A TALE OF TWO CITIES: DRINKING PRACTICES AND PROBLEMS IN TWO METROPOLITAN CITIES IN CHINA, BEIJING AND SHANGHAI

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ABSTRACT

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Alcohol consumption has been common in China. Nonetheless, drinking and drinking-related problems have not been sufficiently described. In this dissertation project, data collected from two metropolitan cities in China, Beijing and Shanghai, have been used to describe drinking behavior and drinking-related problems in these two cities.

Previous studies have provided some basis for speculating that childhood physical punishment (CPA) might be a causal influence on drinking problems, but more research is needed before any causal inference is drawn. In this dissertation project, this possible causal relationship is also inspected in a Chinese context.

Multi-stage probability sampling was used to collect information from 5201 household-dwelling adults in Beijing (n=2633) and Shanghai (n=2568). A version of the World Health Organization Composite International Diagnostic Interview was used to assess drinking behavior and problems, childhood experiences, as well as other relevant variables.

It was found that alcohol is highly accessible in these two metropolitan cities; alcohol consumption is common; heavier drinking is not rare (occurrence ≥ 7%); drinking problems (socially maladaptive drinking and

alcohol dependence) exist at a fairly low occurrence (occurrence <7%). Males and younger people were more likely to be involved with drinking; they are also more likely to have a history of drinking problems.

A positive association was found between CPA and drinking-related problems after taking family history of drinking problems into account. The assumption of exogeneity of covariates was tested using the recursive probit regression method. Estimates from generalized linear models were corrected when there was evidence of endogeneity. Good internal validity and model stability was found by the bootstrap resampling approach. The strengths of associations were stronger for "early onset drinking problem" variables as compared to some other variables.

This dissertation project provided the first epidemiological description of drinking behavior and drinking-related problems in the two biggest cities in China, Beijing and Shanghai. Result implied that the priority of prevention and intervention should be placed on males and young adults. A possible causal relationship between CPA and riskier drinking and drinking problems were found. Limitations of the study were discussed. Directions for future research have been suggested.

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Chapter 1 Aims and objectives

This dissertation is focused on three aims.

Aim 1: To describe epidemiological facets of beverage alcohol involvement in two metropolitan cities in China: Beijing and Shanghai

- Estimation tasks:
  1. For the community populations as a whole, to estimate the cumulative occurrence of opportunity, the first chance to drink alcohol, trying alcohol (ever), pre-teen trying alcohol, alcohol drinking (ever), precocious onset of drinking (<20 years old), heavier drinking, drinking-related social maladaption, clinical features of alcohol dependence, and early onset of alcohol use disorders (AUD, < 23 years old), in Beijing and Shanghai, respectively.
  2. For the community populations as a whole, to estimate the one-year interval prevalence for recent alcohol drinking, heavy drinking, drinking related social maladaption and alcohol dependence, in Beijing and Shanghai, respectively;
  3. For the community populations as a whole, to estimate time-to-event parameters such as the mean and median age of first drink, onset of drinking, and onset of drinking-related problems, and to plot the comparative survival analysis parameter estimates.

Aim 2: To estimate subgroup variation with respect to beverage alcohol involvement.

- Estimation tasks:
  1. Estimation of subgroup-specific cumulative occurrence and interval prevalence of drinking related outcomes with respect to sex (male-female), age groups, and categories of marital status, income level, education attainment, and employment groups.
  2. Estimation of association structure parameters that link drinking and related problems back to their potential sources of variation associated with membership in these subgroups.

Aim 3: To estimate a suspected causal association that links childhood physical punishment to later drinking and drinking problems in order to shed light on the suspected causes, as well as aspects of mechanisms that might lead to alcohol drinking and associated problems.

- Estimation tasks:
  1. To estimate the association between childhood physical punishment and drinking related outcomes, within the context of a more comprehensive conceptual model, fit with the approach of multiple logistic regression, and with a binvariate probit model used to probe into assumptions of the logistic regression model.

2. To estimate the variation in associations across different drinking-related outcomes accounting for the correlation between these outcomes.
3. To estimate the association between childhood physical punishment and stages of alcohol involvement (with respect to the earliest and later stages of alcohol drinking involvement), also within the framework of a more comprehensive conceptual model.

Chapter 2 Background and significance

2.1 Introduction

In this dissertation project, the focus is on the epidemiology of drinking and drinking-related problems in two metropolitan areas of China. In this chapter, the aims are 1) to introduce the concept of alcohol drinking-related problems; 2) to review selected aspects of the history of drinking-with a focus on China; 3) to review each of the 5 rubrics of epidemiology (quantity, location, cause, mechanism, and prevention and control) with respect to drinking practices and related problems; 3) to identify gaps in current knowledge about drinking practices and related problems; 4) to evaluate the potential significance of the dissertation project.

2.2 Drinking-related problems - alcohol dependence and alcohol abuse or harmful use: recent concepts

Alcohol drinking-related problems refer to negative consequences of alcohol consumption, including social and interpersonal problems and a dependence syndrome. In brief, the alcohol dependence syndrome involves (a) neural adaptation to repeated drinking, (b) obsession-like disturbance of the mental life (e.g. craving), and (c) compulsion-like disturbance of behavior (e.g. inability to stop drinking).

Currently, there are two most commonly used mental disorder classification systems, the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) developed by American Psychiatric

Association (APA, 1994) and the International Statistical Classification of Diseases and Related Health Problems, the 10th edition (ICD-10) developed by the World Health Organization (WHO, 1992). Both clearly list alcohol as a psychoactive drug that can cause a dependence syndrome. Two main categories of alcohol use disorders (AUD) defined in the DSM-IV are alcohol dependence and non-dependent alcohol abuse; ICD experts avoided the stigma-laden term 'abuse' and substituted 'harmful use' as a related disturbance.

Alcohol dependence is defined in the ICD-10 glossary and in DSM-IV shown in chart 2.1.

Chart 2.1

ICD-10 glossary

A cluster of behavioral, cognitive, and physiological phenomena that develop after repeated substance use and that typically include

a strong desire to take the drug,

difficulties in controlling its use,

persisting in its use despite harmful consequences,

a higher priority given to drug use than to other activities and obligations,

increased tolerance,

and sometimes a physical withdrawal state.

The dependence syndrome may be present for a specific psychoactive substance (e.g. tobacco, alcohol, or diazepam), for a class of substances (e.g. opioid drugs), or for a wider range of

pharmacologically different subsances

DSM-IV case definition

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

1. tolerance, as defined by either of the following:

a need for markedly increased amounts of the substance to achieve intoxication or desired effect

markedly diminished effect with continued use of the same amount of substance

2. withdrawal, as manifested by either of the following:

the characteristic withdrawal syndrome for the substance

the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms

3. the substance is often taken in larger amounts or over a longer period than was intended

4. there is a persistent desire or unsuccessful efforts to cut down or control substance use

5. a great deal of time is spent in activities to obtain the substance, use the substance, or recover from its effects

6. important social, occupational or recreational activities are given up or reduced because of substance use

7. the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., continued drinking despite recognition that an ulcer was made worse by alcohol consumption)

In ICD-10, harmful use refers to "*A pattern of psychoactive substance use that is causing damage to health. The damage may be physical (as in cases of hepatitis from the self-administration of injected psychoactive substances) or*

mental (e.g. episodes of depressive disorder secondary to heavy consumption of alcohol)." Under DSM-IV, non-dependent abuse is largely characterized by impairment in social functioning, such as socially maladaptive behaviors (e.g., drink-induced violence), or social role impairments (e.g., family or legal troubles), as well as other hazard-laden alcohol-related behaviors such as drunk driving (APA, 1994; WHO, 1992).

These concepts have been refined over the past 50 years. It may be useful to provide some information on the history of the concept of alcohol dependence that has prompted alcohol researchers to differentiate alcohol dependence from harmful drinking or nondependent abuse. In the first and second editions of the DSM, published in 1952 and 1968, respectively, alcoholism (addiction), which was then the term for alcohol dependence, was listed under 'personality disorders and certain non-psychotic mental disorders' (APA, 1952, , 1968). Alcoholism also appeared as a category in the eighth edition of ICD published in 1967 (WHO, 1967).

During the 1960s, the WHO Expert Committee on Drug Addiction-Producing Drugs decided that the term 'addiction' carried too much stigma-laden connotation to be useful in scientific work. Therefore, they re-named themselves the Expert Committee on Drug Dependence, and introduced the definition of 'dependence' that was specific to each drug compound (e.g. dependence of the amphetamine type, dependence of the alcohol/ethanol type. This history is described by the report series of the WHO expert committee on drug dependence, (WHO expert committee on addiction-

producing drugs, 1964)). In the 1970s, diagnostic criteria for 'alcoholism' and 'alcohol dependence' were introduced by Feighner et al. (Feighner et al., 1972) and Edwards and Gross (Edwards & Gross, 1976), respectively, to describe a number of physiological and psychological manifestations of diminished control over alcohol use. In the Edwards-Gross conceptualization, alcohol problems were dimensional; they did not formalize a concept of 'alcohol abuse'. Ten years later, two discrete diagnostic categories specified after the deliberation of a DSM-III committee of experts (Rounsaville, Spitzer, & Williams, 1986). These two conditions were: 1) alcohol dependence, and 2) alcohol abuse, with dependence and abuse allowed to co-occur. In contrast, the ICD-9, published in 1977, used a categorical approach when defining the 'alcohol dependence syndrome' and 'non-dependent abuse' to be mutually exclusive (World Health Organization, 1977). The introduction of the 'non-dependence abuse' category is in response to the clinical observation that some non-alcohol-dependent clients sought out or were referred to medical help for their non-dependent maladaptive behavior due to alcohol drinking. This categorical approach was used in the current DSM-IV and ICD-10 as well (American Psychiatric Association, 1994; WHO, 1992). It is noteworthy that in ICD-10, the term 'alcohol abuse' was replaced by the term 'harmful use' in order to better describe negative health consequences due to alcohol consumption. Evidence has been mixed regarding the question of whether alcohol abuse and alcohol dependence are best represented as distinct dimensions or categories, whether alcohol abuse should be specified as an

intermediate stage along the progression to alcohol dependence, or whether they are merely two labels for the same disorder. Results from a long term follow-up study (60 years) showed there were people who stayed long-term as cases of alcohol abuse without meeting any criterion of alcohol dependence (Vaillant, 2003). On the other hand, cross-sectional research suggests that a noteworthy proportion of individuals with alcohol dependence had never experienced clinical features of alcohol abuse (D. S. Hasin & Grant, 2004). Shorter term follow-up studies also provide evidence that the course of alcohol abuse differs from the course of dependence (D. S. Hasin, Grant, & Endicott, 1990; D. S. Hasin, Van Rossem, McCloud, & Endicott, 1997). Besides evidence on differing natural history and clinical course of these two alcohol-related disturbances, there have also been explorations on this question from other angles. For example, a cross-sectional study in the US has suggested that stronger association can be found between DSM-IV alcohol dependence and alcohol consumption, treatment seeking, alcohol intoxication, and suicidal ideation, as well as family history of alcohol use problems, compared to DSM-IV alcohol abuse (D. Hasin & Paykin, 1999).

Recent advances in methods for latent variable analysis, including computational software, have promoted application of Item Response Theory (IRT), as well as Latent Class Analysis (LCA) models. This work has expanded the scope of the probing into these latent structure questions. Since the early 1990s, a number of some researchers have explored the underlying latent structure of AUD, using items from DSM and ICD constructs for alcohol

dependence, alcohol abuse, and harmful alcohol use, but these analyses have not always yielded consistent evidence. For example, Nelson and colleagues found that latent structures changed when analytic samples changed from the entire sample (drinkers and non-drinkers) drinkers. In specific, a single dimension sufficed as the best model fit to data from their entire sample, whereas two dimensions were needed for the "drinkers only" sub-group. Moreover, dimensions were not completely consistent with the alcohol dependence and abuse conceptualizations (Nelson, Rehm, Ustun, Grant, & Chatterji, 1999). Some investigations have extracted just one underlying dimension (e.g. see (Saha, Chou, & Grant, 2006). Many studies have found cross loadings of items from the dependence domain and the abuse domain (Grant et al., 2007; Muthen, Hasin, & Wisnicki, 1993; Proudfoot, Baillie, & Teesson, 2006). Research using LCA techniques found that items from both DSM-IV alcohol abuse and alcohol dependence differentiate individuals into class memberships (Bucholz et al., 1996; Smith & Shevlin, 2008). Summarized from epidemiological studies, there is evidence of both similarities and differences between DSM-IV alcohol abuse and dependence (D. S. Hasin et al., 2003).

2.3 History of alcohol consumption, with a focus on China

Of course, the history of drinking and related problems did not begin in the 20th century. Evidence of man-made alcohol has been found in pre-historic time in Mesopotamia (e.g. current Iran and Iraq) and in other parts of the world. In ancient Egypt, Greece, and Rome, alcohol had been part of people's

daily diet, as well as served for medicinal and religious purposes (D. B. Heath, 1995). In ancient China, as early as the Shen Nong period of the New Stone Age (approximately 7000 BC), traces of alcohol were found in a wine jar discovered in Jiahu in Hunan province (McGovern & Patrick, 2003). Since then, numerous fairy tales and legends have waltzed around alcohol throughout the Chinese history. For example, there is a beautiful legend story about Du Kang, a boy from a poor farmer family, which tells how alcohol was invented in China. Today Du Kang is a brand of spirit and some Chinese still use the character of his name to indicate alcohol. The invention of alcohol yeast during the period of the Xia-Shang Age (1700 BC) and the reform of storage techniques facilitated the production of alcohol beverages in Chinese history (Cochrane, Chen, Conigrave, & Hao, 2003). Over the years, alcohol drinking has been integrated deeply into the Chinese culture. From the ancient ritual ceremonies to modern parties, alcohol has been regarded as a way to express happiness and to exchange wishes of good luck. Besides daily activities, alcohol has played an important role in Chinese medicine, and it has been tightly associated with art and poetry as well (Hao, Chen, & Su, 2005). For example, a famous Chinese poet Li Bai during the Tang dynasty (618-907) is well-known for his inspiration after drinking alcohol.

Negative consequences of drinking also have been described since ancient times. For example, the origins of present Dram Shop Laws can be traced back to around 2000 BC from Hammurabi's code in ancient Babylon (C. B. Anthony, 1995; D. B. Heath, 1995). Problems induced by overdrinking have

been long recognized in China. For example, excessive drinking of the emperor and his followers has been related to the fall of several Dynasties in the Chinese history with the earliest being the Shang Dynasty in the 11th century B.C. An early epidemic of the use of a combination of alcohol and *hanshi* has been documented in the Han Dynasty (second and third century) in China. Users of this mixture described it as a cause of a mind-opening and thought-clarifying effect from the psychoactive drug mixture, but the chemical identity of *hanshi* remains unknown (Schutz, 1995).

Throughout the Chinese history, there have been multiple efforts at alcohol control in response to drinking problems (Newman, 2002). For example, overdrinking, together with overeating, gambling, and smoking, were recognized as harmful and were listed as the "Four Vices" in China (Cochrane, Chen, Conigrave, & Hao, 2003).

Against this background, it may be understood that social drinking often has been highly accepted and even sometimes is encouraged in the Chinese contexts. Drinking plays important roles in important events, such as Chinese New Year Festival, wedding ceremonies, and birthday celebrations. Ritualized drinking for special events still exists in some areas (Hao, Chen, & Su, 2005).

Nonetheless, drinking behavior has changed markedly as the Chinese market has opened to the outside world, with westernization. As a result, a modified Chinese style of drinking has emerged from an intersection where tradition and modernization meet up. For example, nowadays drinking is used

as a way to ease tension and to facilitate social exchange among Chinese businessmen; it is also believed to help maintain good relationships between employers and employees and among coworkers. On the other hand, the Chinese culture tends to discourages solitary drinking (Williams, 1998) and despite the custom of toasting as a common way to express friendliness, Chinese drinkers tend to avoid overdrinking in social circumstances (Cochrane, Chen, Conigrave, & Hao, 2003; Hao, Chen, & Su, 2005).

Of course, there is some evidence of beneficial health effects of drinking. For example, light-to-moderate alcohol consumption has been associated with lower occurrence of diabetes and cardiovascular diseases (Kloner & Rezkalla, 2007; Room, Babor, & Rehm, 2005; van de Wiel, 2004). Whereas these beneficial effects should not be forgotten, it is the alcohol problems that concern us in the present context. The extent of these problems is the topic of the next section of this chapter.

2.4 The First Rubric of Epidemiology: Quantity

Alcohol drinking is common in many parts of the world, e.g. the Americas, Europe, and Asia. There are approximately 2,000,000,000 alcohol drinkers around the world (Anderson, 2006). Alcohol consumption has been associated with substantial burden of disease, e.g. 1,800,000 deaths per year according to the WHO estimate (WHO, 2004b). Following is a map from the WHO website showing the estimated disease burden from alcohol consumption in each WHO sub-region (WHO, 2009).

Figure 1.1 Burden of disease attributable to: ALCOHOL
(% DALYs in each subregion)

The disease burden comes from various negative health consequences, such as unintentional injuries, AUD and other neuro-psychiatric conditions. It was estimated that AUD accounts for approximately 40% of all disease burden attributable to alcohol consumption (WHO, 2004b). According to the WHO, approximately 125,000,000 people have AUD at any given time point worldwide (WHO, 2004a).

2.5 The Second Rubric of Epidemiology, Location

2.5.1 Stable characteristics

There are stable and sometimes time-invariant characteristics that can be used to map population subgroups variation in the occurrence of alcohol dependence and related problems. In this section, the focus is upon three stable characteristics: (a) sex, (b) year of birth, and (c) family–genetic characteristics.

Consistently, evidence has shown that males are more likely to drink (WHO, 2004b); males are more likely to experience AUD than females across countries and cultures with no notable exceptions to date. However, the degree of the male-associated excess risk can vary dramatically. For example, studies from the USA and other European countries usually show that the occurrence of AUD in males is an estimated 2 to 3 times higher than in females (D. S. Hasin, Stinson, Ogburn, & Grant, 2007; Rehm, Taylor, & Patra, 2006; Wilsnack et al., 2000). In eastern countries such as Korea Japan and China, the male-female ratio is much larger (Hao et al., 2004; Higuchi, Matsushita, Maesato, & Osaki, 2007; J. T. Park, Kim, & Jhun, 2008; Wei, Derson, Xiao, Li, & Zhang, 1999). Accordingly, the odds ratio and other statistical indices of the strength of association between sex and AUD is larger in these eastern countries as compared to values observed in European countries and North America (Keyes, Grant, & Hasin, 2007; Rehm, Taylor, & Patra, 2006). One argument in the literature is that the male-female difference in AUD is largely due to the smaller amounts of alcohol consumed in women, and that at the same levels of consumption, women drinkers might experience as many or more problems than men drinkers (Ely, Hardy, Longford, & Wadsworth, 1999; Fillmore et al., 1995; Miller, Plant, & Plant, 2005). Contrary to this argument is an observation that many females metabolize ethanol less efficiently than males, which means the dose-response curve is left-shifted in females compared to males. Therefore, adverse effects may result from smaller amounts of alcohol in females as in males. The male excess of AUD is seen in

China, which is the location of this dissertation research. In China, the male excess is especially pronounced (Hao et al., 2004; Wei, Derson, Xiao, Li, & Zhang, 1999).

The association between year of birth (as expressed in age strata) and drinking and AUD is not consistent in the literature. For example, some studies have found that people in younger age strata are more likely to have a history of drinking, binge drinking, and AUD (Degenhardt, Chiu, Sampson, Kessler, & Anthony, 2007; Higuchi, Parrish, Dufour, Towle, & Harford, 1994; Kessler et al., 1994; Kim et al., 2008; Naimi et al., 2003; Serdula, Brewer, Gillespie, Denny, & Mokdad, 2004); while some other studies find higher likelihood of drinking and AUD in middle age groups (Rehm, Room, van den Brink, & Jacobi, 2005; Wilsnack et al., 2000; X. Zhou et al., 2006). Explanations for these age-related variations include: chronicle age of the person, period effects, and cohort effects. For example, AUD usually starts to emerge during adolescent to early adulthood. Surveys among pre-teen population may find very low occurrence of AUD because they have not started to drink alcohol yet. During some periods in the history, policies and regulations may influence the availability of alcohol, e.g. the US National prohibition of alcohol (1920-33). The occurrence of AUD may be different for these periods compared to others. People in some cohorts may be more or less likely to be abstainers, e.g. "baby boomers". However, these three factors are highly intertwined with each other. It is especially difficult to tease them out from cross-sectional studies when survival of drinkers may also play a role in

estimates. Nevertheless, up-to-date data on AUD in different age strata provide information about the distribution of disease burden of AUD in population subgroups. In China, the patterns of drinking also are seen to vary across age strata (Hao et al., 2004): current drinking increases with age peaking in middle-age group (36-50), and then declined in older age groups.

For centuries, it has been observed that the AUD (including alcoholism) tend to show familial aggregation (Merikangas, 1990; Radouco-Thomas et al., 1979; Schuckit et al., 2001). Recently, studies equipped with advanced techniques have found that AUD cases differ from controls in selected genotypes. Some studies have been able to pinpoint mechanisms of the family influence down to the level of Single nucleotide polymorphism (SNP) (Edenberg et al., 2004; Schuckit, Smith, & Kalmijn, 2004; Zlotnick et al., 2006). These AUD-associated genotypes and SNPs are involved in various biological and pharmacological functions including neuro transmitters, ethanol metabolism, cell adhesion, etc (Schuckit, Smith, & Kalmijn, 2004). As the technology and knowledge about molecular genetics of AUD continues to evolve, our understanding of these observed locational differences will be clarified to the point that we will regard some genotypes as causal influences on AUD. However, at the present time, these observations remain associational in nature, and the evidence of causal influence is not yet fully developed. Notwithstanding the molecular genetic pathways of family-genetic influence, there also are other mechanisms of note. For example, social learning mechanisms can foster drinking behavior (e.g. offspring of

abstainers are more likely to be abstainers as well (Harburg, DiFranceisco, Webster, Gleiberman, & Schork, 1990)). In addition, studies have provided evidence that some cultures tolerate drinking-related misbehavior more than others (Donovan & Molina, 2008; Room, 2006).

2.5.2. Cross-country or cross-region variation

Theoretically, country or region is time-variant. However, compared to the rate of occurrence of AUD, country and region are relatively stable characteristics. Thus, we treat these characteristics as time-invariant in this section of the background.

Summarized from 55 studies from around the world before 2000, the point prevalence estimate of AUD varied widely from country to country and region to region. For example, estimates of point prevalence in countries in the America (North and South) are typically higher than those in Islamic countries. In some of African countries (e.g. Nigeria and Ethiopia), the estimate is close to zero while in other African countries (e.g. South Africa, Zambia, and Zinbabwe). The same pattern can be seen in incidence as well (C. Mathers & Ayuso-Mateos, 2000). There is an obvious unbalance in literatures written in English regarding the occurrence of AUD from different regions or countries in the world. For example, the US population is much more frequently studied than populations in some other regions and countries, especially lower-income and non-English speaking countries (e.g. countries in Africa, the middle-east, and Asia). For this reason, we summarize studies in the US first and then expand to evidence from other countries.

In the US, the tradition of community surveys of drinking dates back to the 1950s and 1960s (e.g. see W. B. Clark & Hilton, 1991). Using these surveys and national sales and tax records about alcohol, in some countries the alcohol use disorders (AUD) have become quite common. For example, the 2001-2002 US National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) is a cross-sectional study assessing the occurrence of alcohol drinking and related problems based upon a nationally representative sample of household-dwelling individuals. Estimates suggest that an estimated 30% of the US population age 18 and older had a lifetime history of AUD, defined to include both DSM-IV alcohol dependence and DSM-IV alcohol abuse (D. S. Hasin, Stinson, Ogburn, & Grant, 2007).

As noted by Anthony and colleagues (J.C. Anthony & Van Etten, 1998), epidemiological measures based upon the lifetime history include lifetime cumulative incidence proportion (of those who survived to be assessed). This same proportion is sometimes referred to as "lifetime prevalence", but, it deviates from epidemiological definition of a prevalence measure as one that varies with both the incidence of the condition and the duration of the condition (Gordis, 2004). Hereinafter, this measurement will be called by the term "cumulative occurrence", which avoids the conundrum where the "lifetime prevalence" term is used, and is in keeping with a contemporary proposal to send the concept of "lifetime prevalence" into retirement (J.C. Anthony & Van Etten, 1998).

Whereas Hasin and colleagues found that the lifetime occurrence of AUD was about 30% overall, the estimated 12 month prevalence of AUD was 8.5% (D. S. Hasin, Stinson, Ogburn, & Grant, 2007). Two prior large-scale epidemiological studies in the US, the Epidemiologic Catchment Area (ECA) program and the National Comorbidity Study (NCS), yielded AUD 12 month prevalence estimates of about six percent and seven percent, respectively (Kessler et al., 1994; Robins, Locke, & Regier, 1991), not appreciably distant from the corresponding NESARC estimate.

Besides the occurrence of AUD in the entire population, another pertinent set of estimates involves the occurrence of AUD in the sub-population of drinkers. For many reasons, (e.g. religion, personal choice), many people in the population are lifetime abstainers, who thereby remain not at risk of developing AUD. Estimated from the 1994 NCS, about 15% of those who ever consumed alcohol had developed alcohol dependence (J. C. Anthony, Warner, & Kessler, 1994). Studying current drinkers who participated in the 2001 National Household Survey on Drug Abuse in the US, age 12 years old and above, Harford and colleagues found that among young adult users (18-23 years old), one in three male current drinkers and one in five female current drinkers had experienced at least one of the clinical features of alcohol dependence during the 12 months prior to the assessment. "Tolerance" and "a great deal of time spent on alcohol" were the most common clinical features among current drinkers. With respect to the DSM-IV alcohol abuse, one in four male and one in eight female current drinkers, age

18-23, had experienced at least one of the clinical features. Hazard-laden use was the most common clinical feature in this group (Harford, Grant, Yi, & Chen, 2005).

As for other countries of the world, a number of research groups have contributed to an evidence base showing that alcohol problems are not rare (Borges et al., 2005; Demyttenaere et al., 2004; McGovern et al., 2004; Rehm, Room, van den Brink, & Jacobi, 2005; Rehm, Taylor, & Patra, 2006; Soueif, Yunis, & Taha, 1986). In some Latin American countries (e.g. Brazil, Chile, and Mexico), the estimated cumulative occurrence of AUD ranged from 5% to 20%, 12-month prevalence from 4% to 10% (Andrade, Walters, Gentil, & Laurenti, 2002; Barros, Botega, Dalgalarrondo, Marin-Leon, & de Oliveira, 2007; Medina-Mora, Borges, Benjet, Lara, & Berglund, 2007; Vicente et al., 2006). Rehm and colleagues (Rehm, Room, van den Brink, & Jacobi, 2005) attempted to summarize all pertinent epidemiological studies from European countries, as published since 1990. The cumulative occurrence of DSM-IV alcohol dependence ranged from 2% to 14% in males, with substantially smaller estimates for females. As for DSM-IV alcohol abuse, the corresponding ranges were 1% to 14% (males), and 1% to 3% (females). Even in countries of Africa, such as Nigeria, where there are many of the Islamic faith who abstain from alcohol, Gureje and colleagues used a similar methodology as the NCS approach. In their Nigerian Mental Health Survey (NMHS), they found a lifetime cumulative occurrence estimate of 2.8% in a general population sample (Gureje, Lasebikan, Kola, & Makanjuola, 2006). In Australia, estimates

measured for the occurrence of alcohol dependence have been similar to the US estimates (McBride et al., 2008). In Japan and China, the values have tended to be lower than those in Europe, the US, and Australia (Demyttenaere et al., 2004; Kawakami, Shimizu, Haratani, Iwata, & Kitamura, 2004). It has been reported that in eastern countries, there is a specific alcohol-metabolizing pathway, involving ADH and ALDH, rests upon a genetic scaffold that arguably affects drinking behavior (Couzigou, Coutelle, Fleury, & Iron, 1994; Peng, Chen, Tsao, Wang, & Yin, 2007). More details of the pathway can be found in section 2.6.2. In brief, It has been found that polymorphisms in genes encoding enzymes to metabolize ethanol causes a larger proportion of Asians to have flushing effects at a lower amount of alcohol intake (Schuckit, 2009a; Y. C. Shen et al., 1997). In Japanese and Chinese, it has been argued to be a protective factor against excessive drinking. In Korean, however, the drinking culture is believed to be "drinking through flushing" (R. C. Johnson et al., 1984; J. Y. Park et al., 1984). The occurrence of AUD is almost equivalent to Europe, the US, and Australia (J. T. Park, Kim, & Jhun, 2008).

Since the population under this study belongs to the Chinese population, this paragraph provides a summary of previous studies of alcohol drinking and related problems in China. Hao et al. summarized nine epidemiological studies on alcohol-related disorders in China from 1984 to 1994. The overall occurrence of alcohol dependence varied substantially from virtually zero percent up to the 5-6% level (Hao, Chen, & Su, 2005). Although it is difficult to conduct meta-analysis of estimates from these studies due to the differences

in sampling methodology and diagnostic criteria, the occurrence of AUD has tended to be greater in studies conducted more recently compared to earlier studies. One recent WHO sponsored survey on alcohol use conducted in five areas during 2001, (n=24992), found that DSM-III-R defined alcohol used disorders (AUD) may be more common than other studies in China. The prevalence estimate for the AUD was 9% among males (7% alcohol dependence, 2% alcohol abuse) but was indistinguishable from zero in females (Huang, Zhang, Momartin, Cao, & Zhao, 2006). A study conducted in a metropolitan city in Hebei province yielded 22% overall point prevalence of alcohol abuse with 30% and 5% for males and females, respectively (Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004). However, results from these studies are not directly comparable because of differences in assessments. For example, in two studies conducted by Hao et al., DSM-III criteria were used to assess cases of AUD. The Hebei study by Jiafang et al. used a screening test that included a combination of quantity of alcohol consumption and some alcohol induced problems: a score of 8 and above qualified the drinker as a case of alcohol abuse. These differences in case definition preclude direct comparison of results between these studies and other studies.

With respect to secular trends in China, summarizing the three studies conducted during 1993 to 2001 by Hao et al., thre appears to be a quite stable trend of AUD (Hao et al., 2004; Wei, Derson, Xiao, Li, & Zhang, 1999; Wei et al., 1995). In summary, for China, the occurrence of AUD is around 8% in males and is quite rare among females.

2.5.3 Time-varying characteristics

There are time-varying characteristics that have been found to be associated with drinking-related problems as well, including marital status, occupation, income level, educational attainment, and religion. The association between AUD and these characteristics has not been entirely consistent (Andrews, Henderson, & Hall, 2001; J. C. Anthony, Warner, & Kessler, 1994; Crum, Chan, Chen, Storr, & Anthony, 2005; Crum, Helzer, & Anthony, 1993; Crum, Storr, & Anthony, 2005; Gureje, Lasebikan, Kola, & Makanjuola, 2006; D. S. Hasin, Stinson, Ogburn, & Grant, 2007; Huang, Zhang, Momartin, Cao, & Zhao, 2006). In addition, the real meaning of these characteristics can vary under different contexts. As such, special caution is required when comparing these characteristics from one society to another. Islamic religion has been associated with abstinence from alcohol in both individual level and etiological level studies (Michalak, Trocki, & Bond, 2007; WHO, 2004b).

Despite inconsistencies, some general patterns emerge in specific contexts. For example, with respect to marital status, AUD seem to occur more frequently among the separated-divorced, and among the never married; there is some evidence of late-life incidence of AUD, perhaps in connection with the experience of becoming a widower (Power, Rodgers, & Hope, 1999; Prescott & Kendler, 2001). With respect to educational attainment, the work of Professor Rosa Crum indicates that dropout in high school is associated with higher incidence of AUD (Crum, Chan, Chen, Storr, & Anthony, 2005; Crum,

Helzer, & Anthony, 1993; Crum et al., 2006). With respect to occupation, Mandell et al, in the US, found several occupations with especially high prevalence of AUD (e.g. construction and transportation), and some with especially low prevalence (e.g. white-collar occupations). Reed et al. linked drug problems to psychosocial dimensions of the work environment (Reed, Anthony, & Breslau, 2007). With respect to income, in the US, there is an inverse association between income level and alcohol dependence (D. S. Hasin, Stinson, Ogburn, & Grant, 2007; Keyes & Hasin, 2008). In China, there is little evidence on these associations, and the present investigation will be one of the first to provide empirical estimates on these topics. However, Zhao et al. found that being married and divorced (compared to never married), workers and government officials (compared to students) are more likely to be a current drinker (X. Zhou et al., 2006).

Numerous cross-section studies have found that earlier age of the first drink is associated with higher occurrence of later negative drinking-related consequences, e.g. heavy drinking, socially maladaptive drinking, and alcohol dependence (Chou & Pickering, 1992; O'Grady, Arria, Fitzelle, & Wish, 2008; Rothman, DeJong, Palfai, & Saitz, 2008). Follow-up studies also found that earlier age of drinking is associated with higher AUD incidence (Dawson, Goldstein, Patricia Chou, June Ruan, & Grant, 2008; Kendler, Prescott, Neale, & Pedersen, 1997). One limitation of these studies is from the fact that individuals with earlier initiation of drinking are exposed to alcohol effects for a longer time period compared to later-onset individuals, and therefore have

had more time to develop AUD. Time-to-event analytical tools serve better in this context, and have been used in several studies to confirm that earlier onset of drinking is associated with more rapid development of alcohol dependence (DeWit, Adlaf, Offord, & Ogborne, 2000; Hingson, Heeren, & Winter, 2006). DeWit and colleagues found a graded inverse association between age of first drink and the occurrence of alcohol dependence. In a 10 years span after the first drink, the estimated incidence of alcohol dependence was one percent in those who had their first drink after 19, compared to 16% in those who had the first drink when they were 11 or 12 years old (DeWit, Adlaf, Offord, & Ogborne, 2000). It can be argued that the observed higher occurrence of AUD in earlier onset drinkers is due to some background or predisposition not well-controlled in these studies, e.g., family history, childhood adversities, or early mental disturbances. Mixed evidence has resulted in studies that controlled for these possible confounding such as family history (FH). As might be expected, studies with larger sample sizes find a statistically robust age of onset associations with FH controlled (e.g. Dawson, Goldstein, Patricia Chou, June Ruan, & Grant, 2008), but studies with smaller samples have not (e.g. King & Chassin, 2007; Warner & White, 2003). Using latent variable analysis techniques, Kuo and colleagues found in twin-pair data that earlier onset of drinking was associated with greater alcohol problems, and found it predicted younger ages of onset of regular drinking, as well as the first alcohol dependence clinical feature (Kuo, Aggen, Prescott, Kendler, & Neale, 2008).

In some research, AUD and other drinking-related outcomes have been found to occur more often in individuals with adverse experiences during their childhood years, as compared to individuals whose childhood did not include these adversities. Because childhood adversity is one of the main topics of this dissertation, a more detailed literature review on this topic appears in a later section of this dissertation. To the best of the author's knowledge, there have been no China studies on the topic of early age of drinking or childhood adversity and occurrence of AUD.

2.6 The third rubric of epidemiology: cause

2.6.1 Causes in epidemiology

There are four main types of causal inference approaches that are commonly utilized in epidemiological studies, namely graphical models, counterfactual models, sufficient-component cause models, and structural-equations models (Greenland & Brumback, 2002). These four methods are not mutually exclusive. In many circumstances, they are transferable (Flanders, 2006). Each method has its own advantages and disadvantages compared to the others (Parascandola & Weed, 2001). The Scottish philosopher David Hume was the first to explicitly define a cause from the counterfactual perspective as *"if the first object had not been, the second never had existed"* (Hume, 1977). Although this original statement is deterministic, the counterfactual definition of cause can be modified to be probabilistic: "if the first object had not been, the probability of the second would have changed" (Parascandola & Weed, 2001). Over the past century, the counterfactual approach of causal

inference has been serving as the foundation of many quantitative methods used in epidemiological research (Greenland & Brumback, 2002). Guided by this operational definition, this section reviews previous studies on causes of drinking-related problems.

As with many prevalent human diseases that surface to prominence, the causes of drinking-related problems include many genetic and environmental factors, and their interplay. Among various possible causes, alcohol is a necessary cause, but is not sufficient. In fact, none of possible causes will be sufficient by itself, as exposure to alcohol will be a necessity, either as a self-administered exposure or a passive exposure (e.g. drinking by one's mother with a subsequent fetal alcohol spectrum disorder).

2.6.2 Macro-social influences

There is widespread agreement that socially shared macro-social influences can contribute to the individual-level risk and the population-level occurrence of drinking-related problems. For example, Professor Harold Holder maintains that alcohol problems can be controlled via careful manipulation of community-level variables such as alcohol price (or taxation), policies (Andreasson, Holder, Norstrom, Osterberg, & Rossow, 2006; Holder, 2007). These are socially shared "policy instruments" that would have functional significance as "causes of incidence" (Rose, 2001) in that they can account for population-level variation in the occurrence of drinking-related problems, even if they cannot be studied at the level of individuals. Some other examples of macro-social "causes of incidence" variables are divorce

proportion (Fillmore, Golding, Leino, Ager, & Ferrer, 1994), unemployment proportion, crime in the society (Ager et al., 1996), and strictness of law enforcement (Sloan, Reilly, & Schenzler, 1994). A woman's position in the social structure of society has been found to be associated with proportion of drinking problems in women and macro-level forces that influence equality of access and opportunity in the workplace may qualify as causal influences at this level (Rahav, Wilsnack, Bloomfield, Gmel, & Kuntsche, 2006). Recently Room, Schmidt, Rehm, and Mäkelä have argued that increasing affluence at the national level (e.g. due to globalization) will cause increased alcohol consumption and increased incidence of alcohol related hazards (Room, Schmidt, Rehm, & Makela, 2008).

2.6.3 Meso-level influences

Besides macro-level variables, some meso-level variables, which lie between social structural and individual level, have also been suggested as potential influences on drinking behavior and drinking-related problems. Derived from social learning theory (Petraitis, Flay, & Miller, 1995), peer influence has been one of the most commonly studied meso-level variables in adolescent populations. Numerous studies have found that peer alcohol drinking, peer encouragement of drinking, and peer deviance all have possibly causal influences on adolescent drinking behaviors, early-onset drinking, and associated problems (Ary, Tildesley, Hops, & Andrews, 1993; Blackson & Tarter, 1994; Coombs, Paulson, & Richardson, 1991; S. C. Duncan,

Duncan, & Strycker, 2006; Hawkins, Catalano, & Miller, 1992; Quine & Stephenson, 1990; Wu, Lu, Sterling, & Weisner, 2004).

