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Comparing media and family predictors of alcohol use: a cohort study of US adolescents

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ABSTRACT

Objective: To compare media/marketing exposures and family factors in predicting adolescent alcohol use.

Design: Cohort study.

Setting: Confidential telephone survey of adolescents in their homes.

Participants: Representative sample of 6522 US adolescents, aged 10–14 years at baseline and surveyed four times over 2 years.

Primary outcome measure: Time to alcohol onset and progression to binge drinking were assessed with two survival models. Predictors were movie alcohol exposure (MAE), ownership of alcohol-branded merchandise and characteristics of the family (parental alcohol use, home availability of alcohol and parenting). Covariates included sociodemographics, peer drinking and personality factors.

Results: Over the study period, the prevalence of adolescent ever use and binge drinking increased from 11% to 25% and from 4% to 13%, respectively. At baseline, the median estimated MAE from a population of 532 movies was 4.5 h and 11% owned alcohol-branded merchandise at time 2. Parental alcohol use (greater than or equal to weekly) was reported by 23% and 29% of adolescents could obtain alcohol from home. Peer drinking, MAE, alcohol-branded merchandise, age and rebelliousness were associated with both alcohol onset and progression to binge drinking. The adjusted hazard ratios for alcohol onset and binge drinking transition for high versus low MAE exposure were 2.13 (95% CI 1.76 to 2.57) and 1.63 (1.20 to 2.21), respectively, and MAE accounted for 28% and 20% of these transitions, respectively. Characteristics of the family were associated with alcohol onset but not with progression.

Conclusion: The results suggest that family focused interventions would have a larger impact on alcohol onset while limiting media and marketing exposure could help prevent both onset and progression.

ARTICLE SUMMARY

Article focus

- Predictors of drinking during adolescence.
- Particular focus on predicting onset versus binge drinking and media/marketing exposures versus family risk factors.

Key messages

- Somewhat different risk factors exist for alcohol onset versus binge drinking.
- Movie alcohol, alcohol marketing, friend drinking and sensation seeking predicted both outcomes.
- Parent drinking, availability of alcohol at home and parenting predicted alcohol onset, not binge drinking.

Strengths and limitations

- Strengths include longitudinal design, large sample size and analysis that accounted for attrition.
- Limitations include inability to generalise beyond US adolescents or beyond this age bracket.

INTRODUCTION

Underage drinking is prevalent[1 2] and represents an important risk factor for risky sexual behaviour,[3 4] injury and mortality during adolescence[5 6] and subsequent alcohol abuse and dependence.[7 8] Alcohol use or brands are depicted in 80%–95% of movies, and drinking is mostly portrayed positively.[9–13] Previous research on youth in regional samples of US[12] and German adolescents[14 15] has demonstrated an association between viewing alcohol use in movies and early onset of drinking. In the German study, 80% of exposure came from internationally distributed Hollywood movies, so decisions made by US production companies on how alcohol is depicted may impact drinking world wide. Alcohol marketing activities, such as branded merchandise distribution,[16] have also been linked to teen drinking.[17]

This study tests the hypothesis that exposure to movie alcohol use and alcohol-branded merchandise predicts teen alcohol onset and progression to binge drinking. Prior research suggests that predictors of substance use onset may be different from predictors of its progression,[18 19] but this has not been tested for media/marketing exposures. Previously, using data from this longitudinal sample of

US adolescents, we found that the association between movie alcohol exposure (MAE) and drinking frequency was mediated through drinking cognitions[20] and that an association between MAE and alcohol problems was mediated, in part, through quantity of alcohol consumed.[21] We have also noted that Black adolescents and those[22] high in sensation seeking were less responsive to media influence. The present study addresses several issues not addressed in prior research. We test the hypothesis about prediction of onset and progression, and for each transition, we compare the effect of movie/marketing exposure with the effects of family and peer predictors that have been linked with alcohol use by others.[23 24] Moreover, this research addresses the public health importance of mass media by determining the proportions of the drinking transitions that may be attributed to MAE.

METHODS

Participants and procedure

Between June and October, 2003, we conducted a random digit dial telephone survey of 6522 US adolescents aged 10−14 years. The telephone surveys were conducted by trained interviewers using a computer-assisted telephone interview system from Westat (Rockville, Maryland, USA), a national research organisation with survey sites across the US. Interviewers were trained to administer the survey in English or Spanish. We obtained parental consent and adolescent assent prior to interviewing each respondent. To protect confidentiality, adolescents indicated their answers to sensitive questions by pressing numbers on the telephone, rather than speaking aloud. All aspects of the survey were approved by the institutional review boards at Dartmouth Medical School and Westat.

Selection of the sample (online appendix figure 1) involved three stages, through which we identified a list-assisted randomly generated sample of 377 850 residential phone numbers (stage 1), identified households with age-eligible children (stage 2) and enrolled age-eligible adolescents into the study (stage 3). In stage 1, we used an automated system in combination with interviewer calls to purge non-working and business numbers from the list, which reduced the sample to 129 002 known residential telephone numbers. In stage 2, interviewers called each number and successfully completed screener interviews with 69 516 households. Through the screening interviews, we identified 9849 eligible households with adolescents between 10 and 14 years of age. For households with more than one age-eligible adolescent, we randomly selected one for enrolment. In stage 3, we obtained permission from 77% (N=7492) of the parents to interview their child, and 87% (N=6522) of eligible adolescents agreed to participate and completed the survey.

The American Association for Public Opinion Research identifies several ways to calculate survey response.[25] The completion rate (the number of completed interviews (N=6522) divided by the number of eligible households (N=9849)) for this survey was 66%. The response rate is more conservative and includes estimates of eligible households lost during stages 1 and 2 (see online appendix figure 1).[25] Using methods of Brick *et al*,[26] we estimated that 15 057 of the 38 696 non-answered phone numbers in stage 1 were residential. In addition, 59 667 households did not complete the screening interview in stage 2. Assuming that the same proportion of these 74 724 (15 057 + 59 667) unscreened households had age-eligible adolescents as in the screened sample (0.14), we estimate that 10 587 households in stages 1 and 2 could have been eligible for the study. When these households are included in the denominator, our most conservative estimate of the response rate is 32% (6522 interviewed adolescents/an estimated 20 436 (9849 + 10 587) eligible households). Online appendix figure 2 illustrates the geographic coverage of the sampling procedure, which captured adolescents from all 50 US states and which reflects the geographic distribution of the US population. As an additional test of sample representativeness, we assessed the distributions of age, sex, household income and census region in the unweighted sample and found that they were almost identical to percentages approximated in the 2000 US Census (online appendix table 1). Compared with the 2000 US Census, the unweighted sample had a higher percentage of Hispanics and a slightly lower percentage of African−Americans.

The study was originally funded to study smoking and was therefore powered to detect an association between movie smoking and smoking onset. For that outcome, we determined that we needed to successfully follow-up 2200 baseline never-smokers in order to achieve a power of 90% to detect an adjusted OR of 1.4 using a two-sided test with α=0.05.

