES (Figure 4a; Predicted $M = 3.40$ for lower and 3.60 for higher SES children). However, within the context of higher levels of problem drinking among mothers, children from lower SES homes had more frequent long wake episodes ($M = 4.68$) than their higher SES counterparts ($M = 2.68$). Neither simple slope was significant.

A model was fit to examine whether family SES served as a moderator of relations between fathers' problem drinking and children's sleep (while controlling for mothers' problem drinking), $\chi^2(168) = 160.20$, $\chi^2/df = .95$; CFI = 1.00; RMSEA = .00 ns , 95% CI [.00 to .02] (Figure 2). Three interaction effects emerged. SES moderated relations between fathers' problem drinking and children's sleep duration ($B = 127.19$, $\beta = .31$, $p < .001$; $R^2 = .06$). The examination of simple slopes indicated that greater problem drinking among fathers was associated with reduced sleep duration for children from lower SES families (Figure 4b). Surprisingly, more problem drinking among fathers was related to greater sleep duration among children with higher SES. In the context of less problem drinking among fathers, sleep duration was relatively similar for children from lower (Predicted $M = 472$ min) and higher ($M = 459$ min) SES backgrounds. However, in the context of greater problem drinking among fathers, a larger difference in sleep duration was observed between children with lower ($M = 457$ min) and higher ($M = 488$ min) SES.

SES also moderated relations between fathers' problem drinking and children's sleep efficiency ($B = 17.32$, $\beta = .31$, $p < .001$; $R^2 = .06$). Testing of the simple slopes indicated that more problem drinking among fathers was related to reduced sleep efficiency for children with lower SES (Predicted $M = 90\%$ when problem drinking was lower and 86% when problem drinking was higher; Figure 4c). For their higher SES counterparts, the

simple slope was not significant, indicating that fathers' problem drinking was not related to sleep efficiency for children from higher SES families (Predicted $M = 88\%$ when problem drinking was lower and 90% when problem drinking was higher). As shown in the Figure, the lowest level of sleep efficiency was observed for lower SES children who had fathers with greater problem drinking.

SES moderated relations between fathers' problem drinking and children's long wake episodes ($B = -.401$, $\beta = -.39$, $p < .001$; $R^2 = .07$). Testing of the simple slopes indicated that greater problem drinking was related to more frequent long wake episodes only among children from lower SES homes (Figure 4d). For children from higher SES families, fewer long wake episodes were observed in the context of both lower (Predicted $M = 3.12$) and higher (Predicted $M = 2.86$) levels of fathers' problem drinking; fewer long wake episodes were also observed for lower SES children who had fathers with less problem drinking (Predicted $M = 2.78$). The highest number of long wake episodes was found for lower SES children who had fathers with greater problem drinking (Predicted $M = 4.49$).

Lastly, χ^2 tests indicated that the removal of each of the aforementioned moderation effects significantly decreased model fit, thus suggesting the importance of their inclusion. Note that we also fit the path models (both the direct effects and moderation models) with the exclusion of the child and parent mental health variables, parent-child conflict, and inter-partner aggression; findings were identical in nature to those reported in the Results.

Discussion

Toward gaining a more in-depth understanding of sleep within a family context (El-Sheikh & Buckhalt, 2015), relations between fathers' and mothers' problem drinking and school-aged children's sleep were assessed using a community sample. In addition, ethnicity and SES indicated by income-to-needs ratio were examined as moderators of these relations. Supportive of health disparities (Buckhalt, 2011), novel moderation findings show that relations between heightened levels of parental problem drinking and children's shorter sleep duration, reduced sleep efficiency, and more frequent long wake episodes were most evident for African American children and those from lower SES homes. Results explicate that not all children were at equal risk for sleep disruptions and clarify for whom and under which conditions PPD most strongly related to children's sleep. A multi-informant design was used that reduced shared-method variance, sleep was assessed objectively with actigraphy, and potential confounds were controlled, which lends confidence in the findings. Given the important role of sufficient and good sleep quality for positive outcomes in children across many domains, and the negative outcomes associated with poor sleep, findings are of potential significance.