Apparently, this peer influence on drinking behavior is not completely attributable to levels of parental drinking problems, or individual-level predispositions to experience of drinking problems (Bahr, Marcos, & Maughan, 1995; Barnow, Schuckit, Lucht, John, & Freyberger, 2002; Wood, Read, Mitchell, & Brand, 2004). One especially compelling study of peer influence on heavy drinking involved a randomized experiment for which incoming university freshmen were assigned at random to the dormitory roommate. The randomization created pairs of roommates, two freshmen, sometimes both, sometimes one, and sometimes neither of whom entered university with a prior history of heavy drinking. They found that male students with a history of heavy drinking had higher levels of alcohol consumption when pairing with a roommate with a history of heavy drinking as well, as compared to pairing with one without such a history (G. J. Duncan, Boisjoly, Kremer, Levy, & Eccles, 2005). Although there have been suggestions for a more appropriate analytic strategy and more fine-grained methods to better understand the mechanism, this study provided empirical evidence of peer influence on drinking. In addition, several intervention studies have found that peer-led intervention can be effective in reducing adolescent drinking. Inclusion of peers in intervention sessions optimized estimated effects of alcohol reduction programs (Perry et al., 1989; Rowe et al., 2007; Tevyaw, Borsari, Colby, & Monti, 2007).

Other suspected meso-level variables include sibling influence (Trim, Leuthe, & Chassin, 2006), school policies (Desousa, Murphy, Roberts, & Anderson, 2008), and living in dormitories while in residence at college (Barnes, Welte, & Dintcheff, 1992). In the absence of experimental evidence, it is possible that these just-listed researches should be reviewed under the second rubric of epidemiology (location)-that is, until more compelling evidence for causal influence has been gathered. The evidence on these researches is not as compelling as the evidence on peer influence.

2.6.4 Micro-level influences

As reviewed under the heading of the second rubric, it is widely agreed that there is family-genetic predisposition for AUD. For instance, twin studies have found that the concordance of alcohol dependence is greater in monozygotic twins, who share approximately the same individual-level genome, than that in same-sex dizygotic twins, who on average share half of that genome (Kessler, Davis, & Kendler, 1997). In general, twin studies and adoption studies have yielded heritability in the range of 50% to 60% for alcohol dependence (Dick & Bierut, 2006; Merikangas, 1990; Prescott et al., 2005; Schuckit, 2009b). These studies estimate the proportion of AUD variance that can be attributed to genetic factors. However, they do not pinpoint specific loci or region in the human genome that might account for the occurrence of AUD, and that might become future targets for intervention.

Recent advances in genetic engineering have made it possible to manipulate gene polymorphisms in mice. For example, to study functions of

specific genes, the knock-out technique can turn off a specific gene locus to study functional changes (Hooper, Hardy, Handyside, Hunter, & Monk, 1987). In contrast, the knock-in technique can insert a specific gene (Kuehn, Bradley, Robertson, & Evans, 1987). Using various gene targeting techniques, animal studies have found that some specific polymorphisms in selected genes increase or decrease alcohol intake in mice. The most extensively studied genes are those encoding neurotransmitters, such as GABA, dopamine, and serotonin; cell adhesion genes; and protein kinase genes (Crabbe, Phillips, Harris, Arends, & Koob, 2006; Hishimoto et al., 2007; Newton & Messing, 2006; Racz et al., 2003; Werner et al., 2006). It is believed that some of these genes account for loci identified by GWA studies, and there is a convergence in evidence between animal studies and human GWA studies (Uhl et al., 2008). However GWA studies have revealed alcohol dependence associated loci on 17 out of the 23 human chromosomes (Ehlers et al., 2004; Zlotnick et al., 2006). Furthermore, recent discoveries in epigenetics and gene expression have made the understanding of causes of alcohol dependence even more complicated. Therefore, multi-disciplinary effort is needed to draw the complete picture of alcohol dependence, and to trace specific genetic meditational pathways that account for intergenerational "transmission" of susceptibilities for alcohol dependence (J. Liu et al., 2006; Uhl et al., 2008).

Besides genetic factors, environmental factors play important roles in AUD as well. Estimated from twin studies and adoption studies, environmental factors account for more than 40% of the variance (Agrawal & Lynskey, 2008;

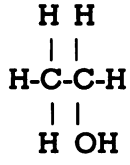
Kendler, Myers, & Prescott, 2007; J. Liu et al., 2006; Prescott et al., 2005). Various environmental factors have been investigated. However, due to underlying heterogeneities and unobserved confounding variables, it is not easy to infer definite cause-effect relationships from observational studies. Many (but not all) studies have found evidence of interactions between genetic and environmental factors, e.g. the 5-HTT genotype and stressful life events (Caspi & Moffitt, 2006; Dai, Thavundayil, Santella, & Gianoulakis, 2007; Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001; Schuckit & Smith, 2006; Schuckit et al., 2005). Among many possible environmental causes, child abuse and neglect has been investigated as a suspected "cause of cases" and determinant of the individual-level risk of becoming a case of AUD by some research teams. The dissertation returns to this topic in section 2.7, which covers the possible role of child abuse/neglect as an early life condition that might account for variation in individual-level risk of AUD and related problems.

2.7 The fourth rubric: mechanism

This section is organized in relation to the following topics: 1) chemistry and pharmacology of ethanol, 2) metabolism and biotransformation of ethanol, 3) reinforcing effects of alcohol, 4) natural history of alcohol problems, 5) comorbid condition, and 6) alcohol-related disabilities and impairment, including secondary social maladaptation and hazard-laden drinking.

2.7.1 Brief introduction of chemistry, pharmacology of ethanol

In chemistry, alcohol can be said to be any organic compound where "a hydroxyl group (-OH) is bound to a carbon atom of an alkyl or substituted alkyl group" (Kopnisky & Hyman, 2002). In simple English, an alcoholic beverage refers to a drink containing the chemical drug compound known as ethanol. Ethanol is the principal, active ingredient in alcoholic beverages, traditionally produced by fermentation (fermentation involving the metabolism of carbohydrates by certain species of yeast under anaerobic conditions). The chemical composition of ethanol is written as



Ethanol generally is colorless, volatile, and water soluble with a mild odor.

Ethanol in humans often shows biphasic responsesin CNS (central nervous system)-mediated behavioral functions, with a more prominent depressant effect to the CNS at higher doses, and sometimes with disinhibition of behavior that can simulates 'stimulant' outcomes at lower doses. These effects seem to be mediated via certain subtypes of the gamma-aminobutyric acid A (GABA-A) receptors and inhibition of NMDA (N-methyl-D-aspartic acid) glutamate receptors. These neurotransmitter mechanisms are intermediaries for relaxation, release from anxiety, sedation, and lowering of inhibitions, with apparent 'stimulation' of behavior-hence, the biphasic response. Nevertheless, higher doses, some other targets are also involved, such as sodium channels, serotonin, as well as dopamine receptors and other

psychopathological pathways, and the CNS depressant response become more salient. Whether there are two separate mediating mechanisms is not yet clear (Boehm et al., 2004; Bowirrat & Oscar-Berman, 2005; Gorwood et al., 2000; Kopnisky & Hyman, 2002; Mihic et al., 1997). At the higher doses, ethanol impairs sensory and motor functions and can slow cognition. An extremely high dose of ethanol can cause unconsciousness and possible death. Meta analysis suggests that the LD50 (half lethal dose) of ethanol is 10,300 mg/kg for rats, 6800mg/kg for mouse and the LD50 in humans is estimated to be 330g (276-455) for a 70kg healthy adult, which translates into approximately 16 bottles of half liter beer with 5% alcohol by volume (Gable, 2004). Thus, ethanol can be lethal; the lethal risk is usually higher for people with a smaller volume of body water, such as children, women, and low body mass individuals. However, clinical significant CNS depressant effects take place at sub-lethal levels; these effects happen much more commonly than lethal overdose, and are more relevant to health conditions than is the lethal threat directly from ethanol. With respect to dos-response relationships, Goldberg evaluated the behavioral and physiological changes in 160 healthy volunteers. Evidence from regression models supports the idea that intoxication can appeare as low as 22mg/DL of blood alcohol concentration in humans (GOLDBERG, 1966). The legal threshold for blood alcohol concentration typically ranges from 50 to 80 mg/DL for most Western countries.

2.7.2 Metabolism and biotransformation of ethanol

Through a series of reactions, complete metabolism of ethanol produces water and carbon dioxide. The major reactions can be depicted as,

C_2H_6O(Ethanol)→C_2H_4O(Acetaldehyde)→$C_2H_4O_2$(Acetic Acid) →Acetyl-CoA→H_2O+CO_2 (Kopnisky & Hyman, 2002)

The enzyme alcohol dehydrogenase (ADH) oxidizes ethanol into acetaldehyde, which is then converted into the relatively harmless acetic acid (vinegar) by acetaldehyde dehydrogenase (ALDH) (Kopnisky & Hyman, 2002). Acetaldehyde, an intermediate product of ethanol metabolism, also is toxic, with negative health effects. Acetaldehyde is listed as a probable human carcinogen by the US Environmental Protection Agency (U.S.EPA, 1994). Ingestion of too much acetaldehyde can cause a cluster of unpleasant effects characterized by facial flushing, dehydration, headache, palpitations, nausea, and vomiting, often characterized to as a "hangover" (Harada, Agarwal, Goedde, Tagaki, & Ishikawa, 1982). As described above, the concentration of acetaldehyde depends on the ethanol intake, the amount of ADH (synthesizing acetaldehyde from ethanol), and the amount of ALDH (degrading acetaldehyde into acetic acid). There is wide variation in the level of ADH and ALDH across individuals, which results in different rates of ethanol metabolism. Low levels of ALDH or high levels of ADH cause acetaldehyde to accumulate through increased synthesis and/or decreased metabolism.

Genetic research has disclosed that the functional variants in genes encoding ADH and ALDH can account for variations in levels of ADH and/or ALDH (Edenberg et al., 2006; Kuo, Aggen, Prescott, Kendler, & Neale, 2008). It

has been widely reported that in a larger proportion of Asian populations (e.g. Chinese, Japanese) these allele variants can cause more rapid and longer-lasting accumulations of acetaldehyde, which in turn can discourage additional alcohol intake due to the soon-appeared unpleasant effects (C. C. Chen et al., 1999; Y. C. Shen et al., 1997; Thomasson et al., 1991). On the other hand, regardless of individual variations in enzymes to digest ethanol, excessive intake of ethanol can cause accumulation of acetaldehyde in the human body.

2.7.3 Reinforcing effect of alcohol

Alcohol is a psychoactive drug that serves reinforcing functions. Via the neurotransmitter mechanisms already discussed, alcohol may disturb the reward circuit by interfering with neurotransmitters and their receptors, such as the D2 dopamine receptors (DRD2), glutamate, serotonin, and the GABA-A receptors (Kopnisky & Hyman, 2002; Lewis, 1996). Whereas the exact mechanisms underlying the reward system are not yet fully understood, it is known that these neurotransmitter systems can work interactively to yield reinforcement of the drinking behavior (Lewis, 1996). This reinforcement might be distinct from so called "natural rewards"- those sought be avoid death from starvation, e.g. associated with hunger. Whereas some scholars have described this reward effect as an acquired "pleasure" from alcohol intake, from the behaviorism perspective, ethanol can function as a positive reinforcer for sustained drinking behavior, operationally defined as an event

that increases the probability of a subsequent event, with no appeal to the lay concept of "pleasure".

Besides positive reinforcement, alcohol also can serve a negative reinforcing function. This function was described by Solomon and Corbit in their 'opponent process theory,' which hypothesizes that the initial drug intake induces a 'hedonic' state. In response, the CNS automatically seeks homeostasis, and with a counteraction to reduce the intensity of the 'hedonic' effect. After the drug wears off, this CNS-mediated counteraction persists, inducing a negative emotional state (Koob, 2006; Solomon & Corbit, 1974). Subsequently, individuals may increase consumption or drug-seeking behavior in order to relieve this acquired negative emotional state. According to the theory, the positive reinforcement that can cause decreased reward thresholds, and with the negative reinforcement, there is an increased self-administration of alcohol. Thereafter, positive and negative reinforcement work together to preoccupy the individual with drug-related activities. Guided by this theory, animal studies find consistent evidence (Koob, 2006).

Alcohol's reinforcing function maybe stronger for some individuals than the drive state required by hunger for food. Similar behavioral mechanisms may influence dependence syndromes that involve other drugs (e.g., cocaine) and behavior functions of the non-drug behavioral repertoire, such as sports activities, or musical performance and practice, and gambling (C. Y. Chen et al., 2004; Wightman & Robinson, 2002). As the exogenous alcohol reinforcers become established, a drinker may develop alcohol dependence.

The "internal" functions and biochemical changes, which coincide with alcohol's reinforcing functions, cannot be observed by unaided naked eyes. They can be studied with brain imaging techniques and can be reflected in behavioral manifestations, such as compulsion-like drinking behavior, tolerance, withdrawal, and the other facets of the alcohol dependence syndrome. The positive reinforcement is believed to be a crucial mechanism behind alcohol tolerance, with negative reinforcement in a similar position with respect to alcohol withdrawal. Compulsive drinking and lost of control over alcohol is a manifestation of both positive and negative reinforcement (Koob, 2006). In addition to neuroadaptational changes that coincide with pharmacological tolerance and withdrawal, there also may be alcohol-related social maladaptation and interpersonal and social problems as well (e.g., drink-induced violence, family or legal troubles, and drunk driving).

2.7.4 Possible natural history of AUD

Our understanding of the natural history of alcohol use and related problems has been advanced with long term longitudinal studies in which attrition has been limited. Due to logistical difficulties, these studies are rare. Nonetheless, these studies have found that the remission of alcohol use in AUDs occurs frequently (Vaillant, 1996; Vaillant & Milofsky, 1982), with little evidence of male-female differences in the course of alcohol dependence (Schuckit, Daeppen, Tipp, Hesselbrock, & Bucholz, 1998). Well designed cross-sectional studies can provide valuable insight into the natural history albeit there can be limitations such as recall bias and incomplete reporting of

past events. For example, findings from the cross-sectional component of the ECA study estimated a median age of onset of AUD to be 21 years and 90% of all AUD cases had experienced their first clinical feature of AUD before the age of 38 years (Helzer JE, 1991). The estimated prevalence of recent AUD decreased across age strata, from about 4% in 18-24 year olds to about 1% among those age 65 years old and above (Regier et al., 1993). A similar trend was found for cumulative occurrence in Caucasians, but not for African Americans. In African Americans, peak prevalence of AUD presented in the middle age stratum, which was 45 to 64 years old (Regier et al., 1993).

2.7.5 Comorbid conditions

Various comorbid conditions have been observed to co-occur with alcohol dependence, including depression, anxiety disorder, and tobacco dependence (Regier et al., 1990; Schuckit, 1985). Many studies have found that childhood conduct disorder, cognitive problems, and attention problems forecast later onset of fully expressed alcohol dependence (Elkins, McGue, & Iacono, 2007; Giancola & Moss, 1998; Gorenstein, 1987; Looby, 2008; Molina, Pelham, Gnagy, Thompson, & Marshal, 2007; Moss & Kirisci, 1995; Myers, Brown, & Mott, 1995). It is also widely documented that antisocial personality is associated with alcohol dependence (Harford & Parker, 1994; Stabenau, 1984). Estimated from clinical patients, onset of antisocial personality occurs about four years earlier than the onset of alcohol dependence (Bahlmann, Preuss, & Soyka, 2002; Stabenau, 1984). There has been evidence that genetic factors play a role in the observed association between these pre-existing

conditions and later alcohol dependence, and might function as confounders (Kendler et al., 2006; Stallings et al., 1997).

Epidemiological studies have also found there is elevated occurrence of mood disorders (e.g. depression and anxiety disorders) in people with alcohol and other drug dependence compared to expectations based upon the general population (J.C. Anthony & Petronis, 1989; Grant & Harford, 1995; Kessler et al., 1994; Regier et al., 1990). The self-medication theory hypothesizes that people drink alcohol to cope with emotional stress or to release their unhappiness (Quitkin, Rifkin, Kaplan, & Klein, 1972). There has been some evidence supporting the self-medication theory (Bolton, Robinson, & Sareen, 2008; Carrigan & Randall, 2003; Robinson, Sareen, Cox, & Bolton, 2009). There are also scholars who argue for common genetic vulnerability underlying these comorbid conditions (Merikangas, Leckman, Prusoff, Pauls, & Weissman, 1985; Merikangas, Risch, & Weissman, 1994; Prescott, Aggen, & Kendler, 2000). In summary, these conditions serve as pre-conditions of alcohol dependence in some groups of people.

2.7.6 Alcohol-related disabilities and impairment, including secondary social maladaptation and hazard-laden drinking

Alcohol dependence accounts for substantial disease burden via alcohol-related mortality and various health consequences including physical, emotional, and social consequences. The WHO global burden of disease project (GBD) found that AUD is one of the leading causes of disease burden in the more established market economies of the world. According to the GBD

data, in 2002, AUD claimed almost 1,800,000 lives worldwide. The peak alcohol-related mortality occurs in the age group 15-44, and the substantial AUD disease burden is due mainly to these deaths (WHO, 2004b). Nevertheless, mortality does not give the complete picture of disease burden because diseases such as AUD also cause disability and dysfunction. The Disability Adjusted Life Years index (DALYs) measures disease burden so as to reflect both premature death and disability (C.D. Mathers et al., 2003). Each AUD DALY represents one lost year of healthy life, either to AUD-caused premature death or to an "AUD-attributable" disability. Despite some limitations, the DALY index has been widely used and has been one of the main measurements of disease burden nowadays (Bastian, 2000; Reidpath, Allotey, Kouame, & Cummins, 2003). According to the WHO, in 2002, AUD was responsible for 58,300,000 DALYs (C.D. Mathers et al., 2003; WHO, 2004b).

It must be mentioned that various other physical and mental health conditions caused by ethanol exposure also contribute to the alcohol-attributable disease burden. Studies have found that alcohol consumption is a possible cause for various physical and mental conditions including cirrhosis of the liver, motor vehicle accidents, drowning, falls, poisonings, self-inflicted injuries and homicide, low birth weight, some cancers, depression, epilepsy, hypertensive disorders (Bazzano et al., 2007; Gu et al., 2007; Huang et al., 2008; Lin et al., 2005; Rehm, Taylor, & Patra, 2006; Ruixing et al., 2006; H. Zhou et al., 2003). As such, alcohol consumption is one of the top determining influences on the burden of disease globally each year. The WHO GBD

project estimated that in 2002, 1,800,000 deaths are attributable to alcohol consumption (WHO, 2004b). One third of these deaths were due to un-intentional injury, e.g. drunk driving, drowning, etc. Some of these deaths are attributable to alcohol dependence, as when persistence of drinking is explained by the presence of alcohol dependence. Moreover, alcohol consumption accounted for 58,300,000 (3.7% of total) DALYs in the same year (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; WHO, 2004b). In European countries, where alcohol consumption is higher than the global average, in 2002 alcohol drinking appears to be responsible for 10-11% of total DALYs (Rehm, Taylor, & Patra, 2006). Unless something occurs to change the current trend, AUD alone will climb up to become the fourth most burdensome disorder within the high-income countries accounting for 4.7% of the total DALYs in 2030 for those countries (C. D. Mathers & Loncar, 2006). As for China, the main focus of this dissertation, alcohol consumption poses large burden of disease as well. According to the WHO estimates, as measured in relation to determinants of DALYs, alcohol consumption ranks high, accounting for 4-8% of DALYs in China (Grimm, 2008).

2.8 The fifth rubric: prevention and control

Some community trials found that alcohol consumption, incidence of drunk driving and assault decreased after the implementations of more restrict local regulations of alcohol, such as encouraging responsible beverage service; limiting access to alcohol, especially to adolescents; and increasing local enforcement of drinking and driving laws (Holder et al., 2000;

Stafstrom, Ostergren, Larsson, Lindgren, & Lundborg, 2006; Treno, Gruenewald, Lee, & Remer, 2007). Other research teams employed intervention strategies aiming at improving social skills, e.g. the good behavior game (Barrish, Saunders, & Wolf, 1969), in school kids. Multiple studies have found that these strategies not only enhance social skills, but also delay the onset of alcohol drinking and reduce drinking-related problems (E. C. Brown, Catalano, Fleming, Haggerty, & Abbott, 2005; Kellam et al., 2008; Poduska et al., 2008; van Lier, Huizink, & Crijnen, 2008). There are reports of other prevention and intervention strategies that was suggested to be effective in reducing drinking and drinking-related problems. These strategies include parent-targeted education (Koutakis, Stattin, & Kerr, 2008), peer-led motivational intervention (Fromme & Corbin, 2004; Tevyaw, Borsari, Colby, & Monti, 2007), social norm education (Turner, Perkins, & Bauerle, 2008), incentive reward (Glindemann, Ehrhart, Drake, & Geller, 2007), etc. Some researchers also found incorporating these strategies with computer-based survey and feedback to be effective (Bewick, Trusler, Mulhern, Barkham, & Hill, 2008; Schinke, Schwinn, Di Noia, & Cole, 2004).

2.9 Possible causal influence of childhood physical abuse (CPA) and drinking-related problems

This section of the dissertation is focused upon a sub-topic of the research, namely, the possibility that childhood physical abuse (CPA) might influence drinking-related problems. Current evidence is reviewed under the nine

guidelines that are used when evaluating the potential causal significance of exposure-disease associations in epidemiology (Gordis, 2004; United States Department of Health, 1964). Table 2.1 lists the nine guidelines and the main issues or questions under each guideline.

Table 2.1. Nine guidelines and corresponding main questions

guidelines	main question
Temporal relationship	Does the exposure occur before the disease/condition?
Strength of association	How strong is the association?
Dose-response relationship	Does the risk of disease/condition increase when the dose of exposure increases?
Replication of the findings	Do different studies yield the same results?
Biologic plausibility	Is it coherent with biologic knowledge?
Consideration of alternate explanations	Could the observed association been explained by confounders? Could the observed association been explained by model misspecification?
Cessation of exposure	Does the risk of disease/condition decline when exposure is reduced or eliminated?
Consistency with other knowledge	Is the finding consistent with findings from other data?
Specificity of the association	Is the association specific to the disease?

2.9.1 Strength of association and replication of findings

Retrospective case-control studies with clinical samples have found that odds of AUD are elevated among patients with history of CPA as compared to controls, and that CPA-associated cases had experienced more drinking-related problems (G. R. Brown & Anderson, 1991; Downs, Capshew, & Rindels, 2004; Kunitz, Levy, McCloskey, & Gabriel, 1998; Swett, Cohen, Surrey, Compaine, & Chavez, 1991). This association also was found in comparisons of AUD adolescents and community controls (D. B. Clark, Lesnick, & Hegedus, 1997). These studies provide initial evidence of the association between CPA

and AUD. Regrettably, these studies may have suffered from a major limitation, usually referred as "Berkson's bias," as can happen when cases are recruited from clinical settings or from intervention programs. The clinical population, consisting of treatment seeking individuals, may differ from the general population in many different ways including demographic characteristics, such as sex, age, and ethnicity, as well as aspects of personal history, such as comorbid illnesses or CPA histories. Unless this "transition bias" or "selection bias" can be taken into account, these differences may lead to biased estimates of the CPA-AUD association. Additionally, individuals with both childhood abuse history and alcohol problems may be more likely to seek treatment than those with only one or none, in a realization of potential Berksonian bias. As such, there is need for more general population-based research on this issue.

In one of the earliest population based studies on the CPA-AUD associations, there was some evidence of a tangible association between earlier CPA and later AUD (Holmes & Robins, 1987, 1988). Later on, estimates from the NCS, with its nationally representative adult sample of US household residents, indicated a weak but statistically robust CPA-AUD association (OR=1.3; 95% CI, 1.1, 1.6; Afifi, Brownridge, Cox, & Sareen, 2006). Based on similar survey methodology, the Ontario Health Survey also found that people with a history of CPA had an elevated odds of AUD (OR=1.8, 95% CI=1.4, 2.3; MacMillan et al., 2001). Besides CPA, a history of slapping and spanking, and milder forms of physical punishment, were also associated with excess odds

of AUD (MacMillan et al., 1999). A smaller CPA-AUD association also has been observed in more restricted non-clinical populations, such as college freshmen, prisoners, lesbians, and Marine recruits (Carrigan & Randall, 2003; Sher, Gershuny, Peterson, & Raskin, 1997; Trent, Stander, Thomsen, & Merrill, 2007).

As now can be summarized from case-control and cross-sectional research with clinical and non-clinical population samples, the strength of the association can be characterized as weak to moderate, with ORs generally in a range from 1.2 to 2.5 (few study provided estimates for Relative Risk and its standard error). Based upon evidence of this type, Simpson & Miller have already concluded that there is evidence of a possible causal relationship between CPA and AUD in females. In males, findings were inconsistent (Simpson & Miller, 2002).

2.9.2 Consideration of alternate explanations

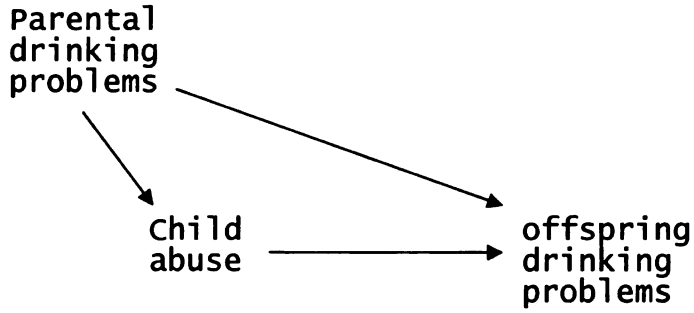
The consideration of alternative explanations guideline is attached to many names. In epidemiology, these "alternative explanations" often are groped under the heading of "confounding" variable. In econometrics, it might be said that alternative explanations are sources of unspecified heterogeneity in the outcome. One of the main plausible confounding variables or source of heterogeneity on the outcome is parental drinking, which might account for the CPA as well as AUD susceptibility. Children from alcoholism-affected families are more likely to be victims of childhood abuse (DiLalla & Gottesman, 1991; Dube, Anda, Felitti, Croft et al., 2001; Widom &

Hiller-Sturmhofel, 2001); AUD is a condition known to aggregate within families and to show heritability (Dick & Bierut, 2006).

In a few of the just cited studies conducted in general population samples, there has been a null association between CPA and risky drinking or AUD, once parental drinking problems are taken into account. The population subgroups studied in this research has included Marine recruits, lesbians, prisoners, and US Indian tribes (Hughes, Johnson, Wilsnack, & Szalacha, 2007; Koss et al., 2003; Libby et al., 2004; Mullings, Hartley, & Marquart, 2004; Young, Hansen, Gibson, & Ryan, 2006). Only one study found with a sample recruited from a primary care setting, found a possibly non-null association between CPA and self-defined alcoholism. In this study, parental drinking problems were collected from offspring (Carrigan & Randall, 2003).

In these studies which took parental drinking problems into account, the generalized linear model (GLM) has been used to estimate a regression coefficient linking CPA to parental drinking problems. One possible problem in this approach comes from an assumption of GLM that covariates are independent. In other words, there might be a violation of an "exogeneity" assumption: error terms are supposed to be independent for each covariate. One plausible relationship between parental drinking problems, CPA, and drinking problems in the offspring is depicted in figure 2.1, such that the exogeneity assumption might be violated (Engle, Hendry, & Richard, 1983).

Figure 2.1 A conceptual relationship between parental drinking problems, CPA, and offspring drinking problems



Therefore, CPA is reasonably an endogenous variable with respect to parental drinking problems, and a simultaneously regression of offspring drinking problem on both parental drinking problems and CPA might have violated the assumption of independence. Violation of this assumption can cause bias as well as inconsistency in estimation (Briscoe, Akin, & Guilkey, 1990; Felitti et al., 1998). Resolutions of this endogeneity problem may include use of instrumental variable methods, which originated and are commonly used in econometrics, as well as structural equation modeling (SEM) (Cameron & Trivedi, 2009; Greenland & Brumback, 2002).

2.9.3 Temporal relationship

In much of the prior research, the retrospective design made causal inference difficult due to uncertainty about of temporal sequencing. This uncertainty can be traced to use of cross-sectional study designs and retrospective methods subject to differential recall biases and differential survivorship (left-censoring). In the few available prospective studies investigating the association between CPA and AUD, the evidence is mixed.

For example, Jasinski et al. showed that CPA predicted heavy drinking in 113 African American childhood victims (OR=8.7, 95% CI, 1.9, 40.0) holding parental relationships, parental drinking, and sexual abuse constant (Jasinski, Williams, & Siegel, 2000). In Horwitz et al. (2001), 908 abused children and 667 non-abused children were identified from court records. The "abused" exposure group and a court-referred control group were matched on sex, age, race/ethnicity, and socio-economic status (SES). Among the 61% followed up for approximately 20 years, the abused children had increased risk of AUD development, statistically robust for females only. In deed, with stressful lifetime events taken into account via regression models, the obserd male childhood experience a lower risk of AUD (Horwitz, Widom, McLaughlin, & White, 2001; Widom, White, Czaja, & Marmorstein, 2007). It is noteworthy that in this study, although children in the control group were referred to the court for reasons other than childhood abuse or neglect, it is possible that some of them also had suffered from childhood abuse or neglect, which might have biased estimates toward the null for both males and females. Jackson and Sher also completed longitudinal research, based on a sample of 489 incoming college freshmen followed for 11 years. They found that the association between childhood stressors and adulthood AUD was attenuated at $p>0.10$ when family history of drinking problems was included in their SEM: the estimate of an effect for childhood stressor on AUD diminished considerably and was not statistically robust after the family history of alcoholism was taken into account. Although the attrition level (22%) was fairly low in this study,

AUD predicted attrition (Jackson & Sher, 2003). In these two studies, childhood abuse (sexual or physical) and neglect were combined into one variable. Thus, the effect of each specific type of experience is not known.

Another longitudinal study with a baseline sample of school-recruited students (n=1634), but substantial attrition (>60%), also suggested a null association between childhood physical or emotional abuse and AUD after taking other childhood adversities, such as parent divorce, family support, and childhood sexual abuse, into consideration (Galaif, Stein, Newcomb, & Bernstein, 2001). However, besides the limitation of high level of attrition, the history of childhood maltreatment in this research was based on retrospective recall at the time of the follow-up assessment, which compromises validity of this study.

The study by Galaif and colleagues raises issues of note. Since the assessment of parenting is concurrent with the assessment of AUD, even if the study design is longitudinal or prospective, this study might be envisioned as a case-control design or cross-sectional design regarding the CPA-AUD association. Nonetheless, an AUD generally develops over a long time, such that follow-up over long spans of time is a requirement if the CPA assessment is to precede the AUD assessment, and this makes the study vulnerable to attrition, with differential attrition as a potentially severe complication.

For this reason, epidemiologists typically will conduct a series of case-control studies, with incidence cases matched to non-cases who passed through the same interval of risk without developing AUD. Then, in accord

with the case-control design, there is a look back (among both cases and controls) to whether an early-life exposure might be observed more frequently among the cases as compared to controls. This design is especially powerful when the early-life experience has a discrete quality and can be placed in time early in life, well before development of the outcome. For example, it is possible to ask cases and controls about their childhood experiences with interview methods that make the study subjects unaware that these experiences will be studied in relation to a specific outcome such as AUD. Then, the occurrence of the outcome can be evaluated for post-childhood years, with knowledge that AUD rarely start during the childhood years. Accordingly, it may be best to postpone longitudinal and perspective research on the CPA and AUD association until after case-control research has been completed to gauge the size of the association, which must be estimated with some fidelity.

Because there are no prospective or longitudinal studies of CPA and AUD in China, and because the logistical problems of epidemiological research on CPA-AUD associations have not been studies, this dissertation involves use of one of the case-control design protocols with an attempt to sort out the temporal sequencing issue by employing reference to time frames in assessments of CPA and AUD: CPA experience "when the respondent was growing up" and the age at onset of AUD and related drinking behaviors. More details are provided in the methods section.

2.9.4 Dose-response relationship

The author is not aware of any study showing the dose-response relationship specifically for CPA to AUD. However, there is evidence that as the number of childhood adversities increases, the risk of alcoholism increases (Felitti et al., 1998).

2.9.5 Biological plausibility

Neurobiological studies have shown the biological plausibility of the long-term effect of childhood adversities. For example, animal studies in monkeys showed that being raised in isolation induces abnormal activities in their hippocampus (R. G. Heath, 1972) and reduces corpus callosum volume (Holder et al., 2000). Studies in rats showed that rats subjected to low levels of maternal care showed alterations in the structure and function of GABA-A receptors (Caldji, Diorio, & Meaney, 2003), and suppression of neurogenesis (Teicher, Tomoda, & Andersen, 2006). People with a history of childhood abuse showed similar changes, such as abnormal electroencephalogram (EEG), smaller volume of hippocampus and prefrontal cortex, altered cortical symmetry in frontal lobes, reduced neuronal density in the anterior cingulate, etc. (Bremner et al., 1997; Teicher, Tomoda, & Andersen, 2006). These changes have also been shown in people with drinking problems, especially the frontal lobes, the limbic system (including hippocampus), and the cerebellum (Oscar-Berman & Marinkovic, 2007). Although the causal relationship cannot be established from these observations in AUD patients, they provided evidence for the biological plausibility of the association between childhood stressors and AUD.

2.9.6 Specificity of the association

With respect to the specificity, Brown & Anderson showed that in clinical patients, AUD is more common in CPA victims compared to sexual abuse victims, while no such difference was found in Axis II disorders and suicidality (G. R. Brown & Anderson, 1991). Green proposed that, compared with victims of sexual abuse, victims of physical abuse had more problems in aggression modulation (Green, 1988). However, population-based studies have shown that CPA is associated with a wide range of mental conditions, including mood disorder, anxiety disorders, suicide ideation, antisocial behaviors, and personality disorders (Dube, Anda, Felitti, Chapman et al., 2001; Kessler, Davis, & Kendler, 1997; MacMillan et al., 1999; MacMillan et al., 2001; Pollock et al., 1990; Windle, Windle, Scheidt, & Miller, 1995). Due to the high comorbidity and the overlap in etiology of mental disorders, it is difficult to infer the specificity of the effect of CPA. And animal studies suggested that childhood stressors cause changes in multiple brain regions and neurotransmitters. To our knowledge, there has been no study showing a specific association between CPA and a single trait or biomarker.

2.9.7 Possible mediating pathway

Several studies have examined the mediating pathway from CPA to drinking and AUD. Based on data from the follow-up study of court record recruits as mentioned above, Schuck & Widom explored the mediating pathway using SEM from CPA to AUD. Results suggested mediation through depression and using alcohol/drug to cope with difficulties. No such

mediation was found through worthless, isolation/loneliness, and low self-esteem (Schuckit et al., 2001). Analysis based upon the NCS showed that the association between CPA and AUD is completely explained by childhood conduct disorder (Kessler, Davis, & Kendler, 1997). Zlotnick et al.'s study in clinical patients suggested that the effect was potentially mediated by posttraumatic stress disorder (PTSD) (Zlotnick et al., 2006). Tarter et al. proposed that early adverse environment causes neurobiological deregulations in children and causes alcohol and drug problems through series manifestations during childhood and adolescent, such as emotional/behavioral deregulations, externalizing difficulties, and antisocial personality (Blackson & Tarter, 1994). Furthermore, studies in adolescents and young adults showed CPA was associated with earlier onset of drinking and heavy drinking (Bensley, Spieker, Van Eenwyk, & Schoder, 1999; Brems, Johnson, Neal, & Freemon, 2004; Riggs, Alario, & McHorney, 1990; Rothman, DeJong, Palfai, & Saitz, 2008).

In summary, with respect to the inference of a causal association links CPA to AUD, it is plausible to investigate CPA as a possible cause of AUD, within the context of a conceptual model in which there is an attempt to specify temporal sequencing with CPA occurring before the onset of the AUD and with attention to parental drinking and other covariates that might structure a biased CPA-AUD association. This work builds from prior evidence of a modest to moderate strength of the CPA-AUD association, mixed evidence on the temporal sequencing, support for biological

plausibility and consistency with other knowledge, replicability, and a possible dose-response or gradient relationship. There is no evidence of specificity, such that CPA causes AUD and only AUD, but this guideline may not be applicable in the context of psychosocial research.

In China, few studies have examined the association between childhood adversities and drinking and related problems. Fairly low occurrence of CPA was shown in one study of factory workers in Shanghai, while high CPA occurrence was shown in another study of high school students in Henan province. A recent publication showed an occurrence of 4.2% of childhood sexual abuse from a representative urban Chinese sample (3.3% in women, 5.1% in men), which is considerably lower than that from US samples (e.g. 32.3% in women, 14.2% in men; Briere & Elliott, 2003; J. Chen, Dunne, & Han, 2006; Luo, Parish, & Laumann, 2008; Ross et al., 2005). In the same research in Chinese high school studnets, childhood sexual abuse was associated with higher likelihood of recent drinking (OR=2.7, 95% CI, 1.5, 5.1), history of being drunk (OR=3.6, 95% CI, 1.9, 6.8), and history of being accidentally injured while drunk (OR=5.36, 95% CI, 2.1, 13.7), but the CPA-AUD association was not investigated.

2.10 Gaps in the epidemiological evidence

In this section, the dissertation returns to the three specific aims under investigation, and places them in a larger context so that the potential significance of the dissertation research may be appreciated. Each aim

addresses a gap in the epidemiological evidence on alcohol dependence and related problems.

2.10.1 Specific aim 1

China is currently the most populated country in the world, hosting one-fifth of the people on Earth. In China, drinking is a common behavior in social contexts and it has been argued that drinking imposes substantial burden of disease (Grimm, 2008). There have been concerns that with the increasing contact with the western drinking culture and the increasing number of automobiles, alcohol related problems are likely to increase (Newman, 2002). In some research, the estimates have suggested that alcohol problems might be on the rise, especially in the urban parts of China in association with China's increasing prosperity (Zhou, et al. 2006; Hao, et al. 1995; Zhang, et al. 1999; Yang, et al. 1999; Cai et al., 1998). Although some studies have studied drinking practices and problems within China, these data may be outdated, and often have not been based upon DSM-IV or ICD-10 criteria to assess AUD (Hao et al., 2004; Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004; Y. C. Shen et al., 2006; Wei, Derson, Xiao, Li, & Zhang, 1999). Furthermore, in past surveys involving multi-stage probability sampling, with individuals nested within sampled households, households nested within sampled cities or villages, the data have been analyzed data as they were collected with a simple random sampling plan. By treating clustered data as simple randomly sampled data, the estimation of variances, standard errors, as well as confidence intervals, can be erroneous (often smaller than they should be), which can disrupt

statistical inference. This problem is especially pertinent in studies on alcohol and drug use since these behaviors are found with significant geographical and local area clustering (Bobashev & Anthony, 2000).

