# Childhood Physical Punishment and Later Alcohol Drinking Consequences: Evidence From a Chinese Context*

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**ABSTRACT. Objective:** The aim of the current study is to estimate a link between early physical punishment in childhood and later alcohol outcomes, taking family history of drinking problems into account, with epidemiological data from China. The yield from previous studies on this relationship is mixed evidence, largely traceable to research design variations, including model specifications that concern parental alcohol or other drug problems (AODPs) that might account for both earlier discipline practices and later drinking problems in the offspring. **Method:** Data are from the World Mental Health Surveys–metropolitan China study, with cross-sectional representative sample surveys of adult household residents living in two metropolitan cities, Beijing and Shanghai. Participants in this general mental health survey were asked about early life experiences (e.g., parental AODP, childhood misbehavior), as well as their own drinking outcomes. Stratification was used to control for parental AODP. **Results:** Logistic regressions found robust associations linking childhood physical punishment with drinking outcomes, even with stratification for parental AODP and childhood misbehavior. **Conclusions:** These results from a cross-sectional survey lay a foundation for future prospective and longitudinal research on possible causal relationships that link childhood physical punishment with later drinking outcomes in China. (*J. Stud. Alcohol Drugs, 72,* 24-33, 2011)

VARIATION IN THE OCCURRENCE OF drinking outcomes generally has been traced to an interplay of both genetic and environmental influences (Liu et al., 2004), including the influence of parental practices on drinking problems. Developmental pathways leading to alcohol problems in the offspring of parents with drinking problems include a possibly attenuated response to alcohol (Schuckit and Smith, 2006) and changes in stress hormones that may interact with coping strategies (Cicchetti and Rogosch, 2007). Social pathways also may link parenting deficiencies to drinking problems, perhaps via incomplete parental supervision that can influence the offspring's access to alcohol (Hamburger et al., 2008), their affiliation with deviant peers (Lloyd and Anthony, 2003), or even childhood misbehavior (CMB), among other precocious antisocial behaviors (Jacob and Johnson, 1997).

Childhood physical punishment (CPP) has been associated with greater occurrence of drinking problems and alcohol use disorder (AUD) in a series of cross-sectional studies with representative samples in the United States and Canada (Afifi et al., 2006; Holmes and Robins, 1987, 1988; MacMillan et al., 1999, 2001). A sturdy CPP–AUD association also has been observed in more restricted clinical and nonclinical population samples of various types, including college freshmen, prisoners, lesbians, and U.S. Marine recruits (Anda et al., 2002; Brown and Anderson, 1991; Clark et al., 1997; Downs et al., 2004; Kunitz et al., 1998; Sher et al., 1997; Swett et al., 1991; Trent et al., 2007). Summarized across these studies, the strength of the association can be characterized as weak to moderate, with estimated odds ratios (ORs) generally in the range of 1.2-2.5.

A few prospective studies have provided estimates for the CPP–AUD association. One prospective study, for example, found a robust CPP–AUD association (Jasinski et al., 2000); two prospective studies found the association in women only (Horwitz et al., 2001; Widom et al., 2007); and one prospective study found a null association (Galaif et al., 2001). In all of these prospective studies, however, there have been substantial levels of attrition over time (40%-75%). This attrition compromises the validity of the study estimates to some unknown extent. Of greater importance are unobserved heterogeneities or confounding, which might be removed if randomization were possible.

Preclinical animal research provides biological plausibility for a causal influence of these childhood stressors on drug use. In rat and primate research, for example, disrupted

Received: January 5, 2010. Revision: July 6, 2010.

*The World Mental Health Survey was supported by U.S. National Institute of Mental Health grant R01MH070884; the John D. and Catherine T. MacArthur Foundation; the Pfizer Foundation; U.S. Public Health Service grants R13-MH066849, R01-MH069864, and R01 DA016558; Fogarty International Center grant FIRCA R03-TW006481; the Pan American Health Organization; the Eli Lilly & Company Foundation; Ortho-McNeil Pharmaceutical, Inc.; GlaxoSmithKline; Bristol-Myers Squibb; and Shire. The work of the authors based at Michigan State University (Hui G. Cheng and James C. Anthony) was supported by National Institute on Drug Abuse grants K05DA015799 and R01DA016558. The Chinese World Mental Health Survey Initiative is supported by the Pfizer Foundation. The content is the sole responsibility of the authors and does not necessarily represent official views of the funding agencies (e.g., within the National Institutes of Health).

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parenting and deprivation of maternal care are followed by abnormal activities in the hippocampus (Heath, 1972), volume reduction in the corpus callosum (Sanchez et al., 1998), alterations in the structure and function of $GABA_A$ receptors (Caldji et al., 2003), and suppression of neurogenesis (Teicher et al., 2006). By the same or similar mechanisms, CPP may influence human brain development. Indeed, changes of this type have been found among people with a history of childhood abuse (Bremner et al., 1997; Teicher et al., 2006) and among those with drinking problems (Oscar-Berman and Marinkovic, 2007).

In the absence of ethical randomized experiments to test the CPP–AUD associations in humans, observational studies must control for potential confounders if causal inference is to be supported. In this context, a parental alcohol or other drug problem (AODP) qualifies as one of the most plausible confounding variables, in that children from AODP-affected families are more likely to have been victims of harsh physical punishment in childhood (DiLalla and Gottesman, 1991; Dube et al., 2001; Widom and Hiller-Sturmhofel, 2001), and AUD aggregates within families (Dick and Bierut, 2006; Kendler et al., 2002). Neglect of these confounders may have produced biased estimates.

Only a few published CPP–alcohol studies have considered parental AODP as a potential confounding variable. Most found a null association between CPP and drinking outcomes once parental AODP was controlled (Hughes et al., 2007; Koss et al., 2003; Libby et al., 2004; Mullings et al., 2004; Young et al., 2006). In two other such studies, there have been positive associations (Dube et al., 2006; Jasinski et al., 2000).

Other limitations that thwart firm conclusions can be found in published studies. First, many studies have used the generalized linear model (GLM), in which an offspring drinking outcome was regressed on both CPP and parental AODP simultaneously (Jasinski et al., 2000; Koss et al., 2003; Libby et al., 2004; Mullings et al., 2004; Young et al., 2006). This approach violates an assumption of the GLM, which requires covariates to be independent and not jointly determined by unobserved sources of variation (Engle et al., 1983). Violation of this assumption can lead to biased or inconsistent estimates (Berg and Mansley, 2004; Briscoe et al., 1990). Moreover, CPP may be an endogenous variable mediating the pathway from AODP to offspring AUD.