No direct associations between PPD and children's sleep emerged. However, PPD (predominantly fathers' problem drinking) interacted with both ethnicity and SES in robust ways, suggesting that the broader sociocultural milieu may influence the association between PPD and children's sleep. Children at greatest risk for sleep problems were AA children (while controlling for SES) and those from lower SES homes (while controlling for ethnicity) who were also exposed to greater PPD. The nature of our findings are consistent

with health disparities (Buckhalt, 2011), as well as dual and cumulative risk perspectives (Evans, 2003; Sameroff, 1983). In the historical and societal milieu of semi-rural Alabama, AA children and those exposed to economic adversity and associated stressors may be at heightened risk for sleep disturbances than their European American and higher SES counterparts. Perceived discrimination, suboptimal housing (e.g., exposure to allergens, noisy and crowded sleeping environments), reduced human capital, and having difficulties making ends meet are among the many variables that may underlie the increased susceptibility of AA and low SES children to sleep problems (Bagley et al., 2015; El-Sheikh et al., 2016). In path models that controlled for potential confounds, one direct effect between SES and sleep (long wake episodes) emerged and none were found for ethnicity. Rather, it is the interaction between PPD and either SES or ethnicity that was more robustly related to sleep problems. Empirical assessments of these tentative explanations are likely to shed light on relations between PPD and sleep in the context of risk.

A growing number of studies have addressed children's sleep in the family context (El-Sheikh & Buckhalt, 2015) and accumulating evidence has indicated that multiple facets of family functioning including parental inter-partner conflict (Kelly & El-Sheikh, 2014), parents' symptoms of depression (Seifer, 2011), and parent-child conflict (Kelly et al., 2014) jeopardize school-aged children's sleep. The current investigation builds on these studies and provides evidence that PPD, a form of family risk that occurs frequently in U.S. homes, relates to children's sleep. Illustrating the importance of contemporaneous considerations of the familial and sociocultural contexts, associations between PPD and children's sleep duration and quality were contingent upon ethnicity and SES. Given that PPD often co-occurs with other family and mental health risk factors, we controlled for inter-partner aggression, parent-child conflict, parents' symptoms of depression, and children's symptoms of anxiety and depression. This conservative approach helped to better isolate the unique relation PPD shares with children's sleep. Overall, findings provide additional insight into understanding children's sleep in the family and broader socioeconomic context.

There are plausible explanations regarding why PPD relates to children's sleep. Exposure to stress that may pose a threat to the child or the family may lead to vigilance, which could disrupt sleep. In this sample, some of the more commonly endorsed items on the Parental Alcohol Experiences scale are drinking alone (26% of mothers and 27% of fathers endorsed this item), drinking to forget troubles (14% of mothers and 10% of fathers), having thoughts about reducing alcohol use (10% of mothers and 18% of fathers), regretting afterward the things that occurred while drinking (6% of mothers and 13% of fathers), getting drunk several days in a row (4% of mothers and 10% of fathers), and passing out from drinking (2% of mothers and 7% of fathers). Further, individuals who are intoxicated also exhibit slurred speech, confusion, and erratic and unpredictable behavior. Collectively, problem drinking may strike at the core of parents' abilities to provide a stable, secure, and comfortable home that promotes restful sleep.

To offer other potential explanations, men with drinking problems have less adequate parenting skills (Eiden, Chavez, & Leonard, 1999) including less monitoring of children's activities (Chassin, Curran, Hussong, & Colder, 1996), which may compromise children's sleep. From a biosocial perspective, PPD is a risk factor for increased cortisol (Keller et al.,

2015), which can disrupt sleep (El-Sheikh, Buckhalt, Keller, & Granger, 2008). Cortisol promotes wakefulness and increased levels may interfere with sleep. Relatedly, family stress is associated with less optimal parasympathetic nervous system regulation (El-Sheikh & Hinnant, 2011), which in turn is linked with sleep problems in youth (El-Sheikh, Erath, & Bagley, 2013). Lastly, sleep problems often co-occur among children and parents (Brand, Gerber, Hatzinger, Beck, & Holsboer-Trachsler, 2009), and researchers have pointed to the potential role of genetic factors that may partially account for this association (Gottlieb, O'Connor, & Wilk, 2007; Heath, Kendler, Eaves, & Martin, 1990). For instance, a heritability estimate of .29 has been proposed for sleep-related problems (Gottlieb et al., 2007). Alcohol consumption and sleep problems co-occur (Crum, Storr, Chan, & Ford, 2004) and thus the link between PPD and children's sleep may be partially attributed to a shared genetic link for sleep problems; note that we controlled for biological parent status in analyses. Overall, we offer these speculations as highly tentative pending future investigations. Examinations of mediating and intervening processes linking PPD with children's sleep will help advance this young literature.