Moreover, in the published literature, the author found no epidemiological description of drinking patterns and behaviors or problems with representative sample from two of the biggest cities in China, Beijing and Shanghai. Nevertheless, it is important to have population-based estimates of drinking practices and problems because the drinking patterns in Beijing and Shanghai today might be the ones that are followed in other cities of China during later years. Of more public health importance is the description of riskier drinking behavior, such as heavier drinking, early onset of alcohol involvement, and socially maladaptive drinking and manifestations of alcohol dependence, topics rarely studied in past research on drinking in China.

2.10.2 Specific aim 2

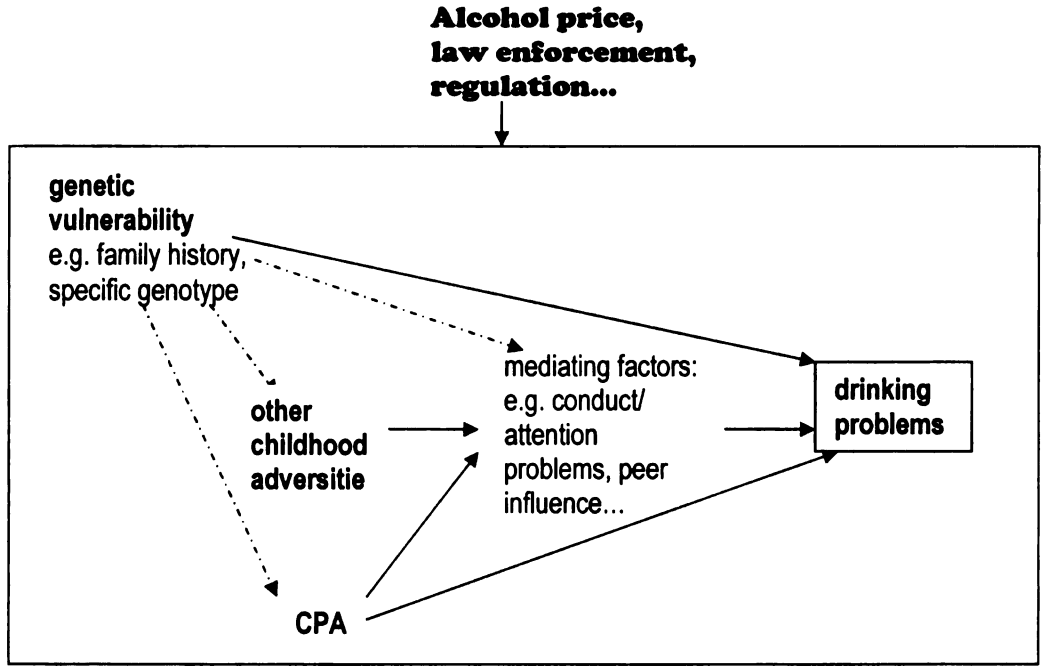
There is one prior study in China on the prevalence of drinking in relation to population subgroups, such as males vs. females, but there is no prior research in China on the prevalence or occurrence of alcohol problems in these subgroups. This dissertation, for the first time in China, will present epidemiological estimates for these public health problems. In addition to the estimation of the sub-group specific parameters, the dissertation involves estimation of the strength of association that links membership in the population subgroups to the occurrence of alcohol problems, first based upon a bivariate analysis and then based upon the multiple logistic regression

model, which is used to evaluate which of the observed associations might be statistically independent of the others.

2.10.3 Specific aim 3

In this section, the dissertation returns to the topic of childhood physical abuse (CPA) as a possible causal influence on the risk of developing drinking-related problems. In many studies, there is evidence that childhood adversities are associated with higher occurrence of adverse drinking outcomes (Galaif, Stein, Newcomb, & Bernstein, 2001; Horwitz, Widom, McLaughlin, & White, 2001; Widom, White, Czaja, & Marmorstein, 2007), but these studies did not focus upon the CPA-AUD association specifically. Here, we argue though that it is of importance to estimate the specific CPA-problem drinking relationship because, as a potentially modifiable characterization, CPA might in theory be manipulated or prevented to relieve some of the disease burden of drinking and related problems. Additionally, as in a "cycle of violence", AUD runs within families as well. Childhood physical abuse may be an important mediating factor in the transmission of AUD from parents to the offspring; it may also be a possible cause of AUD independent of family history of AUD. Figure 2.2 presents a heuristic and conceptual model (figure 2.2) in which CPA is specified to play a role as a cause in a larger multivariable system. This model is presented not as a specification for an econometric or structural equations model, although it conveys how multiple variables might possibly work together to cause alcohol dependence.

Figure 2.2 Conceptual model of the relationship between childhood physical abuse and drinking problems



As depicted in figure 2.2, CPA may be positioned as a possible cause of drinking problems, either independent of family history, or mediating the pathway from family history to offspring drinking problems, or both. Many studies showed a null association between CPA and AUD after introducing parental drinking problems as an independent variable into the model (Hughes, Johnson, Wilsnack, & Szalacha, 2007; Jackson & Sher, 2003; Koss et al., 2003; Libby et al., 2004; Mullings, Hartley, & Marquart, 2004; Young, Hansen, Gibson, & Ryan, 2006), while some others showed a positive association (Carrigan & Randall, 2003; Jasinski, Williams, & Siegel, 2000). However, these studies suffered from some major methodological limitations. First, they were based on samples from special populations, such as prisoners,

US Indian tribes, and marine recruits. The results cannot be generalized to the non-institutional population, where the majority of the AUD cases come from and where drinking behaviors and characteristics of AUD might differ. Second, most studies used the logistic regression to estimate a slope coefficient linking CPA to drinking problems. As stated in section 2.7, many of these studies did not test the independence assumption of logistic regression, and did not take the possible endogeneity problems into account. Moreover, some studies included potential mediators (e.g. lifetime diagnosis of PTSD, depression, individual's education attainment, being a victim of abuse during adulthood) in the multivariable model. The inclusion of these endogenous variables might have artificially biased estimates of CPA toward the null (Libby et al., 2004; Young, Hansen, Gibson, & Ryan, 2006). In summary, there is some basis for speculating that CPA might be a causal influence on drinking problems, but there is reason to complete more research on this topic before any causal inference is drawn.

This dissertation contributes new evidence on the possibility that CPA might merit interpretation as a causal influence on alcohol problems. The dissertation cannot produce definitive evidence on this topic, or settle the question. Nonetheless, its results will help guide future research of a more definitive character and will provide study estimates needed to plan this more definitive research. In addition to estimating the size of the suspected causal association linking CPA with AUD, this dissertation research will help to

clarify whether the association is independent of other associations under study.

Previously published studies are based on samples drawn from Western countries. However, exploring the association between CPA and AUD in non-Western countries is of both theoretical and public health pertinence.

In summary, under these specific aims, the main contribution of this dissertation research will be to add new epidemiological evidence that is pertinent to these guidelines for causal influence about observed associations: strength of the association, consideration of alternate explanations (endogeneity), replication of findings (in the Chinese context), and consistency with other knowledge.

If successful, this dissertation will have scientific and public health significance to the extent that it fills the gaps in evidence outlined in sections 2.10.1 to 2.10.3 of this chapter. In particular, the research will add new estimates on drinking problems in two cities of China. It will contribute new estimates of the size of association that link suspected background characteristics with occurrence and prevalence of drinking problems. Finally, it also will probe into the suspected causal association linking CPA with drinking-related problems.

Chapter 3 Methods

3.1 **Background:** The author of this dissertation research was working in Professor Yueqin Huang's mental health and psychiatry research unit during the period of conceptualization, planning, and field work preparations for the Beijing and Shanghai field surveys of the WMHS initiative. In that context, the author became familiar with the broad outlines of the research and was able to gain the permission of Professor Huang and Professor Shen to make use of the Chinese WMH data for this dissertation research. In addition, the author has developed a deep familiarity with the WMHS research design and analysis issues (e.g. problems of survey design effects) by working between 2005 and 2009 as a data analyst in Professor Jim Anthony's WMHS research group at Michigan State University. As such, this dissertation research project builds from the primary fieldwork experiences that the author gained while completing a master's degree in Peking University, as well as additional data gathering experiences between 2005 and 2009.

3.2 Design: This dissertation involves an analysis of data collected from the World Mental Health Survey-metropolitan China initiative (WMHS-mC), a cross-sectional survey in household-dwelling adults in Beijing and Shanghai, China.

3.3 Sample selection

The WMHS-mC used a stratified multi-stage probability sampling method to select household-dwelling non-institutionalized adults between 18

to 70 years old. In the first stage, the primary sampling unit (PSU) was neighborhood (jumin weiyuanhui) within each metropolitan area. Figure 3.1 is a map of China, showing the location of Beijing and Shanghai. Figure 3.2 shows geographic locations of sampling unit in Beijing and Shanghai.

Figure 3.1 A map of China.

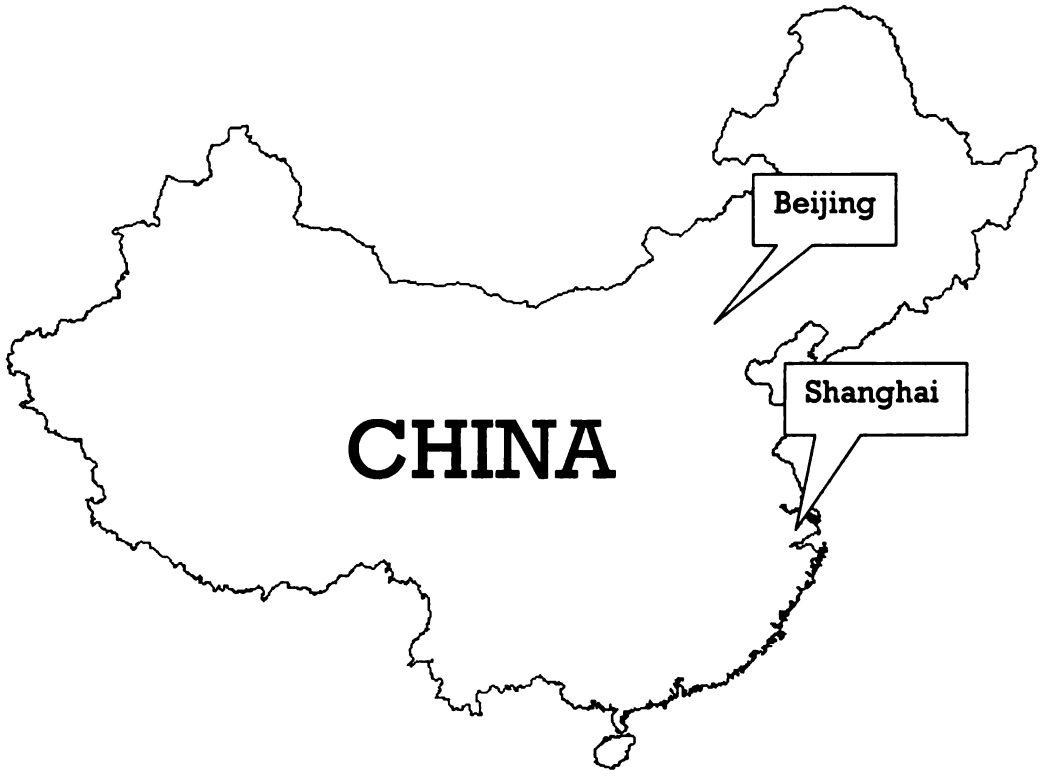
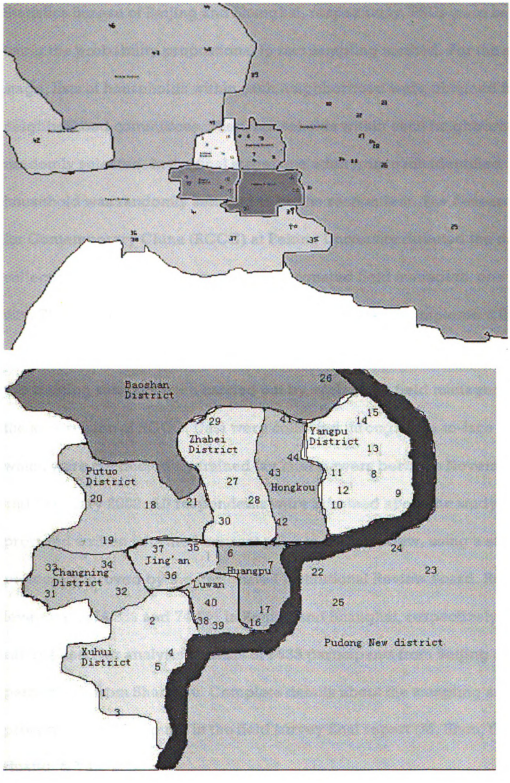


Figure 3.2 Sample geographic maps of the WMH-mC, Beijing (upper) and

Shanghai (lower)



There were 47 PSUs in Beijing and 44 PSUs in Shanghai identified through the Demographic Data for Neighborhoods, 1999, published by the Statistics Bureau of Beijing and Shanghai, respectively. PSUs were selected using the probability proportional to size sampling method. For the second stage, lists of households within each neighborhood were obtained from neighborhood committees. Then, households within each neighborhood 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 (RCCC) at Peking University directed the data collection in both cities through two designated field managers, one for each city. The field manager of each site organized a team to implement the field work following the same survey protocol. Before data collection commenced, two training sessions were carried out by each of the field managers under the supervision of RCCC. Data were collected through face-to-face interviews, which were conducted by trained lay interviewers 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. Response levels were 74.8% and 74.6% in Beijing and Shanghai, respectively. The final sample used for analysis consists of 2633 participants from Beijing and 2568 participants from Shanghai. Complete details about the sampling and field procedures can be found in the field survey final report (M. Shen, Chai, Yang, Huang, & Yan, 2003).

3.4 Measures

3.4.1 Assessments

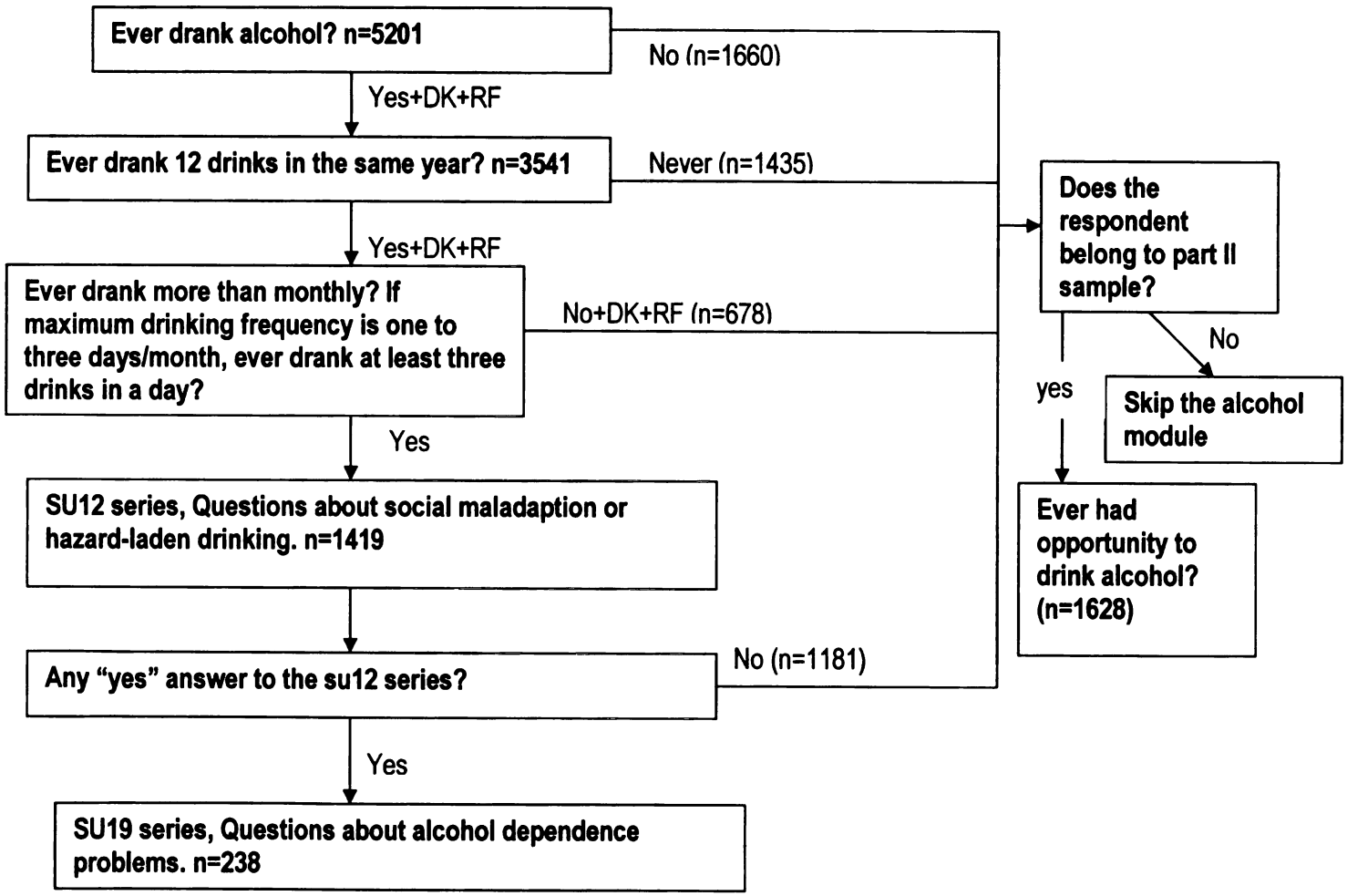
The assessment instrument used in WMH-mC is the World Mental Health Initiative version of the World Health Organization Composite International Diagnostic Interview (WMH-CIDI; Kessler & Ustun, 2004). The WMH-CIDI is a comprehensive, fully structured diagnostic interview designed to be administered by trained lay interviewers to assess clinical features of psychiatric illnesses, and symptoms of mental disorders according to criteria in both the International Classification of Disease, the 10^{th} edition (ICD-10) and the Diagnostic and Statistical Manual of Mental Disorders, the fourth edition (DSM-IV). 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 including tobacco use and extra-medical psychoactive drug use. 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 WMH-CIDI consists of modules on various topics. Each module contains standardized questions relevant to the specific topic. Questions about

lifetime history of alcohol drinking were located at the beginning of the "substance" module and were administered to all respondents. For drinkers ever in lifetime, follow-up questions were asked about recent drinking behavior (in the prior 12 months), the occurrence of socially maladaptive drinking and other clinical features associated with alcohol dependence, as well as the recency of these problems. Figure 3.3 provides a brief description of the logical skip pattern employed in the CIDI. Actual questions in the original English version of the WMH-CIDI, as well as the final Chinese versions of these English language items, and a more detailed diagram of the skip pattern can be found in appendix materials. Two major assumptions are made in the CIDI assessment of alcohol problems. The first is that if an individual has never drunk more than monthly (once a month), then he/she would never qualify as a case of DSM-IV or ICD-10 defined alcohol use disorders (AUD). The second is that if an individual never had experienced socially maladaptive or hazard-laden drinking, he/she never would meet DSM-IV diagnostic criteria for "clinically significant alcohol dependence", with the "clinical significance" criteria as presented in Degenhardt et al. (2007) in their discussion of the DSM-IV concept (Degenhardt, Bohnert, & Anthony, 2007). More detailed discussion about these assumptions can be found in chapter 5 of this dissertation.

Figure 3.3 Skip pattern of the WMH-CIDI alcohol assessment.



There was a WMH-CIDI question about the first opportunity to drink alcohol in Part II of the assessment, so its responses were available for the Part II sample of 1628 respondents. Information about childhood experience was located in the "Childhood" module, which was administered to the Part II sample of 1628 respondents. Information about demographic characteristics was obtained through two separate modules. The "core" demographic module was administered to all participants in Part I (n=5201), while the other more detailed demographic module was administered to the Part II sample only. As a result, data on sex, age, marital status, personal income, and employment status were available for all participants, while data on education attainment were available for the sub-sample only.

3.4.2 Definition of drinking-related variables

In this study, the self-report drinking related variables fall into two main categories, the "drinking behavior" and "indicators of risky drinking".

3.4.2.1 Variables in the "drinking behavior" category

1) Ever had opportunity to drink alcohol;

2) Ever tried alcohol (even a sip);

3) More than minimum (MTM) drinking (>= 12 drinks in a given year);

4) Being a MTM drinker during the year prior to the assessment;

5) Frequency of drinking when drank the most;

6) Frequency of drinking during the year prior to the assessment;

7) Age of first try;

8) Age of onset of drinking;

9) Number of drinks during a typical drinking day when drank the most;

10) Number of drinks during a typical drinking day during the year prior to the assessment.

Opportunity to drink alcohol was assessed by means of the following WMH-CIDI question, which was asked in the first series of Part II questions about drinking experiences of the 1628 Part II respondents: "*The next questions are about the first time you had an opportunity to drink alcohol or to use drugs, whether or not you used them. By "an opportunity to use" I mean someone either offered you alcohol or drugs, or you were present when others were using and you could have used if you wanted to. Please do not include times when a health care provider may have offered you free samples. (Thinking back over your entire lifetime,) about how old were you the very first time you had an opportunity to use (alcohol/drugs)?*" The responses to the item were coded as follows:

the actual number <100 =___years old (n=1167)

997=never (n=366)

998=don't know (n=92)

999=refused (n=3)

For the present study, respondents who gave an age value in response to the question were coded as "yes". Respondents who answered "Never" were firstly coded as "no"; then, if they indicated that they had tried alcohol (from another question, SU1, in the "substance" module), they were recoded as "yes" (n=32). Among 95 respondents who answered "don't know" or

"refused" to the original question, 59 indicated they had tried alcohol. These 59 were then coded as "yes". The remaining 36 were coded as missing. A description of missing values is given in Table 3.1.

The variable "ever tried alcohol" was assessed by means of the following WMH-CIDI question: "*The next questions are about your use of alcoholic beverages, including beer, wine, wine coolers, and hard liquor like vodka, gin or whiskey. How old were you the very first time you ever drank an alcoholic beverage?*" The responses to the question were coded as follows:

the actual number <100 =_____ YEARS OLD (n=3193)

997=NEVER (n=1660)

998=DON'T KNOW (n=348)

999=REFUSED (n=0)

Responses who gave an age value in response to the question were coded as "yes"; respondents who answered "Never" were coded as "no". Among 348 respondents who gave "don't know" or "refused" responses to the original question, 155 of them indicated they have had drunk >=12 drinks in a year. These 155 were therefore recoded as "yes". The remaining 201 respondents were coded as missing.

The variable "ever being an MTM drinker" was assessed by means of the following WMH-CIDI question: "*When I use the word "drink" in the next questions, I mean either a glass of wine, a can or bottle of beer, or a shot or jigger of liquor either alone or in a mixed drink. How old were you when you first started drinking at least 12 drinks in a year?*

IF "ALL MY LIFE" OR "AS LONG AS I CAN REMEMBER," PROBE: Was it before your teens?

IF NO/DK, PROBE: Was it before your twenties?"

The responses to the question were coded as follows:

the actual number <100= _____ YEARS OLD	(n=2049)
12=BEFORE TEENS	
19=BEFORE 20s	
20=NOT BEFORE 20s	
997=NEVER	(n=1433)
998=DON'T KNOW	(n=55)
999=REFUSED	(n=2)

Respondents who gave an age value in response to the question were coded as "MTM drinkers"; respondents who answered "Never" were coded as "non-MTM drinkers". Among 57 respondents who gave "don't know" or "refused" to the original question, 28 indicated that they had drank at least once a month during a year in their lifetime from later questions; these 28 were recoded as "yes". The remaining 29 were coded as missing.

MTM drinking during the year prior to the assessment was assessed by means of the following WMH-CIDI question: "*Think about the past 12 months. In the past 12 months, how often did you usually have at least one drink – nearly every day, three to four days a week, one to two days a week, one to three days a month, or less than once a month?* "

The responses to the question were coded as follows:

1=NEARLY EVERY DAY (n=459)

2=3 - 4 DAYS PER WEEK (n=200)

3=1 - 2 DAYS PER WEEK (n=371)

4=1 - 3 DAYS PER MONTH (n=389)

5=LESS THAN ONCE A MONTH (INCLUDING NEVER DRINK) (n=651)

8=DON'T KNOW (n=34)

9=REFUSED (n=2)

Respondents who gave a value of one through four in response to the question were coded as "yes"; responses of option five were coded as "no". "Don't know" and "refused" were coded as missing values.

Drinking frequency during the year prior to the assessment was assessed by the same question. Options one through five were maintained as they were. "Don't know" and "refused" were coded as missing values.

If the respondent did not have a period of time when they drank more than they did during the 12 months prior to the assessment, their drinking frequency when drank the most was the same as the drinking frequency during the last 12 months. If they indicated that there was a period of time when they drank more than they did during the last 12 months, their drinking frequency when drank the most was assessed by means of the following WMH-CIDI question "*Think about the years in your life when you drank most. During those years, how often did you usually have at least one drink – nearly every day, three to four days a week, one to two days a week, one to three days a month, or less than once a month?*"

The responses to the question were coded as follows:

1=NEARLY EVERY DAY (n=333)

2=3 - 4 DAYS PER WEEK (n=165)

3=1 - 2 DAYS PER WEEK (n=189)

4=1 - 3 DAYS PER MONTH (n=161)

5=LESS THAN ONCE A MONTH (n=369)

8=DON'T KNOW (n=39)

9=REFUSED (n=2)

For this variable, the same logic was used as the past year drinking frequency variables.

A value for the number of drinks per day has been obtained from this question: "*On the days you drank, about how many drinks you usually had per day?*" Separate questions were asked regarding the year prior to the assessment and the period of time when they drank the most. Numbers of drinks were maintained. "Don't know" and "refused" were coded as missing values. For number of drinks per day during the year prior to the assessment, there were 105 missing values, accounting for 9.0%of all answers. For number of drinks per day when drank the most, there were 116 missing values, accounting for 7.6% of all answers.

Table 3.1 Distribution of variables for drinking behavior. Data from the WMH-mC, 2001-2002.

		Entire Sample			Beijing			Shanghai		
		n	%	wt%[2]	n	%	wt%[2]	n	%	wt%[2]
Opportunity[1]	Yes	1258	77.3	77.9	722	79.0	80.4	536	75.1	74.7
	No	334	20.5	19.8	180	19.7	18.6	154	21.6	21.3
	Missing	36	2.2	2.7	12	1.3	0.9	24	3.4	4.0
Ever trying alcohol	Yes	3340	64.2	67.2	1758	66.8	70.1	1582	61.6	64.2
	No	1660	31.9	29.1	832	31.6	28.1	828	32.2	30.2
	Missing	201	3.9	3.7	43	1.6	1.8	158	6.2	5.7
MTM drinking	Yes	2077	39.9	41.7	1146	43.5	45.0	931	36.3	38.4
	No	3095	59.5	57.7	1480	56.2	54.7	1615	65.9	60.8
	Missing	29	0.6	0.6	7	0.3	0.3	22	0.9	0.8
Past year MTM drinking	Yes	1419	27.3	28.0	787	29.9	30.1	632	24.6	25.8
	No	3744	72.0	71.3	1831	69.5	69.4	1913	74.6	73.4
	Missing	36	0.7	0.6	15	0.6	0.5	21	0.8	0.8
Frequency of drinking										
Nearly everyday		610	11.7	10.3	347	13.2	10.9	263	10.2	9.6
3-4 days/week		277	5.3	5.8	146	5.6	6.0	131	5.1	5.5
1-2 days/week		417	8.0	9.0	253	9.6	11.1	164	6.4	6.9
1-3 days/month		395	7.6	8.6	182	6.9	7.7	213	8.3	9.5
< monthly		1817	34.9	36.8	861	32.7	35.9	956	37.2	37.9
No drink		1660	31.9	29.1	832	31.6	28.1	828	32.2	30.2
Missing		25	0.5	0.5	12	0.5	0.4	13	0.5	0.6
Past year drinking frequency										
Nearly everyday		459	8.8	7.3	259	9.8	7.6	200	7.8	7.0
3-4 days/week		200	3.9	4.0	115	4.4	4.4	85	3.3	3.6
1-2 days/week		371	7.1	7.9	218	8.3	9.2	153	6.0	6.7
1-3 days/month		389	7.5	8.8	195	7.4	9.1	194	7.6	8.6
Less than once per month		2084	40.1	42.2	999	37.9	41.3	1085	42.3	43.1
Never drank		1660	31.9	29.1	832	31.6	28.1	828	32.2	30.2
Missing		38	0.7	0.7	15	0.6	0.5	23	0.9	0.9

1. Variable available in Part II only

2. Due to rounding, some values do not sum to exactly 100%

3.4.2.2 Variables in the "indicators of risky drinking" category

Indicators of "risky drinking" in this study were:

1) Early trying alcohol (before teens (<13 years old));

2) Early onset of MTM drinking (before 20 years old);

3) Ever heavier drinking;

4) Heavier drinking during the year prior to the assessment;

5) Past year socially maladaptive or hazard-laden drinking problems;

6) Ever had any alcohol dependence clinical feature;

7) Past year alcohol dependence clinical feature;

8) Early onset of socially maladaptive or hazard-laden drinking (before 23 years old);

9) Early onset of alcohol dependence clinical feature (before 23 years old);

10) The occurrence of five socially maladaptive or hazard-laden drinking problems: responsibility interference, social-interpersonal problems, drinking despite social problems, hazardous use, legal problems;

11) The occurrence of eight clinical features of alcohol dependence: tolerance, withdrawal, difficulty cutting down, giving up activities because of drinking, a great deal of time spent on drinking-related activities, drinking despite physical/emotional problems, and irresistible desire.

Respondents who gave an age value that was less than 13 years, when asked about first trying alcoholic beverages were assigned a "yes" for "early trying alcohol". Respondents, who answered "don't know" or "refused" were deemed as missing values; all others were coded as "no". (The actual

question is shown in section 2.1.) A description of missing values is given in Table 3.3.

A similar procedure was used to recode the "early onset of MTM drinking" values, for which the actual question is shown in section 2.1. The cutoff age of 13 for the first consumption of alcohol is based on previous literature about preteen initiation of drinking (Dube et al., 2006; Hamburger, Leeb, & Swahn, 2008). A cutoff age of 20 was used to designate early onset of MTM drinking. Since there has been no legal age for drinking in China and many children sip alcoholic beverages in celebrations, the age of the first sip does not necessarily mean higher risk of negative consequences in Chinese cultures. Due to this concern, the author created another marker for earlier onset of drinking, "early onset of MTM drinking". No reference age cutoff for MTM drinking was found in the literature. The cutoff point of 20 years old for "MTM drinking" is mainly based on the way of how data were collected. As described above, if the respondent could not recall the exact age to answer the WMH-CIDI question "*How old were you when you first started drinking at least 12 drinks in a year?*", a follow-up question *"Was it before your twenties?"* was asked. If the response is "yes", the age in the dataset is marked as 19. From the de-identified data, there is no way to separate the 19s meaning "before twenties" and the 19s meaning the exact recalled age of 19. Nonetheless, there is evidence that earlier onset of drinking is associated with more negative drinking consequences. This variable serves as a marker for

earlier onset of MTM drinking. Description of these variables can be found in Table 3.3.

This study's "heavier drinking" assessment was derived from questions about number of drinks consumed in a day, for which the actual questions can be found in section 2.1. Lifetime history of heavier drinking and past year heavier drinking were assessed through two separate questions regarding the period when the respondent drank the most and 12 months prior to the assessment, respectively. Heavier drinking was defined as at least five drinks in a typical drinking day for males and at least four drinks for females. This cut-off threshold of five and four is in accordance with the definition of "binge drinking" used by the US National Institute of Alcohol Abuse and Alcoholism (NIAAA) and with the most commonly used definitions for "binge drinking" and "heavy episodic drinking" in previous studies (Jasinski, Williams, & Siegel, 2000; NIAAA, 2004; Trent, Stander, Thomsen, & Merrill, 2007). "Don't know" and "Refused" were coded as missing values. The author is aware of an agreement among alcohol research journal editors to avoid the NIAAA concept of "binge drinking" so that the traditional concept of a "binge" can be retained (e.g. a 'binge' or 'bender' involving multiple days of continuous intoxication). For this reason, the term "heavier drinking" is used in reports on this dissertation research.

Socially maladaptive or hazard-laden drinking was assessed via five consecutive questions. These five questions are based upon criteria of "alcohol abuse" as outlined in DSM-IV. Actual questions appear in table 3.2. In

order to ease the discussion, we labeled them as **"interfere with responsibility", "social problems", "drink despite social problems", "hazard-laden drinking", and "legal problems"**. For all the five questions, respondents who answered "yes" to the original question were coded as "yes"; respondents who answered "no" were coded as "no"; respondents who answered "don't know" or "refused" were coded as missing.

For this dissertation research endorsement of any of the five questions qualified the respondent as a case of "socially maladaptive drinking"; "no" to all five questions qualified the respondent as a non-case of "socially maladaptive drinking". If the respondents answered a combination of "no" and missing ("don't know" or "refused"), he/she was assigned a missing value for "socially maladaptive drinking". Standard algorithms for the WMH-CIDI were used to label the diagnosis of DSM-IV alcohol abuse. Essentially, endorsement of any of the above clinical features, except for "social problems", qualified the respondent as a case of "lifetime DSM-IV alcohol abuse". If any of the clinical features occurred during the 12 months prior to the assessment, the respondent was qualified for a case of "past year DSM-IV alcohol abuse."

Eight clinical features of alcohol dependence, as outlined by DSM-IV and ICD-10, were assessed via 11 consecutive questions. Actual questions appear in table 3.5. Although multiple questions were asked for the clinical features of "withdrawal" and "drink more than intended", the "once endorsed, skip the rest" logic embedded in the assessment testlet precluded us from

studying items within one criterion individually. Therefore, we can only provide estimates for each diagnostic criterion and not for these individual items. In order to ease discussion, we labeled these eight clinical features as **"strong desire", "tolerance", "withdrawal", "more than intended", "cut down", "great deal of time", "give up activities", and "drink despite physical/mental problems"**. For each of the eight criteria, respondents who answered "yes" to the original question were coded as "yes"; respondents who answered "no" were coded as "no"; respondents who answered "don't know" or "refused" were coded as missing. Two questions were asked (SU19b and SU19c) to assess "withdrawal" criterion, a "yes" answer to either question qualified the respondent as a case of "withdrawal"; the same logic was used to recode "more than intended" criterion, where three questions were asked (SU19d to SU19f). One more question (SU32) besides SU19g were used to recode the "cut down" criterion. The question is: "*Starting from the time you first began having any of these problems, how many different times did you ever make a serious attempt to quit drinking?*" If the respondents gave a value of greater than two, they were recoded as "yes" to the "cut down" criterion as well.

Endorsement of any of the eight criteria qualified the respondent as a case of "any clinical feature of dependence"; "no" to all eight criteria qualified the respondent as a non-case. If the respondent answered a combination of "no" and missing ("don't know" or "refused"), he/she was assigned a missing value.

A standardized computerized algorithm for the WMH-CIDI was used to sort drinkers in relation to the diagnosis of DSM-IV alcohol dependence. If the respondents had met at least three of the above criteria, except for "strong desire", and indicated that they had at least three problems in a same year, they qualified as cases of "DSM-IV alcohol dependence"; if the respondents met none of the seven criteria, they qualified as non-cases; if the respondents endorsed a combination of "no" and missing ("don't know" or "refused"), they were assigned a missing value for "DSM-IV alcohol dependence".

Age of the first occurrence of socially maladaptive drinking was obtained from two CIDI questions. The first question is: "*You just reported that your drinking (KEY PHRASE FOR "YES" RESPONSE IN SU12 SERIES). Can you remember your exact age the very first time you had this problem? If so, how old were you?"* Respondent who gave an age value less than 23 to this question was identified as a case of "early onset of socially maladaptive drinking problems" (n=52). The second question is for when the respondent cannot recall the exact age. The question is "*About how old were you (the first time you had [this problem/ (either/ any) of these problems] because of drinking)?*

If "All My Life" Or "As Long As I Can Remember," Probe: Was It Before Your Teens? IF NO/DK, PROBE: Was It Before Your Twenties?"

