Exposure to Parental Alcohol Use Rather Than Parental Drinking Shapes Offspring's Alcohol Expectancies

Koen Smit, Carmen Voogt, Roy Otten, Marloes Kleinjan, and Emmanuel Kuntsche

Background: Alcohol expectancies (AE), that is, the anticipated effects of alcohol, start developing early in childhood and are important predictors of alcohol use years later. Whereas previous research has demonstrated that parental drinking relates to children's AE, this study aims to test whether *exposure* to parental alcohol use mediates the link between parental alcohol use and positive and negative AE among children (6 to 8 years) and early adolescents (12 to 15 years).

Methods: Longitudinal multi-informant family studies were conducted in the Netherlands among children (Study 1 (2015 to 2017): $N = 329$; 48.9% boys; $M_{age} = 4.6$) and adolescents (Study 2 [2015 to 2018]: $N = 755$; 45.6% boys; $M_{age} = 11.3$). Fathers' and mothers' alcohol use in terms of quantity and exposure (i.e., the frequency of alcohol use in 9 family-specific situations), and offspring's AE were collected using online questionnaires.

Results: Structural equation modeling conducted in the full sample and separately by gender revealed the following: For children, no associations were found in the full sample. However, gender-specific results indicated that fathers' exposure was associated with (and mediated) favorable AE. Among adolescents, fathers' exposure was associated with (and mediated) social and coping AE (both boys and girls) and enhancement AE (only boys). Contrastingly, neither mothers' alcohol use nor its exposure was associated with any AE. Although different associations were found by offspring's gender, strong evidence for gender differences was lacking.

Conclusions: This study indicates that, for specific expectancies, exposure to fathers' alcohol use shapes offspring's cognitions about the effects of alcohol, rather than fathers' alcohol use in general. Prevention efforts could focus on lowering the degree to which fathers expose their drinking, which might be more easily changeable than drinking in general.

Key Words: Children, Adolescents, Alcohol Expectancies, Parental Alcohol Use, Exposure to Parental Alcohol Use.

ALCOHOL EXPECTANCIES (AE) are defined as the anticipated positive or negative effects of consuming alcohol (Jones et al., 2001). Evidence suggests that AE develop years before the first alcohol consumption (Jester et al., 2014; Kuntsche, 2017; Voogt et al., 2017a) and that they are important determinants of alcohol-related behavior later on (Campbell and Oei, 2010; Donovan, 2004; Smit et al., 2018b). To illustrate, 1 longitudinal study found that positive AE among 6- to 8-year-olds predicted alcohol initiation and binge drinking (i.e., drinking 5 or more glasses of standard alcohol units on 1 drinking occasion [Wechsler and Nelson, 2001]) even 9 years later (Jester et al., 2014). Therefore, it is crucial to gain further insight about what shapes AE in childhood and in early adolescence. Since parents are the primary socialization agents up to adolescence (Steinberg, 2008), the acquisition of AE has been repeatedly found to depend on factors such as parental drinking (Campbell and Oei, 2010; Smit et al., 2018b). However, the role of parental drinking *in the vision* of their offspring and its influence on AE remain unclear. In response, the current study took a developmental perspective by examining whether *exposure* to parental alcohol use predicts AE among children (6 to 8 years) and young adolescents (12 to 15 years).

Research on AE among young children is limited. A systematic review of studies conducted in the past 40 years on alcohol-related cognitions of children revealed that only 5 cross-sectional studies have focused on 2- to 10-year-old children's AE (Voogt et al., 2017a), with most studies focusing on children aged 8 years and older. Available studies have shown that children as young as 6 years develop AE (Mares

From the, Behavioural Science Institute (KS, CV, RO, EK), Radboud University, Nijmegen, Nijmegen, The Netherlands; Trimbos Institute (KS, CV, MK), Netherlands Institute of Mental Health and Addiction, Utrecht, The Netherlands; Research & Development (RO), Pluryn, Nijmegen, The Netherlands; REACH Institute (RO), Arizona State University, Phoenix, Arizona; Department of General Social Sciences (MK), Utrecht University, Utrecht, The Netherlands; and Centre for Alcohol Policy Research (EK), La Trobe University, Melbourne, VIC, Australia.

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Reprint requests: Emmanuel Kuntsche, PhD, Centre for Alcohol Policy Research, La Trobe University, Health Sciences 2|516, Melbourne, VIC 3086, Australia; Tel.: +61 3 9479 8723; Fax: +61394798711; E-mail: e.kuntsche@latrobe.edu.au

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et al., 2015; Pieters et al., 2010). Moreover, recent studies have shown that children as young as age 3 already have some ideas about the positive (e.g., joyful, relaxed) and negative (e.g., angry, sad) emotional changes that occur when adults drink (Jones and Gordon, 2017; Kuntsche, 2017; Kuntsche and Kuntsche, 2018). Thus, young children already have some understanding of the valence of the effects (positive vs. negative) caused by alcohol (Kuntsche, 2017).

When children grow into adolescence, they develop stronger positive AE and weaker negative AE (Jones and Gordon, 2017; Smit et al., 2018b). It is evident that positive AE are more consistent predictors of alcohol use initiation and subsequent excessive alcohol use (Smit et al., 2018b). This is why we focused on positive AE among adolescents. Learning about the reinforcing effects of alcohol strongly depends on one's own experiences with alcohol (Campbell and Oei, 2010; Smit et al., 2018b). However, as most adolescents before the age of 13 years have not yet initiated alcohol use (Van Dorsselaer et al., 2016), the uptake of these AE depends mostly on the social environment of the youngsters.

Available research has shown that parental alcohol use plays an important role in the acquisition of AE, even early in life. For instance, a study in Switzerland indicated that already at the age of 3 to 6, children's AE correspond to their parents' drinking behavior (Kuntsche and Kuntsche, 2018). Specifically, children of moderately drinking parents developed stronger associations with the positive effects of alcohol (e.g., feeling joyful, funny, and relaxed), whereas children of heavy drinking parents developed stronger associations with the negative effects of alcohol use (e.g., feeling angry, sad, or depressed). The latter finding was confirmed in an earlier study with children from the United States, showing that having an alcoholic family member was associated with less positive AE (Miller et al., 1990). Overall, previous studies have suggested that (moderate) alcohol use by parents may determine the acquisition of AE. Yet, evidence among young children up to 10 years of age remains scarce.

