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Childhood physical punishment and the onset of drinking problems: evidence from metropolitan China

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Abstract

Background—Evidence in support of a suspected causal association linking childhood physical punishment (CPP) and later alcoholic beverage-related disturbances has been found in metropolitan China. Here, the focus shifts to the CPP association with the estimated risk of starting to drink, having the first drinking problem, and transitioning from first drink to the first drinking problem.

Methods—Data are from the World Mental Health Surveys-metropolitan China study, with representative samples of adult household residents living in two metropolitan cities, Beijing and Shanghai. Recalled information was available for early life experiences (including CPP, other childhood adversities, and parental alcohol and drug problems), as well as the onset age of drinking and drinking problems. Survival analyses were used to estimate the hazard ratio. A structural equation modeling approach was used to control for other inter-correlated childhood adversities.

Results—Cox proportional hazards modeling discloses statistically robust associations linking CPP with drinking and drinking problems, as well as more rapid transitions from first drink to first drinking problem, even after accounting for other childhood adversities and parental drinking problems. These associations cannot be attributed to a more general noxious family environment.

Conclusions—These results lay a foundation for future experimental studies on the possible causal relationship linking CPP with the onset of drinking problems and the transition from drinking to drinking problems.

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Contributors

Author HGC designed the study, managed the literature searches, undertook the statistical analysis, and wrote the first draft of the manuscript. JCA advised data analysis and revised the manuscript. All authors contributed to and have approved the final manuscript.

Conflict of Interest

All authors declare no conflicts of interest.

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Keywords

Alcohol use disorder; Childhood physical punishment; China; Epidemiology

1. Introduction

Drinking is one of the leading causes of disease burden globally (Murray and Lopez, 1997). Recent epidemiological surveys have found that drinking is common in China, where drinking problems including alcohol use disorders (AUD) are not rare (Cheng et al., 2010a; Hao et al., 2004). In this context, the identification of possible causes of drinking problems, especially those which may be potentially modified, is of interest because it may provide guidance for prevention and intervention strategies.

This is the third report on a suspected causal association that links harsh childhood physical punishment (CPP) with later drinking problems in metropolitan China. In the first report, a generalized linear model with logit link was used to study a multivariate profile of drinking responses observed in adulthood. The odds of alcohol dependence and associated clinical features in adulthood was found to depend on CPP history, even with family drinking problems taken into account (Cheng et al., 2010c). In the second report, a structural equations modeling (SEM) approach was used to evaluate the degree to which the level of alcohol dependence in adulthood might depend upon the CPP history versus an alternative model in which the most prominent predictive or causal variable of interest is a generally noxious family environment with CPP and other childhood adversities bundled into the noxious environment construct. In that report, levels of alcohol dependence were found to depend on CPP history, even with a generally noxious family environment held constant via an SEM approach (Cheng et al., 2010b).

These investigations left uncertainty about the timing of onset of drinking and the timing of onset of alcohol dependence problems once drinking had started, and did not address some nuances introduced by Sartor and colleagues in their recent studies (Sartor et al., 2007a; Sartor et al., 2007b). For example, an excess odds on level of alcohol dependence might be mediated by earlier onset of the first drink, inasmuch as early-onset drinking per se might account for later excess risk of alcohol dependence (Dawson et al., 2008; DeWit et al., 2000; Kessler et al., 1997; Kuo et al., 2008; McGue et al., 2001; Rothman et al., 2008). Furthermore, alcohol dependence might emerge during the teen years, soon after drinking onset, or might be delayed until many years after the start of drinking. There are questions about the natural history of drinking and drinking problems, or “mechanisms” through which CPP might be linked to greater odds or higher levels of alcohol dependence. Accordingly, in this third report, we seek to answer these questions about the time course from early CPP to (1) later onset of drinking to (2) later onset of alcohol problems.