Responses to this question are:

Table 3.2 Actual WMH-CIDI questions about socially maladaptive drinking and clinical features of alcohol dependence.				
INTERVIEWER INSTRUCTION: IF R PROTESTS OR REFUSES TWO QUESTIONS, CODE ALL UNANSWERED *SU12 SERIES QUESTIONS '9' AND GO TO *SU13.	YES (1)	NO (5)	DK (8)	RF (9)
*SU12. The next questions are about problems you may have had because of drinking. First, was there ever a time in your life when your drinking or being hung over frequently interfered with your work or responsibilities at school, on a job, or at home? (KEY PHRASE: interfered with your work)	1	5	8	9
*SU12a. Was there ever a time in your life when your drinking caused arguments or other serious or repeated problems with your family, friends, neighbors, or co-workers? (KEY PHRASE: caused problems with family, friends or others)	1	5 go to *su12c	8 go to *su12c	9 go to *su12c
*SU12b. Did you continue to drink even though it caused problems with these people?	1	5	8	9
*SU12c. Were there times in your life when you were often under the influence of alcohol in situations where you could get hurt, for example when riding a bicycle, driving, operating a machine, or anything else? (KEY PHRASE: jeopardized your safety because you sometimes drank in situations where you could get hurt)	1	5	8	9
*SU12d. Were you more than once arrested or stopped by the police because of drunk driving or drunk behavior? (KEY PHRASE: resulted in problems with the police)	1	5	8	9
*SU19. (The next questions are about some other problems you may have had because of drinking.) Was there ever a time in your life when you often had such a strong desire to drink that you couldn't resist taking a drink or found it difficult to think of anything else? KEY PHRASE: a strong desire to drink	1	5	8	9
*SU19a. Did you ever need to drink a larger amount of alcohol to get an effect, or did you ever find that you could no longer get a "buzz" or a high on the amount you used to drink? KEY PHRASE: drink a larger amount of alcohol	1	5	8	9
*SU19b. People who cut down or stop drinking after drinking steadily for some time may not feel well. These feelings are more intense and can last longer than the usual hangover. Did you ever have times when you stopped, cut down, or went without drinking and then experienced symptoms like fatigue, headaches, diarrhea, the shakes, or emotional problems? KEY PHRASE: not feel well	1 go to *su19d	5	8	9

Table 3.2 (cont'd)				
*SU19c. Did you ever have times when you took a drink to keep from having problems like these? KEY PHRASE: keep from having problems like these	1	5	8	9
*SU19d. Did you have times when you started drinking even though you promised yourself you wouldn't, or when you drank a lot more than you intended? KEY PHRASE: drank a lot more than you intended	1 go to *su19g	5	8	9
*SU19e. Were there ever times when you drank more frequently or for more days in a row than you intended? KEY PHRASE: more days in a row than you intended	1 go to *su19g	5	8	9
*SU19f. Did you have times when you started drinking and became drunk when you didn't want to? KEY PHRASE: drunk when you didn't want to	1	5	8	9
*SU19g. Were there times when you tried to stop or cut down on your drinking and found that you were not able to do so? KEY PHRASE: tried to stop or cut down on your drinking but not able	1	5	8	9
*SU19h. Did you ever have periods of several days or more when you spent so much time drinking or recovering from the effects of alcohol that you had little time for anything else? KEY PHRASE: little time for anything else	1	5	8	9
*SU19i. Did you ever have a period of a month or longer when you gave up or greatly reduced important activities because of your drinking – like sports, work, or seeing friends and family? KEY PHRASE: gave up or greatly reduced important activities because of your drinking	1	5	8	9
*SU19j. Did you ever continue to drink when you knew you had a serious physical or emotional problem that might have been caused by or made worse by drinking? KEY PHRASE: continue to drink while physical or emotional problem caused by or made worse by drinking	1	5	8	9

a number <100=_____ YEARS OLD

12= BEFORE TEENS

19= BEFORE 20s (n=28)

20= NOT BEFORE 20s

998= DON'T KNOW (n=8)

999= REFUSED (n=1)

Respondents who gave an age value less than 23, but not 20, were coded as cases of "early onset of social maladaptive problems" (n=6). Respondents who had a value of 20 were coded as non-cases because their age of onset was likely to be after 23 (not before the 20s). Age of first occurrence of clinical features of alcohol dependence was recoded in the same manner.

The cutoff age of 23 is based upon previous literature. Previous studies often defined early onset of alcohol problems as before 20 or 25 years old (B. A. Johnson, Cloninger, Roache, Bordnick, & Ruiz, 2000; Watson et al., 1997). Brown and colleagues found that 22.5 is the mean onset age of problem drinking in the more severe group, and 24.3 in the less severe group (J. Brown, Babor, Litt, & Kranzler, 1994). Moss and colleagues' post-hoc analysis with latent class modeling found that the mean age of onset of alcohol dependence clinical features is approximately 23 in the most severe classes, and older in other classes (Moss, Chen, & Yi, 2008). Thus, for this dissertation research, an age of 23 was specified to be cutoff point for early onset of alcohol problems in this study.

Table 3.3 Distribution of variables for risky drinking. Data from the WMH-mC, 2001-2002.

		Entire Sample			Beijing			Shanghai		
		n	%	wt%[1]	n	%	wt%[1]	n	%	wt%[1]
Early onset of trying alcohol	Yes	487	9.4	11.6	201	7.6	10.9	286	11.1	12.3
	No	4366	84.0	81.6	2346	89.1	85.4	2020	78.7	77.6
	Missing	348	6.7	6.8	86	3.3	3.7	262	10.2	10.0
Early onset of MTM drinking	Yes	681	13.1	15.8	382	14.5	19.3	299	11.6	12.1
	No	4463	85.8	86.2	2230	84.7	79.9	2233	87.0	86.6
	Missing	57	1.1	1.0	21	0.8	0.8	36	1.4	1.3
Heavy drinking	Yes	1265	77.7	79.9	726	79.4	80.9	539	75.5	75.5
	No	334	20.5	19.8	180	19.7	18.6	154	21.6	21.3
	Missing	29	1.8	1.7	8	0.9	0.5	21	2.9	3.2
Past year heavy drinking	Yes	147	2.8	3.2	77	2.9	3.5	70	2.7	3.0
	No	4949	95.2	94.3	2507	95.2	94.3	2442	95.1	94.3
	Missing	105	2.0	2.5	49	1.9	2.3	56	2.2	2.7
DSM-IV alcohol abuse	Yes	221	4.3	4.7	145	5.5	6.3	76	3.0	3.0
	No	4959	95.3	94.7	2475	94.0	93.0	2482	96.7	96.5
	Missing	23	0.4	0.6	13	0.5	0.7	10	0.4	0.5
Interfere with responsibility	Yes	111	2.1	2.4	76	2.9	3.5	35	1.4	1.3
	No	5072	97.5	97.2	2548	96.8	96.1	2524	98.3	98.3
	Missing	18	0.4	0.4	9	0.3	0.5	9	0.4	0.4
Social problems	Yes	97	1.9	1.8	65	2.5	3.5	32	1.3	1.1
	No	5085	97.8	97.7	2559	97.2	96.1	2526	98.4	98.5
	Missing	19	0.4	0.5	9	0.3	0.5	10	0.4	0.5
Drink despite social problems	Yes	62	1.2	1.0	42	98.3	1.4	20	0.8	0.6
	No	5137	98.8	98.9	2589	1.6	98.4	2548	99.2	99.4
	Missing	2	0.0	0.1	2	0.1	0.2	0		0.0

Table 3.3 (cont'd)

Hazard-Laden drinking	Yes	131	2.5	2.8	86	3.3	3.6	45	1.8	2.0
	No	5050	97.1	96.7	2537	96.4	95.9	2513	97.9	97.5
	Missing	20	0.4	0.5	10	0.4	0.5	10	0.4	0.5
Legal problems	Yes	10	0.2	0.2	9	0.3	0.4	1	0.0	0.0
	No	5170	99.4	99.3	2613	99.2	99.1	2557	99.6	99.5
	Missing	21	0.4	0.5	11	0.4	0.5	10	0.4	0.5
Tolerance	Yes	50	1.0	1.1	35	1.3	1.6	15	0.6	0.6
	No	5148	99.0	98.9	2595	98.6	98.4	2553	99.4	99.4
	Missing	3	0.1	0.0	3	0.1	0.0	0	0.0	
Withdrawal	Yes	46	0.9	0.9	28	1.1	1.2	18	0.7	0.6
	No	5148	99.0	99.0	2602	98.8	98.8	2546	99.1	99.2
	Missing	7	0.1	0.1	3	0.1	0.0	4	0.2	0.2
More than intended	Yes	150	2.9	3.0	102	3.9	4.2	48	1.9	1.9
	No	5045	97.0	96.9	2529	96.1	95.8	2516	98.0	97.9
	Missing	6	0.1	0.1	2	0.1	0.0	4	0.2	0.2
Cut down	Yes	100	1.9	2.0	69	2.6	2.7	31	1.2	1.2
	No	5095	98.0	97.9	2561	97.3	97.1	2534	98.7	98.7
	Missing	6	0.1	0.1	3	0.1	0.1	3	0.1	0.1
Great deal of time	Yes	30	0.6	0.6	21	0.8	0.8	9	99.6	0.3
	No	5168	99.4	99.4	2610	99.1	99.2	2558	0.4	99.7
	Missing	3	0.1	0.0	2	0.1	0.0	1	0.0	0.0
Give up activities	Yes	28	0.5	0.5	20	0.8	0.7	8	0.3	0.3
	No	5169	99.4	99.4	2611	99.2	99.2	2558	99.6	99.7
	Missing	4	0.1	0.0	2	0.1	0.0	2	0.1	0.0
Drink despite physical or mental problem	Yes	66	1.3	1.4	44	1.7	1.8	22	0.9	0.9
	No	5128	98.6	98.5	2584	98.1	98.1	2544	99.1	99.0

Table 3.3 (cont'd)

	Missing	7	0.1	0.1	5	0.2	0.1	2	0.1	0.0
Strong desire	Yes	71	1.4	98.6	43	1.6	1.8	28	1.1	1.0
	No	5128	98.6	1.4	2588	98.3	98.2	2540	98.9	99.0
	Missing	2	0.0	0.0	2	0.1	0.0	0	0.0	0.0
DSM-IV alcohol	Yes	41	0.8	1.0	32	1.2	1.6	9	0.4	0.4
dependence	No	5153	99.1	98.9	2599	98.7	98.4	2554	99.5	99.5
	Missing	7	0.1	0.1	2	0.1	0.0	5	0.2	0.2
Early onset of socially	Yes	80	1.5	2.0	51	1.9	2.9	29	1.1	1.1
maladaptive drinking	No	5093	97.9	97.2	2568	97.5	96.4	2525	98.3	98.1
	Missing	28	0.5	0.8	14	0.5	0.8	14	0.6	0.7
Early onset of alcohol	Yes	26	0.5	0.6	16	0.6	0.9	10	0.4	0.3
dependence	No	5171	99.4	99.4	2614	99.3	99.1	2557	99.6	99.6
	Missing	4	0.1	0.1	3	0.1	0.1	1	0.0	0.1
Past year DSM-IV	Yes	57	1.1	1.3	40	1.5	2.0	17	0.7	0.6
alcohol abuse	No	5121	98.5	98.1	2580	98.0	97.3	2541	99.0	98.9
	Missing	23	0.4	0.6	13	0.5	0.7	10	0.4	0.5
Past year DSM-IV	Yes	40	0.8	0.9	24	0.9	1.2	16	0.6	0.6
alcohol dependence	No	5156	99.1	99.0	2605	98.9	98.7	2551	99.3	99.4
	Missing	5	0.1	0.1	4	0.2	0.1	1	0.0	0.1

1. Weighted percentage. Due to rounding, some volumes do not sum to exactly 100%.

3.4.3. Covariates under study

With respect to the aim 1, there is no covariate of interest.

As for aim 2, subgroup variations in drinking behavior and outcomes, covariates of interest are sex, age, marital status, education attainment, income level, and employment status.

Sex was dichotomized into male and female based upon the observation of the interviewer. Age was based upon self-report by participants. Participants were further divided into four age groups: "born before 1949", "1949-1965", "1966-1977", and "born after 1977". The division is based on significant events in contemporary Chinese history. The year 1949 is the year of the establishment of the government of the People's Republic of China; the "great leap and culture revolution" started in 1966; the open policy and the only child policy were implemented in 1978. Table 3.4 provides a description of these variables.

Current marital status was categorized as married or cohabitating, no longer married (separated/divorced/widowed), and never married, based on the self-report of participants. Original questions in WMH-CIDI are:" *Are you currently married?*", "*Are you currently living with someone in a marriage-like relationship?*", and "*Have you ever been married?*"

Responses to these items were coded as following:

1= currently married or cohabiting (n=4035)

2= have been married but currently not (n=318)

3= never married (n=848)

Current education attainment was grouped into four categories based on self-report of years of education received by the time of assessment. The original WMH-CIDI question is:" *How many years of school have you completed? (IF NEC: Please include any years of higher education.)*"

Responses to this item were coded as following:

1= "0-6 years" (n=168)

2= "7-9 years" (n=425)

3= "10-12 years" (n=580)

4= "> 12 years" (n=455)

These four categories correspond to "less than or finished elementary school", "some or finished middle school", "some or finished high school", and "some college and above", respectively.

Information about income was obtained from respondent self-report about personal income in the last month. The original question is: "*How much might your own personal earnings income be in last month? including wage, prize, extra income from another job, present from relatives or friends, yield from investment such as stocks etc and all other sources of income)*"

Responses to this item were coded as following:

a number <99990= _____ Yuan (¥) (n=5101)

99998= DON'T KNOW (n=14)

99999= REFUSED (n=77)

Personal income was obtained for 5101 out of 5201 respondents (2605 out of 2633 in Beijing and 2496 out of 2568 in Shanghai). Respondents who

answered "don't know" or "refused" were coded as missing values. Personal income was further categorized into high (>1500 Yuan), high average (1001-1500 Yuan), low average (601-1000 Yuan), and low income (<=600 Yuan) based on the percentile distribution of the personal income variable.

The employment status variable has two categories, "working" and "not working", based upon the respondents' self-report of current employment status at the time of assessment. The original WMH-CIDI question is:" *What about your current employment situation -- are you working now for pay, self-employed, looking for work, disabled, temporarily laid off, retired, a homemaker, a full-time or part-time student, or something else?"*

Responses to this item were coded as following:

1= working now or self-employed (n=2931)

2= unemployed (looking for work), temporarily laid off, retired, homemaker, student, maternity leave, illness/sick leave, disabled (n=2266)

98= don't know (n=0)

99= refused (n=4)

Table 3.4 provides a description of these variables.

With respect to aim three, the main suspected causal variable is "childhood physical punishment" (CPP). It is assessed by a question in the "childhood" module and was assessed for all Part II respondents (n=1628). The question is:" *When you were growing up, how often did someone in your household do any of the things (on the list on page 38 in your booklet) to you – often, sometimes, rarely, or never?"*

Table 3.4 Distribution of sociodemographic variables. Data from WMH-mC, 2001-2002.

	Entire Sample			Beijing			Shanghai		
	n	%	weighted %[2]	n	%	weighted %	n	%	weighted %[2]
Sex									
Female	2668	54.3	47.4	1366	51.9	47.2	1302	50.7	47.6
Male	2533	48.7	52.6	1267	48.1	52.8	1266	49.3	52.4
Age groups									
Born before 1949	1385	26.6	18.7	767	29.1	17.8	618	24.1	19.6
1949-1965	2493	47.9	40.1	1264	48.0	36.4	1229	47.9	43.8
1966-1977	811	15.6	25.2	400	15.2	27.3	411	16.0	23.0
Born after 1977	512	9.8	16.05	202	7.7	18.5	310	12.1	13.6
Education attainment [1]									
<= 6 years	168	10.3	6.7	105	11.5	6.4	63	8.8	7.1
7-12 years	425	26.1	22.7	237	25.9	21.9	188	26.3	23.9
13-15 years	580	35.6	38.1	302	33.0	34.9	278	38.9	42.2
>15 years	455	28.0	32.5	270	29.6	36.9	185	25.9	26.8
Personal income level									
Low	1396	27.4	29.0	672	25.8	29.3	724	29.0	28.7
Low-average	1581	31.0	27.0	815	31.3	25.6	766	30.7	28.5
High-average	871	17.1	17.0	478	18.4	17.7	396	15.9	16.3
High	1250	24.5	27.0	640	24.6	27.5	610	24.4	26.5
Marital Status									
Married/cohabiting	4035	77.6	69.1	2144	81.4	68.2	1891	73.6	70.0
No longer married	318	6.1	4.2	164	6.2	3.8	154	6.0	4.5
Never married	848	16.3	26.8	325	12.3	28.1	523	20.4	25.5
Employment									
Working	2931	56.4	61.4	1489	56.6	63.0	1442	56.2	59.8
Not working	2266	43.6	38.6	1141	43.4	37.1	1125	43.8	40.3

1. Variable available in part 2 only

2. Due to rounding, percentages may not add up to 100%.

Responses to the question are:

1= often	**(n=50)**
2= sometimes	**(n=185)**
3= rarely	**(n=330)**
4= never	**(n=1046)**
8= don't know	**(n=12)**

9= refused (n=5)

Deeds of physical punishment included "pushed, grabbed or shoved", "threw something", or "slapped, hit, or punched." More severe forms of physical punishment, such as burning and hanging, were not assessed due to a concern about upsetting the respondent. Thus, this variable is slightly different from a variable that appears in some of the previous literature on physical abuse in childhood. For this reason, we labeled this variable "childhood physical punishment". In order to gain statistical efficiency, we created a new binary variable for CPP. Respondents who answered "often" and "sometimes" were coded as "yes"; "rarely" and "never" were coded as "no"; "don't know" and "refused" were coded as missing.

Other covariates as possible confounders include "parental alcohol/drug problem", "parental mental problems" and "childhood conduct problems" as well as sex and age of the respondent. "Parental alcohol/drug problem" was derived from two separate questions about alcohol/drug problems regarding mother and father, respectively. Original questions are: "Did (MAN WHO RAISED R) ever have a problem with alcohol or drugs?" and "Did (WOMAN WHO RAISED R) ever have a problem with alcohol or drugs?" Due to the low frequency of mother alcohol/drug problems, we combined maternal problems and paternal problems into one variable to represent alcohol/drug problems of both parents. "Don't know" and "refused" were coded as missing values. Responses were coded as the following

1= either father or mother or both had alcohol or drug problems (n=100)

0= neither father or mother had alcohol or drug problems (n=1494)

missing values (n=34)

"Parental mental problems" included mother/father depressive mood, anxiety, and suicide attempt. Original questions for the woman who raised the respondent are: "*During the years you were growing up, did (WOMAN WHO RAISED R) ever have periods lasting 2 weeks or more where she was sad or depressed most of the time?*", "*during the time you were growing up, did (WOMAN WHO RAISED R) ever have periods of a month or more when she was constantly nervous, edgy, or anxious?*", and "*Did (WOMAN WHO RAISED R) ever attempt to commit suicide?*" Three such questions were asked regarding the man who raised the respondent as well. Due to the low occurrence of the individual condition, we coded the "parental mental problem" variable as "yes" when the respondent reported any of the three conditions for either mother or father. "Don't know" and "refused" were coded as missing values. Responses were coded as the following:

1= either mother or father had above conditions (n=303)

0= neither mother or father had above conditions (n=1135)

missing (n=190)

"Childhood conduct problems" were obtained from two sets of consecutive questions about conduct problems. These questions about conduct problems were asked to respondents younger than 40 years among the sub-sample of 1628 respondents (n=570). Actual questions are listed in the following table.

Table 3.5 Actual CIDI questions assessing conduct problems
*CD1a. As a child or teenager, did you often tell lies to trick people into giving you things or doing what you wanted them to do?
*CD1b. As a child or teenager, did you often get out of doing things you were supposed to do by fooling people or lying to them?
***CD1c**. As a child or teenager, did you often stay out much later at night than your parents wanted?
*CD1d. As a child or teenager, did you often skip school without permission?
*CD1e. As a child or teenager, did you ever shoplift or steal something worth at least $10?
*CD1f. As a child or teenager, did you ever steal money or other things from your parents or the other people you lived with?
*CD1g. As a child or teenager, did you ever break into someone's locked car, or a locked home or building?
*CD1h. As a child or teenager, did you ever set a fire to try to cause serious damage?
*CD1i. (Other than by setting fires,) As a child or teenager, did you ever deliberately damage someone's property by doing something like breaking windows, slashing tires, vandalizing, or writing graffiti on buildings?
*CD1j. As a child or teenager, did you ever run away from home and stay away for at least four days?
*CD1k. As a child or teenager, did you run away from home overnight more than once?
*CD16b. As a child or teenager, did you often get involved in physical fights?
*CD16c. As a child or teenager, did you ever use a weapon on another person, like a baseball bat, glass bottle, knife, gun, or brick?
*CD16d. As a child or teenager, were you ever physically cruel to an animal and hurt it on purpose? (IF NEC: This does not include hunting or getting rid of pests like rodents or insects.)
*CD16e. As a child or teenager, were you ever physically cruel to a person and hurt them on purpose?
*CD16f. As a child or teenager, did you ever force someone to give you something like money, jewelry, or clothing by threatening them or causing them injury?
*CD16g. As a child or teenager, did you ever steal someone's purse, wallet, luggage, package or bag by grabbing it from them? (IF NEC: This does not include stealing from someone who wasn't aware of the theft, such as stealing a piece of luggage when the owner wasn't watching.)
*CD16h. As a child or teenager, did you ever make anyone do something sexual by either forcing, intimidating, or threatening them?

Respondents who answered "yes" to any of the above question were coded as "yes". Respondents who answered "no" to all of above questions were coded as "no". "Don't know" and "refused" answers were coded as missing values. Responses were coded as the following:

1= ever had conduct problem(s) (n=88)

0= never had above conditions (n=458)

missing (n=24)

3.5 Analysis Plan

In order to address the study aims and fulfill corresponding estimation tasks, several statistical techniques have been used for this project. A standard "explore, analyze/estimate, explore" three step cycle was used for analysis. Described below are the statistical methods for each study aim.

Aim 1: To describe beverage alcohol involvement in two metropolitan cities in China: Beijing and Shanghai

1. Frequency tables and estimated proportions were used to study the lifetime occurrence of drinking-related variables including opportunity to use alcohol, trying alcohol, alcohol drinking (>=12 drinks/year), frequency of drinking when drank the most, heavier drinking, as well as diagnosis of alcohol abuse/harmful use and alcohol dependence as outlined in DSM-IV and ICD-10. Additionally, the estimated occurrence of clinical features of socially maladaptive drinking and alcohol dependence as outlined in DSM-IV and ICD-10 were plotted. Further, lifetime occurrence estimates wsere derived for early onset of trying alcohol (< 13 years), early onset of MTM drinking (< 20 years), early onset of socially maladaptive drinking (< 23 years), and early onset of the first clinical feature of alcohol dependence (< 23 years).

2. In the next, interval prevalence was estimated for the period of 12 months prior to the assessment with respect to alcohol drinking, heavier drinking, frequency of drinking, as well as diagnosis of alcohol abuse or harmful use and alcohol dependence as outlined in DSM-IV and ICD-10. Frequencies and estimated proportion have been used to describe the recency of the clinical features of alcohol abuse or harmful use and alcohol dependence.

3. Finally, Kaplan-Meier methods have been used to describe the age of first trying alcoholic beverages, the age of onset of MTM drinking, the age of first socio-maladaptive drinking problem, and the age of the first clinical feature of alcohol dependence. Range, means, and medians have been presented for these variables as well as number of drinks during a typical drinking day when the respondent drank the most and during the 12 months prior to the assessment, respectively.

These estimates have been presented for both specific sites and for the entire sample.

Aim 2: Estimation of subgroup variations with respect to alcohol drinking and related problems

Subgroup specific estimation of lifetime occurrence and 12-month prevalence of drinking-related variables have been presented with respect to sex, age groups, marital status, education attainment, income level, and employment status. Subgroup variations in drinking behaviors and drinking-associated problems of these variables were addressed via generalized linear

models with a logit link (logistic regression). The strength of association is estimated in the form of odds ratios (OR). The statistical robustness is evaluated by the p value with a $p<0.05$ considered to be statistically significant by conventional standards. The following equation depicts logistic regression: $$\log(\frac{p}{1-p}) = \beta_0 + \sum \beta_i x_i$$

p is the probability of the occurrence of the outcome;

β_0 is the log odds of the occurrence of the outcome when all covariates, X, are zero;

β_i is the log odds of the occurrence of the outcome for each unit increase in x_i relative to the log odds of the outcome when $x_i=0$.

Multiple covariates can be entered into logistic regression in order to hold others constant. In this study, both unadjusted OR (uOR) and adjusted OR (aOR) are presented. To estimate the aOR for sex, age was held constant; to estimate the aOR for age groups, sex was held constant; and to estimate the aOR for all other variables, sex and age were held constant.

Aim 3: To estimate the association between childhood physical punishment (CPP) in riskier drinking and associated problems in order to shed light on the suspected causes of drinking problems.

1. First, estimates wre produced for a line chart description of the occurrence of drinking associated outcomes for individuals with CPP experience and without CPP experience, respectively.

Next, logistic regressions were used to estimate the association between CPP and drinking-related outcomes with a focus on indicators of riskier drinking behavior. These indicators included lifetime occurrence of "heavier drinking", "socially maladaptive drinking", "DSM-IV alcohol dependence", "any alcohol dependence clinical feature", "early try (<13)", "early MTM drinking (<20)", "early socio-maladaptive problems (<23)", "early dependence problems (<23)", and "early any problem (<23)". A series of logistic regression models was fit to assess the strength of association between CPP and drinking-related outcomes as well as the statistical independency of the association from other covariates: the participant's sex, age, parental alcohol/drug problems, parental mental problems, and history of conduct problems.

Due to the cross-sectional nature of the study design, it is difficult to pin down temporal sequencing. For example, parent may punish children physically when children start to drink too early or had problems with drinking. Therefore, an attempt was made in order to probe into this issue. First, the variable "onset of problems after 16" was created, where individuals with an onset of problems before 16 years old were excluded. Second, in one of the models for each outcome, we restricted the analytical sample to individuals who started MTM drinking after 16 years old.

Subsequently, possible misspecification of logistic regression models was explored via an adapted version of the Lemeshow-Hosmer test for goodness-of-fit for complex survey data (K.J. Archer & Lemeshow, 2006). The

Lemeshow-Hosmer test evaluates the departure of predicted probability from observed probability. The first step of Lemeshow-Hosmer test consists of sorting observations into *g* (e.g. *g*=10) groups according to their predicted probability. The observed number of cases in the in the *d*th decile is given by O_{1d}; the observed number of non-cases in the *d*th decile is given by O_{0d}; similarly, expected numbers of cases and non-cases are given by E_{1d} and E_{0d}, respectively. The Lemeshow-Hosmer test is calculated by

$$\hat{C}_g = \sum_{h=0}^{1}\sum_{d=1}^{g}\{\frac{O_{hd} - E_{hd})^2}{E_{hd}}\}$$

. The test statistic follows a chi-square distribution with *g*-2 degree of freedom (DF; (Hosmer & Lemeshow, 1980). However, this test is only suitable for random samples. It is not suitable for data collected from multi-stage sampling, where within cluster variations are usually smaller than between cluster variations and a probability weight is involved. To overcome this, Archer and Lemeshow proposed a F-corrected Wald test to test goodness-of-fit for multi-stage survey data (K.J. Archer & Lemeshow, 2006; K. J. Archer, Lemeshow, & Hosmer, 2007). The Wald test statistic for *g* groups is given by

$$\hat{Q}_M = \frac{(f-g+2)}{(fg)}\hat{M}^t\hat{V}(\hat{M})^{-1}\hat{M}$$

, where *f* =(number of PSU-number of strata), *g* =number of groups, $\hat{M}$ is the sum of mean residuals by deciles, $\hat{V}$ is estimated variance-covariance matrix, the estimation of which

involves a Taylor series linearization. The statistic follows an F distribution with g-1 numerator DF and f-g+2 denominator DF (K.J. Archer & Lemeshow, 2006).

As described in the introduction, CPP might be influenced by parental drinking/drug problems and parental mental disturbances. Thus, estimates from logistic regression might have been biased or inconsistent due to a possible endogeneity problem. In other words, the assumption of independent error terms might have been violated. In this context, recursive probit regression and instrumental variable methods were used to explore the possible endogeneity of CPP (Avery, 2005). The bivariate probit with dummy endogenous variable, or recursive probit model, was first introduced by Heckman (Heckman, 1978). In this model, two probit equations are fit simultaneously to the data:

$$y_2^* = x_1'\beta_1 + \varepsilon_1 \text{ , (4.1)}$$

$$y_1^* = x_2'\beta_2 + \gamma_2 y_2 + \varepsilon_2 \text{ , (4.2)}$$

$$COV[\varepsilon_1, \varepsilon_2 | x_1, x_2] = \rho .$$

Thus, the parameter rho represents the correlation between error terms of the two equations. Non-zero rho indicates the existence of correlation. Using this approach, possible endogeneity of CPP from parental variables as well as "conduct problem" have been explored. The log-likelihood function is

given by $LnL = \sum_{j=1}^{n} w_j \ln \Phi_2 (q_1 \xi_j^{\beta}, q_2 \xi_j^{\gamma}, \rho_j^*)$ (StataCorp LP., 2005). Instead of directly estimating ρ, $a \tanh \rho = \frac{1}{2} \ln(\frac{1+\rho}{1-\rho})$ is estimated from this maximum likelihood estimation. Although the likelihood function treats the two equations as if they are exogenous, it can be shown that when y2 is observed as binary, the likelihood function is the same (Wooldridge, 2002). Thus, the γ_2 is a legitimate estimate of the CPP-drinking outcome association taking the endogeneity into account (if there is any).

Therefore, a bootstrap re-sampling approach with replacement was used to investigate the internal validity and stability of the model-based estimates. This approach was encouraged by Royston and Sauerbrei (2008) in their text on multivariable model building and evaluation. As part of the third step in the analysis approach for this dissertation research, the bootstrap was used to draw 600 bootstrap samples of size 1628 from the WMH-mC dataset. Then, the final model was fit to these 600 bootstrap samples in order to produce a distribution of estimates of the CPP-alcohol association. The actual empirical distribution of estimates was used to generate the 95% confidence interval. Two models were evaluated for internal validity and stability: estimates for the occurrence of socially maladaptive drinking when adjusting for sex, age, parental alcohol/drug problems; the occurrence of

early onset of socially maladaptive drinking when adjusting for sex, age, parental alcohol/drug problems.

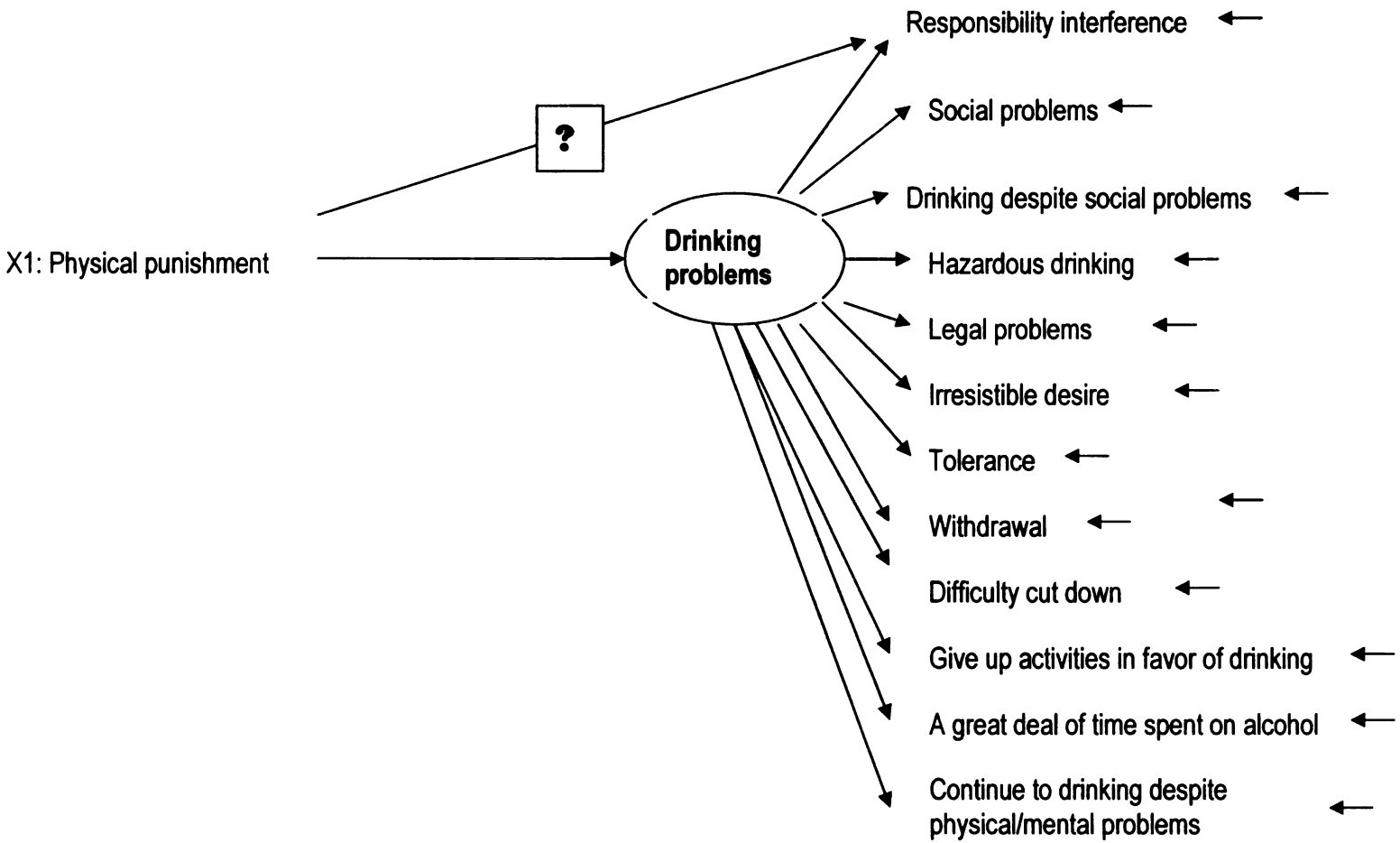
Next, to investigate this possible causal relationship between CPP and drinking problems, there was probing into questions of whether there might be variations in the associations across different outcomes. Comparing the confidence intervals from logistic regression leads to loss of power because it ignores the correlation between outcomes. For example, a person with history of heavier drinking has a higher probability to have socially maladaptive problems than a person with no history of heavier drinking. Hence, comparing confidence intervals is too conservative and may lead to erroneous inference when correlated outcomes are present. The Generalized Estimating Equations (GEE) proposed by Liang and Zeger (Zhao et al., 2004) takes both within-cluster and between cluster information into account in estimating coefficients. GEE also provides direct statistical comparison in order to answer the question proposed above. Thus, in the next steps of analysis, the GEE was used to examine variations across different drinking outcomes in associations between CPP and drinking outcomes, taking the inter-correlation of these outcomes into account. The GEE model is a marginal model and does not require specification of a specific joint distribution. When the study interest is the population mean, GEE provides an efficient and valid tool for this estimation problem.

The GEE model requires independency between clusters (individuals in this case), which might not be met due to the complex study design of this

study. Thus, a variant of GEE, the Alternating Logistic Regression (ALR) is used. ALR is capable of modeling multiple levels of clustering by alternating between the marginal logistic regression and the log odds ratio within clusters (Carey, Zeger, & Diggle, 1993; Katz, Carey, Zeger, & Sommer, 1993). Here we treated neighborhood as the level of geographical clustering because it is the final unit of sampling of the study. The ALR approach can be time-consuming and sometimes has difficulty in convergence. The GEE has the advantage of computational efficiency over the ALR.

Lastly, when robust variations in these associations were found, the Multiple Indicator Multiple Cause **(MIMIC)** model has been used to probe into whether the observed variation might be due to differential item-level responses, holding the level of problematic drinking constant. The MIMIC model is within the framework of latent variable analysis (Muthen & Muthen, 2007). The diagram of the model is depicted in Figure 3.4. In the MIMIC model, information was gathered from multiple clinical features of alcohol dependence to create a dimensional latent variable. The existence of CPP-associated differential item functioning can be tested by simultaneously regressing the latent alcohol dependence variable and specific clinical features on CPP. If the estimated path from CPP to an item response is non-null, it indicates reporting biases. The technique has been previously used in the study of psychiatric conditions including alcohol and drug dependence (C. Y. Chen & Anthony, 2003; Gallo, Anthony, & Muthen, 1994; Harford & Muthen, 2001).

Figure 3.4. *Diagram of the MIMIC model*



One goal has been to find out whether CPP affects every stage of alcohol involvement, or only a subset of stages. The statistical tool is logistic regression. Stages of alcohol involvement include

1) the opportunity to drink alcohol,
2) ever tried alcohol given the opportunity
3) ever drinking at least 12 drinks per year given tried
4) ever heavier drinking given drinking
5) ever socio-maladaptive problems given drinking
6) ever clinical features of alcohol dependence given drinking

Analyses consist of a series of logistic regressions to show the crude OR and adjusted ORs after holding potential confounders constant. Since the occurrence of the first stages of alcohol involvement is quite high (>50%), the OR does not simulate relative risk (RR) well. To ease interpretation, we used the formula $RR = \frac{OR}{(1-P_0)+(P_0 \times OR)}$ to convert OR to RR (Zhang & Yu, 1998).

Special issues regarding variance estimate

As a consequence of the multi-stage sampling procedure, participants are not completely independent. That is, an individual is more likely to be similar to an individual from the same neighborhood than one from another neighborhood. In addition, in this study, participants had different probabilities to enter part II of assessment depending on their response to part I. To address these issues, sample weights were generated to account for differential participation and part I/part II selection. A Taylor series

linearization method was used for variance estimation, with due attention to variation in sample selection probabilities, post-stratification adjustment factors, and the nested strata created as part of the multi-stage probability sampling procedures. The purpose of doing this is to minimize possible estimation bias and make results more generalizable to the source population, non-institutional household dwelling adults living in Beijing and Shanghai, China.

Analyses have been implemented using statistical software Stata (version 9.2, StataCorp LP), Mplus (version 5.1, Muthén and Muthén), and SAS (version 9.1, SAS Institute Inc).

Chapter 4 Results

This chapter presents the results of analyses corresponding to each of the study aims.

4.1. Lifetime occurrence and 12 month prevalence of drinking-related outcomes

4.1.1. Lifetime occurrence

Lifetime occurrence of drinking behavior and drinking-related outcomes are provided in table 4.1.1. Results show that, for the population under study as a whole, alcohol is very accessible. Only a small fraction of people had never had a chance to drink alcohol and the majority of people have tried alcohol at least once (71.9% in Beijing and 69.9% in Shanghai). And less than half of people had ever consumed 12 drinks in a given year during their lifetime up until the assessment (45.3% in Beijing and 39.2% in Shanghai). For the third of people who drank, the frequency of drinking is quite evenly distributed across four categories of frequencies. For both sites, about 10% of people drank nearly everyday when drank the most. The median number of drinks consumed is two for the periods when the respondent drank the most. Histogram of number of drinks in a day is presented in figure 4.1.1 for the period when the respondent drank the most.

More serious drinking, by contrast, is relatively rare. Less than 10% of people had ever drunk heavily (8.0% in Beijing and 6.9% in Shanghai). Less than 7% of people ever had socially maladaptive problems because of

drinking and only 2% of people met criteria of alcohol dependence posed by DSM-IV or ICD-10. One thing merits mention is that the occurrence of drinking-induced problems is consistently higher in Beijing than in Shanghai. The occurrence of drinking-related problems in people living in Beijing is approximately twice of that in those living in Shanghai. The difference is statistically significant at a 0.05 level. This difference remains robust even after holding drinking frequencies, history of heavier drinking, as well as smoking status, constant (OR=0.5, 95%, 0.3, 0.8).

With respect to the age of onset of alcohol involvement, the median age of the first sip of alcohol is 18 years old; the median age is 20 for the onset of MTM drinking (>=12 drinks in a year) among MTM drinkers. The median age of onset of drinking-related problems is in the early to mid 20s. Details about the distribution of these variables are presented in table 4.1.2 and figure 4.1.3 to figure 4.1.6. For the population as a whole, slightly more than 10% of people tried alcohol before teens (11.3% in Beijing and 13.7% in Shanghai); and 19.5% of people started drinking before 20 years old in Beijing, 12.3% in Shanghai. In Beijing, 2% of people initiated socially maladaptive problems before 23 years old, 1.1% in Shanghai; and 0.9% had the first dependence clinical feature before 23 in Beijing, 0.3% in Shanghai.

