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Harsh physical punishment as a specific childhood adversity linked to adult drinking consequences: Evidence from China

Hui G. Cheng^{1,2}, James C. Anthony², and Yueqin Huang¹

¹ Peking University Institute of Mental Health, Key Laboratory of Ministry of Health (Peking University)

² Department of Epidemiology, Michigan State University, B601 West Fee Hall, East Lansing, Michigan 48824, USA

Abstract

Aims—The aim of the current study is to estimate the association between childhood physical punishment (CPP) and level of alcohol use disorder (AUD), using two different approaches to take other childhood adversities into account.

Design and Setting—Population survey using face-to-face interviews to a representative sample of non-institutionalized adult residents of Beijing and Shanghai, China.

Participants—A total of 5201 participants aged 18 to 70 years old.

Measurements—A version of the World Mental Health Composite International Diagnostic Interview was used. Standardized assessments covered early life experiences of childhood physical punishment, other childhood adversities, parental drinking problems, childhood conduct problems, and clinical features of AUD.

Findings—A robust association linking CPP and level of AUD was found, holding other childhood adversities constant (probit coefficient=0.70, 95% CI=1.40, 1.00) via covariate terms in Structural Equations Modeling. Furthermore, there was evidence that CPP might exert an additional influence on level of AUD over and above a generally noxious family environment (probit coefficient=0.20, 95% CI=0.02, 0.38).

Conclusion—There appears to be a robust association between reports of harsh punishment in childhood and alcohol dependence in adulthood adjusting for a range of possible confounding factors. Whether the association is causal or whether both are related to a common underlying factor or recall bias needs to be investigated further.

1. Introduction

Alcohol use disorders (AUD) are among the most commonly occurring mental disorders in China, as in many other countries of the world, and are associated with substantial disease burden (1–3). Twin studies suggest that environmental factors are prominent in the etiology of AUD (4). As such, the identification of modifiable AUD risk-associated environmental factors may help foster prevention strategies to reduce the occurrence of AUD and the associated disease burdens.

In a previous work, our research team described a hypothesized association that links harsh childhood physical punishment (CPP) with later drinking outcomes such as AUD, as well as supportive theory and findings from pre-clinical experiments (5). The CPP-AUD hypothesis

is especially compelling in the context of prevention research for metropolitan China, where alcoholic beverages are widely available throughout the cities, when disposable income levels are increasing, and since parenting practices are potentially modifiable environmental processes (e.g., via parent education and collective child guidance activities). Moreover, in prior research, we and others have found statistically robust CPP-associated excess occurrence of adverse drinking outcomes, even with parental history of drinking problems held constant (5–7). Nevertheless, harsh physical punishment sometimes can be just one facet of childhood adversities, possibly due to a more generally noxious family environment. These childhood adversities include parental divorce, chronic tension in the household, observed inter-parent violence, and child neglect. These adversities also have been associated with the occurrence of AUD (6,8–12). Since these adversities are inter-correlated, it is reasonable to argue that the estimate for a single childhood adversity without considering others might be artificial (13). Given this possibility, some studies have controlled for other childhood adversities and found no specific CPP-AUD associations (8,14–16), while some other studies found the opposite (7,17).

There are several important methodological issues to consider. First, most of the previous reports on the CPP-alcohol relationship were not epidemiological in nature; they lacked predefined source populations. The result is uncertainty about whether the findings can be applied to community populations, not to mention populations in China, one of the two most populous countries of the world. Second, some studies have used the single-equation generalized linear model (GLM), in which AUD was regressed on various childhood adversities simultaneously (7,8,16,18–20). Given that childhood adversities are likely to be inter-correlated and may be jointly determined (13), the GLM assumption of independent covariates may have been violated; the result might have been biased or inconsistent (21–23). This problem has been further complicated by the inclusion of family history of drinking problems in the GLM, which can create a problem known as endogeneity, in that the harsh physical punishment and the family history might be jointly determined. Third, to the best of our knowledge, no study has explored whether CPP, as a specific adversity, is related to the occurrence of AUD over and above a more general influence of a noxious family environment. As noted above, CPP is potentially modifiable (unlike some of the other adversities). Hence, research on CPP-specific effects on alcohol outcomes may help guide the design of future AUD prevention strategies.