Research on the role of parental alcohol use in the acquisition of AE among adolescents is more clear-cut. Evidence indicates that parental alcohol use is an important predictor of predominantly positive AE among drinking (Smit et al., 2018b) and nondrinking adolescents (Ting et al., 2015). It seems that even when parental alcohol use is more problematic, adolescents develop more positive AE but no negative AE (Colder et al., 1997). In addition to parental alcohol use, engaging in family activities (e.g., social activities and birthdays) is also associated with more positive AE among adolescents (Goldstein et al., 2013). Parents' drinking behavior seems to be important in the acquisition of AE in children and adolescents. Especially, the drinking behavior of fathers has a big effect on alcohol-related cognitions and use (Pettersson et al., 2009; Smit et al., 2018a), probably because men drink more than women do (Holmila and Raitasalo, 2005) and have a nonrestrictive attitude toward alcohol-related behaviors (Pettersson et al., 2009). Together, the existing findings indicate that modeling behavior is important for the acquisition and development of AE among children and adolescents. However, all studies used the drinking of the parents but not the offspring's perception of this behavior as a predictor.

To investigate the role of parental drinking behavior on children's and adolescent's AE, previous studies have used quantity and/or frequency measures of parental alcohol use. Although these measures are generally deemed valid to assess and capture the effects of drinking behavior (Room, 2000), parents may not necessarily drink in the direct presence of their offspring. For instance, some parents might drink frequently but only when the children are in bed (zero exposure), while other parents might drink less frequently but always when having dinner (daily exposure; Smit et al., 2018a). In accordance with the Social Learning Theory, *exposure to parental alcohol use* (the frequency of alcohol use in family-specific situations) might be a more proximal measure for predicting AE in children and adolescents compared to the amount of parental drinking. It is likely that more alcohol use by parents leads to a higher exposure of alcohol use to offspring, which is subsequently associated with offspring's AE. Frequent and excessive parental drinking would have little effect on children when happening outside of their vision, for example, with colleagues after work. Thus, a *part* of the effect of parental drinking is likely to run via actual exposure to alcohol use, which we expect to be a more proximal predictor of AE. Following this reasoning, exposure to parental alcohol use should function as a mediator, as parental drinking potentially leads to exposure. This in turn affects children's AE rather than parental alcohol use per se. The development of AE through exposure to parental alcohol use could be interpreted as an underlying mechanism.

Although most previous studies were cross-sectional (Voogt et al., 2017a), a longitudinal study would allow us to draw firmer conclusions on whether exposure mediates the association between parental drinking and youth AE. In terms of prevention, longitudinal evidence can help identify which offspring are at higher risk of acquiring stronger positive and weaker negative AE in early childhood and in early adolescence, thereby putting these children at risk for early alcohol initiation and subsequent excessive alcohol use (Campbell and Oei, 2010; Donovan, 2004; Smit et al., 2018b). As a response, we used 2 different samples (i.e., young children aged 6 to 8 years who are likely to develop AE and early adolescents aged 12 to 15 years who are increasingly likely to initiate alcohol), which made it possible to examine the role of exposure to parental alcohol use and its association with AE across 2 developmental milestones. Thus, to (i) better understand the process that ultimately leads to the endorsement of AE and (ii) better target prevention campaigns, the main aim of this study was to test the mediating role of exposure to fathers' and mothers' alcohol use, that is, drinking *in the presence* of the child, in the relation between parental alcohol use and AE among children (6 to 8 years old), among adolescents (12 to 15 years old), and among their parents.

Hypotheses

Based on prior evidence, we tested 3 hypotheses. First, we expected that greater exposure to parental alcohol use would lead to more positive and less negative AE among 6- to 8-year-olds and 12- to 15-year-olds over time. Second, we expected that parental alcohol use will be associated with increased exposure, which will then predict children's and adolescent's AE 1 year later (Kuntsche and Kuntsche, 2018). Third, considering the previously found stronger modeling effects of fathers (Pettersson et al., 2009; Smit et al., 2018a), we expected that any links between exposure and children's and adolescents' AE were stronger for fathers' alcohol use compared to mothers' alcohol use. In addition, there are differences in associations between parental alcohol use and offspring AE by gender of the offspring. Two studies among early adolescents in the United States and among 3- to 6-year-olds in Switzerland showed that paternal alcohol use was associated with AE for sons, but not for daughters (Handley and Chassin, 2009; Kuntsche and Kuntsche, 2018), whereas the opposite was found among 6- to 9-year-olds in the Netherlands (Mares et al., 2015; Voogt et al., 2017a). Considering these mixed findings of parental alcohol use and AE among boys and girls, we tested the hypotheses for the full sample and separately by gender, but we did not formulate any gender-specific hypotheses.

MATERIALS AND METHODS

Two multi-informant cohort studies were used to study the mediating role of exposure to parental alcohol use in the relation between parental alcohol use and AE in childhood and early adolescence. Study 1 was a longitudinal family design involving children who were between 4 and 6 years old at baseline and then followed for 2 years. Study 2 utilized a similar design, focusing on early adolescents who were between 10 and 13 years old at baseline and subsequently followed for 3 years.

Methods Study 1

Procedure. To obtain a nationally representative sample, parents and their 4- to 8-year-old children were recruited through primary schools (2014 to 2015). Schools were randomly selected from 5 provinces in the Netherlands. After agreeing to participate, schools were requested to distribute invitation letters to parents of children in the first and second grades. To participate in the study, parents were asked to provide active consent either via a secured website (http://www.vol-onderzoek.nl) or on paper. Details about the fieldwork are described in Voogt and colleagues (2017b).

The data of this multi-informant longitudinal family study were collected during yearly home visits (2015 to 2017, T0 to T2). Children completed a task on a tablet, while parents completed online questionnaires. At the end of the home visit, a small gift was provided to the child (e.g., a pencil or stickers) and the parents received 1 gift voucher of 10€ (approximately $11). Moreover, participating families were entered into a drawing for €100 (approximately $110). The Ethical Committee of the Faculty of Social Sciences of the Radboud University (ECSW2014-2411-272) approved this study's procedures, as described in Voogt and colleagues (2017b).