It has been widely documented that childhood adversities are associated with drinking problems (see Cheng et al., 2010c). Possible developmental pathways included changes in the structure of the brain and the function of certain neurotransmitters and their receptors (e.g. GABA, Bremner et al., 1997; Teicher et al., 2006), as well as hormonal responses to stress (Carpenter et al., 2010; Heim et al., 2000). Social pathways also may link parenting deficiencies to drinking problems, perhaps via affiliation with deviant peers (Lloyd and Anthony, 2003), and other precocious antisocial behaviors (Jacob and Johnson, 1997).

Numerous studies have provided supportive evidence on the possibility that early CPP might accelerate time to drinking onset (e.g., Anda et al., 2002; Cheng et al., in press; Dube et al., 2006; Hamburger et al., 2008; Kaufman et al., 2007; Rothman et al., 2008). Dom et al.

(2007) and Kendler et al. (2002) found earlier onset of drinking problems linked back to childhood adversity in general and to parental loss or separation, but did not study CPP in specific (Dom et al., 2007; Kendler et al., 2002). In this context, there are two alternative possibilities for the observed association linking CPP with early onset AUD. The first is that CPP is associated with both early initiation and more rapid progression; the second is that CPP is associated with early drinking initiation only. That is, once drinking starts, CPP is not associated with the progression from drinking to AUD. For example, the observed CPP-AUD association might be due to longer duration of drinking for people who experienced CPP. The latter scenario has been found by Sartor and colleagues in relation to childhood sexual abuse and parental divorce with respect to drinking outcomes. These adversities were associated with earlier initiation of drinking but not the progression from drinking to AUD (Sartor et al., 2007a; Sartor et al., 2007b). To the best of the authors' knowledge, there has been no such study focused on CPP specifically in relation to drinking outcomes.

Against this background, the aims of the study are 1) to estimate the association linking CPP with the time of onset of drinking and of drinking problems for the population as a whole; 2) to estimate the association between CPP and the time from drinking initiation to the onset of drinking problems (i.e., via study of time to drinking problems once drinking has started). In this study, data are from the World Mental Health- metropolitan China survey (WMH-mC), a cross-sectional survey of household-dwelling adults in Beijing and Shanghai, China.

As indicated in many previous studies, childhood adversities are inter-correlated. Hence, work that is focused on one aspect may be artificial (Cheng et al., 2010b; Dong et al., 2004). For this reason, in this study, the research approach is one that couples SEM with survival analyses methods in order to take the inter-correlation into account in a fashion that does not violate assumptions of generalized linear models, which otherwise might introduce bias or inconsistency in estimation.

We alert readers that in some parts of the world the term “child abuse” might apply to experiences we have characterized as “physical punishment,” and these readers are invited to make a mental substitution of the term “child abuse” in our sentences that refer to “physical punishment.” Nonetheless, we note cross-national and sometimes within-country cultural variations in the use of these terms, which are not completely interchangeable. For example, in some parts of the world, whipping a child's buttocks with a paddle, a belt, or switches (a sheaf of small tree branches) is a usual and customary disciplinary practice that would not qualify as “child abuse” as defined in child protection laws, but it would qualify as a form of “harsh physical punishment” in virtually all parts of the world. However, we should clarify that our use of the term “punishment” does not imply that the child has violated a rule or is being punished for rule-breaking. In this context, our usage of the term “punishment” is in conformity with the Oxford American Thesaurus of Current English (Lindberg, 2002), in which “to punish” encompasses serious forms of “abuse,” but also harsh “smacking” and “slapping” with no implication that the child is being punished for breaking a rule. In some instances, the “punishment” might simply be the aggressive lashing out toward a child when one parent has been frustrated by the other parent. We invite our readers to conceptualize the “punishment” in this more general sense, mentally re-casting it as “child abuse” if the result is enhanced comprehension of the study evidence.

2. Methods

2.1 Study design and sample selection

Prior publications give detailed methods overviews (Shen et al., 2003; Shen et al., 2006). Here we provide a brief description.

Data were collected for the cross-sectional WMHS-mC, which used a stratified multi-stage probability sampling method to select household-dwelling adults, aged 18 to 70 living in Beijing and Shanghai. The Research Center for Contemporary China at Peking University directed the field work in both cities, which involved face-to-face interviews between November 2001 and February 2002. All respondents were informed about the study and provided written informed consent prior to the interview using a study protocol approved by the designated Institutional Review Board. Participation levels were approximately 75% in both cities (Shen et al., 2003).