Given this background, the current study aimed (1) to estimate CPP-alcohol relationships, taking into account parental drinking problems and other childhood adversities, and (2) to explore whether CPP is specifically associated with these outcomes after controlling for a general noxious family environment during childhood. The research approach involves multivariate structural equations and measurement models for cross-sectionally gathered data with clear temporal sequencing from earlier childhood adversities to later adult-onset alcohol outcomes. This approach has been used in prior research on parenting and health outcomes, as in (24–26). That is, efforts are made to constrain the possibility that early-onset (childhood) drinking problems have occurred before harsh physical punishment.

2. METHODS

Prior publications give detailed methods overviews (5,27). Here we provide a brief description.

2.1 Study design and sample selection

Data were collected for the cross-sectional World Mental Health Survey-metropolitan China initiative (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 75% in both cities (28).

2.2 Assessments

A Chinese version of the World Health Organization World Mental Health Composite International Diagnostic Interview was used in the WMH-mC (WMH-CIDI; 29). It is a fully structured diagnostic assessment that measures clinical features of psychiatric illnesses according to criteria in both the International Classification of Disease and Related Health Problems 10th Revision (ICD-10) and the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV).

The WMH-CIDI has two parts and starts with a core diagnostic assessment (Part I) for all 5201 respondents in China. Part II includes questions about suspected correlates or determinants as well as additional topics, including childhood adversities. In China, Part II was administered to all respondents with a history of past or recent core mental disorders assessed in Part I, plus a probability sample drawn from the rest of respondents, yielding an analysis sample of 1628 respondents for this research. Sampling weights have been used to take probability sampling and non-response into account, as explained in section 2.5.

2.3 Response variables

In the current study, the main response variable was a latent “level of AUD” variable derived via confirmatory factor analysis (CFA) and measured by CIDI items on clinical features of ICD-10 and DSM-IV defined AUD, including ICD-10 harmful use of alcohol (or DSM-IV alcohol abuse) and alcohol dependence. Thirteen clinical features associated with AUD were included: “tolerance”, “withdrawal”, “unable to cut 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”, “irresistible desire to drink”, “responsibility interference”, “social problems”, “continue to drinking despite social problems”, “hazard-laden drinking”, and “legal problems”. The CFA with all 13 items did not converge due to collinearity between ‘unable to cut down’ and other items. Thus, the ‘unable to cut down’ item was dropped from the main analysis in this study.

2.4 Covariates

The main covariate of this study was “childhood physical punishment” (CPP), as measured via the CIDI item: “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.” The original CPP variable had four categories: “often,” “sometimes,” “rarely,” and “never.” Nonetheless, the small number of cases in each category precluded precise estimates. Thus, the original variable was dichotomized. Respondents who answered “often” and “sometimes” were coded as “yes”; “rarely” and “never” were coded as “no”. Therefore, the CPP variable represented the 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, ever being beaten badly by parents, and a lot of tension in the household. The being beaten badly by parents as a child variable was coded as ‘yes’ when the respondent

had such experience, even once, and there was no frequency measures for this variable. Authors consider that in China the experience of being beaten badly by parents was rare, usually once or twice, during childhood, and often in certain circumstances e.g. when children did something potentially dangerous. Thus, this variable was included as one of the other childhood adversities rather than the CPP variable, the main interest of this study.

In this report, we illustrate two approaches that can be used to inspect whether there is a specific CPP-AUD association: (1) inclusion of a vector of the aforementioned inter-correlated childhood adversities as covariates, and (2) creation of a single “noxious family environment” latent dimension, measured by items on all adversities (including the CPP item).