Sample. Of 831 schools contacted, 92 schools (11.1%) agreed to participate. From these schools, 329 children, 234 fathers, and 301 mothers agreed to participate. Additional information regarding motivation for nonparticipation of schools and participants can be found in Voogt and colleagues (2017a). At baseline, the child sample ranged in age from 4 to 6 years (M = 4.78, Standard Deviation (SD) = 0.78) and included 48.9% boys. Most children (98.1%) were of Dutch origin. Retention analyses on demographic characteristics (gender, age, and ethnicity) revealed that completers (n = 316: children who completed T0, T1, and T2) differed from noncompleters (n = 13) only in terms of age ($t(326) = 2.26$, $p = 0.03$), as completers were younger on average (M = 4.76, SD = 0.73) compared to noncompleters (M = 5.23, SD = 0.83).

In terms of education level of fathers (M_{age} = 39.8, SD = 5.78), 12.6% completed primary school, lower secondary, 45.4% completed higher secondary, vocational school, and 44.1% completed college or university. For mothers (M_{age} = 37.1, SD = 4.72), the percentages were 5.3, 42.4, and 52%, respectively. Although retention rates were high (78% of the fathers and 89% of the mothers participated across all 3 waves), independent-samples t-tests and chi-square tests were conducted to determine whether those who participated in all waves differed from "dropouts" in terms of age, education, alcohol use, and exposure to alcohol use. No significant differences appeared on any of these variables (all ps > 0.05), except that completers reported higher levels of education compared to those who did not complete all questionnaires ($\chi^2_{df=2} = 13.29$, $p < 0.001$).

Measures. Demographics (T0)—Child's age and gender and parent's age, education level, and ethnicity were reported.

*Parental Alcohol Use Quantity (T1)—The quantity of a*lcohol consumed in the last week was reported by both parents separately (Hajema and Knibbe, 1998; Room, 2000). A description of standard glasses (with 1 glass containing 10 g of pure ethanol; Health Council of the Netherlands, 2006) was provided. Participants indicated how many standard glasses of alcohol they consumed on weekdays, and weekend days, within and outside the home. The quantities indicated in the 4 items were summed to create a total number of drinks.

Exposure to parental alcohol use (T1) was assessed by the frequency of alcohol use in 9 of 18 family-specific situations that parents deemed most common for alcohol use such as a family barbecue or a birthday party.[1] To mitigate the effects of social desirability and underreporting, we asked parents to indicate how often they drank in family-specific situations in which alcohol consumption is common. Such drinking represents higher odds for children to be exposed to parental alcohol use. Both parents reported exposure as the frequency of drinking in family-specific situations. Responses were recorded on a 5-point Likert scale ranging from 0 (*never*) to 4 (*always*). The scale for both paternal exposure and maternal exposure showed a high internal consistency ($\alpha_{father} = 0.90$, $\alpha_{mother} = 0.93$). Two latent constructs were developed, including both paternal and exposure to maternal alcohol use, with factor loadings ranging from 0.54 to 0.90.

Children's AE (T2)—To measure positive and negative AE of children, we used the Dutch version of the AE Scale for Children (Dunn and Goldman, 1996; Pieters et al., 2010). Children were asked about whether alcohol use has positive effects (e.g., "Does a person become friendly when they drink alcohol?") and/or negative effects (e.g., "Does a person become mean when they drink alcohol?"). During home visits, a research assistant read the statements aloud to foster understanding of the children. Responses were given on a 4-point Likert scale ranging from 0 (*never*) to 3 (*always*). The

[1]Results included in a manuscript submitted for publication.

subscales showed high to sufficient internal consistency ($\alpha_{positive} = 0.85$, $\alpha_{negative} = 0.70$).

Analyses. First, for descriptive purposes and for testing whether study variables differed for boys and girls and for fathers and mothers, descriptive statistics, bivariate Pearson's correlations (Table S1), and *t*-tests were calculated in SPSS 24.0, IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp. Independent-samples *t*-tests were used to test differences between boys and girls in both studies. To test differences between parents regarding the degree of parental alcohol use within the families, paired-samples *t*-tests were used.

Subsequently, structural equation modeling (SEM) performed in Mplus 7.4, MPLUS (Version 7.4). [Computer Software]. Los Angeles, CA: (Muthén and Muthén, 2010) was used to test the hypothesized mediation of parental alcohol exposure (T1) on the links from parental alcohol use (T1) to children's AE (T2) (see Fig. 1). The degree to which parents expose their drinking (mediator) theoretically co-occurs with alcohol use (independent variable) at the same point in time and does not necessarily do so 1 year later. Therefore, we chose to assess both parental drinking and exposure at the same time point.

Model fit was examined using the Comparative Fit Index (CFI), the Tucker–Lewis Index (TLI), the Root Mean Square Error of Approximation (RMSEA), and the Standardized Root Mean Square Residual (SRMR). For the CFI and TLI, values >0.90 are considered adequate (Iacobucci, 2010). The RMSEA is a noncentrality parameter, for which values of 0.01, 0.05, and 0.08 are considered excellent, good, and mediocre, respectively (MacCallum et al., 1996). For the SRMR, values as high as 0.08 are deemed acceptable (Hooper et al., 2008).

Mediation was tested using the indirect command in Mplus and included bootstrapping with 1,000 random draws (MacKinnon et al., 2007). Missing data patterns were not related to any of the study outcomes. Therefore, we used full information maximum likelihood (FIML) procedures to account for missing data. In general, FIML and similar missing data techniques, such as multiple imputation, can provide good results, particularly if predictors can be found for missing values. Including age as a confounder, the models were estimated for the full sample and separately for boys and girls. First, the full sample results are presented in Table 2 and subsequently the gender-specific results in Table 4. We tested gender differences in 2 ways. First, in the overall model, we examined whether the paths of interest combined (i.e., the paths that differed in significance for boys and girls) are generally different between boys and girls. Second, independent of overall gender differences, we inspected whether the paths differed in significance and the effect size of specific paths differed substantially (i.e., the coefficient is at least twice as high in one group compared to the other). We used a smaller model to test gender differences in the specific paths, because the overall model was apparently too insensitive to detect specific differences. In both cases, we examined whether the model fit was significantly better when tested gender-separately by simultaneously constraining the path(s) to equivalence between the groups. Using the Satorra–Bentler scaling-corrected chi-square difference test (Satorra & Bentler, 2001), we examined whether the chi-square of the freely estimated model significantly differed from the constrained model.