As for context, China is currently the world's most populated country, in which a considerable part of disease burden can be attributed to drinking alcohol (World Health Organization [WHO], 2004). Published evidence suggests that harsh physical punishment and sexual abuse in childhood are not rare in China (Chen et al., 2006; Luo et al., 2008; Ross et al., 2005). One study of Chinese high school students found a robust association between childhood sexual abuse and risky drinking (OR = 2.7-5.4 for various outcomes), but no CPP–AUD association was investigated (Chen et al., 2006). Another study of middle school students in Hong Kong found that students beaten by a family member during the 6 months before the assessment were more likely to consume alcohol during that interval (OR = 3.0, $p < .05$; Lau et al., 2003). In this research, however, the temporal sequence from the beating to the drinking outcome has been uncertain; drinking might have preceded the harsh punishment.

Intended to fill gaps in evidence on CPP–AUD relationships in China, the current study involves a research approach that attempts to sort out the temporal sequencing issue via the use of explicit developmental time frames in assessments of CPP and AUD. This cross-sectional approach can be especially powerful when childhood experience has a discrete quality and can be placed in time early in life, well before development of the outcomes of interest; it is important to note that alcohol drinking and AUD rarely start during childhood in China (e.g., see Degenhardt et al., 2008). Longitudinal studies can produce more definitive evidence, but it may be better to postpone longitudinal research until after cross-sectional research has been completed in order to gauge the size of the association. This association must be estimated with some fidelity to estimate sample size requirements for longitudinal research. The present investigation was designed to produce estimates for China that could lay a foundation for a more probing prospective and longitudinal study of the CPP–AUD association in this context.

## Method

### *Study design and sample selection*

Data were collected from the World Mental Health Surveys–metropolitan China initiative (WMHS-mC), a cross-sectional survey of household-dwelling adults in Beijing and Shanghai, China. The WMHS-mC used a stratified multistage probability sampling method to select household-dwelling adults between 18 and 70 years old. In the first stage, neighborhood-level primary sampling units were selected using the probability proportional to size sampling method. For the second stage, households within each primary sampling unit were randomly selected. In the final stage, one adult from each identified household was randomly selected to be the respondent. The Research Center for Contemporary China at Peking University directed the fieldwork in both cities (Shen et al., 2003).

Data were collected through face-to-face interviews between November 2001 and February 2002. All respondents were informed about the study and provided written informed consent before the interview using a study protocol approved by the designated institutional review board. Participation levels were 75% in both cities. Complete details about the sampling and field procedures can be found in the field survey final report (Shen et al., 2003).

*Assessments*

The WMH-mC assessment instrument is a version of the WHO World Mental Health Composite International Diagnostic Interview (WMH-CIDI; Kessler and Ustun, 2004). The WMH-CIDI offers a comprehensive, fully structured diagnostic assessment designed to be administered by trained lay interviewers. It measures clinical features of psychiatric illnesses according to criteria in both the International Classification of Diseases and Related Health Problems, 10th Revision (ICD-10; WHO, 1994), and the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994). The Chinese version of the WMH-CIDI 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 suspected correlates or determinants, as well as additional topics. Part II was administered to all respondents who were discovered to have a history of past or recent core mental disorders, as assessed in Part I, plus a probability sample drawn from the rest of respondents. A total of 5,201 participants completed Part I; 1,628 completed Part II. The analytical sample is the subsample of 1,628 respondents for whom childhood experience was assessed.

*Response variables: The drinking-related outcomes of interest*

In the current study, drinking-related outcomes are "precocious" involvement with alcohol (trying alcohol before the teen years, consuming at least 12 drinks in a year before the age of 20); heavier or "heavy episodic" drinking (≥5 drinks/day for men, ≥4 for women); socially maladaptive (and often hazard-laden) drinking (e.g., manifestations of DSM-IV defined alcohol abuse); DSM-IV alcohol dependence; and alcohol-dependence clinical features (occurrence of any clinical features of DSM-IV or ICD-10 alcohol dependence). The variable termed "heavier drinking" was assessed via the WMH-CIDI question "On the days when you drank the most, about how many drinks would you usually have per day?" The cutpoints for heavier drinking coincide with the conceptualizations of "binge drinking" that have been advanced by the U.S. National Institute on Alcohol Abuse and Alcoholism, as well as definitions for "binge drinking" and "heavy episodic drinking" described in previous studies (Jasinski et al., 2000; National Institute on Alcohol Abuse and Alcoholism, 2004; Trent et al., 2007).

Five manifestations of socially maladaptive or hazard-laden drinking were studied, in alignment with criteria of DSM-IV alcohol abuse. They were "responsibility interference," "social problems," "drink despite social problems," "hazardous drinking," and "legal problems." Eight clinical features associated with contemporary criteria for alcohol dependence were studied: "strong desire to drink," "tolerance," "withdrawal," "drink more than intended," "difficulty cutting down," "giving up activities because of drinking," "a great deal of time spent on drinking-related activities," and "drinking despite physical/emotional problems."

A major assumption was made in the WMH-CIDI assessment of alcohol-dependence problems: If an individual never had experienced socially maladaptive or hazard-laden drinking, the WMH-CIDI measurement assumption was that the drinker could never fully qualify for the DSM-IV diagnostic criteria for "clinically significant alcohol dependence" as explained elsewhere (e.g., see Degenhardt et al., 2007). This measurement assumption will be addressed in the Discussion section as a potential limitation of this research, but the assumption is not operative with respect to the majority of drinking outcomes under study (e.g., early involvement, heavier drinking). In acknowledgment of ICD influences, we counted "irresistible desire to drink" in the more general "any clinical feature of alcohol dependence" construct.

Numerous studies have found that a precocious onset of drinking is associated with higher occurrence of AUD (e.g., Kessler et al., 1997; Kuo et al., 2008; Rothman et al., 2008). Thus, early alcohol involvement was included in the current study as a marker for a "precocious drinking" outcome. The cutoff age of 13 for early-onset alcohol consumption is based on previous literature about preteen initiation of drinking (e.g., see Dube et al., 2006; Hamburger et al., 2008). The cutoff age for early-onset socially maladaptive drinking and early alcohol-dependence clinical features is 23 years old, based on previous literature and a sample-specific consideration of how many cases would be needed to ensure sufficient statistical precision in estimation of the CPP–AUD association before stratified analyses were undertaken, as described below (Brown and Anderson, 1991; Moss et al., 2008; Watson et al., 1997).

*Covariates under study*

The primary covariate under study is a CPP variable derived from a CIDI assessment: "When you were growing up, how often did someone in your household do any of these things to you—often, sometimes, rarely, or never?" Deeds of physical punishment included "pushed, grabbed, or shoved"; "threw something"; or "slapped, hit, or punched." Respondents who answered "often" and "sometimes" were coded as "yes" (1); "rarely" and "never" were coded as "no" (0). There were just 56 respondents who characterized their histories as "often" involving CPP; results described below did not differ appreciably when these 56 respondents were excluded from the analyses.