There were few missing data for items on the baseline questionnaire; for example, at baseline, 6520 of 6522 participants answered the question about ever binge drinking. After the baseline questionnaire, the adolescents were followed up every 8 months for three more telephone surveys (n=5503, 5019 and 4575 for waves 2, 3 and 4, respectively). Attrition analyses indicated that adolescents lost to follow-up were more likely to be non-Caucasian; were from families with lower parental education and income, rented versus owned their residence; had poorer school performance and had higher levels of sensation seeking. Baseline drinking status (ever vs never tried alcohol) did not predict attrition, but to account for attrition bias related to other variables, estimation was carried out after multiple imputation using the standard missing at random assumption (ie, missing data are assumed missing at random conditional on observed predictors included in the model).[27] The imputation model included all the predictors in the alcohol models plus a number of auxiliary variables that were not of direct theoretical interest but were nonetheless predictive of missingness so as to improve

the quality of the imputations and make the missing at random assumption more plausible.[28]

Movie alcohol exposure measurement

Exposure to movie alcohol use was assessed using the previously validated Beach method.[29] The top 100 US box office hits for each of the 5 years preceding the baseline survey (1998–2002, N=500) and 32 movies earning >$15 million in gross US box office revenues during the first quarter of 2003 were selected. Each adolescent survey included 50 movies randomly selected from the larger sample of 532, stratified by the Motion Picture Association of America rating, so that the distribution of movies in each list reflected the distribution in the full sample of movies (19% G/PG, 41% PG-13 and 40% R). Respondents were asked (Yes/No) whether they had ever seen each movie title on their individual list. We have previously shown that adolescents correctly remember movies they have seen with high reliability.[29]

The movies were content analysed by trained coders who timed the number of seconds of on-screen alcohol use (mean κ for coding reliability on a 10% subsample of movies was 0.86). Alcohol use was defined as a character's actual or implied consumption or the purchase of alcohol. The measure of MAE was based on the summed total of timed alcohol use in the films that each adolescent had seen.

Ownership of alcohol-branded merchandise

Ownership of branded merchandise is a key item in the measurement of receptivity to marketing as developed by Pierce and colleagues for tobacco marketing.[30] This risk factor was not measured at baseline but was included at T2, T3 and T4 surveys. Thus, the hazard estimates are determined over two and not three periods as was the case for the other variables. It was assessed through the question, 'Do you own something with the name of a beer, wine, or liquor brand on it, like a t-shirt or a hat?'

Other predictor variables

The analyses also included age, race/ethnicity (three binary variables for Black, Hispanic and other ethnicity, coded with Whites as the reference group), gender, household income and parental education, media-viewing habits—hours watching television on a school day and how often the participant viewed movies together with his/her parents—and receptivity to alcohol marketing (based on whether or not the adolescent owned alcohol-branded merchandise at waves 2–4).[31] Family predictors included perceived inhome availability of alcohol, subject-reported parental alcohol use (assessed at the 16 M survey and assumed to be invariant) and perceptions of authoritative parenting (α=0.80).[32] Other covariates included school performance, extracurricular participation, number of friends who used alcohol, weekly spending money, sensation seeking (4-wave Cronbach's α range=0.57–0.62)[33] and rebelliousness (0.71–0.76).[34] All survey items are listed in table S1.

Adolescent alcohol use

Alcohol use onset was assessed at each wave by the question: "Have you ever drunk alcohol that your parents did not know about? By alcohol we mean beer, wine, wine coolers or liquor, like whisky, vodka, or gin" (Yes/No), defined in this way to exclude parentally sanctioned sips of alcohol. Binge drinking was assessed by asking "Have you ever had 5 or more drinks of alcohol in a row, that is, within a couple of hours?" (ever-binge drinker) and "Did you have 5 or more drinks of alcohol in a row during the past month?" (30-day binge drinker). To ensure confidentiality in these home-based surveys, subjects indicated responses by pressing numbers on the telephone.

Statistical analysis

From the three drinking outcomes, we estimated the relation between baseline assessment of MAE and covariates with time to event for two survival models: an alcohol onset model for the transition from never-drinker → ever-drinker or ever-binge drinker and a progression-to-binge-drinking model for ever-drinker → ever-binge drinker and ever-binge drinker → 30-day binge drinker. We tested for within-subject correlation between the two transition processes and found none, that is, time to onset was not associated with time to progression, net of covariates. The MICE procedure in the R statistical software package[35] was used to stochastically impute missing data.[36] For descriptive statistics, we averaged across the 20 imputations to obtain a best estimate for each missing data point.

Discrete time hazard survival models[37] were fit to each of the 20 imputed complete data sets using a complementary log-log regression routine in R and following standard procedures for pooling the estimates and obtaining SEs.[36] All predictors were entered in the model simultaneously. The measure of the association is the adjusted hazard ratio (AHR), which assesses time to onset of the outcome and may be interpreted like a relative risk. To aid in comparison of the AHRs, continuous covariates were scaled such that 0 corresponded to the 5th percentile and 1 to the 95th percentile for their distributions, with extreme values in either direction recoded to 0 or 1 to minimise outlier influence. Ordinal variables were scaled so that the lowest value was equal to 0 and the highest value was equal to 1. Continuous and ordinal variables that were protective (eg, authoritative parenting, family income) were reversed (to unskilled parenting, low family income involvement), so that all HRs were ≥1.0. This rescaling procedure allowed for comparison of the effect sizes between continuous, dichotomous and ordered categorical variables. For all models, results for main effects were judged significant if $p<0.05$.

Attributable fraction calculations (adjusted for covariate effects) were carried out after model fitting by

Table 1 Description of the sample at baseline

Variable	N (%)
Sociodemographics	
Age	
10 years	1186 (18)
11 years	1303 (20)
12 years	1338 (21)
13 years	1418 (22)
14 years	1277 (20)
Race/ethnicity	
Caucasian	4037 (62)
African–American	704 (11)
Hispanic	1222 (19)
Other	559 (9)
Sex	
Male	3350 (51)
Female	3172 (49)
Family income (×1000)	
<$20	475 (7)
$20–$29	722 (11)
$30–$49	804 (12)
$50–$74	1360 (21)
$75–$99	1296 (20)
≥$100	1865 (29)
Parent education	
≤9th grade	402 (6)
9th–11th grade	478 (7)
12th grade	260 (4)
HS diploma	1481 (23)
Voc/Tech	234 (4)
Some college	1127 (17)
Associate degree	550 (8)
Bachelor's degree	1197 (18)
Postgraduate education	793 (12)
Family and friends	
Parent alcohol use*	
Never	1270 (19)
Once per year	1913 (29)
Once per month	1872 (29)
Once per week	1103 (17)
Daily use	364 (6)
Home availability of alcohol (could you get alcohol from home without your parents knowing?)	
Definitely no	4641 (71)
Probably no	936 (14)
Probably yes	688 (11)
Definitely yes	257 (4)
Peer alcohol use	
None	5055 (78)
Some	1215 (19)
Most	252 (4)
Media and marketing	
Television viewing	
None	360 (6)
<1 h/day	1261 (19)
1–2 h/day	3041 (47)
3–4 h/day	1323 (20)
>4 h/day	537 (8)
Movie viewing with parents (How often do you watch movies with parents?)	
Most of the time	152 (2)
Sometimes	1705 (26)
Once in a while	2448 (38)
Never	2217 (34)
Receptive to alcohol marketing (owns alcohol-branded merchandise)†	
No	4895 (89)
Yes	597 (11)
Adolescent characteristics	
School performance	
Below average	181 (3)
Average	1625 (25)
Above average	2734 (42)
Excellent	1982 (30)
Weekly spending money	
None	937 (14)
$1–$5	764 (12)
$6–$10	1551 (24)
$11–$15	1652 (25)
$16–$20	920 (14)
$21–$50	568 (9)
$50+	130 (2)