We also provided line chart for a site-specific criterion-wise description of the lifetime occurrence for each socially maladaptive problem and clinical features of dependence (figure 4.1.6). The lifetime occurrence is consistently lower in people living in Shanghai than that in people living in

Beijing. "Hazard-laden drinking" and "drink more than intended" are the most common clinical features for both sites. "Interfere with responsibility" is also one of the most common clinical features in Beijing. The least occurred clinical feature is "legal problems".

Table 4.1.1. Lifetime cumulative occurrence of alcohol drinking related variables. Data from the WMH-mC, 2001-2002.

		Entire Sample				Beijing				Shanghai				
		n	%	wt%[1]	wt% s.e.	n	%	wt%[1]	wt% s.e.	n	%	wt%[1]	wt% s.e.	p 2
Opportunity*	Yes	1258	79.0	79.7	1.5	722	80.0	81.2	1.6	536	77.7	77.8	2.9	0.29
	No	334	21.0	20.3		180	20.2	18.8		154	22.3	22.2		
Ever tried alcohol	Yes	3340	66.8	69.8	1.2	1758	67.9	71.4	1.4	1582	65.6	68.0	1.9	0.16
	No	1660	33.2	30.2		832	32.1	28.6		828	34.4	32.0		
Early Trying alcohol	Yes	487	10.0	12.5	0.7	201	7.9	11.3	1.1	286	12.4	13.7	1.0	
	No	4366	90.0	87.6		2346	92.1	88.7		2020	87.6	86.3		
MTM drinking	Yes	2077	40.2	42.0	1.1	1146	43.6	45.1	1.7	931	37.6	38.9	1.6	0.01
	No	3095	59.8	58.0		1480	56.4	54.9		1615	62.4	61.3		
Early onset of MTM drinking	Yes	681	13.2	15.9	0.8	382	14.6	19.5	1.3	299	11.8	12.3	0.8	p<0.01
	No	4463	86.8	84.1		2230	85.4	80.5		2233	88.2	87.7		
Heavy drinking	Yes	349	6.8	7.4	0.6	189	7.2	8.0	0.8	160	6.3	6.9	0.8	
	No	4815	93.2	98.6		2431	92.8	92.0		2384	93.7	93.1		
Frequency of drinking														
nearly everyday		610	11.8	10.3	0.6	347	13.2	10.9	1.0	263	10.3	9.7	0.6	p<0.05
3-4 days/week		277	5.4	5.8	0.5	146	5.6	6.1	0.7	131	5.1	5.5	0.6	
1-2 days/week		417	8.1	9.1	0.6	253	9.7	11.1	0.9	164	6.4	6.9	0.7	
1-3 days/month		395	7.6	8.6	0.6	182	6.9	7.7	0.7	213	8.3	9.5	1.0	
< monthly		1817	35.1	37.0	1.0	861	32.9	36.0	1.4	956	37.4	38.1	1.6	
no drink		1660	32.1	29.3	1.2	832	31.7	28.2	1.4	828	32.4	30.3	1.8	
DSM-IV alcohol abuse	Yes	221	4.3	4.7	0.4	145	5.5	6.3	0.7	76	3.0	3.0	0.4	p<0.01
	No	4957	95.7	95.3		2475	94.5	93.7		2482	97.0	97.0		
ICD-10 alcohol abuse	Yes	238	4.6	5.1	0.4	157	6.0	6.9	0.7	81	3.2	3.2	0.5	p<0.01
	No	4942	95.4	94.9		2465	94.0	93.1		2477	96.8	96.8		

Table 4.1.1 (cont'd)														
ICD-10 alcohol dependence	Yes	37	0.7	0.9	0.2	27	1.0	1.3	0.3	10	0.4	0.4	0.1	p<0.01
	No	5153	99.3	99.1		2604	99.0	98.7		2553	99.6	99.6		
DSM-IV alcohol dependence	Yes	41	0.8	1.0	0.2	32	1.2	1.6	0.4	9	0.4	0.4	0.1	p<0.01
	No	5153	99.2	99.0		2599	98.8	98.4		2554	99.7	99.6		
Ever had any alcohol dependence clinical feature	Yes	91	1.8	1.9	0.3	62	2.4	2.7	0.5	29	1.1	1.1	0.2	p<0.01
	No	5108	98.3	98.1		2569	97.6	97.3		2539	98.9	98.9		
Onset of any alcohol abuse clinical features	<=22	80	1.6	2.0	0.3	51	2.0	2.9	0.5	29	1.1	1.1	0.3	p<0.01
	>22	148	2.9	2.7		101	3.9	3.7		47	1.8	1.7		
	No	4945	95.6	95.2		2467	94.2	93.4		2478	97.0	97.1		
Onset of any alcohol dependence clinical features	<=22	26	0.5	0.6	0.2	16	0.6	0.9	0.3	10	0.4	0.3	0.1	p<0.01
	>22	63	1.2	1.2		45	1.7	1.8		18	0.7	0.7		
	No	5108	98.3	98.2		2569	97.7	97.4		2539	98.9	99.0		

[1] Weighted percentage. Due to rounding, percentages may not add up to 100.0%.

[2] p value of between site variations.

Figure 4.1.1 Number of drinks per day when drank the most. Data from WMH-mC. 2001-2002

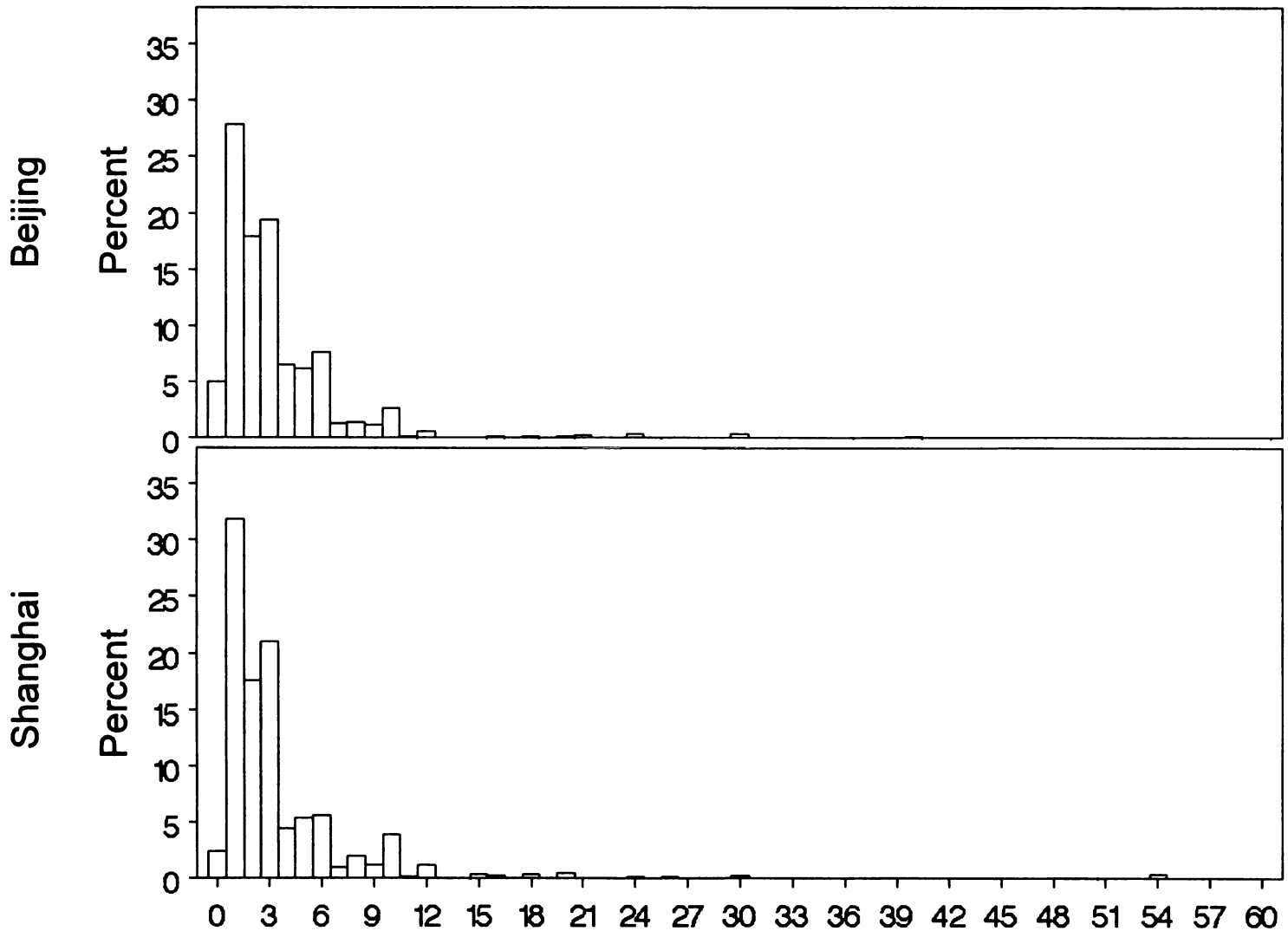
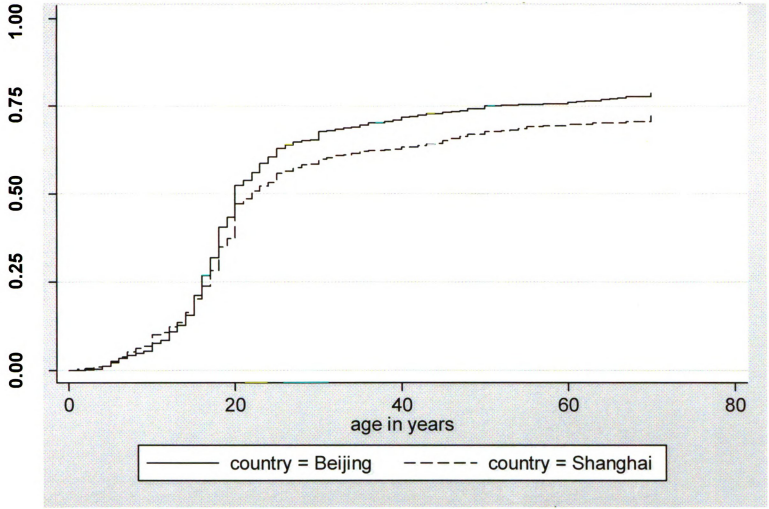


Figure 4.1.2 Kaplan-Meier failure function of the age of trying alcoholic beverages



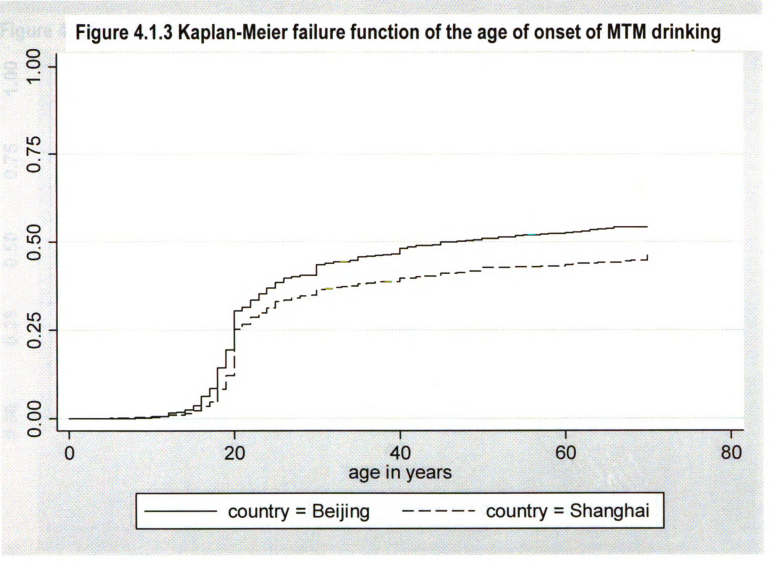


Figure 4.1.3 Kaplan-Meier failure function of the age of onset of MTM drinking
0.00
0.25
0.50
0.75
1.00
0
20
40
60
80
age in years
country = Beijing
country = Shanghai

Figure 4.1.4 Kaplan-Meier failure function of the first socially maladaptive drinking

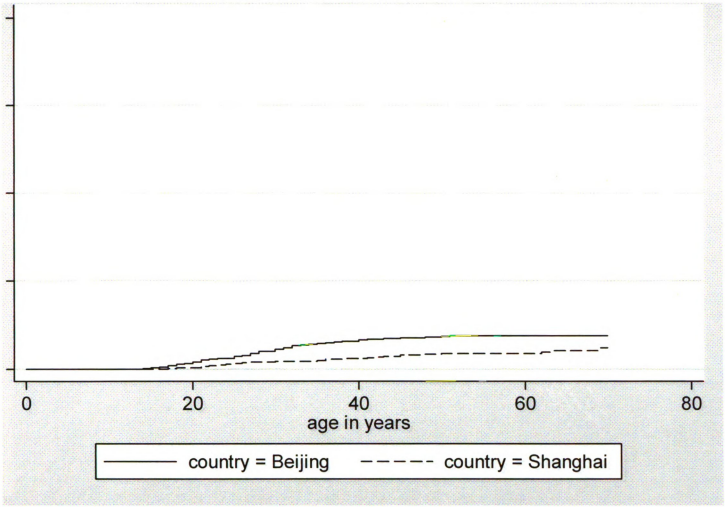


Figure 4.1.5 Kaplan-Meier failure function of the first dependence problems

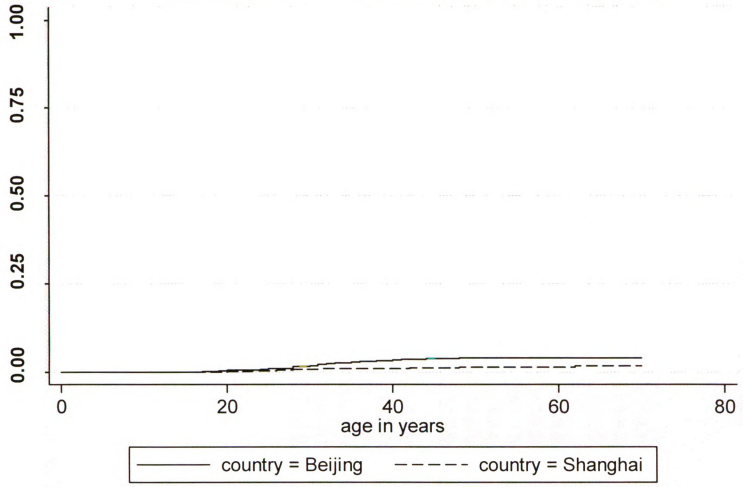
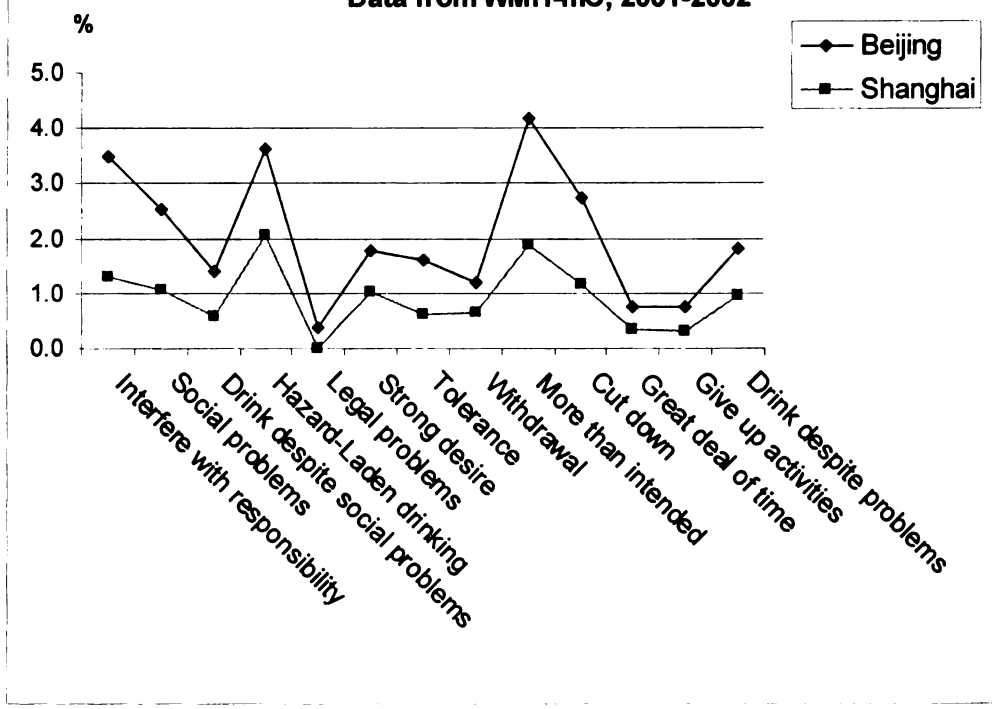


Table 4.1.2. Description of alcohol drinking related variables. Data from the WMH-mC, 2001-2002

	Entire Sample					
age	n	range	mean	median	weighted mean (s.d.)	weighted median
first try	3193	0, 66	17.7	18	16.8 (7.8)	18
onset of drinking	2049	3, 67	22.9	20	21.8 (7.3)	20
1st social maladaptive problem	200	10, 52	27.5	26	25.6 (7.0)	23
1st dependence problem	73	15, 48	27.8	28	26.6 (4.3)	24
# of drinks/day when most	1593	0, 60	3.3	3	3.5 (4.5)	2
# of drinks/day during last year	1314	0, 60	2.4	2	2.5 (2.7)	2
			Beijing			
first try	1715	0, 66	18.2	18	16.8 (8.6)	18
onset of drinking	1132	3, 65	23.3	20	21.7 (7.3)	20
1st social maladaptive problem	134	10, 52	27.3	26.5	25.0 (7.2)	23
1st dependence problem	49	15, 40	28.3	30	26.5 (3.4)	24
# of drinks/day when most	886	0, 60	3.2	2	3.4 (4.1)	2
# of drinks/day during last year	738	0, 60	2.4	2	2.5 (2.8)	2
			Shanghai			
first try	1478	1, 54	16.9	18	16.8 (6.5)	18
onset of drinking	917	3, 67	22.3	20	22.0 (7.2)	20
1st social maladaptive problem	66	10, 48	27.7	25	27.1 (6.2)	23
1st dependence problem	24	17, 48	26.8	24.5	26.8 (6.1)	23
# of drinks/day when most	707	0, 54	3.4	3	3.6 (4.9)	2
# of drinks/day during last year	576	0, 54	2.4	2	2.5 (2.5)	2

Figure 4.1.6. Lifetime occurrence of drinking-related problmes. Data from WMH-mC, 2001-2002



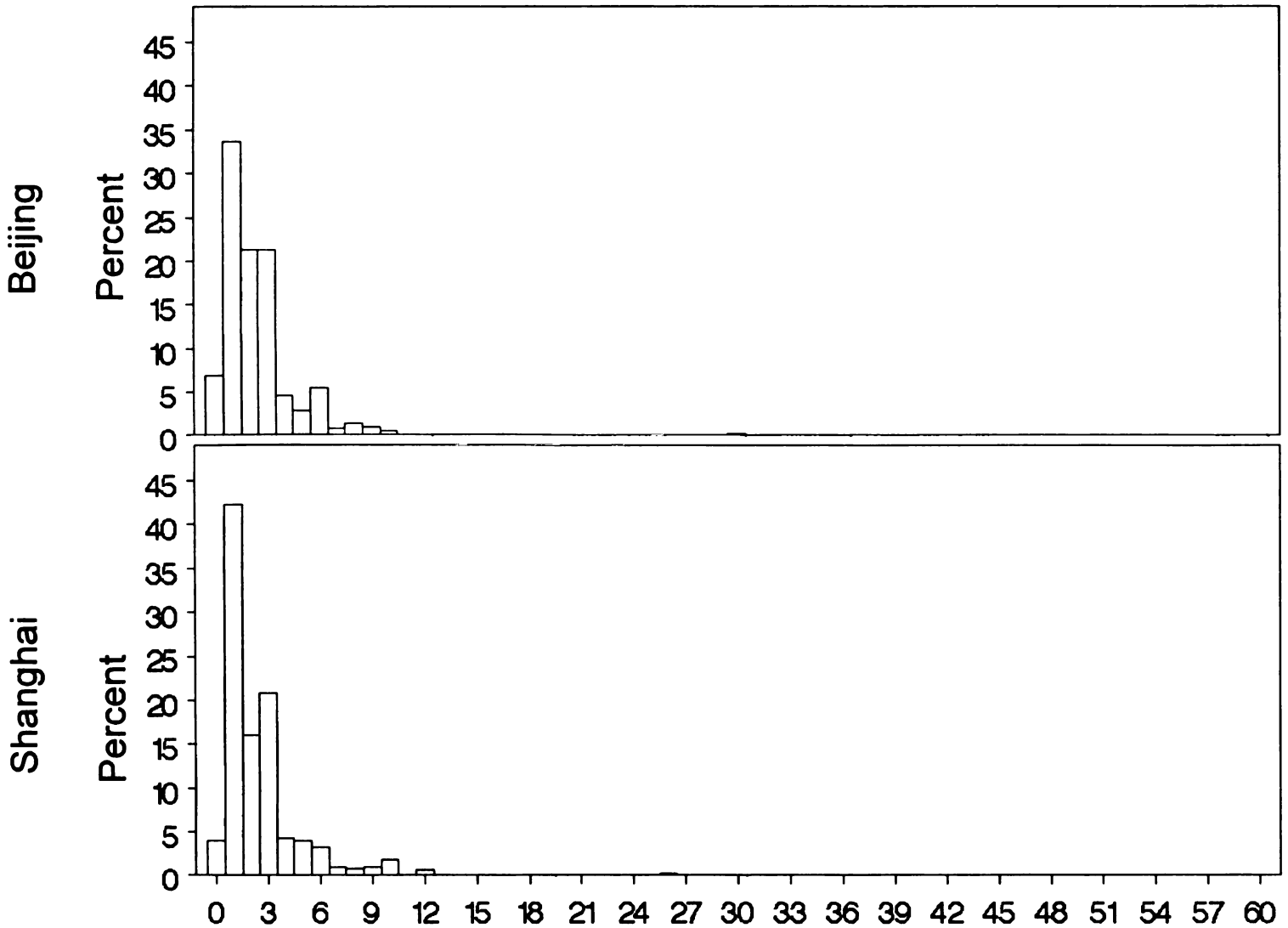
4.1.2 12-month prevalence of drinking-related outcomes

Drinking in general is not an overly common phenomenon during the 12 months prior to the assessment. Approximately one third of the population did not drink alcohol during the past 12 months (28.2% in Beijing and 30.4% in Shanghai), and another 40% drank less than monthly (41.5% in Beijing and 43.5% in Shanghai). An estimated one in thirteen people in Beijing and one in fourteen people in Shanghai drank everyday during the 12 months prior to the assessment. Only 3% of people drank heavily (>=5 drinks in a day for males and >=4 drinks in a day for females) during the past 12 months. The prevalence proportions of drinking-related problems are also low. The 12-month prevalence of any socially maladaptive problem is 2.1% in Beijing and 0.7% in Shanghai; the prevalence of alcohol dependence is less than 1% according to either DSM-IV criteria or ICD-10 criteria. Details can be found in table 4.1.3. The histogram of the number of drinks in a typical drinking day is provided in figure 4.1.7.

Table 4.1.3. Twelve-month prevalence of alcohol drinking related variables. Data from WMH-mC, 2001-2002.

		Entire Sample				Beijing				Shanghai				*p* value of between site variation
		n	%	wt%	wt% s.e.	n	%	wt%	wt% s.e.	n	%	wt%	wt% s.e.	
MTM drinking	Yes	1419	27.5	28.2	0.8	787	30.1	30.3	1.2	632	24.8	26.1	1.0	p<0.01
	No	3744	72.5	71.8		1831	69.9	69.7		1913	75.2	74.0		
Heavy drinking	Yes	147	2.9	3.3	0.4	77	3.0	3.5	0.5	70	2.8	3.1	0.4	p>0.05
	No	4949	97.1	96.7		2507	97.0	96.5		2442	97.2	96.9		
Frequency of drinking														
nearly everyday		459	8.9	7.3	0.4	259	9.9	7.6	0.7	200	7.9	7.0	0.5	p>0.05
3-4 days/week		200	3.9	4.0	0.3	115	4.4	4.4	0.5	85	3.3	3.7	0.4	
1-2 days/week		371	7.2	8.0	0.5	218	8.3	9.2	0.9	153	6.0	6.7	0.6	
1-3 days/month		389	7.5	8.9	0.6	195	7.5	9.1	0.9	194	7.6	8.6	0.8	
< monthly		3744	72.6	71.8	0.8	1831	70.0	69.7	1.2	1913	75.1	73.9	1.0	
DSM-IV alcohol abuse	Yes	57	1.1	1.3	0.2	40	1.5	2.0	0.4	17	0.7	0.6	0.1	p<0.01
	No	5121	98.9	98.7		2580	98.5	98.0		2541	99.3	99.4		
ICD-10 harmful use	Yes	63	1.2	1.4	0.2	43	1.6	2.1	0.5	20	0.8	0.7	0.1	p<0.01
	No	5115	98.8	98.6		97.91	98.4	97.9		2538	99.2	99.3		
DSM-IV alcohol dependence	Yes	23	0.4	0.6	0.2	16	0.6	0.9	0.3	7	0.3	0.3	0.1	p<0.05
	No	5171	99.6	99.4		2615	99.4	99.1		2556	99.7	99.7		
ICD-10 alcohol dependence	Yes	22	0.4	0.5	0.1	14	0.5	0.8	0.2	8	0.3	0.3	0.1	p<0.05
	No	5172	99.7	99.5		2617	99.5	99.2		2555	99.7	99.7		

Figure 4.1.7 Number of drinks per day during the past year. Data from WMH-mC, 2001-2002



4.2. Subgroup variations of lifetime occurrence and 12 month prevalence of drinking-related outcomes

4.2.1. Male is associated with higher likelihood of drinking-related outcomes.

Being male is associated with higher likelihood of having experienced all drinking-related outcomes studied (table 4.2.1). The strongest associations were found in more serious and problematic drinking, such as heavier drinking, socially maladaptive drinking, and alcohol dependence clinical features. For example, compared with about 1% females who ever drank heavily (>= 4 drinks in a day), approximately 13% males ever drank heavily (>=5 drinks per day); less than 10% females drank at least monthly, while more than 40% males drank at least monthly during the 12 months prior to the assessment.

Interestingly, males are more likely to ever have opportunity to drink alcohol (OR=2.5, 95% C.I. 1.8, 3.6). Further analysis showed there are age-related subgroup variations in the male-female difference in opportunity to drink alcohol. The sex-specific proportions of opportunity to drink alcohol in each age group (from the oldest to the youngest) are 85.7% in males vs. 64.9% in females (aOR=3.2 after holding age constant, $p<0.01$), 92.6% in males vs. 70.8% in females (aOR=5.3, $p<0.01$), 85.8% in males vs. 85.8% in females (aOR=1.0, $p>0.05$), and 82.9% vs. 84.8% in females (aOR=0.85, $p>0.05$).

There is no between site variation in associations between sex and these drinking outcomes except for ever tried alcohol. The association is slightly stronger in Shanghai compared with that in Beijing.

Table 4.2.1. The association between drinking-related outcomes and sex. Data from WMH-mC, 2001-2002

			n of cases	wt%[2]	OR	95% CI		aOR[3]	95% CI	
opportunity to drink alcohol [1]		female	549	71.9						
		male	709	86.8	**2.6**	**1.8**	**3.6**	**2.5**	**1.8**	**3.6**
ever tried	Beijing	female	733	61.4						
		male	1025	80.1	**2.5**	**1.8**	**3.5**	**2.5**	**1.8**	**3.5**
	Shanghai	female	612	52.8						
		male	970	81.5	**3.9**	**3.3**	**4.6**	**4.0**	**3.3**	**4.7**
MTM drinking		female	491	19.6						
		male	1586	62.0	**6.7**	**5.6**	**8.1**	**6.9**	**5.7**	**8.3**
ever heavy drinking		female	29	1.2						
		male	320	13.1	**13.2**	**9.0**	**19.5**	**11.9**	**7.6**	**18.4**
ever socially maladaptive problem		female	15	0.7						
		male	223	9.1	**17.1**	**10.1**	**29.0**	**15.0**	**7.0**	**32.3**
ever dependence clinical feature		female	6	0.2						
		male	85	3.4	**15.4**	**6.7**	**35.3**	**14.7**	**5.5**	**39.4**
past year MTM drinking		female	228	8.7						
		male	1191	45.7	**9.5**	**8.2**	**11.2**	**9.3**	**7.6**	**11.2**
past year heavy drinking		female	11	0.4						
		male	136	6.0	**14.0**	**7.6**	**26.0**	**14.1**	**7.3**	**27.5**
past year socially maladaptive problem		female	5	0.2						
		male	58	2.5	**12.5**	**5.0**	**31.3**	**10.9**	**3.9**	**30.7**
early try		female	172	9.3						
		male	315	15.2	**2.1**	**1.7**	**2.5**	**1.7**	**1.4**	**2.2**
early MTM drinking		female	94	4.8						
		male	587	26.0	**8.3**	**6.6**	**10.4**	**7.3**	**5.8**	**9.2**
early alcohol abuse or dependence		female	5	0.3						
		male	79	3.8	**17.3**	**7.0**	**42.7**	**11.3**	**4.1**	**30.6**

1. Variable available in part 2 only

2 Proportion of cases of all female/male participants.

3 aOR: OR after holding age constant

4.2.2. Subgroup variation in drinking outcomes with respect to age groups

Due to small numbers of cases in sub-group in drinking outcomes "past-year socially maladaptive problems" and "early onset of drinking problems", we combined the two older groups and the two younger groups. Also, analyses were restricted in the subsample of males for outcomes "ever socially maladaptive problems", "ever dependence clinical feature", "past-year socially maladaptive problems", and "early onset of drinking problems" due to too few female cases in each sub-group. Compared with the oldest group (born before 1949), later groups presented higher occurrence of most of the drinking outcomes studied here (table 4.2.2). More individuals in the youngest group tried alcohol before teens and initiated MTM drinking before 20, as compared to the two oldest groups ($p<0.01$). Twenty percent of individuals in the youngest group tried alcohol before teens, and 28.3% started to drink at least 12 drinks in a year before age 20. After collapsing two younger groups and two older groups, the younger group had higher occurrence of "early onset of alcohol problems": 5.7% in the younger group vs. 2.4% in the older group. No cross-sites variations were found in any outcome studies here.

Table 4.2.2. The association between drinking-related outcomes and age categories. Data from WMH-mC, 2001-2002

		n of case	wt%	OR	95% CI		aOR[2]	95% CI	
opportunity to drink alcohol [1]	before 1949	282	70.5						
	1949-1965	636	80.3	**1.7**	**1.1**	**2.7**	**1.7**	**1.1**	**2.6**
	1966-1977	207	86.9	**2.8**	**1.4**	**5.3**	**2.7**	**1.4**	**5.2**
	born after 1977	133	81.9	1.9	1.0	3.7	1.8	0.9	3.7
ever tried	before 1949	704	56.0						
	1949-1965	1721	72.3	**2.0**	**1.7**	**2.5**	**2.1**	**1.7**	**2.5**
	1966-1977	571	76.6	**2.6**	**2.0**	**3.4**	**2.6**	**2.0**	**3.5**
	born after 1977	344	68.6	**1.7**	**1.2**	**2.5**	**1.7**	**1.1**	**2.5**
MTM drinking	before 1949	413	31.5						
	1949-1965	1133	46.1	**1.9**	**1.6**	**2.1**	**2.0**	**1.7**	**2.3**
	1966-1977	352	47.3	**1.9**	**1.6**	**2.4**	**2.1**	**1.6**	**2.6**
	born after 1977	179	35.5	1.2	0.9	1.6	1.1	0.8	1.6
past year MTM drinking	before 1949	278	21.8						
	1949-1965	814	33.4	**1.8**	**1.5**	**2.1**	**1.9**	**1.6**	**2.3**
	1966-1977	232	31.2	**1.6**	**1.3**	**2.1**	**1.6**	**1.3**	**2.1**
	born after 1977	95	18.0	0.8	0.6	1.1	0.7	0.5	1.0
ever heavy drinking	before 1949	68	5.4						
	1949-1965	185	7.9	**1.5**	**1.0**	**2.2**	1.4	1.0	2.1
	1966-1977	68	9.2	1.8	1.0	3.2	1.7	0.9	3.1
	born after 1977	28	6.1	1.1	0.7	1.9	1.0	0.6	1.8
past year heavy drinking [3]	before 1949	22	4.0						
	1949-1965	75	5.8	1.5	0.7	2.9			
	1966-1977	32	8.6	2.3	1.0	5.1			
	born after 1977	11	4.3	1.1	0.4	2.9			

Table 4.2.2 (cont'd)									
ever socially maladaptive problem [3]	before 1949	20	3.1						
	1949-1965	134	10.2	**3.6**	**1.9**	**6.8**			
	1966-1977	49	12.3	**4.4**	**2.2**	**8.7**			
	born after 1977	20	7.9	**2.7**	**1.3**	**5.7**			
ever dependence clinical feature [3]	before 1965	59	3.2						
	born after 1965	26	3.5	1.1	0.7	1.7			
past year socially maladaptive problem [3]	before 1965	32	1.5						
	born after 1965	26	3.7	**2.5**	**1.3**	**4.7**			
early try	before 1949	80	6.3						
	1949-1965	177	7.9	1.3	0.9	1.8	1.3	0.9	1.7
	1966-1977	131	19.5	**3.6**	**2.5**	**5.3**	**3.6**	**2.4**	**5.2**
	born after 1977	99	20.1	**3.8**	**2.4**	**5.8**	**3.7**	**2.4**	**5.7**
early MTM drinking	before 1949	68	5.1						
	1949-1965	319	12.6	**2.7**	**1.9**	**3.8**	**2.6**	**1.8**	**3.8**
	1966-1977	151	21.3	**5.1**	**3.4**	**7.5**	**5.3**	**3.5**	**7.9**
	born after 1977	143	28.3	**7.4**	**4.9**	**11.2**	**7.9**	**4.9**	**12.5**
early alcohol abuse or dependence [3]	before 1966	40	2.4						
	Born after 1965	39	5.7	**2.5**	**1.4**	**4.7**			

1. Variable available in part 2 only

2. aOR: OR after holding sex constant

3. Estimates are based on the subsample of males.

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4.2.3. The association between marital status and drinking-related outcomes

Some subgroup variations in drinking-related outcomes are found across different marital status at the time of assessment (table 4.2.3). Compared with people who were married, those who were no longer married (divorced/widowed/separated) are more likely to have a history of heavier drinking. Between-site variations were found for the variable "ever tried alcohol". The difference lies in that "no longer married" people in Beijing were more likely to have opportunity and have tried alcohol than people who were married (table 4.2.3).

4.2.4 The association between education attainment and drinking-related outcomes

Table 4.2.4 presents the association between education attainment by the day of assessment and drinking outcomes. After holding sex and age constant, people with higher education levels are more likely to have tried alcohol and have a history of MTM drinking. In contrast, for riskier drinking behavior and drinking problems, no subgroup variations were found across education levels. No between-site variation was found in any of drinking-associated outcomes.