Other covariates included two important background characteristics that might jointly determine the values of CPP and the later alcohol outcomes under study—namely, paren-

tal AODP and CMB. In this research, parental AODP was formed by combining information from two separate survey items on these problems as known to and reported by the respondent. Because of the low frequency of mother's AODP, we combined maternal and paternal problems into one variable to summarize AODP of both parents. For all variables, "don't know" and "refused" answers were coded as missing values. In the China survey, the CMB was assessed for Part II participants younger than 40 years old ($n$ = 570) via 10 standardized test items designed to tap the DSM-IV criteria for childhood conduct disorder. The temporal sequencing of the CMB and the CPP was not assessed and remains unknown in this research.

*Analysis plan*

In the first steps of analysis, distributions of response variables were examined. Next, logistic regressions were used to assess the strength of association between CPP and drinking-related outcomes adjusting for sex and age. In the estimation of the CPP–AUD association, a special effort was made to ensure appropriate temporal sequencing of earlier CPP and later AUD. Namely, we created a new variable "onset of alcohol problems after 16," to exclude individuals with an onset of problems before age 16. Another effort was to restrict the analytical sample to individuals with limited alcohol experience (<12 drinks per year) before age 16.

The statistical modeling of the CPP–alcohol associations becomes complex when background constructs such as parental AODP and CMB are considered, mainly because the values of the CPP variable, the parental AODP construct, and the CMB construct all might be jointly determined by unobserved background characteristics; also, temporal sequencing of CPP relative to CMB remains uncertain. Under these circumstances, the regression approach is constrained by a plausible violation of standard assumptions about the independent or "exogenous" character of covariates, and alternative approaches are advised. In this context, the approach chosen by our research team is one of stratification, which effectively holds constant the potentially confounding influence of parental AODP and, separately, the possibly confounding influence of CMB, by forming more homogeneous subgroups for estimation of the CPP–alcohol associations under study. Within the subgroup, specifically, that had no history of parental AODP and within the subgroups defined by history of CMB, there can be no confounding of the CPP–outcome association by parental AODP or CMB as measured in this study.

After initial analyze/estimate steps, individuals with and without parental AODP were studied to re-estimate the size of the CPP–alcohol associations with this variable held constant via stratification. Because of the small number of cases (≤5), no stable estimates could be produced based on the subsample of individuals who initiated drinking after 16 years old and those with parental AODP. Thus, regarding individuals who initiated drinking after 16, these postestimation exploratory estimates are presented for those without an AODP-affected parent, which effectively thwarts confounding of the CPP–alcohol association by parental AODP.

A postestimation exploration step also addressed history of CMB in an analogous fashion, via stratification, and here again, there is no way to sort out whether the CMB preceded, followed, or co-occurred with CPP in these cross-sectional data; for this reason, CMB was not modeled as a covariate in the primary regression model. Nonetheless, the analyses involved stratification by presence of CMB, and the CPP–alcohol associations were presented for persons with and without a history of CMB.

In all analyses, sample weights were used to account for differential participation levels, probabilities of sample selection, and Part I/Part II selection probabilities. A Taylor series linearization method was used for variance estimation, with due attention to variation in sample selection probabilities and poststratification adjustment factors. Analyses have been implemented using statistical software Stata Version 10.0 (StataCorp LP, College Station, TX).

## Results

As presented in Table 1, individuals who were in the CPP group were more likely to have experienced precocious drinking and other alcohol outcomes, as compared with those in the non-CPP group. Estimated ORs were moderately strong for the link from earlier CPP to early onset of AUD and to the occurrence of the more general alcohol-dependence clinical features construct (OR = 4.2-8.2), and the ORs were more modest for other drinking outcomes (OR = 2.1-3.6). With covariate adjustment for sex and age, the estimates did not change appreciably; all remained statistically robust at $p < .05$ (Table 1).

Stratified analysis disclosed that for individuals without AODP, the point estimates of ORs remained statistically robust, ranging from 2.1 for early onset of more than minimum drinking to 4.9 for early onset of socially maladaptive drinking (Table 2). Among individuals with a history of parental AODP, the OR point estimates tended to be larger when compared with estimates for individuals without parental AODP. As a result of smaller numbers of cases (<10), however, some of these estimates no longer were statistically significant by conventional standards (i.e., $p > .05$). In specific, statistically significant ($p < .05$) estimates were found for the CPP association with early onset of clinical features of AUD; significant ($p < .05$) associations were found neither for precocious drinking nor for occurrence of socially maladaptive drinking. Possible moderation by AODP was evaluated by the inclusion of a CPP by AODP product term. The observed $p$ values for the CPP–AODP product term never exceeded .12. Among individuals who had limited drinking

TABLE 1. Estimated associations linking earlier childhood physical punishment with alcohol outcomes; data from World Mental Health Surveys–metropolitan China, 2001-2002

| Variable | Childhood physical punishment | *n* of cases | Weighted % | uOR | 95% CI | aOR | 95% CI |
|---|---|---|---|---|---|---|---|
| Precocious drinking | No (*n* = 1,376) | 121 | 9.9 | | | | |
| (drinking onset ≤ 12 years) | Yes (*n* = 235) | 39 | 19.1 | **2.1** | **1.2, 3.9** | **2.0** | **1.1, 3.8** |
| Early more than minimal drinking | No | 194 | 14.3 | | | | |
| (≥12 drinks in 1 year at ≤19 years) | Yes | 66 | 28.9 | **2.4** | **1.6, 3.8** | **2.3** | **1.4, 3.8** |
| Early socially maladaptive drinking | No | 26 | 1.2 | | | | |
| (onset ≤ 23 years) | Yes | 18 | 7.8 | **7.0** | **2.5, 19.1** | **6.2** | **2.2, 17.7** |
| Early dependence problem | No | 12 | 0.3 | | | | |
| (≤23 years) | Yes | 9 | 2.4 | **8.2** | **2.7, 24.8** | **7.7** | **2.5, 23.7** |
| Early any problem | No | 28 | 1.3 | | | | |
| (onset ≤ 23 years) | Yes | 19 | 7.9 | **6.8** | **2.5, 18.3** | **6.0** | **2.1, 16.9** |
| Ever heavier drinking | No | 112 | 7.7 | | | | |
| | Yes | 40 | 19.4 | **2.9** | **1.8, 4.7** | **2.7** | **1.6, 4.7** |
| Ever socially maladaptive drinking | No | 85 | 4.0 | | | | |
| | Yes | 38 | 13.3 | **3.7** | **1.9, 7.2** | **3.4** | **1.7, 6.6** |
| Ever DSM-IV alcohol dependence | No | 27 | 0.7 | | | | |
| | Yes | 15 | 3.2 | **4.9** | **1.9, 12.4** | **4.0** | **1.6, 9.9** |
| Any dependence clinical feature | No | 44 | 1.5 | | | | |
| | Yes | 24 | 5.9 | **4.2** | **1.6, 11.2** | **3.6** | **1.4, 9.6** |
| Onset of problem after age 16 years | No | 81 | 3.9 | | | | |
| | Yes | 35 | 12.7 | **3.6** | **1.8, 7.0** | **3.3** | **1.7, 6.5** |