Continuous variables	Median	IQR
Authoritative parenting	2.4	2.1–2.7
Movie alcohol exposure (h)	4.5	2.2–8.0
Sensation seeking	1	0.5–1.5
Rebelliousness	0.5	0.3–0.8
Extracurricular involvement	1.8	1.5–2.2

*Assessed at 16 months, imputed for baseline numbers, time invariant.
†Assessed at 8 months, used as predictor from 8 months on.

obtaining the model-predicted number of events with the observed data and the model-predicted number of events when levels of MAE in our sample were altered along two scenarios. For the first scenario, the 'intervention' scenario, we lowered all scores for MAE by 25% to model the results of an intervention that successfully reduced MAE exposure. For the second scenario, the 'full effect' scenario, we lowered all scores for MAE to the 5th percentile level to indicate what might happen if alcohol was completely removed from all movies the adolescents had watched. For each of the 20 imputations, we obtained estimates and SEs for the attributable fractions using 100 bootstrap replications. The bootstrap estimates and SEs were then pooled across the 20 multiple imputation models.

RESULTS

Description of the cohort

Table 1 describes the predictor variables at baseline. Age and gender were equally represented. Race/ethnicity and other demographic variables were broadly reflective of the US population, with 11% Black and 19% Hispanic ethnicity. Some 18% of families were low income, with

Table 2 Alcohol use and binge drinking in the cohort

Survey	Drinking outcome/transition (%) Tried drinking (%)	Ever binge drink	30-day binge drink (%)
Prevalence			
Baseline	11	4	1
8 months	16	6	2
16 months	20	10	4
24 months	25	13	6

Incidence	Never → tried	Never → ever binge	Tried → ever binge	Ever binge → 30-day binge
B → 8 months	8	3	24	29
8 → 16 months	6	2	22	25
16 → 24 months	7	3	15	23

7% having incomes of $20 000 or less and 11% having income between $20 000 and $29 000/year. At baseline, the median estimated MAE from the pool of 532 movies was 4.5 h, and at T2, 11% of the respondents reported owning alcohol-branded merchandise. Friend alcohol use was reported by 23%, parental alcohol use (greater than or equal to weekly) by 23% and could obtain alcohol from home by 29% of respondents.

Alcohol use in the cohort

Over the course of the study, the prevalence of tried drinking increased from 11% to 25% (table 2). The incidence categories show data for transitions. Among never-drinkers for each 8-month observation period, 6%–8% transitioned to ever drinking and 2%–3% transitioned to binge drinking. The risk of a transition escalated for ever-drinkers, among whom 15%–24% transitioned to binge drinking and 23%–29% transitioned to 30-day binge drinking over each 8-month period.

Hazard model: time to onset of drinking

Crude and AHRs for time to drinking onset are reported in table 3 and compared in figure 1, where they are sorted by magnitude, with all variables scaled so the AHR >1. Four variables had AHRs >2.0: peer alcohol use, AHR=2.88 (95% CI 2.35 to 3.53), age (2.24 (1.81 to 2.77)), MAE (2.13 (1.76 to 2.57)) and sensation seeking

Table 3 Crude and adjusted hazard ratios for time to onset of alcohol use

Predictor variable	HR initiation Crude	Adjusted
Sociodemographics		
Oldest versus youngest	5.35 (4.49 to 6.37)	2.24 (1.81 to 2.77)
African–American versus Caucasian	1.05 (0.87 to 1.27)	0.99 (0.80 to 1.23)
Hispanic versus Caucasian	1.04 (0.91 to 1.19)	0.98 (0.84 to 1.14)
Other non-Caucasian versus Caucasian	0.87 (0.71 to 1.08)	0.84 (0.68 to 1.05)
Female	1.01 (0.91 to 1.12)	1.10 (0.99 to 1.23)
High parent education	0.84 (0.71 to 1.27)	1.03 (0.81 to 1.29)
Low family income	1.10 (0.93 to 1.30)	1.09 (0.87 to 1.38)
Family and friends		
Parent alcohol use	2.12 (1.78 to 2.52)	1.43 (1.17 to 1.75)
Alcohol available at home	3.47 (2.96 to 4.06)	1.45 (1.21 to 1.74)
Unskilled parenting	5.56 (4.55 to 6.67)	1.76 (1.41 to 2.20)
High peer alcohol use	8.69 (7.34 to 10.3)	2.88 (2.35 to 3.53)
Media and marketing		
Low TV viewing	0.67 (0.53 to 0.84)	1.11 (0.87 to 1.42)
High movie alcohol exposure	5.50 (4.62 to 6.55)	2.13 (1.76 to 2.57)
Views movies without parents	2.04 (1.67 to 2.50)	1.22 (0.99 to 1.50)
Receptive to alcohol marketing	2.63 (2.19 to 3.15)	1.44 (1.19 to 1.74)
Characteristics of adolescent		
High sensation seeking	5.97 (4.98 to 7.15)	2.08 (1.67 to 2.59)
High rebelliousness	4.08 (3.43 to 4.86)	1.55 (1.25 to 1.92)
Poor school performance	2.86 (2.33 to 3.45)	1.32 (1.05 to 1.65)
Low extracurricular involvement	1.6 (1.38 to 2.03)	1.11 (0.91 to 1.37)
High spending money	3.97 (2.98 to 5.29)	1.46 (1.11 to 1.92)