The pattern of the interaction effects may reflect meaningful differences in sleep. For European Americans, relatively high levels of sleep duration and quality were observed regardless of PPD. However, for African Americans, there was a 30-min difference in sleep duration for those exposed to high (451 min per night) versus low (481 min per night) levels of fathers' problem drinking. A daily average of a 30-min difference in sleep time could accumulate and result in chronic sleep insufficiency. In an experimental study, extending or restricting sleep duration by 40-min for 3 consecutive nights led to robust differences in memory and attention among school-aged children (Sadeh, Gruber, & Raviv, 2003). In another experimental study, restriction of sleep duration by 54-min resulted in decreased alertness and poor emotion regulation (Gruber, Cassoff, Frenette, Wiebe, & Carrier, 2012). In addition, AAs who experienced low levels of fathers' problem drinking had relatively good sleep efficiency (93% per night). However, sleep efficiency was substantially lower for these children when exposed to high levels of fathers' problem drinking (85% per night); sleep efficiency below 90% is considered an indicator of poor sleep quality (Sadeh et al., 2000, 2002). Further, AAs who experienced low levels of fathers' problem drinking had relatively fewer long wake episodes (1.72 per night) in comparison to those who were exposed to high levels of such drinking (4.68 per night). This difference is of relevance because more than 3 long wake episodes per night is considered a characteristic of poor sleep (Sadeh et al., 2000, 2002). European Americans tended to have relatively adequate sleep duration and quality regardless of parents' problem drinking.

Similarly, important differences in the various sleep parameters were detected in the context of economic adversity and PPD. Children from higher SES homes tended to have relatively long sleep duration and high sleep quality regardless of PPD. Conversely, those from lower SES homes who were also exposed to high levels of fathers' drinking tended to have the poorest sleep quality (sleep efficiency = 86% per night, long wake episodes = 4.49 per night) and had the shortest sleep in the sample (457 min). Further, one interaction effect emerged for mothers' problem drinking. Long wake episodes were most common among children from lower SES homes who had mothers with high levels of problem drinking (4.68 per

night). Overall, study findings indicate that dual exposure to PPD and sociocultural disadvantage disrupt children's sleep duration and quality in meaningful ways.

We considered both mothers' and fathers' problem drinking, which is less common in the literature than examining problem drinking in one parent. To better assess the unique influence of each, we controlled for mothers' problem drinking while examining fathers' problem drinking and vice versa. Analyses provided robust evidence that fathers' but not mothers' problem drinking interacted with ethnicity and SES to predict children's sleep. Specifically, all six moderation effects examined pertaining to fathers were significant in comparison to only one (out of six) for mothers. Alcohol use impairs the interpretation of social cues and men who have consumed alcohol are especially likely to interpret the behavior of others as threatening and to respond in more hostile ways toward others (Ogle & Miller, 2004). With regard to sleep, the more hostile and unpredictable nature of men's problem drinking might have more greatly contributed to an inadequate sleeping environment for children. It is also possible that different effects for mothers' and fathers' problem drinking emerged because men reported more problem drinking and 12% of fathers, in comparison to 3% of mothers, surpassed the clinical cut-off for alcohol use disorders. The higher levels of fathers' than mothers' problem drinking in the sample is characteristic of drinking in the population (Keller et al., 2011). Study findings are consistent with others that have found fathers' problem drinking to have a more deleterious influence on children's development (Keller et al., 2008). Overall, our inclusion of both parents in the current study builds on a recognized small body of literature that has considered fathers' influence on children's sleep (Bernier et al., 2013; Tikotzky, Sadeh, & Glickman-Gavrieli, 2011).

Our findings have several implications. Practitioners who treat children's sleep problems may want to consider the home environment including parental alcohol use. Results indicate that even sub-clinical levels of parents' problem drinking should not be overlooked. Further, certain populations may be more greatly impacted by family risk and prevention and intervention efforts for children should be tailored based on the family's resources and the challenges facing them. Lastly, on average, children in the sample may not have had adequate sleep. Although there is no consensus regarding the amount of needed sleep (Lumeng, 2010), average amount of time spent asleep was 7 hr and 37 min, which is far under the National Sleep Foundation's (2011) recommendation of allotting 10 to 11 hours for sleep per night (i.e., time spent in bed; average time allotted for sleep in our sample was 8 hr and 37 min). Given the importance of sleep for adequate health, educational programs focused on improving sleep among youth may be beneficial.