*Notes:* **Bold** numbers indicate that the estimate was statistically significant ($p < .05$). uOR = unadjusted odds ratio; CI = confidence interval; aOR = adjusted odds ratio, adjusted for sex and age; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.

experience before age 16 years and no parental AODP, the estimates were not as precise, and some of the associations were not statistically robust at the .05 level. Associations linking earlier CPP with later occurrence of AUD, however, were statistically robust ($p < .05$). Moreover, the OR point estimates in this restricted analysis were not appreciably different from estimates based on the entire sample of individuals without parental AODP.

Consistent with our three-step approach, the postestimation data exploration included re-estimation for subgroups large enough to merit their own estimates. The goal in these postestimation steps was to clarify whether we might best understand the observed CPP–alcohol associations as manifestations of other susceptibility-enhancing characteristics. In terms of stratification by the history of CMB, for example, which was possible only for the respondents younger than age 40 years, the CPP–alcohol associations were generally stronger in the stratum with early CMB (Table 3). It is possible that the CPP–alcohol associations are non-null only when there is a background history of co-occurring childhood conduct problems (e.g., as might be required to evoke harsh physical punishment). Nonetheless, when submitted to a

TABLE 2. Estimated associations between childhood physical punishment and alcohol outcomes, stratified by history of parental alcohol and other drug problems; data from World Mental Health Surveys–metropolitan China, 2001-2002

| | Individuals with parental AODP (*n* = 100) | | Individuals without parental AODP (*n* = 1,496) | | Individuals who initiated drinking after age 16 without AODP (*n* = 543) | |
|---|---|---|---|---|---|---|
| Variable | aOR | 95% CI | aOR | 95% CI | aOR | 95% CI |
| Precocious drinking (drinking onset ≤ 12 years) | 3.2 | 0.6, 16.7 | 1.7 | 0.8, 3.4 | 0.9 | 0.3, 2.4 |
| Early more than minimum drinking (at least 12 drinks in a year, ≤19 years) | 3.7 | 0.9, 15.8 | **2.1** | **1.3, 3.6** | 1.3 | 0.6, 2.6 |
| Early socially maladaptive problem (≤23 years) | **7.3** | **1.1, 48.9** | **4.9** | **1.6, 14.9** | **13.5** | **3.8, 48.0** |
| Early dependence problem (≤ 23 years) | —[a] | | **3.6** | **1.0, 12.5** | 3.0 | 0.6, 15.0 |
| Early any problem (≤23 years) | **7.5** | **1.1, 49.5** | **4.8** | **1.6, 14.3** | **11.7** | **3.4, 40.9** |
| Ever heavier drinking | 3.4 | 0.8, 15.0 | **2.3** | **1.3, 4.1** | 1.5 | 0.6, 3.5 |
| Ever socially maladaptive problems | 5.1 | 0.9, 27.1 | **2.9** | **1.5, 5.7** | **2.9** | **1.3, 6.5** |
| Ever dependence | —[a] | | **2.8** | **1.2, 6.4** | **2.4** | **1.04, 5.7** |
| Any dependence clinical feature | —[a] | | **3.3** | **1.3, 8.2** | 2.5 | 0.9, 7.4 |
| Onset of problem after age 16 years | 3.9 | 0.5, 25.5 | **3.0** | **1.5, 5.9** | **2.9** | **1.3, 6.6** |

*Notes:* **Bold** numbers indicate that the estimate was statistically significant ($p < .05$). All models held sex and age constant. AODP = parental alcohol or other drug problems; aOR = adjusted odds ratio, adjusted for sex and age; CI = confidence interval. [a]Cannot be estimated because of too few cases.

TABLE 3. Estimated associations between childhood physical punishment and alcohol outcomes, stratified by history of childhood misbehavior (CMB); data from World Mental Health Surveys–metropolitan China, 2001-2002

| | Those without CMB ($n$ = 488) | | Those with CMB ($n$ = 82) | |
|---|---|---|---|---|
| Variable | OR | 95% CI | OR | 95% CI |
| Precocious drinking (≤12 years) | 1.7 | 0.6, 4.8 | 1.0 | 0.3, 3.4 |
| Early more than minimum drinking | 1.9 | 0.8, 4.5 | 3.6 | 1.0, 13.1 |
| Early socially maladaptive problem | 2.3 | 0.4, 12.3 | 5.3 | 0.8, 34.9 |
| Early dependence problem | 3.4 | 0.6, 19.8 | **10.2** | **1.8, 58.7** |
| Early any problem | 2.2 | 0.5, 10.0 | 5.3 | 0.8, 34.9 |
| Ever heavier drinking | 1.6 | 0.4, 6.0 | 3.0 | 0.9, 10.0 |
| Ever socially maladaptive problems | 1.4 | 0.4, 5.3 | 4.2 | 0.9, 19.4 |
| Ever dependence | 0.9 | 0.2, 5.0 | **6.2** | **1.2, 31.2** |
| Any dependence clinical feature | 2.0 | 0.4, 10.2 | **8.5** | **1.9, 37.9** |
| Onset of problem after age 16 years | 1.4 | 0.4, 5.4 | 4.1 | 0.9, 18.7 |

*Notes:* **Bold** numbers indicate that the estimate was statistically significant ($p < .05$). OR = odds ratio; CI = confidence interval.

formal statistical evaluation of whether more than one slope estimate is required to summarize the CPP–alcohol associations across strata defined by CMB history, the observed $p$ values for the CPP–CMB product term never exceeded 0.12 (data not shown). Statistical power to detect modest associations involving the CPP–CMB combination and later alcohol outcomes was low, and the resulting absence of evidence on statistical interaction cannot be considered definitive. The available evidence indicates, however, that the OR based on "common regression slope" estimates in Table 1 serve well to summarize the CPP–alcohol associations under study for individuals with a history of CMB and for those without a history of CMB, and there is no need to produce separate stratum-specific slopes in this sample. (To reject the more parsimonious "common slope" model, the CPP–CMB product term should have been non-null.)