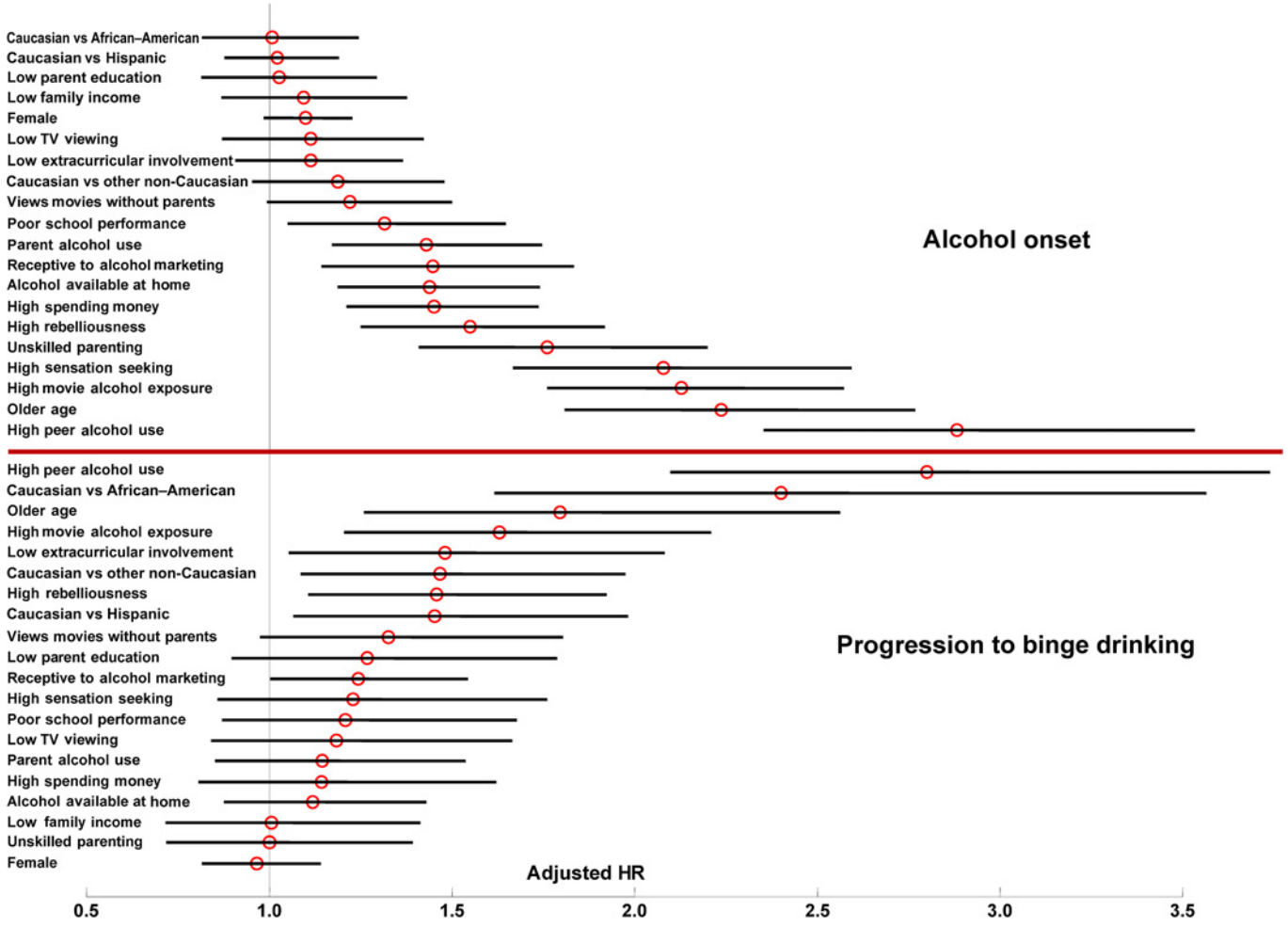

Figure 1 Adjusted hazard ratios (AHRs) for time to alcohol onset among alcohol never users (top panel) and for progression to binge drinking among alcohol experimenters (bottom panel). Each panel sorts the AHRs by size, allowing comparison of media, family, and other risk factors. Error bars represent 95% confidence intervals. Race/ethnicity dummy contrasts from Tables 3 and 4 were reverse-scaled to represent excess risk of being Caucasian compared to other race/ethnicity groups so that all AHRs are >1 to facilitate comparison with other continuous and ordinal risk factors.

(2.08 (1.67 to 2.59)). Other variables with statistically significant AHRs included parenting, rebelliousness, weekly spending money, alcohol availability at home, receptivity to alcohol marketing, parent alcohol use and school performance.

The attributable fraction modelling estimated the proportion of drinking onset transitions prevented if MAE were reduced. An intervention that reduced MAE by 25% across the population would decrease drinking onset by 8% (adjusted attributable fraction (AAF)=−0.08 (−0.09, −0.07)). Eliminating MAE entirely would decrease drinking onset by 28% (AAF=−0.28 (−0.30, −0.25)).

Hazard model: time to progression to binge drinking

Results for the multivariate hazard model for transitions to binge drinking among ever-drinkers are illustrated in figure 1, with numeric values for HRs shown in table 4. High peer alcohol use had an AHR >2 (2.80 (2.10, 3.74)) as did White race (vs Black) (2.40 (1.62, 3.56)). Variables significantly associated with progression included race and ethnicity (higher AHR for Caucasians vs Hispanic or other non-Caucasians), age, MAE (1.63 (1.20, 2.21)), extracurricular involvement, rebelliousness and receptivity to alcohol marketing. An intervention that reduced MAE by 25% across the population would reduce the proportion of adolescent drinkers transitioning to binge drinking by 6% (AAF=−0.06 (95% CI=−0.08 to −0.03)) and eliminating the exposure entirely would reduce it by 20% (AAF=−0.20 (95% CI=−0.28 to −0.13)).

Contrasts: time to onset versus time to progression to binge drinking

Several variables predicted *both* alcohol onset and progression to binge drinking—peer alcohol use, age, MAE, receptivity to alcohol marketing, and rebelliousness. In contrast, some variables played a role in alcohol onset but not binge drinking or vice versa. Notably, none of the family predictors of alcohol onset were significant predictors of progression to binge drinking. For unskilled parenting and sensation seeking, the HR was

Table 4 Crude and adjusted hazard ratios for time to onset of binge drinking

	HR progression	
Predictor variable	**Crude**	**Adjusted**
Sociodemographics		
Oldest versus youngest	3.48 (2.58 to 4.71)	1.80 (1.26 to 2.56)
African–American versus Caucasian	0.37 (0.26 to 0.55)	0.42 (0.28 to 0.62)
Hispanic versus Caucasian	0.37 (0.59 to 0.91)	0.69 (0.55 to 0.88)
Other non-Caucasian versus Caucasian	0.81 (0.60 to 1.10)	0.68 (0.51 to 0.92)
Female	0.95 (0.81 to 1.10)	0.96 (0.82 to 1.14)
High parent education	1.58 (1.21 to 2.05)	1.27 (0.90 to 1.79)
Low family income	0.59 (0.46 to 0.75)	1.01 (0.72 to 1.41)
Family and friends		
Parent alcohol use	1.70 (1.29 to 2.23)	1.14 (0.85 to 1.54)
Alcohol available at home	1.90 (1.53 to 2.37)	1.12 (0.88 to 1.43)
Unskilled parenting	2.08 (1.54 to 2.78)	1.00 (0.72 to 1.39)
High peer alcohol use	4.68 (3.68 to 5.96)	2.80 (2.10 to 3.74)
Media and marketing		
Low TV viewing	1.32 (0.95 to 1.82)	1.18 (0.84 to 1.67)
High movie alcohol exposure	2.47 (1.86 to 3.27)	1.63 (1.20 to 2.21)
Views movies without parents	1.89 (1.41 to 2.50)	1.33 (0.97 to 1.80)
Receptive to alcohol marketing	1.74 (1.43 to 2.12)	1.24 (1.00 to 1.54)
Characteristics of adolescent		
High sensation seeking	2.56 (1.92 to 3.43)	1.23 (0.86 to 1.76)
High rebelliousness	1.90 (1.51 to 2.40)	1.45 (1.07 to 1.98)
Poor school performance	1.67 (1.25 to 2.22)	1.21 (0.87 to 1.68)
Low extracurricular involvement	2.04 (1.48 to 2.80)	1.48 (1.05 to 2.08)
High spending money	1.91 (1.35 to 2.69)	1.14 (0.80 to 1.62)

significantly higher for onset than for binge drinking by factors of 1.76 and 1.69, respectively. Race/ethnicity did not predict onset, however, White adolescents were more likely to transition to binge drinking, with the effects for African–Americans and Hispanics being significantly stronger for binge drinking than onset by factors of 2.38 and 1.42, respectively.