Results Study 1

Descriptives. Pairwise *t*-tests demonstrated that fathers consumed more alcohol compared to mothers in the last week (Table 1). Similarly, fathers reported more alcohol exposure compared to mothers. Boys and girls did not significantly differ in levels of positive and negative AE. Further, children reported significantly stronger positive ($M = 0.78$, $SD = 0.77$) compared to negative ($M = 0.56$, $SD = 0.55$) AE, $t(291) = 3.80$, $p < 0.001$.

Mediation Model. The model showed an adequate fit ($\chi^2_{df=446} = 685.44$; CFI = 0.940; TLI = 0.933; RMSEA = 0.057; SRMR = 0.058). The gender-specific constrained versus unconstrained model for children was significant ($\Delta\chi^2 = 18.42$, $\Delta df = 3$, $p < 0.001$), showing that the results of the paths differed between boys and girls. The results for the entire sample can be found in Table 2, next to the gender-specific results. The full group model did not show any significant associations of exposure with AE. However, when looking at boys and girls separately, a different pattern appeared.

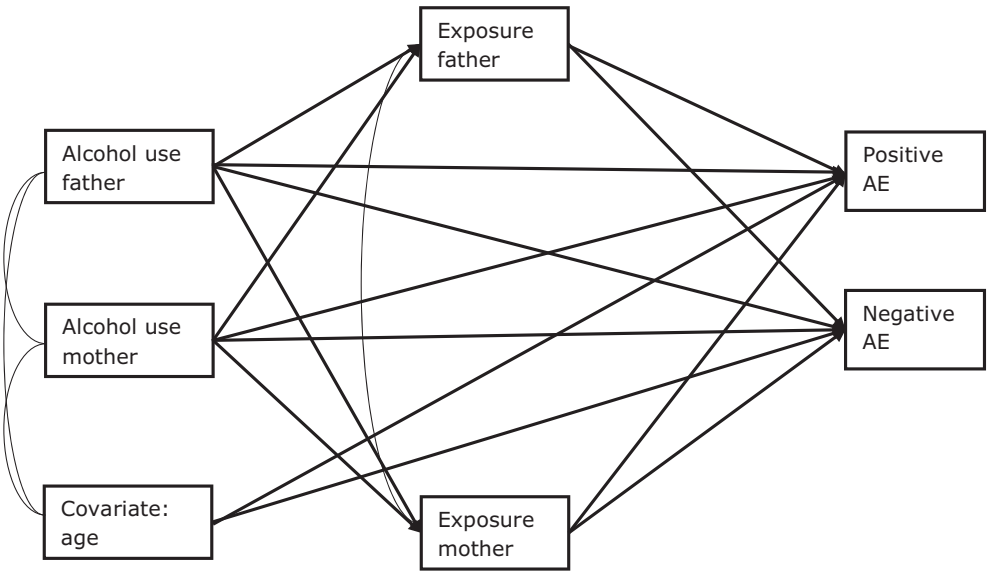

Fig. 1. Conceptual model testing the mediating effect of exposure to parental alcohol use on the relation between parental alcohol use and children's AE.

Table 1. Means (Standard Deviations) of Alcohol-Related Behaviors of Parents and AE of Their Children Separately by Gender (Study 1)

	Range	Male	Female	*t*-value	*p*	Cohen's *d*
Parents						
Alcohol quantity	[0 to 37][a]	7.04 (6.73)	3.08 (3.89)	9.92	<0.001	0.72
Alcohol exposure (T1)	[0 to 4]	2.01 (0.80)	1.54 (0.91)	8.54	<0.001	0.55
Children						
Positive expectancies (T2)	[0 to 3]	0.73 (0.76)	0.83 (0.78)	−1.01	0.315	0.12
Negative expectancies (T2)	[0 to 3]	0.58 (0.58)	0.55 (0.53)	0.50	0.615	0.05

Descriptive statistics are reported in means (standard deviations in brackets); alcohol use quantity (T1) and exposure (T2) were reported by parents, AE (T2) were reported by children.
[a]Empirical range based on data file.

Table 2. Parental Alcohol Use and Exposure as Predictors of Children's Positive and Negative AE 1 Year Later (Separately by Gender and for the Total Group)

	Boys		Girls		Total group	
	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers
Positive AE						
Quantity (c′)	−0.054 (0.099)	0.031 (0.134)	−0.472 (0.104)***	−0.070 (0.118)	−0.231 (0.078)**	−0.017 (0.082)
Exposure (b)	−0.074 (0.116)	0.115 (0.150)	0.290 (0.117)*	0.122 (0.109)	0.103 (0.084)	0.111 (0.084)
Quantity via exposure (a*b)	−0.039 (0.061)	0.076 (0.102)	0.154 (0.067)*	0.069 (0.060)	0.053 (0.044)	0.068 (0.052)
Covariate: age	−0.118 (0.099)		−0.064 (0.078)		−0.104 (0.058)	
Negative AE						
Quantity (c′)	0.001 (0.125)	−0.081 (0.144)	0.128 (0.136)	0.092 (0.090)	0.036 (0.080)	0.010 (0.083)
Exposure (b)	−0.301 (0.139)*	0.100 (0.137)	−0.002 (0.106)	−0.218 (0.130)	−0.148 (0.085)	−0.044 (−0.085)
Quantity via exposure (a*b)	−0.158 (0.075)*	0.066 (0.092)	−0.001 (0.057)	−0.123 (0.075)	−0.076 (0.044)	−0.027 (0.052)
Covariate: age	0.156 (0.086)		0.289 (0.071)***		0.219***	

Effects are standardized betas (standard errors in brackets); alcohol use quantity (T1) and exposure (T2) were reported by parents; AE (T2) were reported by children.
*$p < 0.05$; **$p < 0.01$; and ***$p < 0.001$.