## Discussion

In this study, predicted associations between CPP and most drinking outcomes were found. After taking parental AODP into account via stratification, many associations remained sturdy, including estimates based on participants without a history of parental AODP. As for history of CMB, the estimated associations for CPP–alcohol outcomes were visibly stronger for individuals with a history of CMB; nonetheless, the estimated OR reported in Table 1, based on a "common slope" model, seem to serve well to summarize the CPP–alcohol associations for both subgroups with and without a history of childhood conduct problems.

### *Strengths and limitations*

The major contributions of this study are as follows. First, it provides evidence supporting the plausibility of a suspected causal association linking earlier CPP with later alcohol outcomes in the Chinese context. Our OR estimates now provide an evidentiary basis for the design of future prospective and longitudinal studies of CPP as a potential cause of drinking-related problems in China. Second, our estimates are based on a community sample. Possible biases in estimation were minimized by efforts to adjust for clustering and selection probabilities, as well as nonresponse patterns. Thus, results are applicable to the household-dwelling adults living in the two Chinese cities. Third, using the stratification strategy, we introduced a way to address possible confounding influences from two sources: parental alcohol and other drug problems, and CMB that might evoke harsh physical punishment.

Several limitations of the study merit attention. With respect to the assessment of outcomes, our major concern is the self-report character of the survey data, but in large-scale epidemiological survey research, there are few alternatives to the self-report approach when the research task concerns cumulative occurrence and lifetime history of alcohol outcomes. With respect to the alcohol-dependence outcomes (but not with respect to the many other alcohol outcomes under study), the "gated" approach described in the Method section deserves mention as a limitation, as our research group has previously examined thoroughly (e.g., Degenhardt et al., 2007). This type of measurement assumption is quite common in contemporary alcohol-dependence research. Available evidence suggests that the WMH-CIDI "gating approach" may yield attenuated estimates of the population prevalence of alcohol dependence as defined in terms of DSM-IV standards, to the extent that the "gate" based on history of socially maladaptive and hazard-laden drinking may exclude some mildly impaired dependent individuals who have experienced no alcohol-associated social maladaptation (e.g., Degenhardt et al., 2007; Hasin and Grant,

2004). One might speculate that this "gating approach" also differentially filters out alcohol-dependent persons who have no background history of CPP, although we are aware of no evidence on this form of bias. If this bias is present, however, then this study's estimates on the several alcohol-dependence outcomes might be affected; this would not be the case for precocious drinking as an outcome or the other alcohol outcomes under study that do not reference the WMH-CIDI alcohol-dependence items. In summary, in our general research approach, with an assessment of an array of alcohol outcomes, we find a general pattern of CPP–alcohol associations, even if open questions remain about the CPP–alcohol dependence associations.

We also note that duration of drinking might be important in future research on these relationships. We do not characterize our lack of data on duration of drinking as a limitation, because duration of drinking actually might be influenced by the early history of CPP. As such, we should not control for duration of drinking as a confounding variable. At best, we might be able to stratify on this variable (if it had been measured), or perhaps it might be modeled as a correlated outcome variable in a multiple equations estimation system. This is a possibility best examined in the context of future longitudinal research described below.

With respect to the assessment of covariates, our major concern involves the assessment of parental AODP, here based on self-report from the respondent, not that of parents or other sources. No clear definition of "drinking problems" or "drug problems" was provided to the respondent. Thus, this assessment may be subjective and fairly coarse-grained relative to the fine-grained and more objective alternatives. Several studies have probed this question. These studies suggested that information about parental drinking problems collected from offspring can be pertinent and accurate, and offspring report may be the only viable method of assessing parental problems, given that alternatives are constrained in cross-sectional household survey research (Prescott and Kendler, 1999; Rhea et al., 1993; Sher and Descutner, 1986). It is not clear, however, if this conclusion holds in the Chinese context. (We note that many of the survey respondents had parents living in rural areas far distant from the study sites in Beijing and Shanghai, as a result of massive post-1970s migration of families from rural China toward metropolitan areas.)

Childhood adversity (e.g., parental divorce, tension in the household) and CPP tend to co-occur (Anda et al., 2002; Briere and Elliott, 2003). Because of the observational nature of this study, it is not clear that the observed association is a presentation of a generally noxious childhood family environment, rather than CPP per se. Future studies are needed to clarify this possibility. Also, because of the observational nature of the study, the observed CPP–alcohol associations might be confounded by other unmeasured variables. Nonetheless, we controlled for what are arguably the two most obvious confounding influences: parental alcohol and other drug problems, and CMB.

We also must admit that cross-sectional studies are fraught with limitations traced to differential recall and survival bias. It is possible, for example, that individuals who suffer from drinking problems may search their memories more thoroughly for experiences of CPP. However, previous studies have found good to excellent validity and reliability of assessment of CPP (Bremner et al., 2007; Hyman et al., 2005; Walsh et al., 2008). We attempted to address this problem by grouping 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. It is possible that the CPP–alcohol association was presented in the "often" group only. Unfortunately, the small number of individuals in the "often" group ($n = 50$) precluded any precise estimate. Exploratory inspection, however, found point estimates for the "often" group were not distant from the "sometimes" group. We also note that individuals who suffered from CPP may be less likely to survive, especially in cases of severe forms of abuse (Arias et al., 2003). As such, in a series of birth cohorts, some people with CPP experience may not be captured in any cross-sectional study (nor would they be captured in longitudinal research of childhood samples with follow-up into adulthood). This factor may bias estimates toward the null because previous studies have found that severe forms of abuse are strongly associated with heavy drinking and alcohol dependence (Simpson and Miller, 2002; Widom et al., 2007).

Temporal sequencing is another usual concern for observed associations from retrospective studies with cross-sectional sampling. It is possible that the occurrence of precocious drinking and related problems could be a manifestation of a continuity of childhood conduct problems. These problems may be the reason that parents have physically punished the offspring. To explore this question in a postestimation exploratory analysis, we restricted the subsample to individuals with no more than minimal drinking experience before 16 years old. Estimates remained sturdy.

As it happens, retrospective studies with good internal and external validity may well have advantages over prospective studies when the exposure variable has a discrete nature and can be placed early in time, well before the occurrence of the outcome. Indeed, a study from the Netherlands has found that the estimated associations that link early parenting deficiency (lack of care and overprotection) and cumulative occurrence of AUD lifetime history (at baseline cross-sectionally) were essentially the same as estimated associations that linked early parenting deficiency with becoming an incident case of AUD as observed during prospective follow-up (Overbeek et al., 2007).