DISCUSSION

This study compared movie alcohol and alcohol marketing exposures with family factors and other variables as predictors of alcohol use onset separately from transition to binge drinking. We found that movie and marketing exposures predicted both transitions. After control for multiple covariates, MAE accounted for 28% of the alcohol onset and 20% of the binge drinking transitions observed in this cohort, making it a risk factor with important public health implications and arguing for policy approaches to prevention of MAE. These results are consistent with a German study that also found an association of MAE with alcohol onset and binge drinking,[14] which adds cross-cultural validation to the findings. In contrast, family characteristics—availability of alcohol at home, parental drinking, and parenting practices—predicted alcohol onset but not the transition to binge drinking.

We think the results could reflect two types of processes. For onset, drinking is a proscribed behaviour for adolescents and initiating requires that a youth go against cultural and legal norms. Adolescents who are older and who seek new sensations and experiences are less influenced by these norms hence are more likely to try alcohol. However, parents can communicate norms about alcohol use, and the likelihood of onset is reduced when parents have a warm relationship with children, monitor their behaviour and make alcohol unavailable in the home. Once a youth has tried alcohol, progression to problem drinking probably depends on a substance-using peer environment, a lower level of attachment to conventional social institutions, greater involvement in drinking culture (determined in part by marketing and entertainment media) and the belief that alcohol has positive effects in several areas.[38] Peers may facilitate alcohol use initially through encouraging trial and subsequently through providing an alternative norm structure that reinforces deviant behaviour; adolescents who are more rebellious and less involved in conventional activities should be most susceptible to this kind of influence.

Movie alcohol exposure and alcohol marketing may contribute to both of these processes though for somewhat different reasons. Movie exposure may facilitate onset through providing examples of persons drinking and promoting the belief that alcohol use is common and acceptable. The effect of movie exposure on progression, we suggest, derives from the fact that alcohol use in movies is typically modelled in positive situations, without negative effects, and often shown with

alcohol brands,[9] which consolidates both the adolescent's identity as a drinker and brand allegiance. Acquisition of alcohol-branded merchandise, an article of clothing with an alcohol brand on it, furthers this process. Moreover, wearing alcohol-branded merchandise in public engages the adolescent in the actual marketing campaign, as the adolescent is seen by others as an endorsement of use of the brand. We note that in contrast to the present findings for alcohol, one recent study suggests that movie influence on smoking onset is larger than that on progression,[39] perhaps because for smoking, nicotine addiction drives progression to a greater extent than other types of influences.[40]

Limitations

Consistent with other contemporary random digit dial household surveys, the response rate for this study was moderate and should be considered for the generalisability of the results, though the sample appeared to be representative with respect to most sociodemographic categories. Also, there was attrition from the panel, and although the multiple imputation procedure minimised attrition bias, attrition reduces power, and this should be recognised as a limit to the ability to generalise to minority groups more likely to drop out of the study. As with any observational study, the possibility of an unmeasured confounder needs to be considered. The covariate for television viewing may not have adequately captured exposure to alcohol depictions in television programming[41 42] and the one for alcohol marketing did not capture television or internet alcohol advertising exposures.[43] Finally, further research should be conducted to determine how media exposures are related to alcohol use in late adolescence and emerging adulthood.

Implications for parents, families and clinicians

The findings raise the question about what parents could do to limit MAE and alcohol marketing exposures. One approach to limiting MAE could be through parental restriction on certain types of media, for example, R-rated movies, which contain high levels of drinking (90%) and brand placement (61%).[9] Indeed, parental movie restrictions have been associated with lower risk for alcohol and tobacco use,[44–46] and parental media management merits greater emphasis by clinicians and intervention researchers. Additionally, this and other research strongly indicates that parents should not allow alcohol-branded merchandise in their homes[16 31 47–51]; this type of alcohol marketing seems particularly problematic because adolescents become promotional vehicles as they wear their merchandise in schools and other public places, another point clinicians can make when discussing substance use prevention in office visits. The study also suggests that parents may limit onset of alcohol use by being responsive and setting limits, by promoting extracurricular involvement, by keeping home alcohol in a secure location or by not drinking frequently themselves.

Public health considerations

Product placement in movies is forbidden for cigarettes in the USA but is legal and commonplace for the alcohol industry, with half of Hollywood films containing at least one alcohol brand appearance, regardless of film rating.[9] To the extent that alcohol product placement serves to increase prevalence of movie drinking scenes, limits on movie alcohol product placement could also reduce MAE. Moreover, movie smoking has declined since it became a public health issue and movie studios began monitoring its prevalence[52]; MAE may deserve similar emphasis.

Finally, the global health implications of risk behaviour depiction in Hollywood movies should be mentioned. For some 20 years now, more than half of the revenues for Hollywood movies come from overseas.[53] The main importers of Hollywood products are European countries, but Japan and Canada, Australia, Brazil and South Korea are also important markets. The fact that adolescents who view these movies may also be influenced though visual images from movie exports is underlined by the German studies mentioned above.[15 54] Like influenza, images in Hollywood movies begin in one region of the world then spread globally, where they may affect drinking behaviours among adolescents everywhere they are distributed.

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Competing interests None.

Contributors All authors contributed to the conception and design of the study and were part of the development of the surveys deployed in the study. MS conducted the data analysis. All authors had a hand in the interpretation of the data, the critical revision of the manuscript and all approved of the final version.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement Statistical code and a partial analysis data set (with variables pertaining to this manuscript) are available from the corresponding author. Informed consent was not obtained for data sharing, but the data that would be shared have no personal identifiers. Requests for use of the aforementioned data will not be granted without approval by the Dartmouth and the recipient's human subjects committees.