2.2 Assessments

The instrument used in WMH-mC was the World Mental Health Initiative version of the World Health Organization Composite International Diagnostic Interview (WMH-CIDI, Kessler and Ustun, 2004). The WMH-CIDI was a comprehensive, fully structured diagnostic interview designed to be administered by trained lay interviewers to assess clinical features of psychiatric conditions including AUD. The Chinese version of the WMH-CIDI used in WMH-mC was derived using standard protocols of iterative translation, back translation, and harmonization conducted by panels of bilingual experts.

The interview was administered in two parts. Part I included the core diagnostic assessment. Part II included questions about suspected correlates or determinants as well as additional topics. Part II was administered to all respondents who were suspected to have a history of past or recent core mental disorders, assessed in Part I, plus a 25% random sample drawn from the rest of respondents. A total of 5201 participants completed Part I; 1628 completed Part II. The analytical sample is the subsample of 1628 respondents because childhood experience was available for the sub-sample of 1628 respondents only. An inverse probability weight for the Part II sample was created to adjust for the over-sampling of Part I mental disorder cases.

2.3 Outcome variables

In the current study, there were three sets of outcome variables. The first set is the age of the first drink of alcoholic beverage. The second set is focused upon (a) time to first occurrence of socially maladaptive and/or hazard-laden drinking (abbreviated SMD), and (b) time to first occurrence of clinical features of an alcohol dependence syndrome (abbreviated CFAD). The concept of SMD is in line with ICD-10 harmful use of alcohol or DSM-IV alcohol abuse. The five measured clinical features of SMD were 1) interference with responsibilities due to alcohol drinking, 2) social problems, 3) drink despite social problems, 4) hazard-laden drinking (e.g., drunk driving), and 5) legal problems due to drinking. The eight CFAD included: tolerance, withdrawal, difficulty cutting down, drink more than intended, giving up activities because of drinking, a great deal of time spent on drinking-related activities, drinking despite physical/emotional problems, and irresistible desire. The third set of outcomes included time from the first alcoholic beverage to the onset of SMD and CFAD, respectively.

One aspect of this research that merits attention is its focus upon CFAD with “clinical significance” gauged in relation to whether the drinker had experienced SMD (Degenhardt et al., 2007). In essence, individuals who had never experienced SMD were not assessed for CFAD. Thus, there might be an undercounting of individuals with CFAD. However, we are aware of no evidence that CPP-alcohol associations become biased when there is undercounting of cases of DSM-IV alcohol dependence in the community survey context. This issue has been included in our discussion of study limitations.

2.4 Covariates under study

The primary covariate under study is “childhood physical punishment” (CPP) variable, derived from responses to a CIDI question of the following form: “*When you were growing up, how often did someone in your household do any of the things to you – often, sometimes, rarely, or never?*” Deeds of physical punishment (“things” in the question) included “pushed, grabbed or shoved”, “threw something”, or “slapped, hit, or punched.” Respondents who answered “often” and “sometimes” were coded as “yes”; “rarely” and “never” were coded as “no”. Therefore, the global CPP variable included all types of CPP and has been conceptualized as a form of chronic stress from physical punishment during the childhood.

Other binary coded (yes/no) covariates included in the model were: parental alcohol/drug problems, and other childhood adversities including parental divorce, not living with both parents before 16 years of age, violence between parents, child neglect, poor parental relationship, and a lot of tension in the household. All variables were based on self-report of standardized survey questions from the respondent.

As in prior reports, two approaches were used in this study to estimate the relationship of CPP and the onset of drinking problems: (1) inclusion of a vector of inter-correlated childhood adversities as covariates, and (2) creation of a single “noxious family environment” latent dimension, measured by items of all adversities (including the CPP item; Cheng et al., 2010b).