For boys, children's exposure to fathers' alcohol use predicted *less negative* AE 1 year later (Table 2). Additionally, children's exposure to fathers' alcohol use mediated the relation between fathers' alcohol use and boys' negative AE. Thus, fathers' alcohol use was positively associated with exposure, which was associated with less negative AE. Fathers' alcohol use did not directly predict any AE. The relation between fathers' alcohol exposure and positive AE was not significant. For mother, neither alcohol use nor the alcohol exposure predicted any of the boys' AE.

For girls, exposure to fathers' alcohol use predicted *more positive* AE 1 year later (Table 2). In contrast to boys, fathers' alcohol use predicted less positive AE among girls. Fathers' alcohol exposure mediated the relation between fathers' alcohol use and girls' positive AE. Thus, fathers' alcohol use was positively associated with exposure, which was associated with more positive AE. The relation between fathers' alcohol exposure and girls' negative AE was not significant. For mother, neither alcohol use nor the exposure predicted any of the girls' AE.

Methods Study 2

Procedure. Data were derived from a multi-informant 7-wave longitudinal family study conducted in parallel with the recruitment and data collection of Study 1. Six graders (10 to 13 years old) and their mothers were recruited by research assistants who visited 104 primary schools in the Netherlands (for details, see Smit et al., 2018a). Written active consent was obtained from all participants through the study website. The baseline questionnaires for adolescents were administered in classrooms. Mothers were requested to complete the questionnaires online. In the following period of 3 years, online questionnaires were sent to the adolescents every 6 months and their mothers every 12 months. Yearly monetary incentives (€10) were provided to both adolescents and their mothers. From this more comprehensive study (2015 to 2018, T0 to T6), data were drawn from the second follow-up (T2) and 1 year later (T4; for details, see Smit et al., 2018a).

Participants. Of the 765 participants who signed up, 755 adolescents (45.6% boys, $M_{age} = 11.27$, SD = 0.56) and 755 mothers ($M_{age} = 42.57$, SD = 4.66) who also reported on the alcohol use of 709 fathers (93.9%) completed the baseline questionnaires. Most participants were born in the Netherlands (97.6% of adolescents). Most adolescents lived with both parents (78.5%). Regarding education level, 13.3% of mothers completed primary, 45.0% completed secondary, and 41.8% completed college education. Retention rates were high (94% of adolescents and 93% of mothers participated in both data collection waves). Attrition analyses revealed no differences in age, education, alcohol use, and exposure to alcohol use (all $ps > 0.05$).

Measurements. Parents' Alcohol Use Quantity and Exposure (T2)—Similar alcohol use quantity and exposure measurements were used as those in Study 1. One difference was that mother reported on both mothers' and fathers' alcohol use quantity. Additionally, adolescents reported on exposure to parental alcohol use, which showed a high internal consistency ($\alpha_{father} = 0.92$, $\alpha_{mother} = 0.92$). Two latent constructs were developed, including exposure to both paternal and maternal alcohol use, with factor loadings between 0.57 and 0.86.

Adolescents' AE (T4)—Alcohol expectancies were assessed using 12 items derived from the Drinking Motives Questionnaire Revised

Short Form (Kuntsche and Kuntsche, 2009), which were transferred from motives (i.e., *I drink to achieve X*) into expectancies (i.e., *After drinking, I expect X to occur for people in general*; Kuntsche et al., 2010). Crossing the dimensions valence (i.e., positive or negative) and source (i.e., internal or external) of the expected effects, 4 AE factors can be distinguished (Cooper, 1994; Cox & Klinger, 1988; 1990): enhancement (i.e., to obtain positive feelings), coping (i.e., to avoid or reduce negative feelings), social (i.e., to obtain social rewards), and conformity (i.e., to avoid social rejection). Response categories ranged from 0 (very unlikely) to 4 (very likely). Internal consistencies were sufficient to high ($\alpha_{enhancement} = 0.68$, $\alpha_{social} = 0.81$, $\alpha_{coping} = 0.85$, $\alpha_{conformity} = 0.75$).

Analyses. The same strategy for analysis was applied to both studies. For details, please see Study 1. First, descriptive analyses were conducted. Bivariate correlations can be found in Table S2. Second, SEM was used to test the hypothesized mediation of parental alcohol exposure (T2) on the links from parental alcohol use (T2) to adolescents' AE (T4) (see Fig. 2).

Results Study 2

Descriptives. Pairwise *t*-tests demonstrated that fathers consumed more alcohol compared to mothers in the last week (Table 3). In addition, adolescents *reported more alcohol exposure* by fathers compared to mothers. Boys reported stronger social AE compared to girls, but no differences appeared for the other dimensions.

Mediation Model. The model showed an adequate fit ($\chi^2_{df=546} = 968.51$; CFI = 0.965; TLI = 0.959; RMSEA = 0.045; SRMR = 0.045). The gender constrained model was not significantly different from the unconstrained model ($\Delta\chi^2 = 5.50$, $\Delta df = 5$, $p = 0.358$). Therefore, we presented the full group results in Table 4 and give a brief outline of the gender-specific results.

For the full sample, exposure to fathers' alcohol use mediated the associations between fathers' alcohol use and social and coping AE. Thus, fathers' alcohol use was associated with exposure, which subsequently predicted more social and coping AE. Additionally, fathers' alcohol use quantity was associated with weaker enhancement AE, but not with any other AE. For mother, neither alcohol use nor exposure to alcohol use predicted any AE.

Albeit not significantly different based on the overall chi-square test, several associations were more than twice as strong for boys or girls. For boys, exposure to fathers' alcohol use predicted more enhancement AE 1 year later ($\Delta\chi^2 = 4.00$, $\Delta df = 1$, $p = 0.045$), whereas for girls, fathers' alcohol quantity was associated with weaker enhancement AE ($\Delta\chi^2 = 4.58$, $\Delta df = 1$, $p = 0.032$). For girls, the only additional finding was that fathers' alcohol use quantity appeared associated with weaker coping AE, but this association did not differ significantly between boys and girls ($\Delta\chi^2 = 3.58$, $\Delta df = 1$, $p = 0.058$).