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STROBE Statement—Checklist of items that should be included in reports of ***cohort studies***

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract: Title: "**A Cohort Study of U.S. Adolescents**
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found. √**We think the abstract is balanced**
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported √**Paras 1 and 2 of the intro do that we think**
Objectives	3	State specific objectives, including any prespecified hypotheses √**Para 2 intro: "**This study tests the hypothesis that exposure to movie alcohol use and alcohol branded merchandise predicts teen alcohol onset and progression to binge drinking"
Methods		
Study design	4	Present key elements of study design early in the paper √ **See Overview in Methods section**
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection. √**We have included a**
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up √**pp 6-7 of the ms**
		(*b*) For matched studies, give matching criteria and number of exposed and unexposed N/A
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable √**pp 8-9 of the ms**
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group √**pp 8-9**
Bias	9	Describe any efforts to address potential sources of bias √**p9,** To ensure confidentiality in these home-based surveys, subjects indicated responses by pressing numbers on the telephone.
Study size	10	Explain how the study size was arrived at √**p9,** The study was powered to detect an association between movie smoking and smoking onset. For that outcome, we determined that we needed to have successfully follow up 2,200 baseline never smokers in order to achieve a power of 90 percent to detect an adjusted odds ratio of 1.4 using a two-sided test with alpha=0.05.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why √**pp 9-10, statistical analysis section**
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding √**pp 9-10**
		(*b*) Describe any methods used to examine subgroups and interactions √**main effects examined only**
		(*c*) Explain how missing data were addressed pp 9-10, imputation described
		(*d*) If applicable, explain how loss to follow-up was addressed pp9-10, imputation

		(*e*) Describe any sensitivity analyses N/A
Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed √**see page 7, After the baseline questionnaire, the adolescents were followed up every 8 months for three more telephone surveys (n = 5503, 5019, and 4575 for waves 2, 3, and 4 respectively). Attrition analyses indicated that adolescents lost to follow up were more likely to be non white; were from families with lower parental education and income, rented vs. owned their residence; had poorer school performance; and higher levels of sensation seeking.**
		(b) Give reasons for non-participation at each stage Unable to contact by phone
		(c) Consider use of a flow diagram **have included a flow diagram as an appendix, explaining sample selection**
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders √**See table 1**
		(b) Indicate number of participants with missing data for each variable of interest Data for missing was imputed
		(c) Summarise follow-up time (eg, average and total amount) √**This is evident from the loss to follow up by wave numbers**
Outcome data	15*	Report numbers of outcome events or summary measures over time √**See table 2**
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included See tables 4 and 5
		(*b*) Report category boundaries when continuous variables were categorized √ Statistical methods: **To aid in comparison of the adjusted hazard ratios, continuous covariates were scaled such that zero corresponded to the 5th percentile and 1 to the 95th percentile for their distributions, with extreme values in either direction recoded to 0 or 1 to minimize outlier influence. Ordinal variables were scaled so that the lowest value was equal to 0 and the highest value was equal to 1. Some variables that were protective (e.g., authoritative parenting, extracurricular involvement) were reversed (unskilled parenting, low extracurricular involvement), so that all hazard ratios were ≥ 1.0. This rescaling procedure allowed for comparison of the effect sizes between continuous, dichotomous and ordered categorical variables.**
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period √**See attributable risk estimates**
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses √**No subgroups analysis done**
Discussion		
Key results	18	Summarise key results with reference to study objectives √**We feel that the discussion does this.**
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or

		imprecision. Discuss both direction and magnitude of any potential bias √**We feel that the discussion does this**
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence √**We think the influenza comparison is valid,understand that you may think it an overstatement**
Generalisability	21	Discuss the generalisability (external validity) of the study results √ SEE LIMITATIONS: Consistent with other contemporary random digit dial household surveys, the response rate for this study was moderate and should be considered for the generalizability of the results, though the sample was representative with respect to most sociodemographic categories. Also there was attrition from the panel, and although attrition effects were considered in the imputation, this should be recognized as a limit to the ability to generalize to minority groups more likely to drop out of the study.
Other information		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based √**Done**

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at http://www.strobe-statement.org.

To the Editor:

In moving this manuscript from BMJ to BMJ Open, we thought it would be helpful if we responded to the comments of the two reviewers, in order to facilitate the review. Reviewer 2 asked for additional statistical input, which you may want to consider as well. We would be happy to publish a technical appendix to our report that details the analysis and also gives our output.

Reviewer: 1
Comments:
1) An interesting and well written article, which I fear may be - due to its length, findings, and epidemiological detail may be just outside the remit of a BMJ publication. Such an article is likely to be snapped up by a specialist journal but quite a bit of the methodological content may be outside the interest of the majority of BMJ readership. It adds to the literature on drivers of alcohol use but is the areas of epidemiological investigation are more relevant for health policy readers . Some small concerns regarding methodology, which the authors may wish to take into account on submission to a specialist journal - in the limitations they maintain that the participants were representative while they note in the methodology that attrition between surveys was higher in the families with lower parental education and income.

RESPONSE: We reread the limitations statement and it seems to cover the concerns raised by the reviewer; the term representative referred to the similarity between the demographics of the survey participants compared with the US census. It has been amended to read, Consistent with other contemporary random digit dial household surveys, the response rate for this study was moderate and should be considered for the generalizability of the results, though the sample was representative with respect to most sociodemographic categories. Also there was attrition from the panel, and although the multiple imputation procedure minimized attrition bias, nevertheless attrition reduces power and this should be recognized as a limit to the ability to generalize to minority groups more likely to drop out of the study."

2) More details are required on the way children are questioned by telephone, and their non-responses to specific questions which was rather scantily addressed at the expense of lengthy sections devoted to indicators and analytic techniques.

RESPONSE: There were few missing items on the baseline questionnaire. We cover this with the following sentence, "There were few missing data for items on the baseline questionnaire; for example, at baseline 6520 out of 6522 participants answered the question about ever binge drinking."

3) It was unclear whether exposures to alcohol in movies and branding was 'ever' or since the last survey interview, and perhaps needs to be accounted for.

RESPONSE: We are sorry to have been unclear about such a key issue. We assessed the relation between exposure and covariates at baseline and time to onset. The opening sentence of the Statistical Methods section now reads: "From the three drinking outcomes, we estimated the relation between baseline assessment of movie alcohol exposure and covariates with time

to event for two survival models: an alcohol onset model for the transition from never-drinker to ever-drinker or ever-binge drinker, and a progression-to-binge-drinking model for ever-drinker to ever-binge drinker and ever-binge drinker to 30-day binge drinker."

4) I would have liked some discussion on participants responses by house phones when, observing my own adolescents, it seems adolescents converse more freely on their own cell phones.

RESPONSE: These were surveys that required parental consent. We did not contact adolescents on their mobile phones unless directed to do so by the parent. In all cases, we made sure that the parent was not within hearing distance once the adolescent was on the line. Adolescents tend to under-report substance use in household surveys but tend to over-report in the school setting. I know of no study of how adolescents respond on mobile vs. landlines, but as mentioned above, we were precluded from contacting the adolescents directly by our human subjects committee.

5) Lastly, it was unclear why the study was powered to detect an association between movie smoking and smoking onset.

RESPONSE: The study was funded to examine smoking. A second grant was obtained to enrich the survey with alcohol questions, but the sample size calculation was done for the original study. We have now made that clear.

Reviewer: 2

Originality: This study investigates factors that may influence the onset of alcohol use in young people and occurrence of binge drinking using a cohort of US adolescents aged 10-14 years. Predictor variables included movie alcohol exposure, ownership of alcohol branded materials and family factors. Overall the paper is well written though it lacks detail with regards to the statistical analysis.