2.5 Analysis Plan

Prior to analysis, discrete age and age of onset values, assessed retrospectively at cross-section, were used to synthesize the time to event datafiles for drinking onset and for the SMD and CFAD outcomes, with a Tukey-style exploratory data analysis step for consistency and range checks and to evaluate marginal distributions of the variable values. Thereafter, survival analysis models were used with person-year as the unit of analysis. All covariates (including childhood adversities, sex, age, and parental drinking problems) were treated as time-invariant variables. Initially, Kaplan-Meier estimators were used to describe the survivorship experience of (a) the age of onset of drinking and (b) the age of onset of drinking problems, as well as (3) the time between first drinking to the onset of drinking problems for CPP and non-CPP groups, respectively.

Thereafter, a Cox proportional hazards model was used to estimate the association linking CPP with drinking outcomes. A single-equation model was used to hold constant sex and age. Due to the inter-correlation of various childhood adversities, estimates from single equation linear regression models may be biased or inconsistent due to the violation of the assumption of independent covariates (Heckman and Singer, 1986). Therefore, we turned to the SEM approach in order to hold constant these inter-correlated covariates. In the first SEM model, other childhood adversities were held constant via covariate terms which were allowed to be inter-correlated in order to produce an estimate of the CPP–drinking relationship (Figure 1). Hereinafter, this model is labeled “multi-adversity SEM.” Next, a “CPP-specific” model was used to inspect whether the CPP–drinking association might be attributed to a generally noxious family environment, as measured by all childhood adversity items. In this model, CPP was allowed to have a direct influence on the outcome variable, over and above the influence of the “noxious family environment” variable (see Figure 2, arrows on the left represent error terms of the observed variables that were included in the latent variable). That is, in this model, a regression slope estimate was produced for the direct path drawn from the latent “noxious family environment” to the outcome variable; at the same time, another regression slope was produced for the direct path from CPP to the

outcome. In both approaches, “parental alcohol and drug problems” was then introduced as a potential confounding variable; sex and age were held constant via covariate terms. Evidence in support of the unidimensionality of the latent “noxious family environment” is described in a prior report (Cheng et al., 2010b). These models were fit for the onset of drinking problems and time from the first drink to the first drinking problem, respectively. Estimates were presented in the form of Hazard Ratio (HR). The HR estimate represents the relative hazard of experiencing the outcome over time for individuals who experienced CPP compared to those without CPP.

One might suspect the observed CPP-associated hazard for the onset of drinking or drinking problems might be attributed to the greater proportion of drinkers in the CPP group. Thus, these models were fit for the population sample as a whole, as well as the subpopulation of drinkers ($n=1059$). To help tease out the temporal relationship of CPP and the onset of drinking or drinking problems, models were also fit for the subpopulation of drinkers who started drinking after 16 years of age ($n=668$). In a cross-sectional context, this may be important (especially for the onset of drinking): parents might physically punish their children due to the child’s early initiation of drinking.

After initial analysis/estimation steps, the proportional hazards assumption of the Cox model was checked via the inclusion of product terms: i.e., CPP crossed with categorical failure time (Cleves et al., 2008). In this approach, a null product term is consistent with acceptability of the proportional hazards assumption. Model specifications for single-equation models were checked via the link test.

2.6 Special issues regarding variance estimation

Sampling weights were used to account for variations in sample selection probabilities, differential participation, and CIDI Part I/Part II selection described above. Detailed description about the weights can be found in previous publications (Kessler et al., 2004; Kessler and Merikangas, 2004). A Taylor series linearization method was used for variance estimation, with due attention to variation in sample selection probabilities and post-stratification adjustment factors. Analyses have been implemented using statistical software Stata (version 11.0, StataCorp LP) and Mplus (version 6.0, Muthén and Muthén).

3. Results

Table 1 provides a description of sex and age of the sample, as well as means of the onset age of drinking and drinking problems for the CPP and non-CPP group, respectively. Individuals who experienced CPP are slightly younger ($\bar{x}=37.9$ for the CPP group vs. $\bar{x}=41.5$ for the non-CPP group, $p=0.02$). A slightly larger proportion of males is found in the CPP group compared to the non-CPP group (59.7% vs. 51.4%, $p=0.08$).