Table 4.2.3. The association between drinking-related outcomes and marital status. Data from WMH-mC, 2001-2002

			n of cases	wt%	OR	95% CI		aOR[2]	95% CI	
opportunity to drink alcohol [1]	married/cohab		944	77.9						
	no longer married		100	81.3	1.2	0.6	2.5	1.6	0.8	3.2
	never married		214	83.9	1.5	0.9	2.5	0.7	0.4	1.2
ever tried		married/cohab	1417	71.3						
	Beijing	no longer married	117	76.3	1.3	0.8	2.0	**2.0**	**1.3**	**3.1**
		never married	224	70.0	1.0	0.6	1.5	0.7	0.5	1.2
		married/cohab	1143	66.5						
	Shanghai	no longer married	78	58.3	0.7	0.5	1.1	1.0	0.7	1.6
		never married	361	73.9	**1.4**	**1.1**	**1.8**	1.0	0.8	1.4
MTM drinking	married/cohab		1637	43.0						
	no longer married		108	35.4	0.7	0.6	0.9	1.1	0.8	1.5
	never married		332	40.2	0.9	0.7	1.2	0.7	0.5	1.0
past year MTM drinking	married/cohab		1137	29.8						
	no longer married		75	26.5	0.9	0.6	1.1	1.3	0.9	1.9
	never married		207	24.4	**0.8**	**0.6**	**1.0**	0.8	0.6	1.2
ever heavy drinking	married/cohab		271	7.4						
	no longer married		23	10.5	1.5	0.8	2.5	**2.3**	**1.2**	**4.3**
	never married		55	7.0	0.9	0.7	1.3	0.6	0.3	1.0
past year heavy drinking [3]	currently or ever married/cohab		108	5.8						
	never married		28	6.4	1.1	0.7	1.8	1.2	0.6	2.2
ever socially maladaptive problem [3]	currently or ever married/cohab		184	9.6						
	never married		39	7.8	0.8	0.5	1.3	0.5	0.2	1.0

Table 4.2.3. (cont'd)									
ever dependence clinical feature [3]	currently or ever married/cohab	70	3.9						
	never married	15	2.3	0.6	0.3	1.2	0.4	0.1	1.4
past year socially maladaptive problem [3]	currently or ever married/cohab	43	2.4						
	never married	15	2.5	1.1	0.5	2.3	0.3	0.1	1.6
early try	married/cohab	307	9.5						
	no longer married	17	6.4	0.7	0.3	1.3	0.9	0.4	1.7
	never married	163	21.3	**2.6**	**2.1**	**3.2**	1.4	0.9	2.1
early MTM drinking	married/cohab	444	12.6						
	no longer married	37	11.7	0.9	0.6	1.4	**1.6**	**1.0**	**2.5**
	never married	200	25.2	**2.3**	**1.8**	**3.1**	0.8	0.5	1.2
early alcohol abuse or dependence [3]	currently or ever married/cohab	51	3.0						
	never married	28	5.7	**2.0**	**1.0**	**3.9**	0.5	0.1	1.7

1. Variable available in part 2 only

2. aOR: OR after holding sex, age, and age groups constant

3. Estimates are based on the subsample of males.

Table 4.2.4. The association between drinking-related outcomes and education attainment. Data from WMH-mC, 2001-2002

		# cases	wt%	OR	95% CI		aOR[2]	95% CI	
opportunity to drink alcohol [1]	1-6 yrs	107	64.4						
	7-9 yrs	335	81.5	**2.4**	**1.4**	**4.2**	1.6	0.9	2.9
	10-12 yrs	455	82.3	**2.6**	**1.6**	**4.1**	1.6	0.8	3.0
	>12 yrs	361	81.7	**2.5**	**1.4**	**4.3**	1.6	0.9	2.9
ever tried	1-6 yrs	81	47.1						
	7-9 yrs	278	69.0	**2.5**	**1.6**	**4.0**	1.6	1.0	2.5
	10-12 yrs	386	70.7	**2.7**	**1.7**	**4.2**	1.6	0.9	2.9
	>12 yrs	314	71.6	**2.8**	**1.7**	**4.6**	**1.8**	**1.1**	**3.1**
MTM drinking	1-6 yrs	47	24.9						
	7-9 yrs	181	40.0	**2.0**	**1.1**	**3.5**	1.1	0.6	2.0
	10-12 yrs	253	44.4	**2.4**	**1.5**	**3.9**	1.4	0.7	2.6
	>12 yrs	197	41.1	**2.1**	**1.3**	**3.4**	1.4	0.7	2.5
past year MTM drinking	1-6 yrs	35	18.6						
	7-9 yrs	133	27.4	1.6	0.9	3.0	0.8	0.4	1.7
	10-12 yrs	188	31.9	**2.0**	**1.2**	**3.4**	1.1	0.6	2.1
	>12 yrs	132	27.9	1.7	1.0	2.9	1.0	0.5	2.1
ever heavy drinking	1-6 yrs	10	4.8						
	7-9 yrs	42	7.6	1.6	0.5	5.0	0.7	0.2	2.7
	10-12 yrs	66	12.3	2.8	1.0	8.0	1.1	0.3	3.5
	>12 yrs	35	7.7	1.7	0.6	4.7	0.7	0.2	2.4
past year heavy drinking [3]	0-9 yrs	18	3.3						
	>9 yrs	41	8.4	**2.7**	**1.1**	**6.8**	2.3	0.9	5.6
ever socially maladaptive problem [3]	1-9 yrs	39	9.2						
	>9 yrs	75	9.0	1.0	0.5	1.9	0.7	0.4	1.5
ever dependence clinical feature [3]	0-9 yrs	25	3.9						
	>9 yrs	38	3.2	0.8	0.4	1.5	0.6	0.3	1.2
past year socially maladaptive problem [3]	0-9 yrs	15	2.0						
	>9 yrs	23	3.1	1.6	0.6	3.9	1.1	0.4	3.0
early try	0-9 yrs	36	6.1						
	>9 yrs	126	13.5	**2.4**	**1.1**	**5.0**	1.5	0.7	3.3
early MTM drinking	1-6 yrs	9	4.6						
	7-9 yrs	62	13.9	**3.4**	**1.1**	**9.9**	1.0	0.3	3.1
	10-12 yrs	103	19.5	**5.1**	**1.7**	**15.0**	1.2	0.4	3.7
	>12 yrs	86	17.5	**4.4**	**1.5**	**13.2**	1.3	0.4	3.9
early alcohol abuse or dependence [3]	0-9 yrs	11	2.5						
	>9 yrs	33	4.2	1.7	0.6	5.2	1.2	0.5	2.5

[1]. Variable available in part 2 only; [2]. aOR: OR after holding sex and age constant; [3]. Estimates are based on the subsample of males.

4.2.5. The association between personal income level and drinking outcomes

Table 4.2.5 presents associations between drinking outcomes and categorical personal income during the month prior to the assessment. After holding sex and age constant, individuals in the higher personal income categories were more likely to be involved in drinking, such as opportunity to drink alcohol, ever tried alcohol, and ever MTM drinking, compared with people in the low personal income category (less than 600 Yuan). They are also more likely to drink during the past year; and people in the highest personal income category are more likely to drink heavily during the past year (OR=2.0, 95% CI, 1.3, 3.3). In contrast, income level does not appear to be associated with riskier drinking and drinking problems.

4.2.6. The association between drinking outcomes and employment status

Individuals who had a job at the time of the assessment were associated with higher likelihood of drinking involvement, including experiences during lifetime and past year. For riskier drinking and drinking problems, no employment-status-associated subgroup variations were found (table 4.2.7).

Table 4.2.5. The association between drinking-related outcomes and the personal income level.

			# cases	wt%	OR	95% CI		aOR 2	95% CI	
opportunity to drink alcohol 1	Low		333	74.4						
	Low-average		355	80.3	1.4	0.8	2.4	**1.8**	**1.1**	**3.0**
	High-average		221	77.8	1.2	0.7	2.2	1.2	0.7	2.1
	High		324	86.4	**2.2**	**1.3**	**3.7**	**2.0**	**1.2**	**3.4**
ever tried	Low		789	61.6						
	Low-average		945	66.6	**1.2**	**1.0**	**1.5**	**1.5**	**1.3**	**1.8**
	High-average		603	73.8	**1.8**	**1.4**	**2.2**	**1.8**	**1.4**	**2.3**
	High		938	79.0	**2.4**	**1.8**	**3.1**	**2.0**	**1.5**	**2.7**
MTM drinking	Low		462	34.8						
	Low-average		576	37.8	1.1	0.9	1.4	**1.3**	**1.1**	**1.6**
	High-average		382	46.8	**1.7**	**1.3**	**2.1**	**1.6**	**1.2**	**2.1**
	High		616	50.4	**1.9**	**1.5**	**2.4**	**1.5**	**1.2**	**1.9**
past year MTM drinking	Beijing	Low	168	23.8						
		Low-average	230	27.2	1.2	0.8	1.7	1.3	0.9	1.9
		High-average	142	30.9	1.4	0.9	2.2	1.5	0.9	2.5
		High	240	39.4	**2.1**	**1.5**	**3.0**	**1.7**	**1.2**	**2.4**
	Shanghai	Low	119	16.6						
		Low-average	173	23.5	**1.5**	**1.2**	**1.9**	**1.7**	**1.3**	**2.2**
		High-average	126	35.4	**2.7**	**2.1**	**3.6**	**2.4**	**1.8**	**3.1**
		High	196	33.3	**2.5**	**2.0**	**3.2**	**1.8**	**1.4**	**2.4**
ever heavy drinking	Beijing	Low	52	8.8						
		Low-average	48	6.0	0.7	0.4	1.1	0.7	0.4	1.2
		High-average	31	8.5	1.0	0.5	1.7	0.9	0.5	1.6
		High	54	8.2	0.9	0.5	1.7	0.7	0.4	1.2
	Shanghai	Low	32	4.9						
		Low-average	39	5.7	1.2	0.7	2.1	1.3	0.7	2.3
		High-average	28	7.7	1.6	0.8	3.0	1.2	0.7	2.4
		High	59	10.2	**2.2**	**1.4**	**3.4**	1.5	0.9	2.5
past year heavy drinking 3	Low		31	4.6						
	Low-average		26	4.1	0.9	0.5	1.6	0.9	0.4	1.9
	High-average		25	6.0	1.3	0.7	2.5	1.3	0.6	2.7
	High		51	8.6	**1.9**	**1.3**	**3.0**	1.7	1.0	3.1
ever socially maladaptive problem 3	Low		63	10.1						
	Low-average		58	8.0	0.8	0.5	1.3	0.9	0.5	1.5
	High-average		30	8.5	0.8	0.5	1.4	0.8	0.5	1.4
	High		70	9.8	1.0	0.6	1.5	0.8	0.5	1.4
ever dependence clinical feature 3	Low		31	5.2						
	Low-average		18	2.4	**0.5**	**0.2**	**1.0**	0.5	0.2	1.0
	High-average		10	2.1	0.4	0.1	1.1	0.3	0.1	1.0
	High		25	3.4	0.6	0.3	1.4	0.5	0.2	1.2

Table 4.2.5. (cont'd)									
past year socially maladaptive problem [3]	Low	21	3.8						
	Low-average	14	1.7	**0.4**	**0.2**	**0.9**	0.7	0.3	1.4
	High-average	5	1.0	**0.3**	**0.1**	**0.8**	0.3	0.1	1.0
	High	18	2.8	0.7	0.3	1.8	0.7	0.2	2.1
early try	Low	123	12.5						
	Low-average	108	9.1	**0.7**	**0.5**	**1.0**	1.0	0.7	1.5
	High-average	79	10.6	0.8	0.6	1.2	1.0	0.7	1.4
	High	161	16.1	1.3	1.0	1.9	1.4	1.0	1.9
early MTM drinking	Low	185	16.8						
	Low-average	158	11.0	0.6	0.5	0.8	1.0	0.7	1.4
	High-average	114	16.6	1.0	0.7	1.4	1.3	0.9	1.8
	High	214	19.8	1.2	0.9	1.7	1.3	0.9	1.7
early alcohol abuse or dependence [3]	Low	28	5.1						
	Low-average	10	1.7	**0.3**	**0.1**	**0.8**	0.5	0.2	1.3
	High-average	12	4.7	0.9	0.4	2.0	1.4	0.6	3.4
	High	29	4.0	0.8	0.4	1.4	1.1	0.5	2.2

1. Variable available in part 2 only;
2. aOR: OR after holding sex and age constant;
3. Estimates are based on the subsample of males.

Table 4.2.7. The association between drinking-related outcomes and current employment status. Data from WMH-mC, 2001-2002

		n of cases	wt%	OR	95% CI		aOR[2]	95% CI	
opportunity to drink alcohol [1]	not working	517	732						
	working	740	85.1	**2.1**	**1.4**	**3.1**	**1.6**	**1.0**	**2.4**
ever tried	not working	1239	60.3						
	working	2099	75.7	**2.0**	**1.8**	**2.4**	**1.4**	**1.2**	**1.7**
MTM drinking	not working	697	32.4						
	working	1379	48.0	**1.9**	**1.7**	**2.2**	**1.4**	**1.1**	**1.7**
past year MTM drinking	not working	436	20.0						
	working	983	33.7	**2.1**	**1.8**	**2.4**	**1.5**	**1.2**	**1.9**
ever heavy drinking	not working	105	5.6						
	working	244	8.6	**1.6**	**1.2**	**2.2**	1.1	0.7	1.7
past year heavy drinking [3]	not working	39	4.4						
	working	97	6.7	**1.6**	**1.0**	**2.4**	1.3	0.7	2.3
ever socially maladaptive problem [3]	not working	67	8.1						
	working	156	9.6	1.2	0.8	1.8	0.7	0.5	1.2
ever dependence clinical feature [3]	not working	29	3.8						
	working	56	3.1	0.8	0.5	1.5	**0.4**	**0.2**	**0.8**
past year socially maladaptive problem [3]	not working	20	3.0						
	working	38	2.2	0.7	0.4	1.5	0.5	0.2	1.1
early try	not working	183	11.3						
	working	304	13.2	1.2	0.9	1.5	0.9	0.7	1.1
early MTM drinking	not working	207	12.3						
	working	474	18.2	**1.6**	**1.3**	**2.0**	1.1	0.9	1.5
early alcohol abuse or dependence [3]	not working	27	4.0						
	working	52	3.7	0.9	0.5	1.6	0.8	0.4	1.6

1. Variable available in part 2 only

2. aOR: OR after holding sex and age constant

3. Estimates are based on the subsample of males.

4.3. The impact of childhood physical punishment on alcohol drinking outcomes

4.3.1. The associations between childhood physical punishment (CPP) and drinking outcomes.

4.3.1.1. Estimation using logistic regressions

As shown in the figure 4.3.1.1, figure 4.3.1.2, and table 4.3.1.1, individuals, who suffered childhood physical punishment (CPP), are more likely to have experienced undesirable drinking outcomes in both cities. ORs were mostly moderate. No between site variation in the association between CPP and drinking outcomes were found. After adjusting for sex and age, estimates are not appreciably different; all estimates remained statistically robust (table 4.3.1.1).

Figure 4.3.1.1. Lifetime occurrence of drinking outcomes stratified by experience of childhood physical punishment in Beijing.

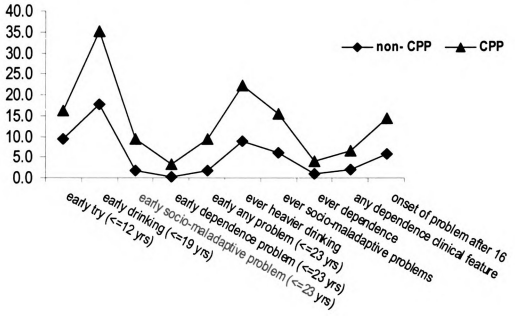


Figure 4.3.1.2. Lifetime occurrence of drinking outcomes stratified by experience of childhood physical punishment in Shanghai.

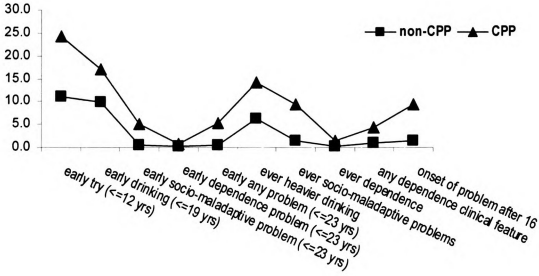


Table 4.3.1.1. Associations between childhood physical punishment and riskier drinking and problems. Data from WMH-mC, 2001-2002

	CPP	n of cases	weighted %	uOR	95% C.I.		p^1	aOR[2]	95% C.I.		p^1
early try (<=12 yrs)	no	121	9.9								
	yes	39	19.1	**2.1**	**1.2**	**3.9**	0.60	**2.0**	**1.1**	**3.8**	0.77
early MTM drinking (<=19 yrs)	no	194	14.3								
	yes	66	28.9	**2.4**	**1.6**	**3.8**	0.53	**2.3**	**1.4**	**3.8**	0.32
early socially maladaptive drinking (<=23 yrs)	no	26	1.2								
	yes	18	7.8	**7.0**	**2.5**	**19.1**	0.62	**6.2**	**2.2**	**17.7**	0.83
early dependence problem (<=23 yrs)	no	12	0.3								
	yes	9	2.4	**8.2**	**2.7**	**24.8**	0.15	**7.7**	**2.5**	**23.7**	0.11
early any problem (<=23 yrs)	no	28	1.3								
	yes	19	7.9	**6.8**	**2.5**	**18.3**	0.54	**6.0**	**2.1**	**16.9**	0.73
ever heavier drinking	no	112	7.7								
	yes	40	19.4	**2.9**	**1.8**	**4.7**	0.75	**2.7**	**1.6**	**4.7**	0.52
ever socially maladaptive drinking	no	85	4.0								
	yes	38	13.3	**3.7**	**1.9**	**7.2**	0.20	**3.4**	**1.7**	**6.6**	0.24
ever DSM-IV alcohol dependence	no	27	0.7								
	yes	14	3.2	**4.9**	**1.9**	**12.4**	0.72	**4.0**	**1.6**	**9.9**	0.71
any dependence clinical feature	no	44	1.5								
	yes	24	5.9	**4.2**	**1.6**	**11.2**	0.71	**3.6**	**1.4**	**9.6**	0.70
onset of problem after 16	no	81	3.9								
	yes	35	12.7	**3.6**	**1.8**	**7.0**	0.19	**3.3**	**1.7**	**6.5**	0.22

1. p value of between site variation

2. Adjusted for sex, age, and age categories.

ORs remained robust after holding sets of potential confounders (parental alcohol or drug problem, parental mental disturbances, and conduct problems of the respondent) constant (table 4.3.1.2 and 4.3.1.3), although points estimates decreased. In the subpopulation of individuals, who initiated drinking (at least 12 drinks in a year) after 16, estimates were not as precise and a few associations were not statistically robust at 0.05 level (possibly due to a lack of power); nonetheless many associations remain robust and point estimates are not distant from estimates based on the entire part II sample.

No between-site variations in these associations were found. Since no evidence of site variations was found from above analysis, samples from the two sites were combined for later analysis in order to gain statistical efficiency.

Table 4.3.1.2. The association between childhood physical punishment and alcohol drinking outcomes. Data from WMH-mC, 2001-2002

	Model 1[1]				Model 2[1]				Model 3[1](n=589)						
	aOR	95% C.I.		p[2]	aOR	95% C.I.		p[2]	CPP	n of cases	wtd%	aOR	95% C.I.		p[2]
early try (<=12 yrs)	1.9	1.0	3.7	0.75	1.3	0.6	2.8	0.90	yes	15	12.7	0.5	0.2	1.8	0.39
									no	51	14.1				
early MTM drinking (<=19 yrs)	**2.3**	**1.4**	**3.7**	0.35	**2.6**	**1.6**	**4.5**	0.40	yes	47	40.3	1.7	0.9	3.4	0.74
									no	137	30.9				
early socially maladaptive problem (<=23 yrs) [2]	**5.1**	**1.7**	**15.6**	0.64	**4.2**	**1.2**	**15.0**	0.42	yes	13	12.7	**11.4**	**2.6**	**49.8**	0.54
									no	12	1.1				
early dependence problem (<=23 yrs)	**5.9**	**2.2**	**15.8**	0.18	**6.2**	**2.0**	**19.4**	0.37	yes	5	3.1	**5.3**	**1.2**	**24.5**	0.36
									no	7	0.6				
early any problem (<=23 yrs)	**5.0**	**1.7**	**14.9**	0.55	**4.1**	**1.2**	**14.5**	0.33	yes	13	12.7	**10.2**	**2.5**	**42.1**	0.68
									no	14	1.3				
ever heavier drinking	**2.5**	**1.4**	**4.4**	0.70	**2.5**	**1.4**	**4.6**	0.95	yes	32	30.2	1.7	0.8	3.7	0.33
									no	89	20.8				
ever socially maladaptive problems	**3.1**	**1.6**	**6.0**	0.16	**2.4**	**1.1**	**5.2**	0.14	yes	29	22.2	**3.2**	**1.4**	**7.2**	0.27
									no	61	9.1				
ever dependence	**3.4**	**1.5**	**7.6**	0.61	**2.9**	**1.4**	**6.0**	0.29	yes	12	5.2	**2.4**	**1.1**	**5.4**	0.27
									no	23	1.7				
any dependence clinical feature	**3.5**	**1.4**	**8.8**	0.67	**2.4**	**1.0**	**5.6**	0.45	yes	18	9.6	2.2	0.9	5.3	0.52
									no	32	3.6				
onset of problem after 16	**3.1**	**1.6**	**6.1**	0.16	**2.5**	**1.1**	**5.4**	0.14	yes	29	22.2	**3.2**	**1.4**	**7.4**	0.26
									no	60	8.9				

1. Model1 held sex, age, age categories, and parental alcohol/drug problems constant. Model 2 additionally held parental mental problems constant. Model3 was restricted in the subpopulation of individuals who initiated drinking after 16 with the same set of covariate as in model 2.

2. p value of between site variation

Table 4.3.1.3. The association between childhood physical punishment and alcohol drinking outcomes. Data from WMH-mC, 2001-2002

		Model 4[1] (n=570)				
	CPP	N of cases	weighted% [3]	aOR	95% C.I.	p [2]
early try (<=12 yrs)	yes	23	14.9	1.2	0.5 3.3	0.79
	no	64	21.7			
early MTM drinking (<=19 yrs)	yes	41	35.7	**3.4**	**1.7** **6.9**	0.44
	no	99	20.1			
early socially maladaptive drinking (<=23 yrs)	yes	16	8.9	2.0	0.5 7.9	0.43
	no	16	3.8			
early dependence problem (<=23 yrs)	yes	8	4.0	**7.4**	**1.4** **40.5**	0.61
	no	8	0.4			
early any problem (<=23 yrs)	yes	16	8.9	1.9	0.5 7.4	0.40
	no	17	1.8			
ever heavier drinking	yes	23	21.3	2.5	1.0 6.7	0.85
	no	48	10.1			
ever socially maladaptive problems	yes	23	15.1	1.8	0.7 4.6	0.08
	no	38	5.4			
ever dependence	yes	8	3.6	2.2	0.6 8.4	0.12
	no	15	0.9			
any dependence clinical feature	yes	15	8.0	**2.8**	**1.0** **7.8**	0.14
	no	22	1.9			
onset of problem after 16	yes	20	14.0	1.8	0.7 4.7	0.06
	no	35	5.2			

1. Mode4 additionally held conduct disorder clinical features constant. The analytical sample for model4 consists of individuals who were younger than 40.

2. p value of between site variation

3. Weighted percentage. Due to rounding, percentages may not add up to 100.0%.

Figure 4.3.1.3. ORs of associations between childhood physical punishment and drinking outcomes without covariate.

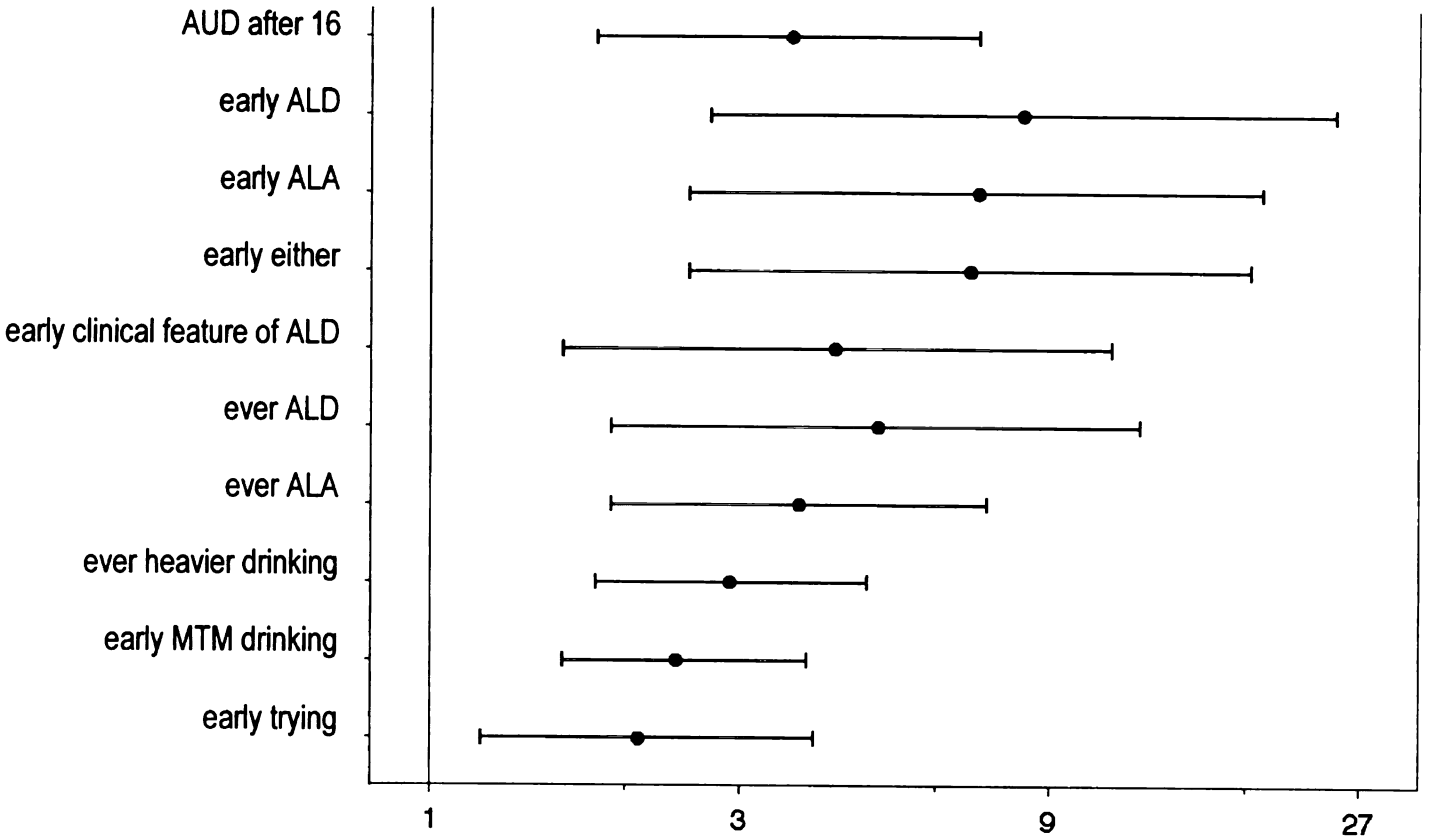
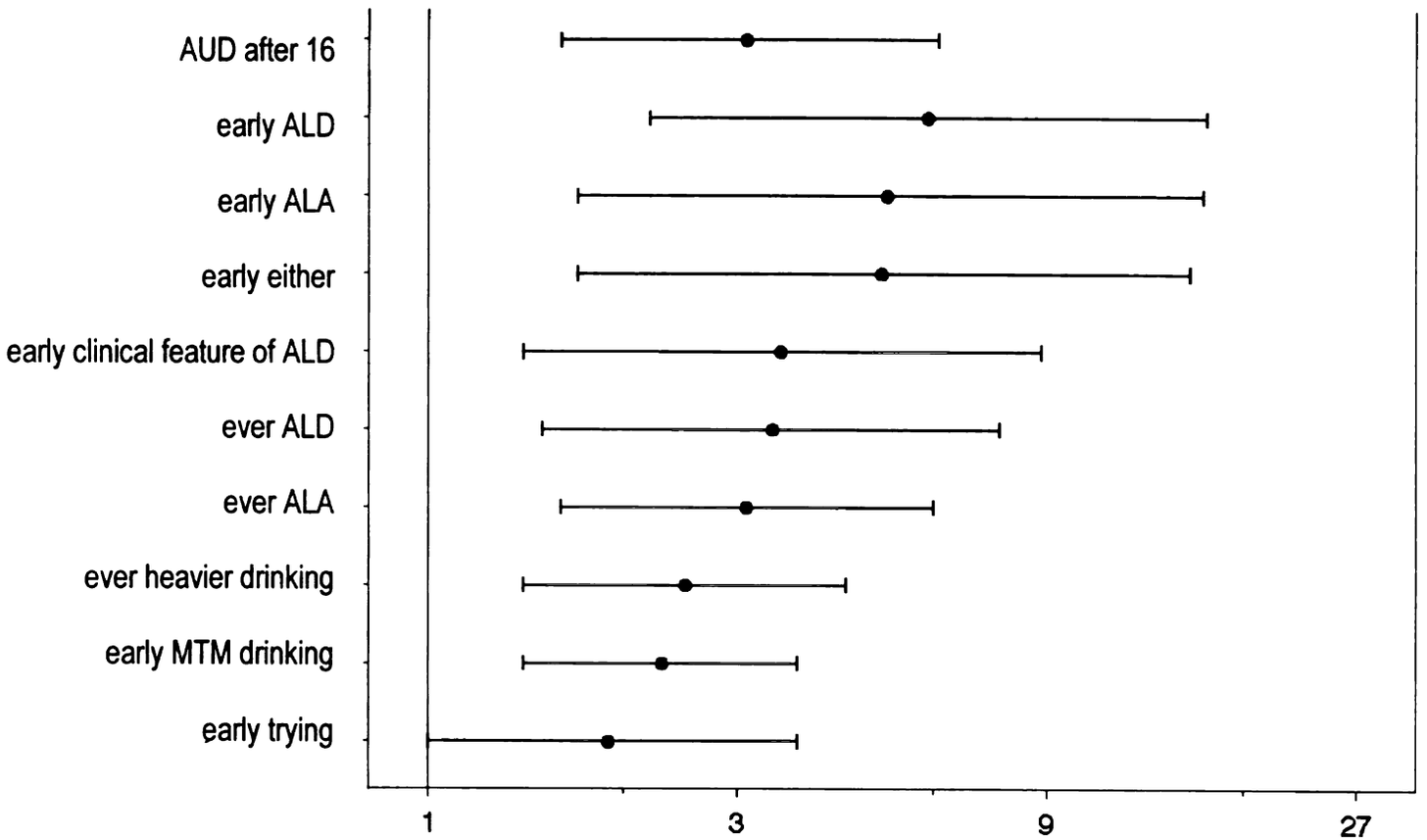


Figure 4.3.1.4. ORs of associations between childhood physical punishment and drinking outcomes holding sex, age, and parental alcohol/drug problems constant.



Many previous studies have found that the association between childhood physical abuse and drinking and drug problems is present in females, but absent in males (Simpson & Miller, 2002). As we stated in chapter 2 (introduction), from an epidemiological perspective, the nature of the association in males is actually largely unknown due to the fact that many previous evidence is based upon subgroups of populations. In this context, in the next steps, above analysis were restricted in the sub-sample of males. Robust CPP-drinking associations were found from the current study.

Table 4.3.1.4.The association between childhood physical punishment and alcohol drinking outcomes in males. Data from WMH-mC, 2001-2002

							Model1[1]			Model2[1]			Model3 (n=448) [1]		
	CPP	# cases	wtd%	uOR	95% C.I.		aOR	95% C.I.		aOR	95% C.I.		aOR	95% C.I.	
early try (<=12 yrs)	no	74	11.2												
	yes	28	17.3	1.6	0.8	3.1	1.5	0.7	3.0	0.9	0.4	1.9	0.4	0.1	1.3
early drinking (<=19 yrs)	no	165	24.3												
	yes	62	45.0	**2.6**	**1.5**	**4.3**	**2.6**	**1.5**	**4.2**	**3.0**	**1.8**	**5.1**	2.0	1.0	4.0
early socially maladaptive problem (<=23 yrs)	no	25	2.3												
	yes	14	12.8	**6.3**	**2.2**	**17.7**	**4.9**	**1.5**	**15.7**	**4.0**	**1.1**	**14.8**	**11.4**	**2.6**	**49.8**
early dependence problem (<=23 yrs)	no	10	0.5												
	yes	8	3.7	**8.2**	**2.6**	**25.4**	**5.5**	**2.0**	**14.8**	**6.0**	**1.9**	**18.2**	**5.7**	**1.0**	**31.3**
early any problem (<=23 yrs)	no	16	2.3												
	yes	18	12.9	**6.3**	**2.3**	**17.5**	**4.9**	**1.6**	**15.4**	**4.0**	**1.1**	**14.6**	**10.8**	**2.5**	**46.4**
ever heavier drinking	no	101	13.8												
	yes	39	32.0	**2.9**	**1.7**	**4.9**	**2.8**	**1.5**	**5.0**	**2.7**	**1.4**	**5.2**	1.7	0.7	4.1
ever socially maladaptive problems	no	79	7.4												
	yes	35	20.5	**3.2**	**1.6**	**6.5**	**2.9**	**1.4**	**6.0**	**2.3**	**1.0**	**5.4**	**3.3**	**1.4**	**8.1**
ever dependence	no	25	1.6												
	yes	15	5.3	**4.8**	**1.8**	**12.4**	**3.6**	**1.6**	**8.2**	**2.9**	**1.4**	**6.2**	**2.5**	**1.1**	**5.7**
any dependence clinical feature	no	41	2.7												
	yes	22	8.4	**3.3**	**1.2**	**9.2**	**3.0**	**1.1**	**8.0**	1.9	0.8	4.8	2.0	0.8	4.9
onset of problem after 16	no	75	7.2												
	yes	32	19.5	**3.1**	**1.5**	**6.3**	**2.9**	**1.3**	**6.1**	2.4	1.0	5.6	**3.4**	**1.4**	**8.2**

1. Model1 adjusted for sex, age, age categories, and parental drinking/drug problems. Model2 additionally adjusted for parental mental disturbances. Model3 was restricted in the subpopulation of individuals who initiated drinking after 16. Model3 was restricted in the subpopulation of individuals who initiated drinking after 16, and held sex, age, parental alcohol/drug problems, parental mental problems constant.

Table 4.3.1.5. The association between childhood physical punishment and alcohol drinking outcomes in males. Data from WMH-mC, 2001-2002

		Model 4[1] (n=303)				
	CPP	n of cases	weighted%	aOR	95% C.I.	
early try (<=12 yrs)	no	39	14.9	0.7	0.2	1.8
	yes	16	20.1			
early MTM drinking (<=19 yrs)	no	80	31.8	**4.4**	**2.0**	**9.6**
	yes	37	56.7			
early socially maladaptive problem (<=23 yrs) [2]	no	16	3.2	1.8	0.5	7.6
	yes	15	15.4			
early dependence problem (<=23 yrs)	no	7	0.7	**8.0**	**1.3**	**49.7**
	yes	7	6.0			
early any problem (<=23 yrs)	no	16	3.2	1.8	0.4	7.6
	yes	15	15.4			
ever heavier drinking	no	41	16.8	**2.8**	**1.0**	**7.9**
	yes	22	37.1			
ever socially maladaptive problems	no	36	9.8	1.5	0.5	4.2
	yes	21	24.2			
ever dependence	no	14	1.6	2.3	0.6	9.7
	yes	8	6.4			
any dependence clinical feature	no	21	3.3	2.2	0.7	6.6
	yes	13	11.6			
onset of problem after 16	no	33	9.4	1.6	0.6	4.3
	yes	18	22.5			

1. Mode4 additionally held conduct disorder clinical features constant. The analytical sample for model4 consists of individuals who were younger than 40.

4.3.1.2. Goodness-of-fit of logistic regressions and exploration of endogeneity

In the next steps, goodness-of-fit was assessed for above models (table 4.3.1.6).

Table 4.3.1.6. p values of F-tests of the Goodness-of-fit. Data from WMH-mC, 2001-2002

	Adjust for sex, age, and age categories	Additionally adjust parental drinking/drug problems	Additionally adjust for parental mental disturbances	Additionally adjust for conduct problems
early try (<=12 yrs)	**<0.01**	0.21	0.01	**<0.01**
early drinking (<=19 yrs)	0.95	0.73	0.64	**<0.01**
early socially maladaptive problem (<=23 yrs)	**<0.01**	**<0.01**	**<0.01**	**<0.01**
early dependence problem (<=23 yrs)	**<0.01**	**<0.01**	**<0.01**	**<0.01**
early any problem (<=23 yrs)	**<0.01**	**<0.01**	**<0.01**	**<0.01**
ever heavier drinking	0.39	**<0.01**	0.07	**<0.01**
ever socially maladaptive problems	**0.01**	**0.02**	**<0.01**	**<0.01**
ever dependence	**<0.01**	**<0.01**	**<0.01**	**<0.01**
any dependence clinical feature	**<0.01**	**<0.01**	**<0.01**	**<0.01**
onset of problem after 16	0.78	0.84	**<0.01**	**<0.01**

Results suggested lack-of-fit for many of the above models. (p values from the F-test for models for the subpopulation of males also suggested lack-of-fit, $p<0.01$.) There are different possible reasons that a model might have been misspecified, such as missing important covariates, measurement errors, incorrectly specified distribution of covariates (categorical vs. continuous) (Begg & Lagakos, 1990). Of our major concern is the possibility that parental drinking/drug problems or mental disturbances and CPP are not independent. Moreover, "conduct problems" may be correlated with CPP. In this scenario, the assumption of independence of error terms for logistic regression is violated. Thus, we used the recursive probit models to (1) explore if there is endogeneity in above models; (2) estimate coefficient for CPP on drinking outcomes taking the endogeneity into account.

Table 4.3.1.7 lists results of the Wald test for the rho parameter from these three models. If the *p* value is greater than 0.05, little endogeneity is suggested and vise versa. Firstly possible endogenous variables were assessed one by one; secondly parental variables were entered together; finally conduct problems were added. The two equations which are to be estimated simultaneously are,

$$\text{CPP} = \alpha_1 + \beta_1 \text{ sex} + \beta_2 \text{ age} + \beta_3 \text{ age categories} + \beta_4 \text{ endogenous variable(s)} + \text{error1} \quad (1)$$

$$\text{drinking outcome} = \alpha_2 + \gamma_1 \text{ sex} + \gamma_2 \text{ age} + \gamma_3 \text{ age categories} + \gamma_4 \text{ CPP} + \text{error2} \quad (2)$$

Table 4.3.1.7. *p* values from Wald tests for endogeneity. Data from WMH-mC, 2001-2002.

	p values of the test of H_0: atanhρ=0				
	Model 1	Model 2	Model 3	Model 4	Model 5
early try (<=12 yrs)	0.52	**0.04**	0.86	**0.03**	0.52
early drinking (<=19 yrs)	**<0.01**	0.35	**0.05**	0.63	0.85
early socially maladaptive problem (<=23 yrs)	0.63	0.88	0.06	0.96	**<0.01**
early dependence problem (<=23 yrs)	**<0.01**	0.13	0.08	0.07	**0.02**
early any problem (<=23 yrs)	0.60	0.85	**0.03**	0.99	**<0.01**
ever heavier drinking	**<0.01**	0.66	**<0.01**	0.86	0.28
ever socially maladaptive problems	0.67	0.32	**0.02**	0.37	**<0.01**
ever dependence	**<0.01**	**0.01**	0.15	**<0.01**	0.10
any dependence clinical feature	0.40	**<0.01**	0.12	**<0.01**	**0.05**
onset of problem after 16	0.90	0.29	0.03	0.32	**<0.01**

1. Model 1 has parental drinking/drug problems as endogenous variable; model 2 has parental mental disturbances as endogenous variable; model 3 has conduct problems as endogenous variable; model 4 has both parental drinking/drug problems and mental disturbances as endogenous variables; model 5 has conduct problem and parental variables as endogenous variables.

Evidence of endogeneity was found using recursive biprobit regression model. Parental variables introduced endogeneity to CPP for outcomes in relation to "dependence"; parental drinking/drug problems also introduced endogeneity to adverse drinking behavior, such as "early initiation of MTM drinking" and "history of heavy drinking"; conduct problem introduced endogeneity to CPP for outcomes in relation to adverse drinking behavior. Corresponding results for the subpopulation of males can be found in the appendix table A4.3.1.7.

Table 4.3.1.8 shows estimates for CPP from recursive bivariate probit models after taking endogeneity into account. Estimates are presented when endogeneity is suggested from above results. Similar results for the subgroup of males can be found in appendix table A4.3.1.8.

One of the disadvantages of probit model is that it is not easy to attach a substantive meaning to the coefficient estimate. For example, the coefficient of 2.0 means CPP is associated with 2 standard deviation increase in the probit function for early MTM drinking. Nevertheless, statistically robust and positive association between CPP and drinking outcomes were found from recursive bivariate probit models after taking the endogeneity into consideration; when endogeneity is suggested, estimates from recursive probit model are greater than those from logistic regression. For all statistically robustly correlated error terms (rho$\neq$0), the correlation is negative, which means unobserved heterogeneity affects CPP and parental variables and conduct problem in different ways.