This is not the first study to look at these influences on alcohol consumption in adolescence although it does uniquely bring them together in the same study: The authors identify some previous studies and in addition Gordon et al 2010 used a cohort study to assess the impact of different types of alcohol marketing on drinking initiation and consumption frequency1; Ferguson and Meehan 2011 used a cohort design to assess the impact of peers on alcohol use, adjusting for media influences2 and Fisher et al, showed that possession of alcohol-branded promotional items is associated with increased likelihood of alcohol initiation. In addition several studies have attempted to identify other influences on alcohol uptake and use in adolescence3-5. However the literature on this topic is not extensive and the current study does distinguish factors that affect alcohol use onset and more hazardous patterns of drinking which adds to the research literature.

RESPONSE: We thank the reviewer for pointing out references we missed in our review of the literature. We cited the Gordon article as an example of a study that links alcohol marketing with drinking. We parenthetically note that the Gordon article found an odds ratio of 1.6 for the association between mums drinking and having tried one or more alcoholic drinks, an odds ratio that is similar to ours (1.4) for the association between parent drinking and drinking onset. Since our study was adequately powered to detect that level of association on drinking onset, the odds ratio of 1.4 was statistically significant, whereas the odds ratio of 1.6 in the Gordon study was not. See more on power in our response to queries about power below. We did not mention Ferguson as that study was cross sectional, had what we consider to be inadequate measures of media exposure and no measures of family or peer alcohol use. We did not mention Melotti for similar reasons. The studies by Noal and Duncan are mentioned as prospective studies that link family alcohol with youth alcohol use. Duncan is especially interesting because in that one parent alcohol use was linked with intercept but not slope in a growth model, a finding similar to ours, in which parent alcohol use was linked with initiation but not progression to binge drinking. We point out that similarity in the opening paragraph of the discussion.

Methods:
1) Research question: The research question is clearly defined i.e. to estimate the association between a range of media and family factors and alcohol use initiation and progression onto binge drinking in adolescents and a cohort study design is appropriate for this.

RESPONSE: We agree.

2) Participants: the authors describe well the process of recruitment. No exclusion criteria appear to have been applied, except for age and consent being obtained which are both appropriate. However there may be some selection biases associated with the sampling method i.e. participants needed to have a landline telephone, which may have selected out poorer households.

RESPONSE: The selection process may have selected out some households, but the appendix table suggests that poorer households was not one of the problems. In fact, there were slightly more households in the poorest household income category compared with the US census figures, and we were pleasantly surprised by this fact. Most households have telephone service in the United States. At this time, many poorer households are served by mobile phones, but at the time of this survey, 2003, this was not the case.

3) Outcome measures: The main outcome measures of time to onset of alcohol use and binge drinking are clear but the STROBE questionnaire did not appear to have been submitted, although correspondence said it had.

RESPONSE: We have included our STROBE statement in the revision application.

Ascertaining alcohol use is notoriously difficult because of recall and social desirability biases (people don't necessarily want to admit to their use) so non face-to-face interviews using keypad pressing to answer sensitive questions is a reasonable method to use, although still subject to some of these biases.

RESPONSE: We agree with the reviewer that ascertaining alcohol use among adults is notoriously difficult, social desirability and denial being big issues with that demographic. Among adolescents there is less social desirability bias to under-report. In fact, alcohol use can improve social standing among adolescents, and that may be why, in the school setting, adolescents tend to over-report their alcohol use.

The authors sought parental consent prior to adolescent consent and in doing so, enrolled 6522 individuals out of a possible 9849. The authors estimated their response rate to be 32% (including non-contactable households in the denominator), which is quite low but to be reasonably representative of the US population with some differences in ethnicity. The comparable US statistics show that the sample is slightly over representative of the higher household incomes and this is important for the study's generalisability given the telephone-based method of data collection and the potential for not recruiting people who move home frequently or do not possess a telephone landline. Other studies have shown conflicting results regarding the importance of socioeconomic status and binge drinking in particular, so this is a limitation of the study.

RESPONSE: Please review the data on household incomes for the appendix Table. In fact, low income households are slightly over represented and affluent families under represented. We wonder if the reviewer is referring to the fact that adolescents from poorer households were more likely to drop out, an issue that we have discussed in our handling of attrition below.

4) Exposure: Movie alcohol exposure was assessed using a previously validated method. However the nature of the classification does not appear to evaluate the nature of the exposure, whether 'positive' or 'negative' in terms of how alcohol use is depicted.

RESPONSE: It is impossible to ascertain how the adolescent reacted to each scene of alcohol use in a movie using our content coding scheme and our method for ascertaining exposure. This is a limitation of our method; we suggest that assessing response to positive and negative alcohol scenes would be a better topic for an experimental research project. We have more complete data on movie tobacco use and found that it didn't matter whether the character depicting tobacco use was a "good" guy or a "bad" guy, that adolescents responded with increased smoking to each exposure.[1] Based on that study, we would be surprised if ascertainment of character valence would make much of a difference for alcohol.

4 continued) Alcohol branded merchandise was not ascertained at baseline which is unfortunate.

RESPONSE: We would have preferred to have assessed ABM at baseline, but it was assessed at the second wave. Since there was an association between ABM ownership and drinking

outcomes, net other exposures, it isn't clear in our mind why this would be a major problem with the study, except that the hazard ratio is averaged over two observation periods rather than three.

4 continued) A range of other predictor variables were ascertained relating to the individual adolescent's and then family predictors included variables shown in other studies to be important in alcohol use initiation including availability of alcohol in the home, alcohol use and parenting authority.

RESPONSE: We agree that the large range of covariates represents a major strength of the study. The association between movie alcohol and alcohol branded merchandise and the outcomes is independent of this large range of covariates.

5) Sample size: The sample size calculation refers to moving smoking and never smokers (p8) which is clearly a careless 'cut and paste' mistake?? The authors need to show what figures they used to calculate the sample size fully and explain why they used these. What was the estimated prevalence of alcohol use at baseline and what did they use as an estimate of movie exposure? Why did they use movie exposure and not ownership of branded materials or parental factors which may be less common?

RESPONSE: It is unfortunate that the reviewer interpreted this sentence pejoratively, as a careless cut and paste mistake. Please see our response to point 5 for reviewer 1. We make clear in the revision why the study was powered on smoking and not alcohol. The questions that follow on point 5 are hypotheticals, but since we did not do a sample size calculation based on alcohol, they seem less relevant. It is worth mentioning here that adolescents start using alcohol about the same time as they start smoking, and that studies powered to study smoking as an outcome during early adolescence are generally adequately powered to study alcohol. It seems rather extraordinary to be facing criticism that our longitudinal study of over 6,000 adolescents is inadequately powered, but that often happens when studies make a point out of a null finding, as we have in this case for family predictors of alcohol use on binge drinking. If the reviewer wishes to explicate the particular variables that the study may be inadequately powered for, we can then look at the estimate and determine how many individuals would have to be in the study in order to detect an effect. For associations like the availability of alcohol in the home on binge drinking (AHR 1.12) the sample size necessary to detect this effect as significant is likely to be in the tens of thousands and clearly beyond the scope of this or any of the other longitudinal studies of alcohol use during adolescence cited in this article. The pertinent question here is, "Is an AHR of 1.12 important from a clinical or public health standpoint?" We would say no.