DISCUSSION

This study examined whether exposure to alcohol use by parents mediated the association between the amount of parental alcohol use and AE in childhood and adolescence 1 year later by using 2 longitudinal multi-informant family studies. Whereas previous studies showed that parental drinking shapes AE among young adolescents (Smit et al., 2018b) and even among young children (Kuntsche and Kuntsche, 2018; Voogt et al., 2017a), this study is the first suggesting that this is directly associated with *exposure* to fathers' alcohol use and not with fathers' alcohol use per se. Moreover, fathers' alcohol exposure mediated the association between fathers' drinking and offspring's AE, indicating that increased drinking is associated with increased exposure for fathers, which subsequently predicted AE. Specifically, for children, no associations were found when looking at the full sample. However, gender-specific results indicated that fathers' exposure was associated with (and mediated) positive AE of young girls and negative AE of young boys. Among adolescents, fathers' exposure was associated with (and mediated) social and coping AE (both boys and girls) and enhancement AE (only boys). Although different associations were found by offspring's gender, strong evidence for gender differences was lacking. Furthermore, the effects were absent for mothers' alcohol use and exposure.

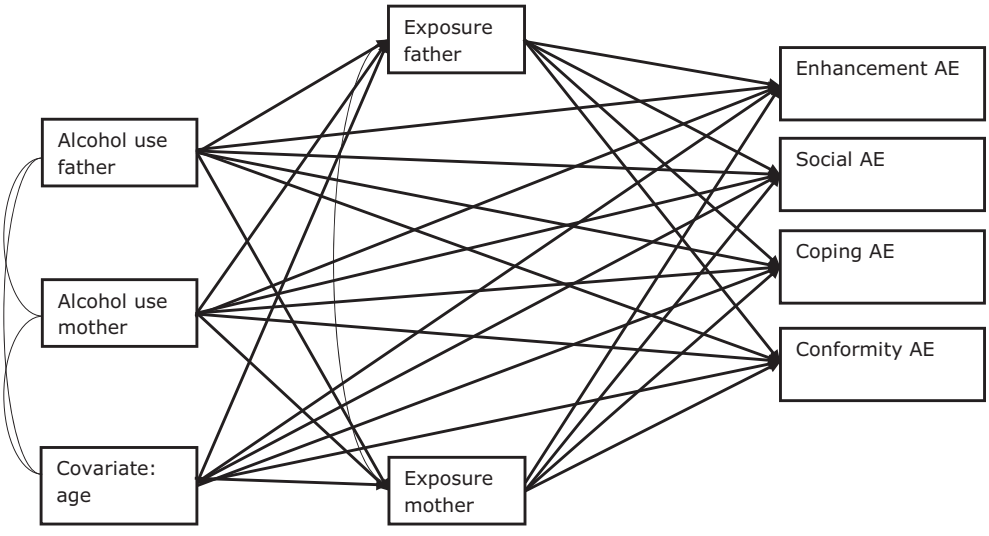

Fig. 2. Conceptual model testing the mediating effect of exposure to parental alcohol use on the relation between parental alcohol use and adolescents' AE.

Table 3. Means (Standard Deviations) of Alcohol-Related Behaviors of Parents and AE of Their Children separately by Gender (Study 2)

	Range	Male	Female	*t*-value	*p*	Cohen's *d*
Parents						
Alcohol quantity	[0 to 60][a]	5.57 (6.74)	2.75 (4.05)	13.12	<0.001	0.51
Alcohol exposure	[0 to 4]	1.53 (0.99)	1.26 (0.93)	7.81	<0.001	0.28
Adolescents						
Enhancement AE	[0 to 4]	2.59 (0.80)	2.63 (0.68)	−0.61	0.540	0.05
Social AE	[0 to 4]	1.90 (1.05)	1.74 (1.00)	2.03*	0.043	0.16
Coping AE	[0 to 4]	2.04 (0.99)	2.00 (0.98)	0.43	0.667	0.04
Conformity AE	[0 to 4]	1.75 (0.89)	1.71 (0.86)	0.79	0.429	0.05

Descriptive statistics are reported in means (standard deviations in brackets); alcohol use quantity (T2) was reported by mothers, exposure (T4) and AE (T4) were reported by adolescents.
[a]Empirical range based on data.

Table 4. Parental Alcohol Use and Exposure, as Predictors of Adolescent's AE 1 Year Later (Separately by Gender and for the Full Group)

	Boys		Girls		Total group	
	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers
Enhancement AE						
Quantity (c′)	−0.045 (0.086)	−0.022 (0.083)	−0.227 (0.114)*	0.092 (0.073)	−0.158 (0.059)**	0.064 (0.060)
Exposure (b)	0.165 (0.079)*	−0.032 (0.081)	0.033 (0.073)	0.053 (0.076)	0.102 (0.056)	0.010 (0.058)
Quantity via exposure (a*b)	0.081 (0.041)*	−0.020 (0.054)	0.017 (0.039)	0.032 (0.045)	0.051 (0.028)	0.006 (0.036)
Covariate: age	−0.059 (0.054)		0.059 (0.220)		−0.003 (0.037)	
Social AE						
Quantity (c′)	0.002 (0.085)	0.024 (0.083)	−0.146 (0.075)	0.089 (0.065)	−0.085 (0.058)	0.077 (0.059)
Exposure (b)	0.249 (0.079)**	0.006 (0.076)	0.185 (0.081)*	0.109 (0.081)	0.220 (0.054)***	0.049 (0.057)
Quantity via exposure (a*b)	0.122 (0.042)**	0.004 (0.048)	0.093 (0.044)*	0.066 (0.050)	0.109 (0.029)***	0.030 (0.035)
Covariate: age	−0.032 (0.049)		−0.034 (0.048)		−0.024 (0.036)	
Coping AE						
Quantity (c′)	0.027 (0.084)	0.022 (0.099)	−0.176 (0.079)*	0.129 (0.071)	−0.098 (0.059)	0.102 (0.060)
Exposure (b)	0.127 (0.083)	−0.044 (0.084)	0.116 (0.077)	−0.009 (0.079)	0.124 (0.055)*	−0.022 (0.058)
Quantity via exposure (a*b)	0.062 (0.042)	−0.028 (0.054)	0.058 (0.041)	−0.006 (0.048)	0.061 (0.028)*	−0.014 (0.036)
Covariate: age	−0.111 (0.058)*		0.012 (0.048)		0.042 (0.037)	
Conformity AE						
Quantity (c′)	0.033 (0.093)	−0.087 (0.108)	−0.129 (0.084)	0.119 (0.081)	−0.073 (0.059)	0.040 (0.061)
Exposure (b)	0.122 (0.089)	−0.052 (0.087)	0.082 (0.083)	−0.074 (0.089)	0.102 (0.056)	−0.063 (0.059)
Quantity via exposure (a*b)	0.060 (0.044)	−0.033 (0.056)	0.041 (0.043)	−0.045 (0.054)	0.051 (0.028)	−0.039 (0.036)
Covariate: age	−0.005 (0.050)		−0.001 (0.052)		0.001 (0.037)	

Effects are standardized betas (standard errors in brackets); alcohol use quantity (T2) was reported by mothers, exposure (T4) and AE (T4) were reported by adolescents.
*$p < 0.05$; **$p < 0.01$; and ***$p < 0.001$.