Table 4.3.1.8 Estimates for CPP from recursive probit models after taking endogeneity into account. Data from WMH-mC, 2001-2002.

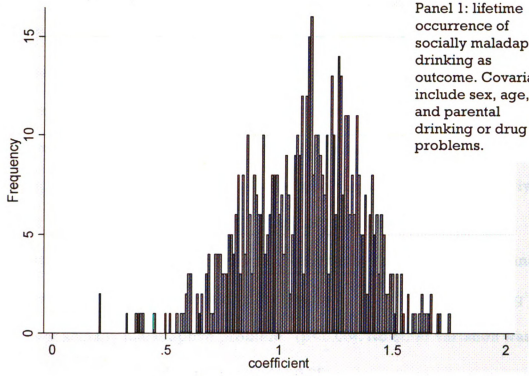
	Model 1		Model 2		Model 3		Model 4		Model 5	
	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.
early try (<=12 yrs)			1.1	0.2 2.0			1.2	0.3 2.1		
early drinking (<=19 yrs)	2.0	1.4, 2.6			2.1	1.4, 2.9			2.0	1.0, 2.9
early socio-maladaptive problem (<=23 yrs)									1.6	0.5, 2.7
early dependence problem (<=23 yrs)	3.1	2.2, 4.1							2.5	1.3, 3.8
early any problem (<=23 yrs)					2.8	1.7 3.8			2.0	1.1, 2.9
ever heavier drinking	2.1	1.4, 2.8			2.2	1.6, 2.8				
ever socio-maladaptive problems					2.3	1.4 3.1			1.9	1.0, 2.7
ever DSM-IV dependence	2.8	1.9, 3.8	2.4	1.4, 3.4			2.4	1.4, 3.3		
any dependence clinical feature			2.1	1.1, 3.0			2.0	1.0, 2.9	2.1	0.8, 3.4
onset of problem after 16									1.9	1.0, 2.8

1. Model 1 has parental drinking/drug problems as endogenous variable; model 2 has parental mental disturbances as endogenous variable; model 3 has conduct problems as endogenous variable; model 4 has both parental drinking/drug problems and mental disturbances as endogenous variables; model 5 has conduct problem and parental variables as endogenous variables.

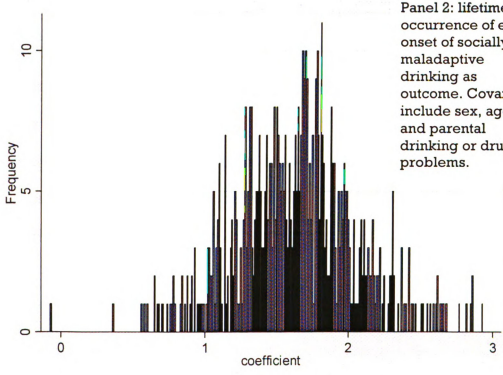
4.3.1.3 Stability of estimates

As described in the Methods chapter, the bootstrap resampling approach was used to look into the stability of the model-based estimates. Two estimates are assessed in this step. The first one is the estimate of "CPP-lifetime occurrence of socially maladaptive drinking" relationship adjusting for sex, age, and parental drinking or drug problems; the second is the estimate of "CPP-early onset of socially maladaptive drinking" adjusting for sex, age, and parental drinking or drug problems. Figure 4.3.1.3 show actual distributions of estimates under the model just described, based upon 600 bootstrap re-samples of size 1628 from the WMH-mC dataset. As a measure of central tendency, the mean value for this distribution is 1.1 and the 95% of the estimates fall within an interval from 0.6 to 1.6 for the first model. The corresponding OR (and its 95% CI) is 3.1 (1.9, 4.8). For the second model, the mean value for this distribution is 1.7 and the 95% of the estimates fall within an interval from 0.8 to 2.6 for the first outcome. The corresponding OR (and its 95% CI) is 5.2 (2.2, 13.1). In summary, this post-estimation data exploration step helps to confirm that the CPP-alcohol association of primary interest is non-null, and provides additional evidence against the null.

Figure 4.3.1.5 Distribution of coefficients from bootstrap resampling procedure.



Panel 1: lifetime occurrence of socially maladaptive drinking as outcome. Covariates include sex, age, and parental drinking or drug problems.



Panel 2: lifetime occurrence of early onset of socially maladaptive drinking as outcome. Covariates include sex, age, and parental drinking or drug problems.

4.3.2. Variations of associations between CPP and drinking across different outcomes

Evidence of variations in the association between CPP and drinking outcomes was found from both GEE and ALR analysis. Compared with the CPP-"early try (before teens)" association, stronger association was found in "early onset of drinking problems" including "early onset of socially maladaptive problems", "early onset of alcohol dependence", and "early onset of any problem" (table 4.3.2.2). Besides "early try", stronger associations were also found when comparing these "early onset" drinking problems with "early MTM drinking (before 20)", "ever heavier drinking", and "ever socially maladaptive problem" ($p<0.05$). No other variation was found across other drinking outcomes ($p>0.05$).

As we stated in the Method section, ALR is able to take the possible clustering of outcomes within the primary sampling unit into consideration. Coefficients yielded from the alternating logistic regression (ALR) model is slightly less precise compared with those from generalized estimating equations (GEE). The statistical inferences are identical. The ALR results suggested that above variations are not due to possible differential distributions of sex and age across geographic sampling units.

Table 4.3.2.2. Variations of the association between childhood physical punishment and drinking outcomes. Data from WMH-mC, 2001-2002

	GEE1 with no covariate				GEE1 with sex and age				ALR with sex and age			
	OR	95% CI		p^1	OR	95% CI		p^1	OR	95% CI		p^1
early try (<=12 yrs)	**2.1**	**1.2**	**3.6**	reference	1.7	0.9	3.2	reference	1.7	0.9	3.2	reference
early MTM drinking (<=19 yrs)	**2.4**	**1.5**	**3.9**	0.64	**2.2**	**1.5**	**3.5**	0.61	**2.2**	**1.4**	**3.5**	0.45
early socially maladaptive problem (<=23 yrs)	**7.0**	**3.0**	**16.6**	**0.02**	**6.2**	**2.5**	**15.5**	**0.02**	**6.5**	**2.5**	**16.9**	**0.01**
early dependence problem (<=23 yrs)	**8.0**	**2.8**	**22.8**	**0.03**	**6.5**	**2.1**	**20.0**	**0.04**	**7.3**	**2.5**	**21.5**	**0.02**
early any problem (<=23 yrs)	**6.8**	**3.0**	**15.8**	**0.02**	**5.0**	**2.5**	**14.8**	**0.03**	**6.3**	**2.5**	**16.2**	**0.01**
ever heavier drinking	**2.9**	**1.6**	**5.2**	0.46	**2.6**	**1.5**	**4.6**	0.46	**2.6**	**1.5**	**4.3**	0.32
ever socially maladaptive problems	**3.7**	**2.0**	**6.7**	0.18	**3.3**	**1.7**	**6.3**	0.19	**3.2**	**1.5**	**6.6**	0.18
ever dependence	**4.8**	**2.1**	**11.2**	0.09	**4.0**	**1.6**	**10.0**	0.13	**4.4**	**1.8**	**10.9**	0.14
any dependence clinical feature	**4.2**	**2.0**	**9.0**	0.14	**3.6**	**1.6**	**8.1**	0.18	**3.7**	**1.4**	**10.1**	0.24
onset of problem after 16	**3.7**	**2.0**	**6.9**	0.16	**3.3**	**1.7**	**6.4**	0.18	**3.2**	**1.5**	**6.8**	0.17

1. *p* value of the product term of CPP•item indicator. A *p* value less than 0.05 suggests variation of ORs for the outcome as compared with "early try".

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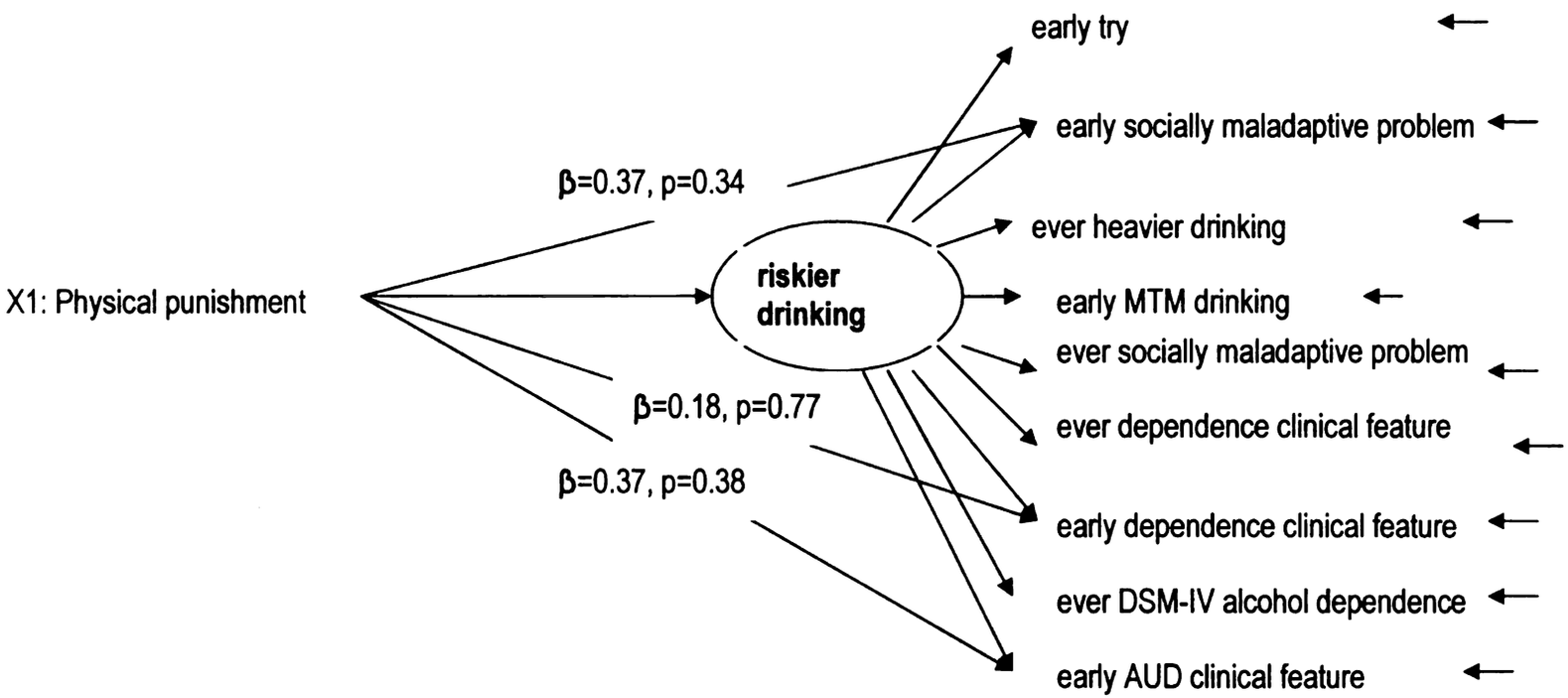
To better infer temporality, we restricted the analysis in the subsample of people who did not initiate drinking (at least 12 drinks in a year) until 16. Similar pattern was found from results (see appendix tableA 4.3.2.3).

Results from the MIMIC model found that at the same level of riskier drinking, individuals who suffered from CPP are no more likely to report "early onset" of drinking problems ($p>0.05$). Thus, the observed variations are not due to differential reporting between CPP and non-CPP groups.

Figure 4.3.2.1. MIMIC model of riskier drinking. Data from WMH-mC, 2001-2002.



Socially maladaptive problems and alcohol dependence problems include multiple correlated manifestations. Figure 4.3.2.1 depicts the lifetime occurrence of each of the problem studied. Individuals who suffered from CPP were more likely to experience each of the drinking problems. According to GEE and ALR models, the association between CPP and "irresistible desire of drinking" is stronger than the one between CPP and "drinking despite physical/mental problems", and so is for the association between CPP and "difficulty cutting down". Furthermore, the association between CPP and "difficulty cutting down" is also stronger than that between CPP and "drink more than intended". No other variation in the strength of associations across different drinking problems was found.

Figure 4.3.2.2. Lifetime occurrence of drinking problems. Data from WMH-mC, 2001-2002

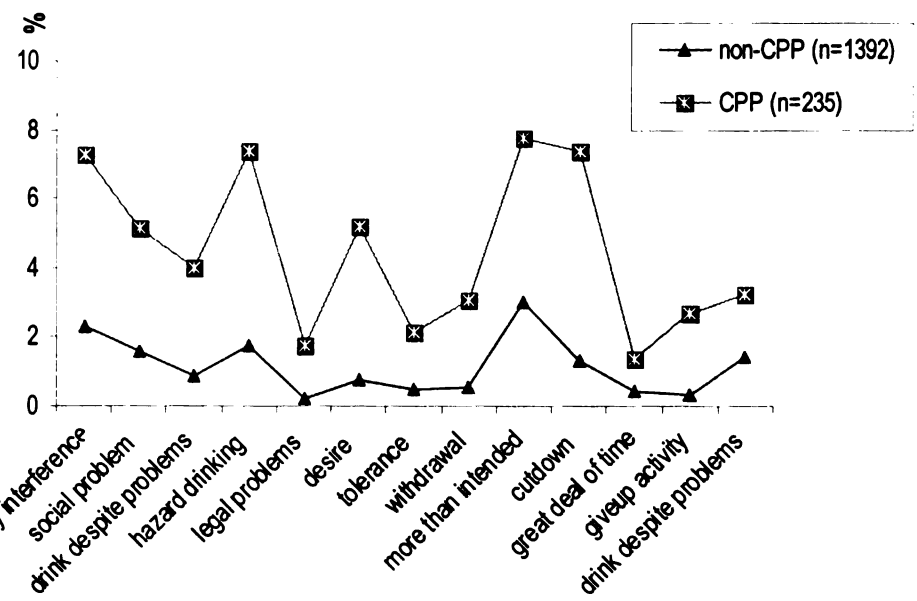


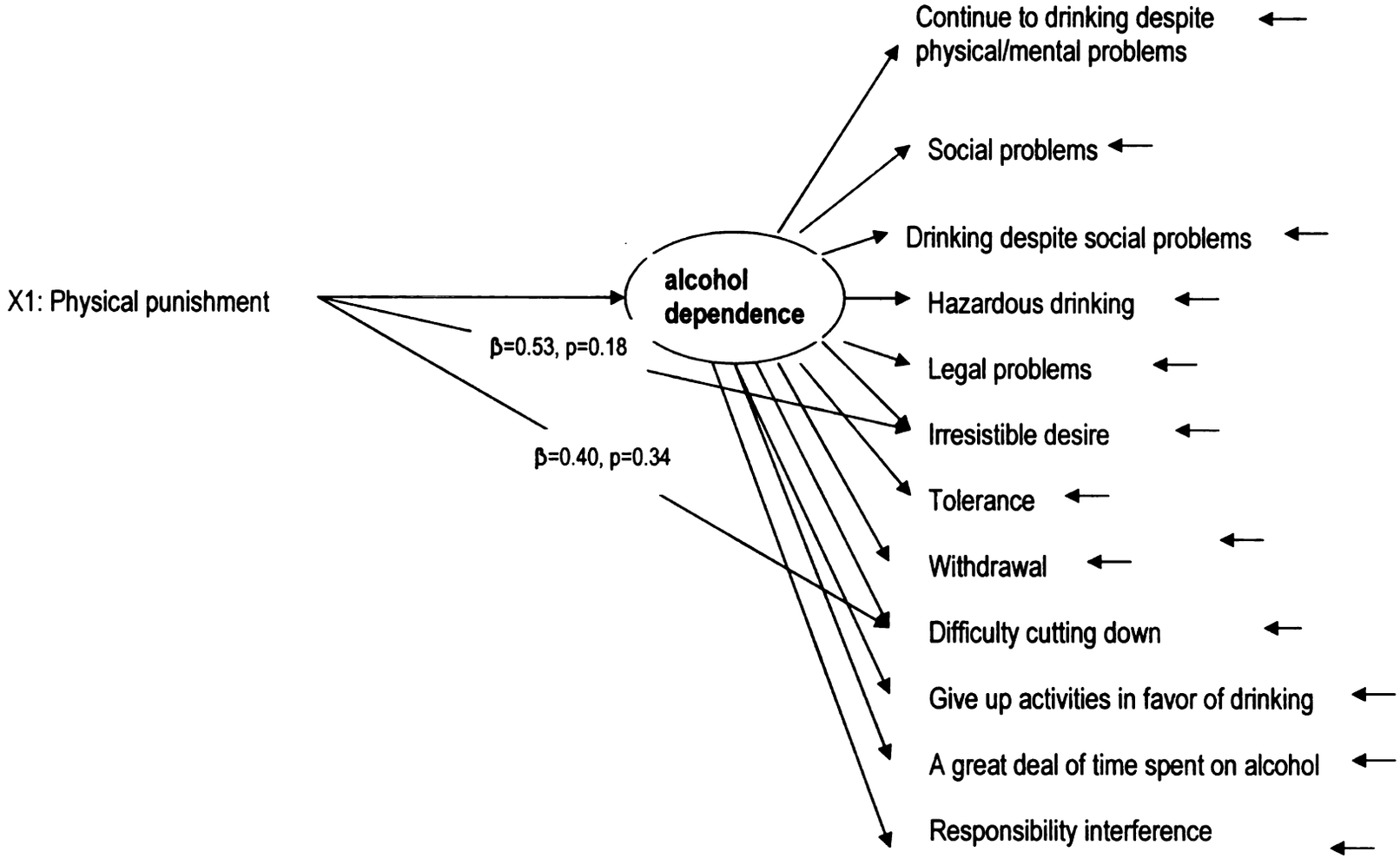
Table 4.3.2.3. Variations of the association between childhood physical punishment and drinking outcomes. Data from WMH-mC, 2001-2002

	GEE with no covariate				GEE with sex and age				ALR with sex and age			
	OR	95% CI		p [1]	OR	95% CI		p [1]	OR	95% CI		p [1]
Responsibility interference	3.3	1.4	7.8	0.47	3.0	1.2	7.3	0.46	2.9	1.1	7.5	0.54
Social problem	3.3	1.4	8.1	0.39	2.9	1.2	7.1	0.38	2.7	1.0	7.5	0.48
Drink despite social problem	4.7	1.6	14.3	0.16	4.2	1.4	12.3	0.16	4.2	1.3	13.5	0.12
Hazardous drinking	4.5	2.1	9.9	0.18	4.1	1.8	9.1	0.18	4.2	1.7	10.6	0.15
Legal problems	9.0	1.2	69.1	0.21	8.0	1.0	60.9	0.21	8.5	1.1	64.5	0.21
Desire	**6.9**	**3.0**	**16.1**	**0.03**	**6.1**	**2.6**	**14.4**	**0.03**	**6.0**	**2.2**	**16.3**	**0.03**
Tolerance	4.2	1.6	11.1	0.33	3.7	1.4	9.8	0.33	3.7	1.1	12.2	0.38
Withdrawal	5.7	2.1	15.5	0.06	5.0	1.8	14.0	0.07	4.7	1.4	16.4	0.13
More than intended	2.9	1.4	6.0	0.55	2.6	1.2	5.7	0.52	2.6	1.0	6.6	0.56
Cut down	**5.9**	**2.5**	**13.7**	**0.03**	**5.3**	**2.2**	**12.6**	**0.03**	**5.0**	**1.8**	**13.9**	**0.05**
Great deal of time	3.1	0.9	10.8	0.62	2.6	0.8	9.4	0.61	2.8	0.7	11.3	0.50
Give up activities	8.9	2.7	28.9	0.07	7.8	2.4	25.3	0.07	8.2	2.3	29.6	0.08
Drink despite problems	2.3	0.9	5.8		2.0	0.8	5.0		1.8	0.5	7.6	

[1]. p value of the product term of CPP×item

Results from the MIMIC model suggested that at the same level of alcohol dependence, individuals who suffered from CPP are no more likely to report either "strong desire" or "difficulty cutting down" ($p>0.05$).

Figure 4.3.2.3. MIMIC model of alcohol dependence. Data from WMH-mC, 2001-2002.



4.3.3. The association of CPP and drinking outcomes with respect to the earliest to the later stages of alcohol involvement

As presented in table 4.3.3.1, in most of the stages of alcohol involvement, individuals experienced CPP were more likely to be involved in alcohol compared with their counterparts without such experience. Endogeneity from parental variables (model 3) was explored. Evidence of endogeneity ($p<0.05$ for the test of rho) was found for the first and the last stage: opportunity to drink alcohol and occurrence of clinical feature of alcohol dependence in drinkers. The estimate from recursive bivariate probit model is 1.8 (95% C.I. =1.5, 2.0) and 2.0 (95% C.I. =1.0, 2.9), respectively. Our result highlights the importance of the stage of the occurrence of alcohol related problems (social maladaption and clinical features of alcohol dependence) after the initiation of MTM drinking, where the largest relative risks are found. For example, the occurrence of socially maladaptive problems in people who experienced CPP is twice of that in people who did not experience CPP given that they both drink.

Table 4.3.3.1. The association between childhood physical punishment and drinking involvement with weight. Data from the WMH-mC, 2001-2002

	CPP	n	wt %	model 1			model 2 [a]			model 3 [a]		
				uRR	95% CI		aRR	95% CI		aRR	95% CI	
alcohol opportunity	no	1345	78.7									
	yes	232	88.0	**1.12**	**1.02**	**1.18**	1.10	0.99	1.17	1.10	0.99	1.17
trying given opportunity	no	1006	85.3									
	yes	200	91.1	1.07	0.95	1.13	1.06	0.91	1.13	1.04	0.87	1.12
drinking given tried	no	860	59.3									
	yes	181	74.8	**1.26**	**1.06**	**1.42**	**1.25**	**1.05**	**1.41**	**1.27**	**1.04**	**1.44**
ever heavy drinking among drinkers	no	521	20.8									
	yes	134	34.4	**1.65**	**1.13**	**2.26**	**1.60**	**1.05**	**2.27**	1.48	0.92	2.20
ever socially maladaptive problems in drinkers	no	531	10.7									
	yes	136	23.5	**2.19**	**1.28**	**3.46**	**2.13**	**1.21**	**3.45**	1.60	0.80	2.92
ever dependence problems in drinkers	no	534	3.9									
	yes	134	10.3	**2.67**	**1.08**	**6.05**	**2.59**	**1.06**	**5.79**	1.74	0.78	3.72

[a] model 2 adjusted for sex, age, and age categories; model 3 additionally adjusted for parental drinking problems and parental mental disturbances.

Chapter 5 Discussion

This chapter discusses results presented in chapter 4 with respect to each study aims in the context of existing knowledge. Pertinent strengths and limitations of the study will be addressed along with each study aim. The final section draws conclusions and discusses implications for future studies.

5.1 The frequency of beverage alcohol involvement in two metropolitan cities in China: Beijing and Shanghai

5.1.1 Summary of results

For the population as a whole, the majority of people in Beijing and Shanghai had the opportunity and had at least tried an alcoholic beverage prior to the assessment. Less than half of the population had ever consumed 12 drinks in a year. Medium number of drinks is three for drinkers when they drank the most. Less than one in ten people had a history of heavier drinking or socially maladaptive drinking. Development of alcohol dependence, as defined in DSM-IV or ICD-10, seems to be quite rare (<2%).

Frequency distribution for the age of onset for the first drink of alcoholic beverages showed a peak during teenage years (13-19). Socially maladaptive drinking had onset starting in the late teen years; some onsets were found throughout the 20s and 30s.

Less than a third of the population in the two metropolitan cities drank at least 12 drinks recently (i.e. during the year prior to the assessment). Medium number of drinks is two during the last 12 months prior to the assessment for recent drinkers. Less than four percent recently drank heavily.

Recent socially maladaptive drinking and alcohol dependence was rare (≤ 2%).

5.1.2 Strengths and limitations

To our knowledge, this is the first study describing drinking behavior and associated problems in the two biggest cities in China: Beijing and Shanghai. Since the late 1970s, alcohol production and consumption have been increasing steadily concurrent with a booming economy and increased affluence (WHO, 2003). As such, it is important to have obtained up-to-date estimates for the frequency of drinking behavior and problems in these two most advanced economy, metropolitan areas of China. These study estimates serve as benchmark values for estimating future trends in the extent of drinking and related problems in contemporary 21st century China.

Furthermore, as urbanization proceeds, with more and more people migrating into cities, there may be change in drinking behaviors and problems in the future for other Chinese cities. As such, one of the strengths of the current study is that the source population is non-institutional household-dwelling adults, where the major drinking-related disease burden tends to occur. Second, although the participation levels are not perfect (~75%), potential bias in point estimates was minimized by applying probability weights and post stratification adjustments in estimation. The possible clustering between individuals, as a result of the multi-stage sampling, was also taken into account to avoid erroneous statistical inference. These methodological refinements help promote the likelihood that these study

results are directly generalizable to the source populations under study. In contrast to ecological level studies, the measurement was at the individual level and was based on what individual said about themselves. As compared to estimates based on clinical populations, this study avoids a possibility that patients seeking clinical care are just a small portion of the entire set of cases in population ("the tip of an iceberg").

Nonetheless, the study does have some counterbalancing limitations. **With respect to the source population**, although the study sample covers the household-dwelling population where most drinking-related disease burden can be found, some important subgroups were not included in this study (for instance, people in the military, homeless people, and adolescents). Future studies in these special populations can be used to supplement to the current study design. The sampling frame of this study only included people who were formally registered in a non-agricultural household. Thus the majority of immigrants, who usually work in Beijing and Shanghai but were not officially registered in households within these two cities, were not included. Nonetheless, drinking patterns and problems in immigrants may bear interesting messages about social-culturally influences on drinking.

With respect to the assessment of drinking, our major concern is that the drinking-related variables have been measured via a retrospective, self-report approach. Recall and reporting bias cannot be ruled out, especially when a long time period has elapsed since the drinking behavior of interest occurred. For this reason, the study did not make extensive use of the age of

onset of drinking variables. Instead, individuals were dichotomized into "early onset" and "later onset" subgroups to constrain the recall bias, meanwhile retaining important information. Additionally, the use of a face-to-face interview approach precluded anonymity of the assessment. The stigma attached to some drinking outcomes, such as clinical features of alcohol dependence and socially maladaptive drinking, may have introduced under-estimations in relation to the occurrence of such variables. Although it has been shown that self-report can be a reliable and valid method of assessing drinking behavior, this argument has not yet been confirmed in the Chinese context (Del Boca & Darkes, 2003), especially for sensitive information, such as legal problems due to drinking. **With respect to the assessment of alcohol dependence**, besides concerns about the reliance on self-report, another notable limitation of study method is "gated" approach as embedded in the version of CIDI used in this study. That is, respondents lacking a history of drinking-related socially maladaptive problems or hazard-laden drinking were not asked about clinical features of alcohol dependence. Evidence from the NESARC suggests that this "gate" may induce decrements in estimation of lifetime occurrence and 12 month prevalence of alcohol dependence (Degenhardt, Bohnert, & Anthony, 2007; D. S. Hasin & Grant, 2004). Though the degree of under-estimation is unknown since there has been no comparison using data from China, our estimates of alcohol dependence may well serve best as starting point, possibly subject to underestimation.

In summary, limitations such as these may have led to under-estimations in cumulative occurrence and 12-month prevalence of drinking-related problems, especially legal problems and alcohol dependence. To a lesser extent, there may have been under-estimation of drinking behavior frequency.

5.1.3 Drinking practices in Beijing and Shanghai

Despite limitations mentioned above, our results are informative in that this study presetns the first comprehensive description of drinking and related problems in contemporary metropolitan China based upon epidemiological samples. First, with respect to experience with alcohol involvement, our study is the first to observe that over 80% of individuals had the opportunity to drink alcohol. Along with results from previous studies conducted in the metropolitan area of Wuhan city in the Hubei province, it demonstrated that alcohol can be highly accessible in metropolitan cities of China (Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004; Zhang, Casswell, & Cai, 2008). Despite this level of accessibility of alcohol, most people only drank occasionally and moderately (less than monthly and less than 4 drinks in a day), which was also found in the Wuhan city study; and the median number of drinks during a typical drinking day is similar to the Wuhan study as well (Zhang, Casswell, & Cai, 2008). Compared with other countries, people in these two Chinese metropolitan cities have been found to have a higher level of lifetime experience with drinking than people in several countries in the Middle-East and South Asia (e.g. India), and lower than people in America,

New Zealand, and European countries (Degenhardt et al., 2008; WHO, 2005). With respect to the age of first trying alcohol and the initiation of MTM drinking, our results pointed out peak values during the late teenage years, with respect to experiences with alcohol. This result is strikingly similar with other countries participating in the WMHS (Degenhardt et al., 2008). Adolescence is a developmental period for sustaining brain maturation, and teen onset of drinking may signed greater susceptibility to developing drinking problems. The implications for timing of effective prevention and intervention programs are obvious (e.g. see O'Brien & Anthony, 2005)).

5.1.4 Drinking problems in Beijing and Shanghai

With respect to drinking-related problems, this study's finding can be compared with results from a 1993 study conducted in three provinces in China, which applied the DSM-III-R criteria. In this comparison, the occurrence of drinking-related problems in Beijing is observed to be higher than that in all three outlying provinces, and the occurrence in Shanghai is intermediate (Wei et al., 1995). This study's results are not directly comparable with more recent studies conducted in other areas of China, since the assessments are distinct from one another (Hao et al., 2004; Zhang, Casswell, & Cai, 2008). Compared with the occurrence of drinking-related problems in other countries, the occurrence is generally lower than that of Western countries, slightly lower or equal to that of some other Asian countries (e.g. Japan and Korea), while higher than that of Nigeria (Gureje, Lasebikan, Kola, & Makanjuola, 2006; Kawakami, Shimizu, Haratani, Iwata, &

Kitamura, 2004; Medina-Mora, Borges, Benjet, Lara, & Berglund, 2007; J. T. Park, Kim, & Jhun, 2008). For example, using the same tools of assessment, surveys in the US and several countries in Europe all showed a lifetime occurrence well above 10% (CPES, 2001; Rehm, Room, van den Brink, & Jacobi, 2005). One possible explanation is that DSM-IV and ICD-10 criteria are largely based on clinical observations in Western countries. The sensitivity of questions might not be as good in the Chinese culture. Nonetheless, the clinical reappraisal study showed fairly good validity of CIDI in assessing drinking-related problems (Huang et al., 2008). Thus, the much lower occurrence is not likely to be completely due to the validity of the assessment.

Difference of this type also can be due to socio-cultural and biological variations across populations. To illustrate with a contextual example, according to data from the Gender, Alcohol, and Culture: an International Study (GENACIS), in some European countries, the bar or pub is an especially common place to drink alcohol; such that males might engage in solitary drinking (without a companion) on 10-20% of drinking days during a year, as compared to females on 5-15% of drinking days (Bloomfield et al., 2005). With respect to China, the 1993 study found that a common reason for drinking is "to celebrate". On most drinking occasions, there is a meal and in general, Chinese drinkers tend to avoid being drunk in front of friends and business partners (Hao, Chen, & Su, 2005; Wei et al., 1995).

As for biological variations, our best example for the Chinese context involves variations in proteins involved in alcohol metabolizing pathways (e.g.

the allele frequencies of genes encoding ADH and ALDH). To illustrate, the ADH2*1 allele presents in over 95% of many European heritage populations By comparison, it is found in only 32% of Chinese Han. ALDH2*2 is virtually absent in the European heritage population and is present in about 20% of Chinese (Goedde et al., 1992; Y. C. Shen et al., 1997; Thomasson et al., 1991). These mutations in ADH and ALDH genotypes result in accumulation of acetaldehyde, which induces the "flushing effect" that functions to punish and dampen future drinking behavior in many drinkers with the flushing phenotype. To the extent that culture does not overcome this pharmaco-genetic process, these mutations may be serving to protect many Chinese people from excessive drinking. In deed, in many European countries and the US, people drink alcohol in a higher frequency and larger quantities (Bloomfield et al., 2005; CPES, 2001). The GENACIS found that considerable proportions of people drank more than five drinks monthly in selected European countries (20-50% males, 5-20% females). Genetic variations mentioned above may play a role behind the higher level of alcohol consumption among Europeans and US. In summary, the interplay of these biological and cultural factors results in the lower occurrence of drinking problems in these two metropolitan cities in China.

Although compared with values observed in metropolitan areas of other countries, drinking is not as common in Chinese metropolitan cities, riskier drinking behavior and drinking problems definitely exists. For example, 7% people had a history of heavier drinking, which is similar to the

results from previous studies (Hao et al., 2004; World Health Organization, 1999), five percent people had a history of socially maladaptive problems related to drinking, and two percent people have had at least one clinical feature(s) of alcohol dependence. Given the large population base, these percentages easily translate into considerable disease burden, not only for individuals, but for their families and societies.

Some observations about the future may be in order. As compared to some European countries and the USA, there is less commercial alcohol beverage market prevention (Degenhardt et al., 2008), and there is still much latent demand for commercial alcohol in China. With increasing affluence, the market forces may address this demand, in processes described by Room et al. (Room, Schmidt, Rehm, & Makela, 2008) and it is very likely that alcohol consumption will grow. There is some evidence that alcohol production and consumption in China are climbing (National Bureau of Statistics of China, 2008) and we might project that drinking-related problems will climb as well. Along with a growing number of automobiles in the cities of China, results from this study add more reason to call for preventions and harm-reduction initiatives in China. Looking to the future, the teenage years have been the key interval for initiation of Chinese drinking, as is true elsewhere (Degenhardt et al., 2008). Moreover, a large proportion of Chinese drinkers in this research were found to have had their first socially maladaptive problem or dependence clinical feature early in that second decade of life. Hence, to be most affective, prevention and intervention programs should be

designed for young people (from teenage to early 20's) in addition to more general regulation prevention effort (e.g. control through taxation, as suggested by the work of Holder, Holder, 2007).

One interesting observation from this study is that a larger proportion of people in Beijing had drinking-related problems compared with people in Shanghai. This greater occurrence of drinking problems in Beijing is not explained by the higher drinking frequency. The 1993 survey of three sites in China also found significant geographical variations in drinking problems (Wei et al., 1995). However, due to the lack of assessment of drinking context and biological factors, as well as the self-report nature of the study instrument, we are much restricted to any explanation beyond pure observation.

5.2 Subgroup variation with respect to beverage alcohol involvement.

5.2.1 Summary of results

With respect to time-invariant variables assessed in this dissertation, sex and age groups, it was found that being male is associated with higher occurrence of both drinking and drinking-related problems. The strengths of associations for drinking-related problems are much higher than that in drinking practices. Compared to people in the oldest age group, those in younger groups are more likely to be involved in alcohol drinking. Individuals in the youngest group are especially more likely to be involved earlier in their life.

With respect to time-variant variables assessed in this dissertation, marital status, education, and personal income, subgroup variations were

found as well. Individuals who were no longer married are more likely to have a history of heavier drinking, but not recent heavier drinking; individuals with higher income are more likely to be involved in drinking, but no more likely to experience drinking-related problems. Individuals who had a job at the time of assessment were more likely to be involved in drinking, as well as experience drinking-related problems; however they were no more likely to initiate drinking early or experience drinking-related problems early.

5.2.2 Strengths and limitations

The main strength of this study is, as stated above, the results are directly applicable to the source population. Not like studies conducted in clinical settings, the source population in this study is household dwelling adults in Beijing and Shanghai. Due to characteristics associated with treatment seeking behavior, e.g. sex, age, severity of the condition, education, and so on, it is precluded the generalization of results from clinical studies to the entire patient population. However, considerable disease burden is from people who never sought clinical aid. Thus, it is critical to have estimates from studies which can be generalized to the population where the major disease burden comes from.

Of major concern of limitations is the "gated" approach in assessing alcohol dependence, as mentioned above. If the "gate" differentially filtered individuals in relation to their characteristics, e.g. sex, age, martial status, and so on, the estimates of subgroup variation in alcohol dependence might have been biased. Degenhadt et al. compared the patterns of associations with

respect to sex and age groups, and did not find appreciable differences in estimates for alcohol dependence (Degenhardt, Bohnert, & Anthony, 2007). Nevertheless, by comparing percentage loss of cases of alcohol dependence across different subgroups, Hasin et al. did show that disproportionally more cases were missed in females and minority groups (African American, and Hispanics), although no statistical test was provided. We are not aware of any such comparisons with respect to marital status, education level, income level, and employment status; neither in the sample of Chinese. Thus, it remains unclear if or how much of the estimates for the occurrence of alcohol dependence is biased because of the "gating" procedure. Nevertheless, alcohol dependence is only one of the various indicators for "riskier drinking" in this study. By assessing the pattern of associations between assessed characteristics and multiple outcomes, we are able to get the profile of these associations.

One thing merits mention is that under this study aim, our goal is not to infer causal relationship, but rather to present the differential distribution of drinking practices and problems in each of the subgroups. By doing this, we intend to prioritize prevention and intervention programs and to provide basis for designing effective prevention and intervention programs according to individual characteristics.

5.2.3 Subgroup variations in drinking outcomes

5.2.3.1. Sex and age

Consistent with findings from previous studies around the world, excessive drinking and problems are found in males and younger age groups. (Degenhardt et al., 2008; D. S. Hasin, Stinson, Ogburn, & Grant, 2007; Higuchi, Parrish, Dufour, Towle, & Harford, 1994; Kessler et al., 1994; Kim et al., 2008; Naimi et al., 2003; J. T. Park, Kim, & Jhun, 2008; Serdula, Brewer, Gillespie, Denny, & Mokdad, 2004; WHO, 2005). This male-female gap in drinking opportunity only exists in the two older age groups. In the two youngest groups, there was no sex-difference in opportunities to drink. Among all outcomes in this study, opportunity to drink alcohol is the only one that is solely affected by socio-cultural factors. This result may serve as a reflection of the diminishing stigma attached to female drinking as the reform of the Chinese society.

Results are consistent with previous studies in China in that males are more likely to be involved with drinking, and males are much more likely to have a history of riskier drinking and drinking-related problems (Hao et al., 2004; Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004; Zhang, Casswell, & Cai, 2008; X. Zhou et al., 2006). Moreover, our results found that although the male-female difference was found in each of the drinking outcomes, it is rather small in early stages of alcohol involvement (e.g. opportunity, ever tried alcohol) and increases dramatically in riskier drinking behavior and drinking-related problems. Drinking-related problems are mainly male phenomenon in these two metropolitan cities in China. Such robust male-female differences

were not likely to be completely accounted for by either differential report or differential survival.