5 continued) Also, why did they select onset of drinking rather than onset of binge drinking – presumably the latter was less frequent (particularly because the sample size is further reduced as it is for ever drinkers) which raises concerns about whether the study is adequately powered to detect factors associated with progression to binge drinking.

RESPONSE: Although onset of binge drinking is less frequent (see Table 2), it does occur at high enough frequency to detect significant associations for a number of variables, including race, age, movie exposure, ownership of alcohol branded merchandise, sensation seeking, and rebelliousness. Because different predictors may predict different drinking transitions, we prefer the current approach, which is similar to using growth models to predict intercept and slope.

6) Statistical analysis: Based on my understanding of survival analysis there are several limitations here. Survival models were used in the analysis with no reference to which specific method they used (Cox?), whether proportional hazards assumptions held true or which statistical software was used.

We stated in the statistical methods section, “Discrete time hazard survival models were fit to each of the imputed complete datasets following standard procedures for pooling the estimates and obtaining standard errors.” The Cox model is a continuous time survival model. Discrete time hazard models are appropriate when data are collected in survey waves, as they are here. We added to the statistical methods section to make it clear that the survival models were fit using the logistic regression routine in the R statistical package. The proportional hazards assumption in this framework implies that the effect of a baseline predictor stays constant throughout the discrete time periods that make up the longitudinal follow up. This amounts to a moderation of a substantive predictor effect by time, which was not of direct theoretical interest for this manuscript. For brevity, we focused this manuscript on the main effects of the predictors.

There is no discussion of how hazard ratios have been adjusted ie. what the modeling strategy was and what covariates were considered for inclusion and why.There is no discussion about collinearity of variables or potential interactions (age is the obvious one). It would be helpful to have indicated how the attributable risk fraction calculations on adjusted data were undertaken.

Response: All variables were entered in the model simultaneously. Covariates were selected to rule out the possibility of a spurious relation for the variables of direct theoretical interest, movie alcohol exposure and owning alcohol branded merchandise. Pathological collinearity of variables was not an issue and moderation due to age, follow up time and other interactions were not the theoretical focus of the manuscript. For brevity, we focused this manuscript on the main effects of the predictors. Unfortunately, the reviewer appears to have overlooked the explanation on page 11, lines 33-54 for the attributable risk calculations. If there are still concerns about the explication of the statistical methods in the manuscript, we would be happy to submit a technical appendix for publication.

Results:
Participants were followed up over three 8 month periods, with approximately 30% attrition by the final phase compared to those eligible, but this still falls within the sample size calculation for onset of drinking. The authors acknowledge that the characteristics of those who dropped out were disproportionately from non-white, poorer groups. The multiple imputation method used to address missing data assumed it was missing at random which clearly it wasn’t.

RESPONSE: Unfortunately, the reviewer misunderstands the common terminology in use for missing data methods, a frequent problem because of the confusing terms applied by experts that study attrition methods. Missing at random, the so called MAR assumption, means missing at random conditional on covariates included in the model. Missing completely at random, MCAR, is the term for missingness that is completely unpredictable. Thus, the fact that predictors included in the model are significantly related to the fact of missingness does not invalidate the MAR assumption. The imputation model included all of the predictors in the model and some auxiliary variables not included in the model that were nonetheless predictive of missingness. This helps to make the MAR assumption more plausible and improves the quality of the imputations.[2] It is also commonly and erroneously assumed that the MAR assumption can be tested. Unfortunately, it cannot unless the missing data can somehow be recovered. Nevertheless, MAR based methods are still the current method of choice because they outperform older methods (in terms of power and bias) based on using only subjects with complete data or single imputation. Much as we would like to include this kind of background information in all our manuscripts to help educate reviewers and readers, space limitations preclude it.

Tables 3 and 4 show adjusted hazard ratios for 20 different variables for the onset of consumption (table 3) and onset of binge drinking (table 4). My concern is that the study may not have been adequately powered to detect factors associated with progression to binge drinking (see above). Inclusion of sample numbers rather than just percentages in table 2 would be useful.

RESPONSE: See our detailed response to power. Again, it would help if the reviewer would explicate exactly which variables she feels the study was inadequately powered to address and why, based on the proportion affected and the adjusted hazard estimates, she feels that particular variable is important from a population or clinical standpoint.

From a public health perspective it is useful to see the attributable risk fractions presented though how these were done on adjusted data was not clear as previously mentioned.

RESPONSE: See our response above concerning the explanation of attributable risk calculations. We think that Reviewer 2 may have overlooked the explanation on page 11, lines 33-54 for the attributable risk calculations.

Notwithstanding some of the concerns over whether the study is adequately powered to detect factors associated with progression to binge drinking, there are interesting associations between for example peer alcohol use, parental alcohol use, parenting skills and alcohol availability and the onset of alcohol use, which are, aside from movie exposure, important risk factors.

Response: Yes, and these associations suggest that power was adequate to detect effects of public health significance.

Discussion and references:
The authors acknowledge that unmeasured confounding is a limitation of the study (as with other observational studies) and recognise that they were not able to disentangle television and movie alcohol exposure. The conclusions were reasonable given the data presented, although again this depends upon whether the sample was adequately powered to detect all associations with progression to binge drinking.

Response: Powering a study to detect all non-zero associations with an outcome is not even remotely practical. Investigators need to make decisions about how big an effect must be to be important and then power studies accordingly. The reviewer needs to be more specific about what effect size her power concerns pertain to in order for us to answer the concern more specifically.

References: were comprehensive but the majority were from several years ago, with little recent literature cited. The STROBE questionnaire did not appear to have been submitted, although correspondence suggested it had.

Abstract: The abstract was an accurate reflection of the paper and key messages were appropriately presented.

Ethics: There is no mention of the ethical approval in the manuscript.

RESPONSE: In the methods section, we included a sentence at the end of the first paragraph, “All aspects of the survey were approved by the institutional review boards at Dartmouth Medical School and Westat.” Is there something additional the reviewer was looking for?

Recommendation on paper
As the paper currently stands I think there is insufficient statistical detail to assess the results in an informed manner. My concerns are that there is no clarity on the multivariate modelling, whether the survival analysis is appropriate and whether the study is powered to detect factors associated with progression to binge drinking. If not, then the differences in associations found between onset of consumption and progression to binge drinking are potentially erroneous. I would welcome further expert statistical review of the methods.

RESPONSE: We hope that our responses to the specific concerns addressed above are satisfactory to assuage concerns about the statistical analysis and power to detect an effect of clinical or public health importance.

REFERENCES

1. Tanski SE, Stoolmiller M, Dal Cin S, Worth K, Gibson J, Sargent JD. Movie character smoking and adolescent smoking: who matters more, good guys or bad guys? *Pediatrics* 2009;124(1):135-43.
2. Graham JW. Missing data analysis: Making it work in the real world. *Annual Review of Psychology* 2009;60:549-76.