Kaplan-Meier estimators and resulting plots of the survival curves disclose that in both CPP and non-CPP groups, drinking onsets tend to emerge early in life (before 10 years of age); there is acceleration of drinking onset rates in the late teens and early 20s (Figure 3). Individuals in the CPP group are found to be more likely to start drinking, irrespective of the age value considered. Regarding the onset of drinking problems, in the CPP group, emergent problems are observed in late teens, with acceleration through the 20s, after which a plateau of the rate is seen (Figure 4). In the non-CPP group, drinking problems emerge in later teens and increase gradually until the late 30s.

Among participants who started drinking after 16 years old, CPP is associated with greater hazard of the drinking onset in this subset ($HR=1.5$, 95% $CI=1.1, 2.0$) holding all other covariates constant (Table 2).

Cox regressions also disclosed that for the population as a whole, CPP is associated with a greater hazard of drinking problems (Table 2). With sex and age held constant as covariates, estimated HRs are 2.9 (95% CI=1.6, 5.2) and 3.4 (95% CI=1.6, 7.1) for onset of SMD, and for CFAD, respectively. After holding other childhood adversities constant under the “multi-adversity SEM” model, point estimates for HRs decrease to 2.0 and 2.7 and remain statistically robust. The “CPP-specific” model disclosed that the CPP-associated excess hazard is not accounted for by the generally noxious family environment (i.e., estimated HRs remain robust for the onset of SMD and also for CFAD). In the analysis restricted to drinkers only, the estimated HRs are generally smaller. After holding other adversities constant under the “multi-adversity SEM” model, point estimates for HRs dropped to approximately 2.0; confidence intervals included the null value. The “CPP-specific” model disclosed robust associations linking CPP with excess hazard of drinking problems.

CPP also is found to be associated with time from the first drink to the onset of drinking problems. After holding constant sex and age, individuals with CPP experience are more likely to develop SMD (estimated HR= 2.4, 95% CI=1.2, 4.8) and CFAD (HR=2.6, 95% CI=1.0, 7.0) once drinking started. Estimated HRs from the “multi-adversity SEM” and “CPP-specific” model are approximately 2.0, although some confidence intervals include the null value. Compared to results from the subpopulation of drinkers, point estimates for those who started drinking after 16 years old are generally greater and not as precise (consistent with the restricted sample size).

Product terms of CPP by categorical failure time were not statistically robust for the four outcomes under study ($p>0.20$). Additionally, the link test produced no evidence of model misspecification for sex and age adjusted models.

4. Discussion

Main results of this study are summarized succinctly as follows. First, for the population under study as a whole, individuals who experienced CPP were found to be at a greater hazard of drinking and developing drinking-related problems (SMD and CFAD in this study). Based on the covariate-adjusted models, this association might not be due to parental drinking problems or other childhood adversities. Moreover, it is not explained by a generally noxious family environment. After accounting for these alternative potentially explanatory constructs, the HRs are estimated to be approximately 2.0. Furthermore, there is evidence that CPP is associated with the transition from drinking to drinking problems once drinking starts, but this association is not robust once other adversities are taken into account.

4.1 Limitations and strengths

Before detailed discussion, several important limitations merit attention. Of major concern is the use of recalled information. With respect to the assessment of drinking problems, under-reporting may be introduced when a long time period has elapsed since the first onset of drinking or drinking problem. Previous studies in the US have found that recalled onset age of drinking problems can provide valid and reliable information for the population as a whole (e.g., Harris et al., 1994). Nonetheless, it is not clear whether there is CPP-associated differential recall or whether the above U.S.-based findings apply to Chinese populations. Some studies have found poor validity for retrospectively recall of age of the first drink (e.g., Bailey et al., 1992). Therefore, it is possible that bias has been introduced in estimates for transition from drinking to drinking-related problems if patterns of recall vary for CPP and non-CPP groups, which, to the best of our knowledge, has not yet been studied. More methodological and longitudinal research in China will be required to address these issues. Regarding the possible under-reporting of CPP, there is evidence from Western countries

that information about CPP collected from respondents is valid and reliable (Bremner et al., 2007; Hyman et al., 2005; Walsh et al., 2008). In China, CPP is not especially stigmatized, and a considerable portion of children experienced occasional physical punishment while growing up. Thus, it is not likely that the estimates were substantially biased due to the under-reporting of CPP.