2.5 Analysis Plan

In the first steps of our standard ‘explore, estimate, explore’ approach, the marginal frequencies of childhood adversities and clinical features of AUD were studied. Next, the CFA for a single dimension was completed. The first step of estimation involved the use of structural equation modeling (SEM) with the ‘level of AUD’ latent variable, with each childhood adversity included as a covariate (see Figure 1 for a description). For ease of discussion, we label this model ‘multiple adversities SEM.’ In this SEM, the AUD measurement model was based on 12 clinical features of AUD. This latent ‘level of AUD’ variable was regressed on childhood adversities, allowing them to be inter-correlated. This approach produced an estimate of the CPP-AUD relationship, with other childhood adversities held constant via covariate terms.

The next approach involved use of the ‘noxious family environment’ dimension, as measured by all childhood adversity items, with CPP allowed to have a direct influence on the latent ‘AUD’ level, over and above the influence of the ‘noxious family environment’ variable (see Figure 2 for a diagram). That is, in this model, a regression slope estimate was produced for the direct path drawn from the latent ‘noxious family environment’ to the latent ‘level of AUD’ variable; at the same time, another regression slope was produced for the direct path from CPP to the latent ‘level of AUD’ variable. For ease of discussion, we name this model ‘CPP-specific SEM.’ In both approaches, a term for ‘parental alcohol and drug problems’ was then introduced as a potential confounding variable; sex and age were held constant via covariate terms.

The SEM has two major advantages. First, since AUD levels cannot be directly observed in the survey context, and can only be inferred from clinical features, it qualifies as a ‘latent’ variable (30). Thus, with the measurement model used to build the latent ‘AUD’ variable, measurement errors can be constrained. Second, the SEM model can be used to produce unbiased estimates even when covariates are correlated or jointly determined (31–33).

The CPP-AUD estimate in the ‘multiple adversities SEM’ measures the CPP-AUD relationship holding other childhood adversities constant, i.e. by regressing them as covariates on the latent AUD variable. In contrast, the theoretical rationale behind the “CPP-specific SEM” model is that there is a latent ‘noxious family environment’ variable, which cannot be directly observed and is causing various childhood adversities, including CPP, to occur. In this context, the CPP-AUD relationship may be due to the general noxious family environment. Thus, this general ‘noxious family environment’ is posited as an influence on the AUD level and the direct path from CPP to AUD measures if CPP exerts an additional influence on the level of AUD.

In the final ‘explore’ steps, the analytical sample first was restricted to individuals without parental alcohol/drug problems. Estimates from this stratification approach were compared

to those from the above modeling approach. (Small sample size precluded such analysis for people with parental alcohol/drug problems.) The purpose of this step is to probe into the issue of possible ‘endogeneity’ involving parental drinking problems. Second, individuals who had any AUD clinical features before 18 years old were excluded for an additional clarification of the temporal sequence from CPP to drinking problems. In addition, the models were re-fit for the subgroup of males because some prior studies have found CPP-AUD associations only for females. Finally, due to the concern that the relationship may be confounded by childhood conduct problems, the history of conduct problems was held constant as a covariate.

All analyses were implemented using statistical software Stata (StataCorp LP, 2009) and Mplus (Muthén and Muthén, 2009) for complex sample survey data. A probit link function was used to estimate regression coefficients in Mplus. Probability weights and post-stratification factors were applied to account for differential participation levels and the Part I/Part II selection probabilities, and any study design effects.

3. RESULTS

Greater occurrence of each AUD clinical feature was found for the CPP group compared to the non-CPP group (Table 1). Childhood adversities occurred more frequently in the CPP group compared to the non-CPP group. Confirmatory factor analysis found good fit for a single dimension of the AUD clinical features (RMSEA=0.026, CFI=0.995, TLI=0.994) with strong and statistically significant factor loadings. A single dimension of the childhood adversity variables (level of noxious family environment) was also acceptable (RMSEA=0.044, CFI=0.940, TLI=0.940) with moderate–strong and statistically significant factor loadings.