In accordance with the first (and third) hypothesis, our findings indicated that exposure to fathers' alcohol use is directly associated with children's and adolescents' AE and that this exposure measure is a more consistent predictor of positive AE compared to fathers' alcohol use per se (Mares et al., 2015; Smit et al., 2018b). Accordingly, we found that the acquisition of these positive associations starts at an early age and is not limited to adolescents when they reach the age of drinking initiation (Van Dorsselaer et al., 2016). However, this was only the case when looking at the children gender-separately. Current findings add to previous studies by showing that fathers' alcohol use is more positively associated with AE in children and adolescents compared to mother's alcohol use (Handley and Chassin, 2009; Mares et al., 2015; Pieters et al., 2010).

One unexpected finding was that the *quantity* of fathers' alcohol use was associated with lower levels of positive AE for young children and lower enhancement and AE for early adolescents. Possibly, excessive drinking among fathers accounts for this association. Previous studies have shown that moderate parental alcohol use is associated with more positive and less negative AE (Mares et al., 2015), whereas excessive parental drinking has been found to be associated with less positive AE (Kuntsche and Kuntsche, 2018). The latter finding may explain the negative association between the quantity of fathers' alcohol use and children's positive AE. Fathers who drink higher quantities might behave "differently" or "unpredictably" in the eyes of offspring (Foster et al., 2017), resulting in less positive AE. In contrast, being *exposed* to fathers' moderate alcohol use in different family-specific situations, such as family barbecues, alcohol use may be perceived as more positive in the eyes of offspring and therefore result in more positive AE. Another explanation for the negative association between the quantity of fathers'

drinking and offspring's positive AE might be that fathers who drink without being seen by their offspring (e.g., with friends in a bar) are more conscious about their alcohol-specific socialization practices. Consequently, offspring might develop fewer positive associations with alcohol. However, this notion is speculative, and as such, it should be further examined in future studies.

In line with the second (and third) hypothesis, we found that only fathers' drinking was associated with exposure, which subsequently predicted stronger AE in both samples. This implies that the quantity of fathers' alcohol use is associated with less favorable AE; however, when fathers *expose* their drinking to their children in situations that are likely to be family-specific, this actually leads to more favorable AE among (adolescent) offspring. The current study provides more evidence in line with the Social Learning Theory (Bandura and McClelland, 1977), suggesting that *exposure* to behavior is associated with more favorable cognitions about alcohol years before and around the age of alcohol initiation. This is important because more positive AE were found to predict early alcohol initiation and more risky alcohol use in adolescence (Donovan, 2004; Morgenstern et al., 2011; Pieters et al., 2014; Smit et al., 2018b).

In accordance with the third hypothesis, we found stronger associations for fathers' drinking than for mothers' drinking. Moreover, it appeared that mothers' drinking was not predictive of any AE in both samples. This is possibly partly explained by fathers drinking more alcohol compared to mothers in general (Holmila and Raitasalo, 2005). This study extends previous literature by showing that fathers also drink more in family-specific situations compared to mothers (Tables 1 and 3). Accordingly, they are more likely to expose their offspring to alcohol consumption, thereby providing more opportunities to observe the emotional consequences of alcohol use. Our findings are in line with previous studies, indicating that fathers' modeling effects are stronger compared to mothers' modeling effects when it comes to alcohol use. The exposure to alcohol use of fathers might explain stronger modeling effects in previous studies (Cabrera et al., 2007; Mares et al., 2012; Seljamo et al., 2006; Van Der Vorst et al., 2013; Yu, 2003). In addition, there may be general differences in parenting between fathers and mothers. Indeed, fathers are thought to be less restrictive with regard to alcohol use and thus hold more liberal rules compared to mothers (Pettersson et al., 2009). This was reflected in previous results showing that fathers more often confirmed that their offspring already tasted alcohol at home (Pettersson et al., 2009; Sharmin et al., 2017).

Specific Findings for Cohort and Gender

Overall, similar patterns of results were found in both cohorts. However, several specific results emerged for children and adolescents. Gender-specific results should be interpreted with care, since we found mixed evidence for the existence of differences between boys and girls.

In Study 1 (children), no associations were found between exposure and offspring's AE when looking at the full sample. However, when looking at the gender-specific results, fathers' alcohol use exposure appeared to be directly associated with less negative AE for boys (e.g., people become *less* mean or unpleasant). Moreover, exposure seems to mediate the association between alcohol use and negative AE. For girls, a similar pattern emerged for positive AE. Exposure to fathers' alcohol use was directly associated with more positive AE (e.g., people become happier or nicer) and partially mediated the association between alcohol use and positive AE. Thus, boys might associate their fathers' alcohol use with reduced negative consequences (e.g., daddy becomes less mean), whereas girls associate it with increased positive consequences (e.g., daddy becomes more fun). However, the gender-specific results should be interpreted with care and further research is needed before any strong conclusions can be drawn regarding the effect of parents' drinking *in the presence* of their children. Preliminary results do imply that exposure to parental alcohol use is associated with *favorable* AE for both boys and girls, suggesting that exposure plays a role in the development of more positive attitudes toward alcohol use.