The assessment of CPP was a global assessment and did not differentiate various forms of CPP. It is possible that the estimates were driven by more severe forms of CPP (e.g. slapping, punching, or hitting) rather than less severe forms (e.g. pushing or grabbing). However, previous studies have found that these behaviors were highly correlated. For example, over 85% of individuals who experienced any form of CPP had experienced another form of childhood adversity (Dong et al., 2004; Hawkins et al., 2010). Furthermore, we grouped people who answered “often” and “sometimes” together and those who answered “rarely” and “never” together. In this way, the CPP variable represents a more chronic stressor during childhood. This study serves as a foundation for future studies on more specific CPP-AUD relationship. Another issue to consider in the interpretation of the estimates is the differential survival of CPP and AUD affected individuals. For example, CPP and non-CPP individuals might have experienced different alcohol-related morbidity and mortality rates. According to previous research, individuals who suffered from severe forms of abuse may be less likely to survive; severe forms of abuse have been strongly associated with heavy drinking and alcohol dependence (Arias et al., 2003; Simpson and Miller, 2002; Widom et al., 2007). Therefore, bias from this source might have driven the study estimates toward the null (i.e., yielded smaller estimates than the true values).

The WMH-CIDI module on drinking problems is not 100% exhaustive in its coverage of all drinking problems, and some affected cases or problem drinkers might have been missed in the survey. Nonetheless, the WMH-CIDI coverage of drinking problems was broad, and the outcomes under study were not selected with any CPP hypothesis in mind. For this reason, one might suspect that a more complete set of questions about drinking problems would lead to similar patterns of CPP association.

The study’s focus was on drinking onset and time to the first CFAD; it was not upon DSM-IV alcohol dependence. The focus on the first CFAD seems to be justified; there is evidence that the first CFAD can be predictive of later development of alcohol dependence and often has clinical significance (Harford et al., 2010; McBride et al., 2009a; McBride et al., 2009b). Last but not least, the source population of this study is non-institutionalized adults living in Beijing and Shanghai, China. Thus, results may not be applicable to other groups of Chinese (e.g. rural residents or institutionalized individuals) before further evidence.

Several counter-balanced strengths of the study are as follows. First, most prior research on this topic has been based on Western population samples, often limited to clinical cases being treated for child abuse or for alcohol problems. Nonetheless, CPP is a culturally sensitive topic, and its definition can vary from place to place. Even when CPP definition is held constant, the influence of CPP may vary from society to society. Here, the estimates for two of China’s metropolitan areas are based upon household respondents during a community sample survey. Possible biases in estimation were minimized by efforts to adjust for clustering and selection probability, as well as non-response patterns. Thus, results are directly applicable to the household-dwelling adults living in the two Chinese cities under study. Epidemiological sampling of this type can be particularly important when and where the majority of individuals with drinking problems do not seek medical attention (Demyttenaere et al., 2004; Shen et al., 2006). Second, the study provides evidence of a possible causal relationship between CPP and the onset of drinking problems in a Chinese context, with a broadening of the published evidence from China to include time to event

outcomes. Using a survival analysis approach, we were able to address questions about the age-specific hazard, building upon prior investigations of excess odds and levels of CPP-associated alcohol outcomes that did not take time-to-event into account. Third, via a SEM approach, we sought to account for parental drinking/drug problems and other inter-correlated childhood adversities. This research approach helped add statistical efficiency and may have ensured more accurate estimates of CPP-drinking associations.