Main estimates for this study were presented in Table 2. Holding constant all other childhood adversities, CPP was associated with a higher level of ‘AUD’ (coefficient=0.70, $p<0.01$, “multiple-adversity SEM”). The regression coefficient can be interpreted as a statistically robust greater mean level of AUD for the CPP group compared to the estimated mean for the non-CPP group, on the probit scale. Estimates for other childhood adversities varied from -0.34 for child neglect ($p>0.10$) to 0.74 for observed violence between parents ($p<0.01$).

Results from the “CPP-specific SEM” suggests that, with the latent ‘noxious family environment’ construct held constant, CPP was associated with an elevated level of ‘AUD’ (coefficient=0.20, $p=0.034$). As such, the influence of CPP might not be completely accounted for by the generally noxious family environment, even though considerable AUD variation could be traced back to the level of the noxious family environment (CPP slope estimate dropped from 0.70 of the ‘multiple-adversity SEM’ to the current 0.20).

In the post-estimation exploration steps, results were approximately the same when individuals with any AUD clinical features before 18 years old were excluded. Similar results were found for the analyses restricted to males only. Model fitting indices for these models can be found in Table 3. Additionally, estimates were not appreciably different for analyses restricted to individuals without parental alcohol/drug problems when compared to the above modeling approach (CPP-AUD slope estimates of 0.55 vs. 0.70 for ‘multiple-adversity SEM’, and 0.19 vs. 0.20 for ‘CPP-specific SEM’). Also, after the ‘unable to cut down’ item was combined with the ‘drink more than intended’ item, all estimates remained approximately the same (e.g. 0.57 vs. 0.60 for ‘multiple-adversity SEM’, and 0.18 vs. 0.20 for ‘CPP-specific SEM’) although fitting indices suggested worse fit. After controlling for the occurrence of conduct problems (which were assessed to individuals younger than 40

years old only, $n=570$), CPP-AUD coefficient was 0.56 ($p<0.01$) and 0.02 ($p>0.50$) for the 'multiple adversity SEM' and 'CPP-specific SEM,' respectively.

DISCUSSION

In this study, childhood physical punishment was found to be associated with an elevated level of AUD even when other childhood adversities were held constant. This estimated relationship apparently was not due to family history of drinking problems, nor sex nor age. Additionally, the CPP-AUD relationship was not completely accounted for by the noxious family environment.

4.1 Limitations

Several limitations of the study merit attention. First, due to the fact that the current study is observational and cross-sectional, it cannot provide definite evidence of a causal link between CPP and AUD because of possible unmeasured confounding variables. An analogue to this limitation is the failure of randomization of exposure (CPP) in experimental studies. The solution to this limitation lies in prevention or intervention trials with successful randomization of CPP as well as a follow-up period long enough for AUD to occur and low attrition rate. This type of research is resource-demanding. In the absence of any convincing evidence of a CPP-AUD relationship in Chinese populations, it is of importance to estimate the magnitude of the association from retrospective or concurrent studies with efforts to control for confounders in order to guide future prospective studies. There have been concerns that the estimates linking any specific childhood adversity to mental disorders may be biased away from the null because various childhood adversities are inter-correlated and that each adversity may pose a risk to the development of mental disorders (13). Here we offer the findings as a rigorous attempt to probe into the specificity of a CPP-AUD relationship, with other childhood adversities held constant. Furthermore, using the SEM approach, we were able to control for parental drinking problems, which are arguably the most important confounders for the CPP-AUD relationship.

With respect to covariates assessment, information about parental drinking problems was based on self-report from the respondents instead of their parents. No clear definition of "drinking problems" was given in the CIDI question on this topic. Thus, the assessment may be subjective and fairly coarse. Several studies in the United States have suggested that information about parental drinking problems collected from offspring can be pertinent and accurate, and may serve as a valid method for assessing parental problems when alternatives are not possible (34–36). Whether this conclusion holds true in the Chinese context is not clear.