*CPP–AUD association*

Despite limitations such as these, our findings are of interest. The results correspond to some previous findings in the more established market economies of the U.S. and Canadian populations, where statistically robust modest to moderately strong associations between CPP and drinking problems have been found (Holmes and Robins, 1987, 1988; Kessler et al., 1997; MacMillan et al., 1999, 2001). The results are in line with evidence from animal models showing chronic exposure to stressful events during early life linked to stress hormone levels, with associated changes in adult behaviors (Heath, 1972; Teicher et al., 2002). Findings from this study are different from previous studies with null associations (Hughes et al., 2007; Koss et al., 2003; Libby et al., 2004; Mullings et al., 2004; Young et al., 2006). The divergence may lie in a clear difference in the study population and in the statistical approach. Here we argue that our results are pertinent because the sampling was of noninstitutionalized household-dwelling adults, where the majority of alcohol-related disease burden resides. Using the stratification approach, we found a possible causal relationship between CPP and drinking outcomes, independent from parental drinking problems. Although results from this study supported the common slope for both AODP and non-AODP groups, it is still of importance for future study to assess the possible violation of the "independent covariates" assumption when using GLM for estimation.

Previous literature has found that CMB is a good predictor of later alcohol dependence with shared environmental causes (e.g., Knop et al., 2009). CMB may evoke recurrent inept harsh CPP, in a coercive process that drives CMB forward and may contribute to precocious drinking and other alcohol outcomes (Fergusson et al., 2008). As such, CMB is not a mere confounder in the CPP–alcohol associations under study. Indeed, CMB and physical abuse may be conceptualized as interdependent manifestations of background vulnerabilities for alcohol outcomes (Brown and Anderson, 1991; Spencer et al., 2005). CMB may well serve as a mediator in pathways from CPP to drinking outcomes; controlling CMB as a mere covariate may bias CPP–alcohol estimates toward the null. In our cross-sectional data, we cannot sort out this sequence, but because our preliminary results are based on CMB stratification and in a model with CPP–CMB product terms, there is still evidence of a CPP–alcohol association for several drinking outcomes, especially those related to alcohol dependence, as presented in common slope estimates (Table 1) that seem to serve well to summarize the CPP–alcohol-dependence relationships for individuals with and without the CMB history in this study sample. In a complementary article, we have sharpened the alcohol-dependence focus of our investigation. In that work, we have taken an alcohol-dependence latent trait dimensional approach, finding that an individual's position on the alcohol-dependence dimension depends to some degree on CPP history specifically, even when a structural equations model "multiple indicators, multiple causes" (MIMIC) approach is used to express CPP as just one facet of an underlying "noxious family environment" construct (Cheng et al., 2010).

Notwithstanding these concerns about its cross-sectional character, this study's evidence supports the idea of a tangible modest moderately strong association linking earlier CPP with later alcohol outcomes in China. Based on cross-sectional findings, a next step might be replication with an improved design that attends to temporal sequencing of CPP, CMB, and parental alcohol and other drug problems, perhaps with improved assessments as well. The systematic replication approach is a typical step in epidemiological investigation when initial observational and retrospective research highlights an interesting and plausible causal hypothesis linking earlier exposure with later disease outcomes. Nonetheless, in present contexts, it might be more prudent, cost-efficient, and informative to design the next cross-sectional study as a foundational baseline assessment for a more elaborated prospective and longitudinal study design, with due attention to developmental issues required to probe into the suspected causal association more completely. In China, the prospective and longitudinal study approach can be used to secure a more fine-grained and broad assessment of co-occurring childhood adversities, as well as risk-enhancing or protective conditions and processes, as might modify the now-observed CPP–AUD association. In this fashion, the "natural history" of the observational research process might be accelerated toward future experimental trials of parenting education programs and other interventions of value in public health practice that seek to reduce the population prevalence and disease burdens associated with alcohol dependence and associated drinking outcomes.

## Acknowledgment

We thank the WMH staff for assistance with instrumentation, fieldwork, and data analysis.

## References

Afifi, T. O., Brownridge, D. A., Cox, B. J., & Sareen, J. (2006). Physical punishment, childhood abuse and psychiatric disorders. *Child Abuse & Neglect, 30,* 1093-1103.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anda, R. F., Whitfield, C. L., Felitti, V. J., Chapman, D., Edwards, V. J., Dube, S. R., & Williamson, D. F. (2002). Adverse childhood experiences, alcoholic parents, and later risk of alcoholism and depression. *Psychiatric Services, 53,* 1001-1009.

Arias, E., MacDorman, M. F., Strobino, D. M., & Guyer, B. (2003). Annual summary of vital statistics—2002. *Pediatrics, 112,* 1215-1230.

Berg, G. D., & Mansley, E. C. (2004). Endogeneity bias in the absence of unobserved heterogeneity. *Annals of Epidemiology, 14,* 561-565.

Bremner, J. D., Bolus, R., & Mayer, E. A. (2007). Psychometric properties

of the Early Trauma Inventory-Self Report. *The Journal of Nervous and Mental Disease, 195,* 211-218.

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., . . . Charney, D. S. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse—a preliminary report. *Biological Psychiatry, 41,* 23-32.

Briere, J., & Elliott, D. M. (2003). Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. *Child Abuse & Neglect, 27,* 1205-1222.

Briscoe, J., Akin, J., & Guilkey, D. (1990). People are not passive acceptors of threats to health: Endogeneity and its consequences. *International Journal of Epidemiology, 19,* 147-153.

Brown, G. R., & Anderson, B. (1991). Psychiatric morbidity in adult inpatients with childhood histories of sexual and physical abuse. *The American Journal of Psychiatry, 148,* 55-61.

Caldji, C., Diorio, J., & Meaney, M. J. (2003). Variations in maternal care alter GABAA receptor subunit expression in brain regions associated with fear. *Neuropsychopharmacology, 28,* 1950-1959.

Chen, J., Dunne, M. P., & Han, P. (2006). Child sexual abuse in Henan Province, China: Associations with sadness, suicidality, and risk behaviors among adolescent girls. *Journal of Adolescent Health, 38,* 544-549.

Cheng, H. G., Anthony, J. C., & Huang, Y. (2010). Harsh physical punishment as a specific childhood adversity linked to adult drinking consequences: Evidence from China. *Addiction, 105,* 2097-2105.

Cicchetti, D., & Rogosch, F. A. (2007). Personality, adrenal steroid hormones, and resilience in maltreated children: A multilevel perspective. *Development and Psychopathology, 19,* 787-809.

Clark, D. B., Lesnick, L., & Hegedus, A. M. (1997). Traumas and other adverse life events in adolescents with alcohol abuse and dependence. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 1744-1751.

Degenhardt, L., Bohnert, K. M., & Anthony, J. C. (2007). Case ascertainment of alcohol dependence in general population surveys: 'gated' versus 'ungated' approaches. *International Journal of Methods in Psychiatric Research, 16,* 111-123.