In Study 2 (adolescents), exposure to fathers' alcohol use predicted social AE among adolescents 1 year later. This is consistent with prior studies on the association between parental alcohol use and adolescent's positive AE (Colder et al., 1997), which suggested that young adolescents observe positive social effects of fathers' alcohol use during family gatherings, such as birthdays. As early adolescents approach drinking age, this result becomes particularly relevant, since social AE are predictors of alcohol use initiation[2] (Jester et al., 2014; Smit et al., 2018b). Additionally, adolescents' exposure to fathers' alcohol use was associated with coping AE, indicating that when adolescents see their fathers drinking more often, they associate alcohol use with drinking to forget problems. This is important, since coping AE are found to be predictive of peak alcohol use in later adolescence (Jester et al., 2015). Fathers' exposure appeared predictive of enhancement AE among boys. Enhancement AE are important predictors of enhancement motives, which have been found to predict heavy drinking in adolescence and young adulthood (Kuntsche and Kuendig, 2012; Kuntsche et al., 2010). Importantly, future studies should seek further evidence on whether different modeling effects exist for boys and girls.

Strengths and Limitations

This study has several strengths. First, the study included 2 large nationwide samples of young children aged 6 to

[2]One could argue that the adolescents' social AE are associated with their own alcohol use. A similar model taking lifetime alcohol use into account, showed that this did not change the results (to be obtained from the author upon request).

8 years (N = 329) and young adolescents aged 12 to 15 years (N = 765). Second, we developed 2 longitudinal multi-informant family designs with parallel time points and variables to examine the role of exposure to parental alcohol use in the acquisition of AE from a developmental perspective. A strength of Study 1 was that the data were collected during home visits, such that children were in a familiar and safe environment (Sweet and Appelbaum, 2004) while the researcher explained to them every expectancy item to help them understand what is being asked of them.

Although several important conclusions can be drawn from this study, this study had several shortcomings that should be mentioned. One limitation is that participation rates during recruitment were low in both schools and families (Smit et al., 2018a; Voogt et al., 2017a). Low rates are, unfortunately, common in substance use research in the Netherlands (Van Loon et al., 2003). In addition, individuals with lower levels of education were underrepresented in both studies (around 10% in current study compared to 30% in the Netherlands; StatLine, 2018) and the sample was predominantly of Dutch origin. These points should be considered when generalizing the results to the Dutch population. Second, although we considered the temporary sequence from alcohol use to exposure to alcohol use to AE in the analyses, we assessed AE only at 1 time point; hence, future studies with more frequent measurements of AE over time are needed to gain further insight into the predictive role of parental alcohol exposure in AE *changes*. Moreover, longitudinal studies are advised: (i) to examine how long the effects of alcohol use exposure are lasting and (ii) to evaluate the transition from AE to alcohol use initiation and subsequently to more risky drinking patterns in adolescence and beyond within the same individuals. Third, in Study 1, we measured exposure to parental drinking by examining the frequency of drinking in family-specific situations. Thus, we did not explicitly ask parents how often they drank in the presence of their offspring in family-specific situations to mitigate the effects of social desirability and underreporting. However, this limits the conclusions on alcohol exposure in Study 1. One final limitation specific to Study 2 is that we relied on mothers' reports of fathers' alcohol use quantity. Practical reasons included the restricted financial means required for incentives (Smit et al., 2018a). Fortunately, the fact that both mothers and adolescents completed the questionnaires enabled us to assess exposure to parental alcohol use as perceived by the young adolescents, which was one of this study's strengths (Smith et al., 1998).

Implications

This is one of the first studies to consider exposure to parental alcohol use as a mediator of the association between parental alcohol use and offspring's AE (Voogt et al., 2017a). In our view, future studies could benefit from including exposure measures (i.e., including questions about drinking in the *presence* of children). This might provide more robust results in terms of modeling parental behavior and therefore may provide stronger explicit guidelines for prevention programs. Currently, the existing antismoking campaigns (e.g., the Smoke-Free Generation campaign in the Netherlands) focus on reducing parental smoking exposure. A similar message may be conveyed to parents regarding alcohol use. For instance, prevention message campaigns could suggest that parents limit their alcohol consumption to specific contexts in which no children are present.

To measure alcohol exposure in a nonconfrontational manner for the children, we asked parents how often they consumed alcohol in situations where there is a high likelihood that children are present. Future research may implement more ecologically valid measures (e.g., ecological sampling methods that more frequently assess parental drinking when their children present) to assess whether actual observations of parental alcohol use are associated with the development of AE over time. Moreover, since we approached exposure as drinking within family situations, one might argue that other forms of exposure (e.g., intoxicated parents) play an important role in learning about alcohol and its consequences. Future research should therefore further unravel the impact of different forms of exposure on specific kinds of AE.

In addition, since we found the association between parental alcohol use and AE development to be true for fathers' alcohol use (exposure) only, future studies should aim to unravel whether, and for what reason, fathers' drinking plays a bigger role in shaping offspring's AE. For young children, future research should consider using more age-adequate assessments of AE, as children's cognitive and language skills are still developing (Berk, 2013; Keenan and Evans, 2009). For instance, the recently developed Alcohol Expectancy Task (Kuntsche and Kuntsche, 2017) accessible on a tablet could foster more age-friendly measurements. Furthermore, longitudinal studies (e.g., Jester, 2014) are advised to evaluate the transition from AE to alcohol use initiation and subsequently to more risky drinking patterns in adolescence and beyond using the same individuals.

CONCLUSION

Two similar studies conducted with different age groups (i.e., 6 to 8 and 12 to 15 years) provided evidence that exposure to fathers' alcohol use is responsible for favorable cognitions about the effects of alcohol in offspring's eyes rather than alcohol use in general. Children's observations of parental alcohol use are crucial, as they can help us better understand the intergenerational transmission of alcohol use habits via cognitions because expectancies about the effects of alcohol are important predictors of alcohol initiation and subsequent risky drinking patterns in adolescence and beyond (Campbell and Oei, 2010; Jester et al., 2014; Smit et al., 2018b). Based on the current study's findings, prevention efforts may benefit from trying to decrease the offspring's exposure to parental drinking, as this behavior

might be easier to change compared to parental drinking in general.

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CONFLICT OF INTEREST

The authors have no conflicts of interest.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Correlations among parental alcohol use, exposure, and children's AE.

Table S2. Correlations among parental alcohol use, exposure, and adolescent's AE