4.2 Childhood physical punishment and the onset of drinking problems

In an extension of evidence from prior studies linking CPP with drinking outcomes (Cheng et al., 2010b; Cheng et al., 2010c), we found individuals who had experienced CPP were an estimated 1.5 times as likely to start drinking even after holding other childhood adversities constant (among those who started drinking after 16 years of age). This finding is in line with previous research from the U.S. (e.g., Bensley et al., 1999; Dube et al., 2006; Hamburger et al., 2008; Lee et al., 2010; Tarter et al., 1999). Moreover, individuals who experienced CPP are approximately three times as likely to develop drinking-related problems compared to individuals without CPP experience after holding other childhood adversities constant. This association cannot be completely attributed to the greater proportion of drinkers among individuals who experienced CPP.

Additionally, this study found some evidence supporting a more rapid transition from drinking to problems for individuals with history of CPP compared to individuals with no such history. After drinking starts, individuals with the past experience of CPP are twice as likely to develop drinking problems after holding other childhood adversities constant. Thus, the higher hazard of drinking problems in CPP individuals may not be completely attributed to their earlier initiation of drinking. This finding may be perceived as somewhat different from previous findings from the U.S., where childhood adversities might not be associated with the transition from drinking to problems (e.g., Sartor et al., 2007a; Sartor et al., 2007b). Several possible explanations for the apparent divergence include the following. First, a difference in findings might be an artifactual consequence of a difference in focus. The present study is particularly focused on CPP, one of the potentially modifiable childhood adversities, while previous studies have studied either childhood adversities as a global construct, or some other specific adversities (e.g., childhood sexual abuse). Second, the source population of the current study is household-dwelling adults living in Beijing and Shanghai, China. Most previous studies are from the U.S. of various populations such as twins (Sartor et al., 2007a; Sartor et al., 2007b). Third, to produce its CPP-specific estimates, the current study controlled for other inter-correlated childhood adversities via SEM. Another possibility is that the current study grouped individuals who “never” and “rarely” had CPP experience together versus “sometimes” and “often” together. In this way, the CPP variable is more linked to chronic rather than acute stress and it might be the chronic stress that has an influence on drinking or development of drinking problems. In this context, we note that, the childhood years through early adulthood represent a crucial developmental period for brain maturation (Tarter et al., 1999). Long-term exposure to environmental stressors such as CPP might alter the maturation processes (e.g., via epigenetic mediator mechanisms not yet fully understood). There has been evidence that different sets of variables may play major roles in various stages of the drinking trajectory (Sher et al., 2004). Previous findings suggested that the influence of childhood adversities was mainly in the stage of drinking initiation and diminished afterwards (Sartor et al., 2007a; Sartor et al., 2007b). Our findings suggest that besides early initiation of drinking, CPP may also influence the transition from drinking to drinking problems.

In summary, the current study found an association linking CPP with time to both onset of drinking and onset of drinking problems. Nonetheless, experimental studies (e.g., intervention trials) may be required to confirm the suspected causal significance of this

association. New observational research may shed light on underlying mechanisms that might account for the observed associations, even if they are not causal. Above research will be needed to open the “epidemiology black-box” represented by the association as now observed.

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Figure 2.
A “CPP-specific SEM” model

Figure 3. Kaplan-Meier estimator of the onset of the first drink for subgroups in relation to experiences of childhood physical punishment (CPP). Data from WMH-mC 2001–2002. Panel 1 (left), Time of the first drink for all persons. Panel 2 (right), Time of the first drink among drinkers.

Figure 4.

Kaplan-Meier estimator of the onset of the first drinking problem for subgroups in relation to experiences of childhood physical punishment (CPP). Data from WMH-mC 2001–2002. Panel 1 (upper left), Onset of socially maladaptive drinking. Panel 2 (upper right), Onset of the first alcohol dependence clinical feature. Panel 3 (lower left), Time from the first drink and the onset of socially maladaptive drinking. Panel 4 (lower right), Time from the first drink and the first alcohol dependence.

Table 1

Sample description. Data from WMH-mC, 2001–2002 (n=1628).