Degenhardt, L., Chiu, W.-T., Sampson, N., Kessler, R. C., Anthony, J. C., Angermeyer, M., . . . Wells, J. E. (2008). Toward a global view of alcohol, tobacco, cannabis, and cocaine use: Findings from the WHO World Mental Health Surveys. *PLoS Medicine, 5* (7), e141.

Dick, D. M., & Bierut, L. J. (2006). The genetics of alcohol dependence. *Current Psychiatry Reports, 8,* 151-157.

DiLalla, L. F., & Gottesman, I. I. (1991). Biological and genetic contributors to violence—Widom's untold tale. *Psychological Bulletin, 109,* 125-129; discussion 130-132.

Downs, W. R., Capshew, T., & Rindels, B. (2004). Relationships between adult women's alcohol problems and their childhood experiences of parental violence and psychological aggression. *Journal of Studies on Alcohol, 65,* 336-344.

Dube, S. R., Anda, R. F., Felitti, V. J., Croft, J. B., Edwards, V. J., & Giles, W. H. (2001). Growing up with parental alcohol abuse: Exposure to childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect, 25,* 1627-1640.

Dube, S. R., Miller, J. W., Brown, D. W., Giles, W. H., Felitti, V. J., Dong, M., & Anda, R. F. (2006). Adverse childhood experiences and the association with ever using alcohol and initiating alcohol use during adolescence. *Journal of Adolescent Health, 38,* 444.e1-444.e10.

Engle, R. F., Hendry, D. F., & Richard, J. F. (1983). Exogeneity. *Econometrica,* 51, 277-304.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008). Exposure to childhood sexual and physical abuse and adjustment in early adulthood. *Child Abuse & Neglect, 32,* 607-619.

Galaif, E. R., Stein, J. A., Newcomb, M. D., & Bernstein, D. P. (2001). Gender differences in the prediction of problem alcohol use in adulthood: Exploring the influence of family factors and childhood maltreatment. *Journal of Studies on Alcohol, 62,* 486-493.

Hamburger, M. E., Leeb, R. T., & Swahn, M. H. (2008). Childhood maltreatment and early alcohol use among high-risk adolescents. *Journal of Studies on Alcohol and Drugs, 69,* 291-295.

Hasin, D. S., & Grant, B. F. (2004). The co-occurrence of DSM-IV alcohol abuse in DSM-IV alcohol dependence: Results of the National Epidemiologic Survey on Alcohol and Related Conditions on heterogeneity that differ by population subgroup. *Archives of General Psychiatry, 61,* 891-896.

Heath, R. G. (1972). Electroencephalographic studies in isolation-raised monkeys with behavioral impairment. *Diseases of the Nervous System, 33,* 157-163.

Holmes, S. J., & Robins, L. N. (1987). The influence of childhood disciplinary experience on the development of alcoholism and depression. *Journal of Child Psychology and Psychiatry, 28,* 399-415.

Holmes, S. J., & Robins, L. N. (1988). The role of parental disciplinary practices in the development of depression and alcoholism. *Psychiatry, 51,* 24-36.

Horwitz, A. V., Widom, C. S., McLaughlin, J., & White, H. R. (2001). The impact of childhood abuse and neglect on adult mental health: A prospective study. *Journal of Health and Social Behavior, 42,* 184-201.

Hughes, T. L., Johnson, T. P., Wilsnack, S. C., & Szalacha, L. A. (2007). Childhood risk factors for alcohol abuse and psychological distress among adult lesbians. *Child Abuse & Neglect, 31,* 769-789.

Hyman, S. M., Garcia, M., Kemp, K., Mazure, C. M., & Sinha, R. (2005). A gender specific psychometric analysis of the early trauma inventory short form in cocaine dependent adults. *Addictive Behaviors, 30,* 847-852.

Jacob, T., & Johnson, S. (1997). Parenting influences on the development of alcohol abuse and dependence. *Alcohol Health and Research World, 21,* 204-209.

Jasinski, J. L., Williams, L. M., & Siegel, J. (2000). Childhood physical and sexual abuse as risk factors for heavy drinking among African-American women: A prospective study. *Child Abuse & Neglect, 24,* 1061-1071.

Kendler, K. S., Sheth, K., Gardner, C. O., & Prescott, C. A. (2002). Childhood parental loss and risk for first-onset of major depression and alcohol dependence: The time-decay of risk and sex differences. *Psychological Medicine, 32,* 1187-1194.

Kessler, R. C., Davis, C. G., & Kendler, K. S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine, 27,* 1101-1119.

Kessler, R. C., & Ustun, T. B. (2004). The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). *International Journal of Methods in Psychiatric Research, 13,* 93-121.

Knop, J., Penick, E. C., Nickel, E. J., Mortensen, E. L., Sullivan, M. A., Murtaza, S., . . . Gabrielli, W. F. (2009). Childhood ADHD and conduct disorder as independent predictors of male alcohol dependence at age 40. *Journal of Studies on Alcohol and Drugs, 70,* 169-177.

Koss, M. P., Yuan, N. P., Dightman, D., Prince, R. J., Polacca, M., Sanderson, B., & Goldman, D. (2003). Adverse childhood exposures and alcohol dependence among seven Native American tribes. *American Journal of Preventive Medicine, 25,* 238-244.

Kunitz, S. J., Levy, J. E., McCloskey, J., & Gabriel, K. R. (1998). Alcohol dependence and domestic violence as sequelae of abuse and conduct disorder in childhood. *Child Abuse & Neglect, 22,* 1079-1091.

Kuo, P. H., Aggen, S. H., Prescott, C. A., Kendler, K. S., & Neale, M. C. (2008). Using a factor mixture modeling approach in alcohol dependence in a general population sample. *Drug and Alcohol Dependence, 98,* 105-114.

Lau, J. T., Chan, K. K., Lam, P. K., Choi, P. Y., & Lai, K. Y. (2003). Psychological correlates of physical abuse in Hong Kong Chinese adolescents. *Child Abuse & Neglect, 27,* 63-75.

Libby, A. M., Orton, H. D., Novins, D. K., Spicer, P., Buchwald, D., Beals, J., . . . the AI-SUPERPFP Team. (2004). Childhood physical and sexual abuse and subsequent alcohol and drug use disorders in two American-Indian tribes. *Journal of Studies on Alcohol, 65,* 74-83.

Liu, I. C., Blacker, D. L., Xu, R., Fitzmaurice, G., Lyons, M. J., & Tsuang, M. T. (2004). Genetic and environmental contributions to the development of alcohol dependence in male twins. *Archives of General Psychiatry, 61,* 897-903.

Lloyd, J. J., & Anthony, J. C. (2003). Hanging out with the wrong crowd: How much difference can parents make in an urban environment? *Journal of Urban Health, 80,* 383-399.