This study is not without limitations. The community sample is largely characterized by sub-clinical levels of PPD and thus, findings may not generalize to clinical populations. However, our results indicate that PPD may not need to be clinically severe to influence children's sleep and that low levels of PPD should not be disregarded. The absence of direct effects in our study may be attributed to sample characteristics including sub-clinical levels of PPD. In addition, the cross-sectional design limits conclusions about directionality of effects between study variables. Past work has indicated that children's sleep problems forecast an increase in marital conflict over time (Kelly & El-Sheikh, 2011), and it is

plausible that children's sleep problems may also precede PPD. We attempted to reduce the common association between ethnicity and income-to-needs ratio, however the two variables were still associated ($r = .40$), which is reflective of community demographics. Thus, although we controlled for income-to-needs ratio while examining ethnic-related effects and vice versa, the two variables were not independent. Lastly, although actigraphy has many strengths, it does not assess some important sleep domains. The inclusion of other assessment tools that can measure other facets of sleep (e.g., use of polysomnography to measure sleep stages) holds promise for advancing this body of work.

Despite these limitations, this multi-method study provides evidence that ethnicity and SES moderate associations between PPD and children's sleep during the school-aged years. The present study highlights the importance of contemporaneous assessments of family risk and the broader sociocultural milieu in relation to children's bioregulatory function.

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Figure 1.

Path model fit to examine ethnicity as a moderator of relations between fathers' problem drinking and children's actigraphy measured sleep. Model fit: $\chi^2(168) = 155.79$ $\chi^2/df = .93$; CFI = 1.00; RMSEA = .00ns, 95% CI [.00 to .02]. Income-to-needs ratio was used to assess socioeconomic status. Residual variances among the sleep variables were allowed to correlate. Significant relations among exogenous variables were allowed to covary. Statistically significant lines are solid and non-significant lines are dotted. Children's sex, pubertal status, age, single-mother status, biological parent status, family composition, asthma, chronic illness, children's symptoms of anxiety and depression, mothers' and fathers' symptoms of anxiety and depression, mother-child conflict, father-child conflict, and inter-partner conflict were included as covariates.

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

Figure 2.

Path model fit to examine socioeconomic status (as assessed by income-to-needs ratio) as a moderator of relations between fathers' problem drinking and children's actigraphy measured sleep. Model fit: $\chi^2(168) = 160.20$ $\chi^2/df = .95$; CFI = 1.00; RMSEA = .00_{ns}, 95% CI [.00 to .02]. Residual variances among the sleep variables were allowed to correlate. Significant relations among exogenous variables were allowed to covary. Statistically significant lines are solid and non-significant lines are dotted. Children's sex, pubertal status, age, single-mother status, biological parent status, family composition, asthma, chronic illness, children's symptoms of anxiety and depression, mothers' and fathers' symptoms of depression, mother-child conflict, father-child conflict, and inter-partner conflict were included as covariates.

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

a.

b.

c.

Figure 3.

Ethnicity as a moderator of relations between parental problem drinking and children's sleep, as measured by actigraphy. For slopes that differ from zero, the p value is presented next to the slope.

Figure 4. Socioeconomic status (as assessed by income-to-needs ratio) as a moderator of relations between parental problem drinking and children's sleep, as measured by actigraphy. For slopes that differ from zero, the p value is presented next to the slope.

Table 1

Mean, Standard Deviations, and Correlations among Primary Study Variables.

	1.	2.	3.	4.	5.	6.
1. Socioeconomic status	-					
2. Mothers' problem drinking	-.05	-				
3. Fathers' problem drinking	.06	.17 ^t	-			
4. Children's sleep duration	.09	-.04	-.00	-		
5. Children's sleep efficiency	.09	-.05	-.05	.81 ^{***}	-	
6. Children's long wake episodes	-.17 [*]	.00	.12	-.59 ^{***}	-.83 ^{***}	-
<i>M</i>	1.71	15.84	16.86	457.02	88.22	3.57
<i>SD</i>	1.04	3.06	4.26	59.23	8.22	2.14

Note. Socioeconomic status was assessed by income-to-needs ratio. For children's sleep duration, 457 min equates to 7 hrs and 37 min.

^t
 $p < .10$.

^{*}
 $p < .05$.

^{**}
 $p < .01$.

^{***}
 $p < .001$.