	Exposed to CPP (n=235) ^I		Non-exposed to CPP (n=1376) ^I		p
Age	n=235	$\bar{x}$ (95% CI)= 37.9 (35.1, 40.7)	n=1376	$\bar{x}$ (95% CI)= 41.5 (40.3, 42.8)	0.02
Male	n=235	% (95% CI)= 59.7 (51.7, 67.5)	n=1376	% (95% CI)= 51.4 (47.4, 55.4)	0.08
Age of first drink	n=175	$\bar{x}$ (95% CI)= 16.3 (14.9, 17.7)	n= 838	$\bar{x}$ (95% CI)= 19.1 (18.3, 19.9)	<0.01
Age of onset of SMD ^I	n= 38	$\bar{x}$ (95% CI)= 23.1 (20.9, 25.4)	n= 85	$\bar{x}$ (95% CI)= 25.6 (22.6, 28.6)	<0.01
Age of onset of CFAD ^I	n= 24	$\bar{x}$ (95% CI)= 24.8 (21.9, 27.7)	n= 44	$\bar{x}$ (95% CI)= 25.7 (23.6, 27.9)	0.02

^I Weighted means ($\bar{x}$) and proportions (%) based on the 1,628 adult respondents in the WMH-mC surveys in Beijing and Shanghai

² SMD, socially maladaptive or hazard-laden drinking; CFAD, the first clinical feature of alcohol dependence

Table 2

Associations between childhood physical punishment and the onset of drinking problems. Data from WMH-mc, 2001–2002.¹

	No other covariate		Adjusted for sex and age		Multiple-adversity SEM ²		CPP-specific SEM ²	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
All persons								
Age of the first drink	1.6	1.3, 2.0	1.4	1.1, 1.8	1.3	0.9, 1.7	1.3	1.0, 1.7
Onset of SMD ³	3.6	2.0, 6.6	2.9	1.6, 5.2	2.0	1.0, 4.0	2.3	1.2, 4.3
Onset of ADCF ³	4.3	2.0, 9.3	3.4	1.6, 7.2	2.7	1.1, 6.9	2.8	1.3, 5.7
Among drinkers (n=1059)								
Age of the first drink	1.4	1.1, 1.7	1.2	0.9, 1.6	1.1	0.8, 1.5	1.2	0.9, 1.6
Onset of SMD ³	3.1	1.6, 5.9	2.5	1.4, 4.7	1.8	0.9, 4.0	2.0	1.04, 3.7
Onset of ADCF ³	3.6	1.6, 8.1	3.0	1.4, 6.5	1.9	0.7, 5.1	2.5	1.2, 5.1
Interval between 1 st drink and SMD ³	3.0	1.5, 6.0	2.4	1.2, 4.8	1.8	0.8, 3.8	2.0	1.03, 3.9
Interval between the 1 st drink and CFAD ³	3.2	1.2, 8.4	2.6	1.0, 7.0	1.8	0.8, 4.4	2.1	0.9, 4.8
Among drinkers who started drinking after 16 years old (n=668)								
Age of the first drink	1.4	1.0, 2.0	1.2	0.8, 1.8	1.5	1.1, 2.0	1.4	1.0, 2.0
Onset of SMD ³	4.0	1.7, 9.3	3.1	1.4, 6.8	1.9	0.6, 5.7	2.2	0.8, 5.7
Onset of ADCF ³	8.5	2.9, 25.0	7.0	2.0, 24.5	4.1	0.8, 22.8	3.7	1.2, 11.3
Interval between 1 st drink and SMD ³	4.0	1.6, 9.9	2.9	1.3, 6.6	1.6	0.5, 5.1	2.1	0.8, 5.4
Interval between the 1 st drink and CFAD ³	4.5	1.3, 15.8	3.7	1.1, 12.9	2.2	0.4, 11.2	2.2	0.8, 6.0

¹ Bold font means statistical significance ($p < 0.05$).

² Adjusting for sex, age, parental drinking problems, and other childhood adversities (including parental divorce, not living with parents before 16 years old, child neglect, a lot of tension in the household, poor relationship between parents, and violence between parents)

³ SMD, socially maladaptive or hazard-laden drinking; CFAD, the first clinical feature of alcohol dependence