Luo, Y., Parish, W. L., & Laumann, E. O. (2008). A population-based study of childhood sexual contact in China: Prevalence and long-term consequences. *Child Abuse & Neglect, 32,* 721-731.

MacMillan, H. L., Boyle, M. H., Wong, M. Y., Duku, E. K., Fleming, J. E., & Walsh, C. A. (1999). Slapping and spanking in childhood and its association with lifetime prevalence of psychiatric disorders in a general population sample. *CMAJ: Canadian Medical Association Journal, 161,* 805-809.

MacMillan, H. L., Fleming, J. E., Streiner, D. L., Lin, E., Boyle, M. H., Jamieson, E., . . . Beardslee, W. R. (2001). Childhood abuse and lifetime psychopathology in a community sample. *The American Journal of Psychiatry, 158,* 1878-1883.

Moss, H. B., Chen, C. M., & Yi, H. Y. (2008). DSM-IV criteria endorsement patterns in alcohol dependence: Relationship to severity. *Alcoholism: Clinical and Experimental Research, 32,* 306-313.

Mullings, J. L., Hartley, D. J., & Marquart, J. W. (2004). Exploring the relationship between alcohol use, childhood maltreatment, and treatment needs among female prisoners. *Substance Use & Misuse, 39,* 277-305.

National Institute on Alcohol Abuse and Alcoholism. (2004). NIAAA Council Approves Definition of Binge Drinking (*NIAAA Newletter,* winter, No. 3, NIH Publication No. 04-5346). Bethesda, MD: Author.

Oscar-Berman, M., & Marinkovic, K. (2007). Alcohol: Effects on neurobehavioral functions and the brain. *Neuropsychology Review, 17,* 239-257.

Overbeek, G., ten Have, M., Vollebergh, W., & de Graaf, R. (2007). Parental lack of care and overprotection: Longitudinal associations with DSM-III-R disorders. *Social Psychiatry and Psychiatric Epidemiology, 42,* 87-93.

Prescott, C. A., & Kendler, K. S. (1999). Age at first drink and risk for alcoholism: A noncausal association. *Alcoholism: Clinical and Experimental Research, 23,* 101-107.

Rhea, S. A., Nagoshi, C. T., & Wilson, J. R. (1993). Reliability of sibling reports on parental drinking behaviors. *Journal of Studies on Alcohol, 54,* 80-84.

Ross, C. A., Keyes, B. B., Xiao, Z., Yan, H., Wang, Z., Zou, Z., . . . Zhang, H. (2005). Childhood physical and sexual abuse in China. *Journal of Child Sexual Abuse, 14,* 115-126.

Rothman, E. F., Edwards, E. M., Heeren, T., & Hingson, R. W. (2008). Adverse childhood experiences predict earlier age of drinking onset: Results from a representative US sample of current or former drinkers. *Pediatrics, 122,* e298-e304.

Sanchez, M. M., Hearn, E. F., Do, D., Rilling, J. K., & Herndon, J. G. (1998). Differential rearing affects corpus callosum size and cognitive function of rhesus monkeys. *Brain Research, 812,* 38-49.

Schuckit, M. A., & Smith, T. L. (2006). An evaluation of the level of response to alcohol, externalizing symptoms, and depressive symptoms as predictors of alcoholism. *Journal of Studies on Alcohol, 67,* 215-227.

Shen, M., Chai, J., Yang, B., Huang, S., & Yan, J. (2003). *The World Mental Health Survey in China: An overview of design and field procedures* (p. 9). Beijing, China: Research Center for Contemporary China, Peking University.

Sher, K. J., & Descutner, C. (1986). Reports of paternal alcoholism: Reliability across siblings. *Addictive Behaviors, 11,* 25-30.

Sher, K. J., Gershuny, B. S., Peterson, L., & Raskin, G. (1997). The role of childhood stressors in the intergenerational transmission of alcohol use disorders. *Journal of Studies on Alcohol, 58,* 414-427.

Simpson, T. L., & Miller, W. R. (2002). Concomitance between childhood sexual and physical abuse and substance use problems: A review. *Clinical Psychology Review, 22,* 27-77.

Spencer, N., Devereux, E., Wallace, A., Sundrum, R., Shenoy, M., Bacchus, C., & Logan, S. (2005). Disabling conditions and registration for child abuse and neglect: A population-based study. *Pediatrics, 116,* 609-613.

Swett, C., Jr., Cohen, C., Surrey, J., Compaine, A., & Chavez, R. (1991). High rates of alcohol use and history of physical and sexual abuse among women outpatients. *The American Journal of Drug and Alcohol Abuse, 17,* 49-60.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., & Navalta, C. P. (2002). Developmental neurobiology of childhood stress and trauma. *Psychiatric Clinics of North America, 25,* 397-426, vii-viii.

Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: Are results from human and animal studies comparable? *Annals of the New York Academy of Sciences, 1071,* 313-323.

Trent, L., Stander, V., Thomsen, C., & Merrill, L. (2007). Alcohol abuse among U.S. Navy recruits who were maltreated in childhood. *Alcohol and Alcoholism, 42,* 370-375.

Walsh, C. A., Macmillan, H. L., Trocmé, N., Jamieson, E., & Boyle, M. H. (2008). Measurement of victimization in adolescence: Development and validation of the Childhood Experiences of Violence Questionnaire. *Child Abuse & Neglect, 32,* 1037-1057.

Watson, C. G., Hancock, M., Gearhart, L. P., Malovrh, P., Mendez, C., & Raden, M. (1997). A comparison of the symptoms associated with early and late onset alcohol dependence. *The Journal of Nervous and Mental Disease, 185,* 507-509.

Widom, C. S., & Hiller-Sturmhofel, S. (2001). Alcohol abuse as a risk factor for and consequence of child abuse. *Alcohol Research & Health, 25,* 52-57.

Widom, C. S., White, H. R., Czaja, S. J., & Marmorstein, N. R. (2007). Long-term effects of child abuse and neglect on alcohol use and excessive drinking in middle adulthood. *Journal of Studies on Alcohol and Drugs, 68,* 317-326.

World Health Organization. (2004). *WHO Global Status Report on Alcohol 2004: Country Profiles-China.* Geneva, Switzerland: Author. Retrieved from http://www.who.int/substance_abuse/publications/en/china.pdf

World Health Organization. (1994). *The International Statistical Classification of Diseases and Related Health Problems 10th Revision* (*ICD-10*). Geneva, Switzerland.

Young, S. Y., Hansen, C. J., Gibson, R. L., & Ryan, M. A. (2006). Risky alcohol use, age at onset of drinking, and adverse childhood experiences in young men entering the US Marine Corps. *Archives of Pediatrics & Adolescent Medicine, 160,* 1207-1214.