4.2 The CPP-AUD association

Despite these limitations such as these, our interesting findings may be important.

In this study, we have provided further evidence of a possible causal relationship between CPP and AUD in the Chinese context (5). Mixed evidence was found from previous studies in the US for the CPP-AUD relationship, with other childhood adversities taken into account (7,8,14–17). The discrepancies might be traced to variations in the study population (e.g. US Marines, child victims recruited from the court, individuals from a primary care clinic, etc.), the statistical tools used (GLM vs. SEM), the study design (concurrent vs. prospective), and the study participation levels (varying from 25% to above 80%). With respect to study populations, the present study was based upon a sample drawn from non-institutionalized adults living in the two metropolitan cities in China. Possible biases in estimation were minimized by efforts to adjust for clustering and selection probability, as well as non-

response patterns. Thus, results are applicable to the household-dwelling adults living in the two Chinese cities. Given there is evidence that the majority of Chinese individuals with AUD have never sought treatment in China (27), it becomes especially important to have estimates from community survey samples. With respect to statistical tools, using the “multiple-adversity SEM” model, we were able to accommodate the inter-correlations among childhood adversities (8,13) and to estimate the relationship between CPP and AUD. Additionally, using a novel “CPP-specific SEM” model, we were able to take into account the background level of noxious family environment during childhood in order to inspect if the CPP-AUD relationship was due to the general noxious family environment.

With respect to study design and response level, although prospective observational studies are superior to cross-sectional studies in clarifying the temporal sequence and controlling for recall bias, prospective studies are resource-demanding. Furthermore, prospective research on the CPP-AUD relationship would necessitate long-term follow-up, which increases the chance of attrition. Indeed, many previous prospective studies suffer from substantial attrition over time (>40%) (7,14,37). Moreover, there has been evidence that participant attrition was positively associated with the occurrence of AUD (38). Concerning recall bias, previous studies have found that retrospective self-report on childhood adversities yielded good to excellent validity and reliability (39–41). Concerning the temporal sequence, it is possible that the occurrence of riskier drinking and related problems could be a manifestation of a continuity of childhood behavioral problems (including early drinking problems). These problems may constitute one reason parents physically punish their offspring. To explore this question, we excluded individuals who had any clinical feature of AUD before 18 years old. No appreciable difference in the estimates was found. The issue of conduct problem is more difficult to address in the context of a cross-sectional research because the temporal sequence of CPP and conduct problems cannot be known. On one hand, childhood conduct problem may serve as a confounder, in that conduct problem has been found to be a good predictor of later alcohol dependence (e.g. 42). On the other hand, conduct problems may well serve as a mediator in the pathway from CPP to AUD because CPP may contribute to the occurrence of conduct problems in children (43); conduct problems may also share causes with alcohol dependence and CPP (possibly PDP, 44–46). Thus, controlling for conduct problem may artificially bias the estimates for CPP-AUD association toward the null. Nonetheless, according to our preliminary results, after controlling for the history of conduct problems, there was still evidence of a CPP-AUD association.

It is noteworthy that the current study found the CPP-AUD association among males. Several previous studies have found robust CPP-AUD associations for females only (37,47). Nonetheless, it has not been clear whether this is true for the general in China or elsewhere. This study has provided the first evidence for a CPP-AUD association among Chinese males living in the two metropolitan cities.

The results from this study provided a solid foundation for future inspections on the CPP-AUD relationship. It may have important implications for designing prevention and intervention strategies for AUD: a large portion of the CPP-AUD relationship may be attributable to a generally noxious family environment; however, in cases when a deficient family is inevitable, modulation of physical punishments may still help modify AUD risk.

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Figure 1.
A “multiple-adversity SEM” model

Figure 2.
A “CPP-specific SEM” model

Table 1

Sample description and results from confirmatory factor analysis. Data from WMH-mC, 2001–2002 (n=1628).

Panel A. Description of sex and age for childhood physical punishment (CPP) exposed group and non-exposed group.				
	Exposed to CPP (n=235)		Non-exposed to CPP (n=1376)	
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)
Male	n=152	% (95% CI) ¹ = 59.7 (51.7, 67.5)	n= 658	% (95% CI) ¹ = 51.4 (47.4, 55.4)

Panel B. The occurrence of clinical features of alcohol use disorders and childhood adversities and factor loading of these variables from confirmatory factor analysis.					
Clinical features of alcohol use disorder			Childhood adversities		
	% among CPP (se) ¹	% among non- CPP (se) ¹	% among CPP (se) ¹	% among non- CPP (se) ¹	Factor loading ²
Responsibility interference	7.3 (2.3)	2.3 (0.5)	3.1 (1.6)	1.6 (0.6)	0.44
Social problems	5.1 (1.6)	1.6 (0.5)	27.2 (3.5)	2.7 (0.6)	0.75
Drinking despite social problems	4.0 (1.4)	0.9 (0.3)	18.3 (3.4)	5.1 (1.0)	0.87
Hazard drinking	7.4 (2.1)	1.7 (0.5)	32.0 (3.8)	4.8 (0.8)	0.47
Legal problems	1.8 (1.4)	0.2 (0.1)	12.3 (3.0)	1.3 (0.3)	0.92
Drinking despite physical/emotional problems	3.2 (1.3)	1.4 (0.5)	10.4 (2.5)	0.8 (0.3)	0.88
Irresistible desire	5.2 (1.6)	0.8 (0.2)	--	--	0.75
Tolerance	2.1 (1.0)	0.5 (0.2)			
Withdrawal	3.1 (1.3)	0.5 (0.2)			
More than intended and difficulty cut down	7.4 (2.2)	1.3 (0.4)			
Great deal of time spent	1.4 (0.7)	0.5 (0.2)			
Give up activities	2.7 (1.3)	0.3 (0.1)			
Fit statistics					
Fitting indices ² : χ^2 (df)= 18.77 (9), RMSEA= 0.026, CFI= 0.995, TLI= 0.994			χ^2 (df)= 24.63 (6), RMSEA= 0.044, CFI= 0.940, TLI= 0.940		

¹Weighted proportions

²Based on confirmatory factor analysis

Estimated probit slope coefficient linking childhood physical punishment and level of alcohol use disorder. Data from WMH-mC, 2001–2002 (n=1628).

Table 2

	“Multiple-adversity SEM” model			“CPP-specific SEM” model		
	Coefficient	95% CI	p	Coefficient	95% CI	p
All persons included ¹	0.70	0.40, 1.00	<0.001	0.20	0.02, 0.38	0.034
Excluding those whose AUD clinical feature occurred before 18 ¹	0.51	0.16, 0.87	<0.001	0.20	0.01, 0.39	0.038
Restricted to males ²	0.62	0.33, 0.91	<0.001	0.36	0.12, 0.59	0.003

¹ Adjust for parental drinking problems, sex, and age.

² Adjust for parental drinking problems and age.

Table 3

Model fitting indices for SEM models presented in Table 2.

	“Multiple-adversity SEM” model				“CPP-specific SEM” model			
	RSMEA	CFI	TLI	TLI	RSMEA	CFI	TLI	TLI
Without adjustment	0.031	0.980	0.981	0.980	0.030	0.978	0.980	0.980
Adjust for parental drinking problems	0.040	0.954	0.964	0.968	0.040	0.968	0.968	0.968
Adjust for parental drinking problems, sex, and age	0.040	0.953	0.958	0.968	0.035	0.968	0.968	0.968
Excluding those whose AUD clinical feature occurred before 18 ¹	0.041	0.943	0.954	0.929	0.054	0.933	0.929	0.929
Restricted to males ²	0.044	0.928	0.933	0.904	0.057	0.911	0.904	0.904

¹ Adjust for parental drinking problems, sex, and age.

² Adjust for parental drinking problems and age.