We found higher alcohol involvement, as well as drinking-related problems, in younger age groups, which is in agreement with previous studies conducted in other areas of China (Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004; Zhang, Casswell, & Cai, 2008; H. Zhou et al., 2003). The higher level of alcohol involvement in younger age groups may well reflect the increasing alcohol production and consumption in National reports (National Bureau of Statistics of China, 2008). There are several alternative interpretations of the observed variations in drinking-related problems across age groups. The first is that it might be due to differential recall. However, we argue that since socially maladaptive problems and dependence are quite distinct experiences during one's lifetime, it is not likely to be forgotten. Second, it is also not likely to be due to differential report because the same pattern of variation was seen in less stigmatized outcomes, such as opportunity to drink and ever tried alcohol. Previous studies have shown that self-report of drinking is fairly reliable (Cumming & Klineberg, 1994; Lee, Whittemore, & Lung, 1992; S. Liu et al., 1996). One remaining possibility is that drinking-related problem affects mortality. However, previous studies do not support it. The study by Hao, et al. found that an individual's health status is not associated with drinking behavior (Hao et al., 2004); follow-up studies in the US failed to show convincing evidence of elevated mortality in heavy drinkers and people with alcohol dependence (Dawson, 2000; Vaillant, 1996, 2003).

One study in Shanghai showed that low to moderate alcohol consumption is associated with lower mortality in middle-aged Chinese (Yuan, Ross, Gao, Henderson, & Yu, 1997). Furthermore, the same pattern was found in early stages of alcohol involvement (e.g. opportunity to drink, ever tried alcohol), which is not very likely to be much associated with mortality. Thus, elevated mortality in alcohol consumers is not likely to account for the consistent and robust variations across age groups.

One thing merits attention is that drinking involvement in the youngest group is more likely to be "right censored", which means by the time of the assessment they had not yet experienced relevant drinking outcomes, but they might later in life. Given that the peak of initiation of MTM drinking is around 20 years old, the lower occurrence of alcohol involvement in the youngest group, compared with the adjacent group, is likely to reflect this "right censorship". With this in mind, it is striking that the occurrence of "early MTM drinking" increases with age groups going from older to younger (5% in the oldest group and almost 30% in the youngest group). Considering the right censorship, the occurrence of early involvement with drinking in the youngest group is likely to be higher if the same survey was to be taken later when they all reached 20 years old. Early initiation of drinking is associated with higher risk of heavy drinking and alcohol dependence (Caamano-Isorna, Corral, Parada, & Cadaveira, 2008; Dawson, Goldstein, Patricia Chou, June Ruan, & Grant, 2008). Due to the cross-national design of this study, we are not able to tease age, cohort, and period effect apart beyond the observed

variations across age groups (Holford, 1991). Nevertheless, these results warn us of the likely increasing disease burden of alcohol drinking if the young people are not educated about responsible drinking.

5.2.3.2. Other variables

With respect to drinking practices, our results agree with some previous studies in the Huaihua area of China and the US national comorbidity survey replication (NCSR) in that people who are "married", "working", and in "higher income levels" are more likely to be involved in recent drinking, and education is not extensively associated with recent drinking (Degenhardt, Chiu, Sampson, Kessler, & Anthony, 2007; X. Zhou et al., 2006). With respect to drinking-related problems, our results are quite different from previous studies in the Wuhan metropolitan area (Jiafang, Jiachun, Yunxia, Xiaoxia, & Ya, 2004) in terms of personal income level. In the current study, no association between income level and drinking problems was found, while the Wuhan study found higher income was associated with higher likelihood of drinking problems after adjusting for several other covariates (no unadjusted OR were given). Several alternative reasons may explain the difference. First, the study population is different. Second, in this study, we held only sex and age constant. In the Wuhan study, many other covariates, such as fellow drinking, parental drinking, attitudes about drinking, were included in the model as well. In the context of exploring subgroup variation, the inclusion of these variables made results difficult to explain and made direct comparisons of results impossible. In the context of exploring potential

cause, some covariates are more likely to be endogenous rather than exogenous due to the cross-sectional nature of the study. Thus, the causal inference for any of the variables is not clear either. Here, we argue that our results are more relevant in terms of exploring subgroup variation and providing basis for intervention programs in that we held only sex and age, which are exogenous to outcomes, constant. By doing this, we seek the possible subgroup variation, which is not due to the differential composition in terms of sex and age.

Our results indicate that drinking and related problems are not evenly distributed in subgroups in the population. Certain subgroups of people should be priorities for intervention and harm-reduction programs. Special effort should be made to target at drinking problems in males, younger age groups. Additional to sex and age, priorities should be given to people who are working. Policies and regulations on work place drinking may need to be created and emphasized.

5.3. The association between childhood physical punishment and drinking and related problems in order to shed light on suspected causes of drinking-related problems

5.3.1 Summary of results

Robust associations between CPP and negative drinking outcomes are found. This association is statistically independent of parental drinking problems, parental mental disturbances, and conduct problems in childhood. After taking possible endogeneity into account, this association is still robust.

Variations in association were found across drinking outcomes and across clinical features of AUD as defined by DSM-IV and ICD-10. Stronger associations were found between CPP and "early onset of drinking-related problems" as compared to some other negative drinking outcomes. Stronger associations were found between CPP and "strong desire" and "difficulty cutting down" as compared to "drink despite physical/mental problems". According to our results, the most important stage to link CPP to drinking problems is after the initiation of MTM drinking.

5.3.2 Strengths and limitations

The major contributions of this study are as follows. First, it provides evidence of a possible causal relationship between childhood physical punishment and adverse drinking outcomes in the Chinese context. Most of previous studies are based on Western populations. Nonetheless, CPP is culturally sensitive. The influence of CPP may vary from society to society. Our study provided initial basis for future studies of CPP as a potential cause of drinking-related problems. Second, our estimates are based upon a community sample. 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. Third, using a novel statistical method, we successfully controlled for endogeneity bias and found robust association after taking the endogeneity into consideration. Fourth, previous studies in special populations found that the association between CPP or CPA and

drinking outcomes only presents in females (Simpson & Miller, 2002). This study found that from a population perspective, the estimated effect of CPP to negative drinking outcomes is robust in males as well.

The study does suffer from some limitations. Of our major concern is the assessment of CPP. The assessment of CPP is "one question for multiple behaviors". Therefore, we are not able to tease out which behavior(s) account for the association. However, these assessed behaviors tend to occur together as an indication of rearing style of the parents. All these forms of physical punishment pose stress throughout the childhood and possibly influence the development trajectory of the child (D. B. Clark, 2004). One thing merits attention is that more severe forms of physical punishment, such as biting, being burnt, being scalded, being hung, etc. were not assessed in this study due to the potentially emotional upset to the respondent. These severe forms of physical punishment may or may not present in individuals under study. Thus, it is possible that estimates were driven by these severe forms of CPA, but not CPP, per se. more studies are needed to clarify this possibility. Also of concern is the assessment of parental drinking problems. The assessment of parental drinking problems is based on the self-report from the respondent instead of by their parents. No clear definition of "drinking problems" was given in the question. Thus, the assessment may be subjective and fairly coarse. Several studies have probed into this question. These studies suggested that information about parental drinking problems collected from offspring is pertinent and accurate, and it serves as a valid method of

assessing parental problems when the alternative is unfeasible (Prescott et al., 2005; Rhea, Nagoshi, & Wilson, 1993; Sher & Descutner, 1986). It is not clear, however, if this conclusion holds to be true in the Chinese context.

5.3.3 Possible causal inference

Despite these limitations, our findings are of interest. To our knowledge, this is the first study to show a possible causal relationship between CPP and riskier drinking and drinking problems in the Chinese culture. The results correspond to some previous findings in US and Canada populations showing a moderate association between CPA and drinking problems (Holmes & Robins, 1987, 1988; Kessler, Davis, & Kendler, 1997; MacMillan et al., 1999; MacMillan et al., 2001). Many previous studies found that after controlling for parental drinking problems, there is no association between CPA and drinking outcomes (Hughes, Johnson, Wilsnack, & Szalacha, 2007; Koss et al., 2003; Libby et al., 2004; Mullings, Hartley, & Marquart, 2004; Young, Hansen, Gibson, & Ryan, 2006), while one small-scaled follow-up study found a robust association between CPP and binge drinking in 113 African American female child abuse victims, after holding parental drinking problems constant (Jasinski, Williams, & Siegel, 2000). A cross-sectional study also suggested an association between childhood adversities and AUD holding parental drinking problems constant; however, no specific estimate was provided for physical abuse in the study (Carrigan & Randall, 2003). Nevertheless, in these studies, as we described in the background section, the possibility that CPP might be an endogenous variable of parental drinking problems was not

taken into account. The endogenous bias might have resulted in inconsistent estimates; ignoring that it may have led to erroneous inference. Additionally, these studies were conducted in special groups of population, such as marine recruits, lesbians, prisoners, and people attending primary care. Thus, results from these studies cannot be applied to the general population. Our study showed from a population perspective that after controlling for parental drinking problems, the possible causal relationship between CPP and drinking problem persists. Additionally, we found that the estimated effect cannot be attributed to parental mental disturbances (depressive mood, anxiety, and suicidal attempt) either.

Due to the cross-sectional and observational nature, this study is more liable to potential biases, such as differential recall and survival bias, which makes the inference of causality more difficult. First, it is possible that people who suffer from drinking problems may search their memory deeper for experiences of CPP. However, it is not supported by previous studies, which have shown good to excellent validity and reliability of assessment of CPP (Bremner, Bolus, & Mayer, 2007; Walsh, Macmillan, Trocme, Jamieson, & Boyle, 2008). The finding has been replicated in cocaine dependent individuals (Kopnisky & Hyman, 2002). We further offset this problem by grouping people who answered "often" and "sometimes" together, and "rarely" and "never" together. In this way, the CPP variable represents chronic stress during the childhood, which is reasonably easy to recall. Second, individuals who suffered from CPP may be less likely to survive,

especially from severe forms of abuse (Arias, MacDorman, Strobino, & Guyer, 2003). As such, less people with CPP experience will be captured in the cross-sectional study. This will only bias estimates toward the null since previous studies have found that a severe form of abuse is highly associated with heavy drinking and alcohol dependence (Simpson & Miller, 2002; Widom, White, Czaja, & Marmorstein, 2007). Thus, our estimates may be an underestimate of the real coefficient. Third, reverse causality is a usual concern for observed associations from retrospective or concurrent studies. 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). These problems may be the reason for parents to physically punish offspring. To explore this question, we performed two series of analysis. In the first one, we restricted our analysis in the subsample of individuals who initiated MTM drinking after 16 years old. Estimates for CPP remained robust. In the second series of analysis, we adjusted for the history of childhood conduct problem. The estimated effect of CPP remains robust as well. Although it cannot be ruled out that the robustness is due to other unmeasured confounders, our results provide a solid basis for further inspections.

Summarized from previous studies, Simpson and Miller concluded that there is evidence of the possible causal relationship between CPA and drinking problems in females and boys, but not in adult males (Simpson & Miller, 2002). In fact, this issue has been rarely studied in the general adult male population. The relationship between CPP and negative drinking

outcomes may be different in special population, e.g. prisoners, court recruits, and clinical patients, from that in the general population. Drinking and related problems are complex human behaviors and conditions with numerous competing causes. In special populations, individuals who were not exposed to CPP/CPA may be more likely to experience other possible causes of AUD (e.g. lack of parental supervision, emotional distress, etc.), compared to the entire population of non-CPP exposed individuals; and eventually develop drinking problems. Thus, we argue that the association in the majority of adult the male population is still largely unknown. Our study has provided first-hand evidence of the possible causal relationship between CPP and riskier drinking and drinking problems in adult males from a population perspective.

Using the novel technique of GEE, we are able to take the inter-correlation of outcomes into consideration. This technique granted us statistical efficiency and ensured accurate statistical comparison of estimated strengths of associations. Results are interesting in that CPP is more strongly associated with "early onset" of drinking problems, compared with "early try" and "early MTM drinking". It suggested that victims of CPP progress more rapidly than people with no history of CPP. Previous studies have shown that victims of CPA are more likely to initiate drinking earlier (Bensley, Spieker, Van Eenwyk, & Schoder, 1999; Dube et al., 2006; Hamburger, Leeb, & Swahn, 2008; Rothman, DeJong, Palfai, & Saitz, 2008). Our results suggested that above the early involvement of drinking (early try and early MTM drinking), CPP may pose additional risk to early onset of drinking problems.

It is in line with the maturation theory of early onset of substance use disorders (Blackson & Tarter, 1994). According to the maturation theory, childhood to early adulthood is the key period for brain maturation. Long-term exposure to environmental stress, e.g. CPP, can push the maturation process to deviate toward non-normality through behavioral epigenetic mechanisms. The study by Rothman et al. found that compared with drinkers with no CPP history, victims of CPP are more likely to use drinking as a tool to cope (Rothman, DeJong, Palfai, & Saitz, 2008). This motive of drinking may get victims of CPP into unhealthy drinking behavior, e.g. binge drinking, which leads to social interpersonal problems, and alcohol dependence sooner. Early onset drinking problems is associated with higher level of severity and worse treatment outcome (J. Brown, Babor, Litt, & Kranzler, 1994). Along with another finding that CPP was more strongly associated with "early onset" of drinking problems than those with "lifetime occurrence" of drinking problems, our results highlighted that one focus of prevention strategies may be placed on fostering coping skills in CPP victims from early on.

Drinking problems in this study covered a broad range of problems, from drinking-related aggression to neuro-adaptation to compulsive drinking. Different mechanisms are involved in different clinical features (Koob, 2006; J. Liu et al., 2006). Previous animal studies have shown that lack of maternal care or adverse environment leads to changes in the GABA-A receptor, which is highly involved with the psychoactive effect of ethanol (Caldji, Diorio, & Meaney, 2003), as well as changes in hippocampus and prefrontal cortex

(Bremner et al., 1997; R. G. Heath, 1972; Teicher, Tomoda, & Andersen, 2006). When these drinking problems were grouped into two categories (socially maladaptive problems and alcohol dependence) it is not clear which problems are actually associated with CPP, and furthermore, whether the magnitudes of associations vary across manifestations. Thus, important messages may be buried when using one variable to represent different manifestations. To our knowledge, there has been a vacancy in literature regarding this issue. Using the GEE technique, we not only generated manifestation-specific estimates, but also compared the magnitude across estimates. Heterogeneous associations in manifestations of alcohol dependence were found. The smallest OR (non-significant) was found in "drink despite physical/mental problems". Compared with the association in "drink despite physical/mental problems", (which is a clinical feature of compulsive drinking) stronger associations were found in "irresistible desire" and "difficulty in cutting down", both of which are clinical features of loss of control over alcohol. One possible explanation is that at the same level of alcohol dependence, individuals who suffered from CPP may be more likely to report these two clinical features. However, results from the MIMIC model did not support this hypothesis: at the same level of problematic drinking, CPP victims are no more likely to report "irresistible desire" and "difficulty cutting down" than other problems. We hope the observed heterogeneity of associations can spur novel hypotheses and promote future studies into this issue, especially the mechanisms behind loss of control over alcohol.

With respect to stages of alcohol involvement, our results demonstrated that the most important stage is arguably the occurrence of drinking problems after the initiation of MTM drinking rather than the initiation of MTM drinking. However, it is still a rather broad stage involving many stages of progression. Due to the cross-sectional design of this study and self-recall of assessment, the ability of inference of temporality is limited. Our results serve as a call for future studies on this particular stage.

5.4. Future research

First, the observation that people in Beijing had more problems with drinking deserves further inspection. A study conducted in four Scottish towns found geographical variations in drinking patterns, and these variations in drinking patterns were closely associated with alcohol-related crime, morbidity, and mortality (Plant & Pirie, 1979). Given these findings, studies about drinking culture with respect to drinking context, people's attitude about social and excessive drinking may bear important messages for designing prevention and intervention strategies. Besides, a previous study showed that the ADH and ALDH genotype and allele frequencies are differentially distributed across different ethnic groups within China (Y. C. Shen et al., 1997). There has been no study exploring geographic variations of these allele frequencies within the Han ethnicity. Here, we argue that since these genotypes directly affect individual drinking behavior, it is of interest to integrate these biological measurements into socio-cultural factors to explore the geographic variations.

Second, we found robust subgroup variations with respect to sex, age, marital status, employment status, and income level. Among these variables, sex and age are possible determinants of drinking and related problems because both are exogenous to drinking. Further investigation into these two variables may provide insights into the etiology and drinking-related problems. For example, studies in Western countries have shown that the male-female difference in drinking problems may be due to higher drinking level in males (Ely, Hardy, Longford, & Wadsworth, 1999; Miller, Plant, & Plant, 2005). Whether it holds in the Chinese drinking culture will complement current knowledge.

It is interesting that a higher level of personal income is associated with higher drinking involvement but not drinking problems. The lack of association between income and drinking problems was also shown in the NESARC study (D. S. Hasin, Stinson, Ogburn, & Grant, 2007). Several possible explanations can be proposed. For example, the phenomenon may suggest that people with higher income have more opportunities to drink, e.g. business-related social activities given that "toasting" has been a popular way of building relationships in modern China (Cochrane, Chen, Conigrave, & Hao, 2003; Hao, Chen, & Su, 2005). It might reflect the infiltration of Western drinking culture as people in the higher income level started to embrace drinking into their daily life. It also might suggest that people in the higher income level are more aware of health benefits of modest drinking. To further

probe the reason behind this observation bears useful information for future education strategies about healthier drinking style.

Third, children from dysfunctional families, including but not limited to alcoholic families, suffer from various childhood adversities, such as family tension or parent divorce, witnessing domestic violence, child neglect, and so on. These clusters of childhood adversities may influence their neurodevelopment and lead to internalizing or externalizing problems, which in turn put them into a higher risk of adulthood physical and mental outcomes (Clemmons, DiLillo, Martinez, DeGue, & Jeffcott, 2003; Edwards & Gross, 1976; Ruchkin, Gilliam, & Mayes, 2008; Tremblay et al., 2004). In this context, it is of interest and importance to tease out the effect of more modifiable factors, e.g. CPP, from less modifiable factors such as parent divorce or family tension.

Additionally, people with drinking problems vary in severity. Disease burden is higher in those who suffer from multiple problems than those with only one problem. Early observations by Tarter, et al. suggested that drinkers in higher severity level differ from drinkers in lower severity level in childhood brain function (Blackson & Tarter, 1994). There has been evidence that CPP is associated with brain function. In this context, it is of interest to probe if CPP is associated with the occurrence of drinking problems only or with severity as well.

With respect to inferring temporality, longitudinal studies with small attrition are needed. Nevertheless, observational studies are low in power to infer causality. Experimental studies are needed to establish the causal

relationship between CPP and drinking problems. Previous studies have found that both genetic and environmental factors influence violent behavior toward children, including physical abuse (DiLalla & Gottesman, 1991; Widom, 1989). In this context, experimental studies are especially of interest to clarify in what degree physical abuse is modifiable. Our study found stronger association between CPP and loss of control over drinking. We also found stronger association between CPP and "early onset of problems". Survival analysis from follow-up data will be essential to tease out if the stronger association is due to earlier onset (longer progression) or due to the influence of CPP on specific functions of the brain. Last but not the least, the incorporation of genetic and epigenetic methodology is essential to study the etiology of drinking-related problems as the developmental process is a complex network of genetic and environmental factors through multiple pathways (Blackson & Tarter, 1994).

Appendix

Questions about drinking

*SU1. The next questions are about your use of alcoholic beverages, including beer, wine, wine coolers, and hard liquor like vodka, gin or whiskey. How old were you the very first time you ever drank an alcoholic beverage?

_____ YEARS OLD

(IF VOL): "NEVER" ..... 997 **GO TO *SU39**
DON'T KNOW 998
REFUSED 999

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*SU2. IF R CAN READ: (RB, PG 17) Please use the table on page 17 in your booklet as a guide in answering the next questions. How old were you when you first started drinking at least 12 drinks in a year?
IF R CANNOT READ: When I use the word "drink" in the next questions, I mean either a glass of wine, a can or bottle of beer, or a shot or jigger of liquor either alone or in a mixed drink. How old were you when you first started drinking at least 12 drinks in a year?
IF "ALL MY LIFE" OR "AS LONG AS I CAN REMEMBER," PROBE: Was it before your teens?
IF NO/DK, PROBE: Was it before your twenties?

_____ YEARS OLD

BEFORE TEENS 12
BEFORE 20s 19
NOT BEFORE 20s 20
(IF VOL): "NEVER" ..... 997 **GO TO *SU39**
DON'T KNOW 998
REFUSED 999

---

*SU3. (RB, PG 17) (Look at page 17 in your booklet.) Think about the past 12 months. In the past 12 months, how often did you usually have at least one drink – nearly every day, three to four days a week, one to two days a week, one to three days a month, or less than once a month?

NEARLY EVERY DAY 1
3 - 4 DAYS PER WEEK 2
1 - 2 DAYS PER WEEK 3
1 - 3 DAYS PER MONTH 4

LESS THAN ONCE A MONTH (INCLUDING NEVER DRINK) .....5 **GO TO *SU8**
DON'T KNOW ..... 8 **GO TO *SU8**
REFUSED ..... 9 **GO TO *SU8**

---

*SU4. (RB, PG 17) (Looking at page 17 in your booklet,) On the days you drank in the past 12 months, about how many drinks did you usually have per day?

_____ NUMBER OF DRINKS PER DAY

DON'T KNOW 998
REFUSED 999

***SU5.** Was there ever a year in your life when you drank more than you did in the past 12 months?

YES ..... 1 **GO TO *SU8**
NO 5
DON'T KNOW 8
REFUSED 9

***SU6.** INTERVIEWER CHECKPOINT: (SEE ***SU3**)

***SU3** EQUALS '4' 1
ALL OTHERS ..... 2 **GO TO SU12**

***SU7.** INTERVIEWER CHECKPOINT: (SEE ***SU4**)

***SU4** IS EQUALS '3' OR MORE ..... 1 **GO TO SU12**
ALL OTHERS ..... 2 **GO TO SU39**

***SU8.** Think about the years in your life when you drank most. During those years, how often did you usually have at least one drink – nearly every day, three to four days a week, one to two days a week, one to three days a month, or less than once a month?

NEARLY EVERY DAY 1
3 - 4 DAYS PER WEEK 2
1 - 2 DAYS PER WEEK 3
1 - 3 DAYS PER MONTH 4

LESS THAN ONCE A MONTH ..... 5 **GO TO *SU39**
DON'T KNOW ..... 8 **GO TO *SU39**
REFUSED ..... 9 **GO TO *SU39**

***SU9.** And on the days you drank during those years, about how many drinks would you usually have per day?

_____ NUMBER OF DRINKS PER DAY

DON'T KNOW 998
REFUSED 999

***SU10.** INTERVIEWER CHECKPOINT: (SEE ***SU8**)
***SU8** EQUALS '4' 1
ALL OTHERS ..... 2 **GO TO SU12**

***SU11.** INTERVIEWER CHECKPOINT: (SEE ***SU9**)

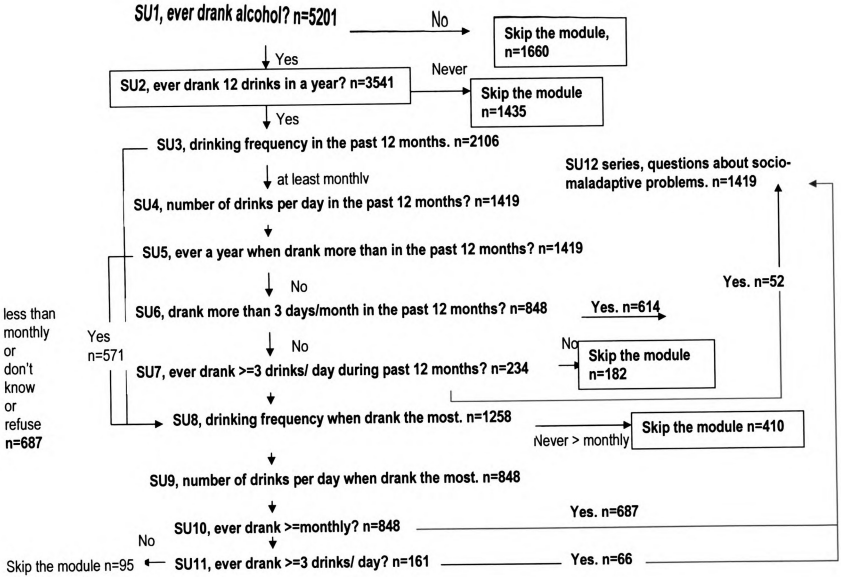
***SU9** EQUALS '3' OR MORE..... 1
ALL OTHERS..... 2 **GO TO *SU39**

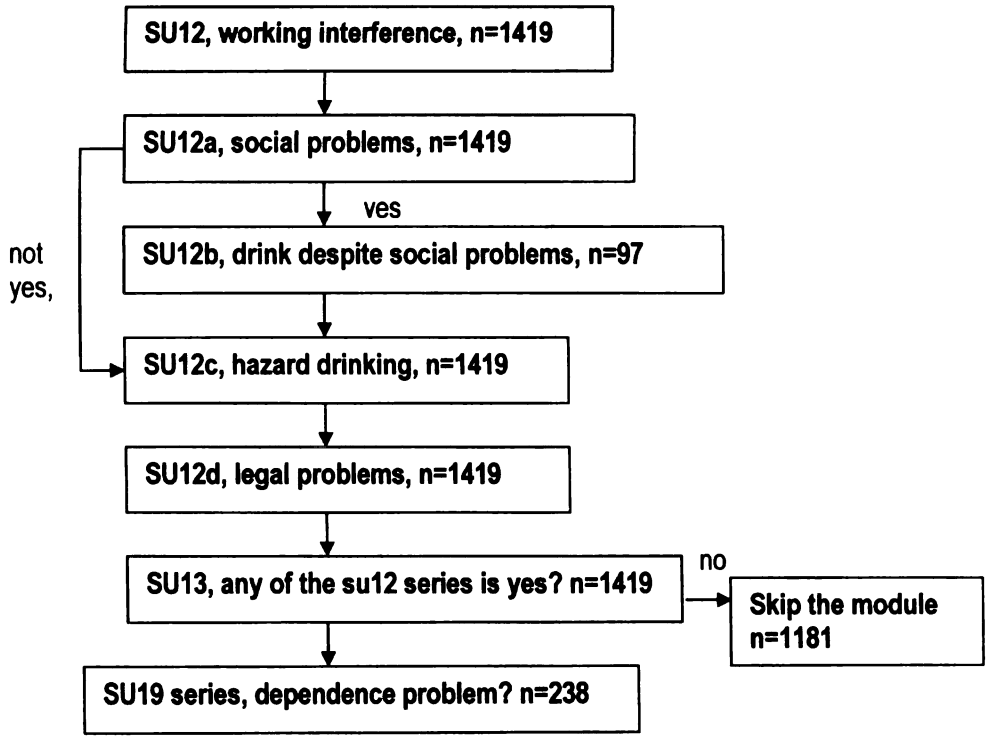
INTERVIEWER INSTRUCTION: IF R PROTESTS OR REFUSES TWO QUESTIONS, CODE ALL UNANSWERED ****SU12 SERIES*** *QUESTIONS '9' AND* **GO TO *SU13.**	**YES (1)**	**NO (5)**	**DK (8)**	**RF (9)**
***SU12.** The next questions are about problems you may have had because of drinking. First, was there ever a time in your life when your drinking or being hung over frequently interfered with your work or responsibilities at school, on a job, or at home? (KEY PHRASE: interfered with your work)	1	5	8	9
*SU12a. Was there ever a time in your life when your drinking caused arguments or other serious or repeated problems with your family, friends, neighbors, or co-workers? (KEY PHRASE: caused problems with family, friends or others)	1	5 **GO TO *SU1 2c**	8 **GO TO *SU1 2c**	9 **GO TO *SU1 2c**
*SU12b. Did you continue to drink even though it caused problems with these people? (NO KEY PHRASE)	1	5	8	9
***SU12c.** Were there times in your life when you were often under the influence of alcohol in situations where you could get hurt, for example when riding a bicycle, driving, operating a machine, or anything else? (KEY PHRASE: jeopardized your safety because you sometimes drank in situations where you could get hurt)	1	5	8	9
*SU12d. Were you more than once arrested or stopped by the police because of drunk driving or drunk behavior? (KEY PHRASE: resulted in problems with the police)	1	5	8	9

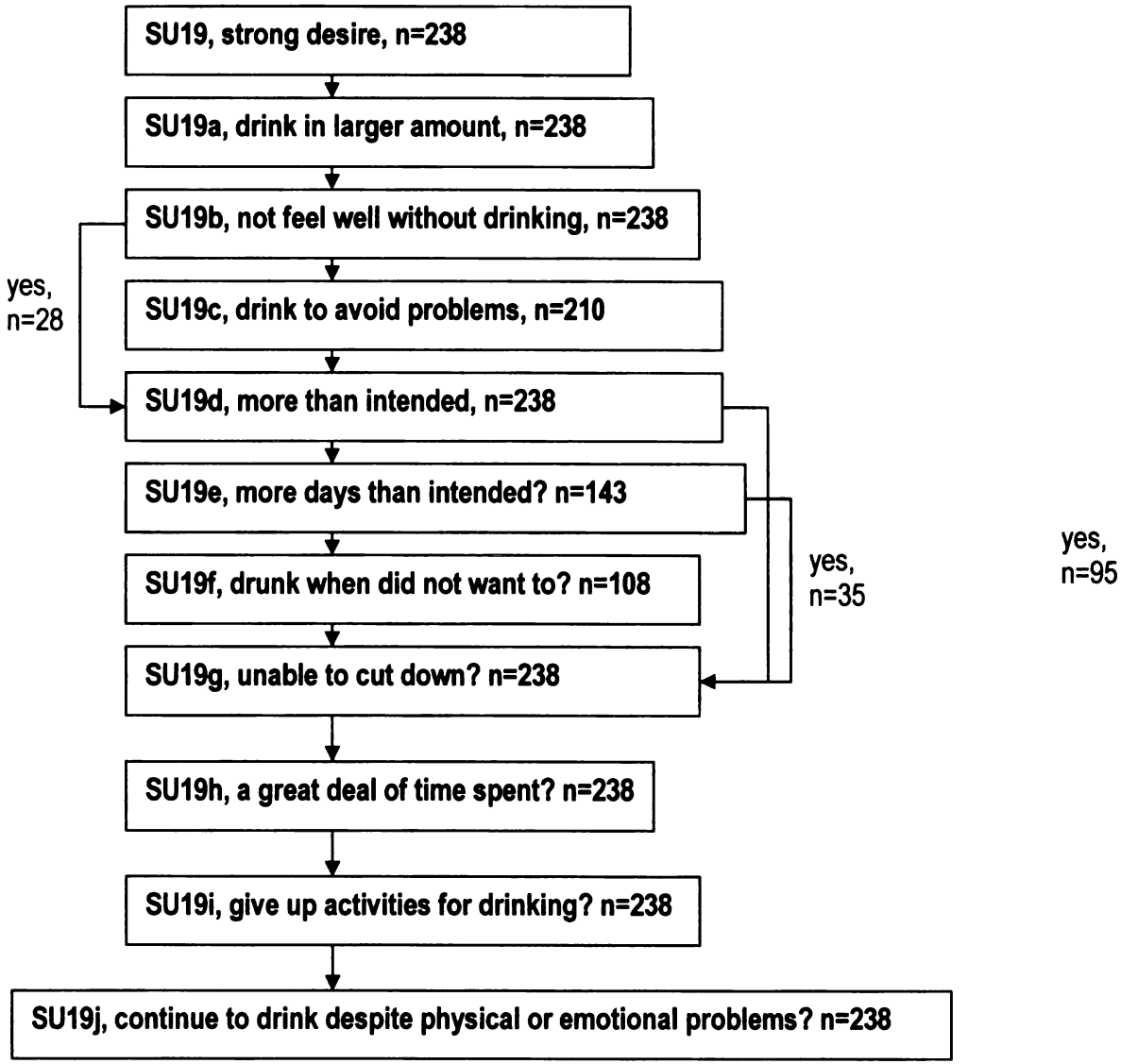
INTERVIEWER INSTRUCTION: IF R PROTESTS OR REFUSES TWO QUESTIONS, CODE ALL UNANSWERED ***SU19 SERIES** QUESTIONS '9' AND **GO TO *SU20**.	**YES (1)**	**NO (5)**	**DK (8)**	**RF (9)**
***SU19**. (The next questions are about some <u>other</u> problems you may have had because of drinking.) Was there ever a time in your life when you often had such a strong desire to drink that you couldn't resist taking a drink or found it difficult to think of anything else? KEY PHRASE: a strong desire to drink	1	5	8	9
*SU19a. Did you ever need to drink a larger amount of alcohol to get an effect, or did you ever find that you could no longer get a "buzz" or a high on the amount you used to drink? KEY PHRASE: drink a larger amount of alcohol	1	5	8	9
*SU19b. People who cut down or stop drinking after drinking steadily for some time may not feel well. These feelings are more intense and can last longer than the usual hangover. Did you ever have times when you stopped, cut down, or went without drinking and then experienced symptoms like fatigue, headaches, diarrhea, the shakes, or emotional problems? KEY PHRASE: not feel well	1 **GO TO *SU19d**	5	8	9
*SU19c. Did you ever have times when you took a drink to <u>keep</u> from having problems like these? KEY PHRASE: keep from having problems like these	1	5	8	9
***SU19d**. Did you have times when you started drinking even though you <u>promised</u> yourself you wouldn't, or when you drank a lot more than you intended? KEY PHRASE: drank a lot more than you intended	1 **GO TO *SU19g**	5	8	9

*SU19e. Were there ever times when you drank more frequently or for more days in a row than you intended? KEY PHRASE: more days in a row than you intended	1 **GO TO *SU19g**	5	8	9
***SU19f. Did you have times when you started drinking and became drunk when you didn't want to?** KEY PHRASE: drunk when you didn't want to	**1**	**5**	**8**	**9**
***SU19g.** Were there times when you tried to stop or cut down on your drinking and found that you were not able to do so? KEY PHRASE: tried to stop or cut down on your drinking but not able	1	5	8	9
*SU19h. Did you ever have periods of several days or more when you spent so much time drinking or recovering from the effects of alcohol that you had little time for anything else? KEY PHRASE: little time for anything else	1	5	8	9
*SU19i. Did you ever have a period of a month or longer when you gave up or greatly reduced important activities because of your drinking – like sports, work, or seeing friends and family? KEY PHRASE: gave up or greatly reduced important activities because of your drinking	1	5	8	9
*SU19j. Did you ever continue to drink when you knew you had a serious physical or emotional problem that might have been caused by or made worse by drinking? KEY PHRASE: continue to drink while physical or emotional problem caused by or made worse by drinking	1	5	8	9

Skip patterns are shown in following flow charts.


SU1, ever drank alcohol? n=5201
No
Skip the module, n=1660
Yes
SU2, ever drank 12 drinks in a year? n=3541
Never
Skip the module n=1435
Yes
SU3, drinking frequency in the past 12 months. n=2106
at least monthly
SU4, number of drinks per day in the past 12 months? n=1419
SU5, ever a year when drank more than in the past 12 months? n=1419
No
SU6, drank more than 3 days/month in the past 12 months? n=848
Yes. n=614
less than monthly or don't know or refuse n=687
Yes n=571
No
SU7, ever drank >=3 drinks/ day during past 12 months? n=234
No
Skip the module n=182
SU8, drinking frequency when drank the most. n=1258
Never > monthly
Skip the module n=410
SU9, number of drinks per day when drank the most. n=848
SU10, ever drank >=monthly? n=848
Yes. n=687
No
Skip the module n=95
SU11, ever drank >=3 drinks/ day? n=161
Yes. n=66
SU12 series, questions about socio-maladaptive problems. n=1419
Yes. n=52


SU12, working interference, n=1419
SU12a, social problems, n=1419
ves
not
yes,
SU12b, drink despite social problems, n=97
SU12c, hazard drinking, n=1419
SU12d, legal problems, n=1419
SU13, any of the su12 series is yes? n=1419
no
Skip the module
n=1181
SU19 series, dependence problem? n=238


SU19, strong desire, n=238
SU19a, drink in larger amount, n=238
SU19b, not feel well without drinking, n=238
yes, n=28
SU19c, drink to avoid problems, n=210
SU19d, more than intended, n=238
SU19e, more days than intended? n=143
yes, n=35
SU19f, drunk when did not want to? n=108
SU19g, unable to cut down? n=238
SU19h, a great deal of time spent? n=238
SU19i, give up activities for drinking? n=238
SU19j, continue to drink despite physical or emotional problems? n=238

yes, n=95

200

Questionnaire in Chinese
物质滥用（SU）

***SU1.** 下面的问题是关于您饮用含酒精饮料的情况，如啤酒，果酒，葡萄酒和烈性酒（如白酒、威士忌、伏特加等）。您第一次饮用含酒精饮料时是多大年纪？

_____ 年龄

(如果主动提出): “从来没有” 997 **进行到*SU39 第 97 页**

不知道 998
拒绝回答 999

***SU2、(如果受访人能够阅读)** 请您使用手册**第 14 页**的酒精类饮品换算表作为回答下列问题的一个度量标准。当您最初开始每年至少饮酒 12 杯（1 杯相当于 1 听啤酒）时，您有多大年纪？

（如果受访人自己不能读）当我在下列问题中使用“一杯”这个词的时候，我的意思是指喝一杯葡萄酒、一听啤酒、或一盅白酒。当您最初开始每年至少饮酒 12 杯时，您是多大年纪？

（如果受访人回答“生下来就这样”或者“很长时间了”，请问：是在十三岁以前吗？

如果受访人回答“不是”或者“不知道”，请问：是在您二十岁以前吗？）

_____ 年龄

十三岁以前 12
二十岁以前 19
二十岁以后 20

(如果主动提出): “从来没有” 997 **进行到*SU39 第 97 页**

不知道 998
拒绝回答 999

***SU3.** 请看手册的 14 页，在过去 12 个月中，您通常是几乎每天至少饮酒 1 次（至少 1 杯才算 1 次），每周 3-4 次、每周有 1-2 次、每月 1-3 次，还是每个月饮酒少于 1 次？

几乎每天 1
每周 3-4 次 2

每周 1-2 次 3
每月 1-3 次..... 4
每月少于 1 次（包括不喝酒）.. 5 **进行到*SU8**
不知道..... 8 **进行到*SU8**
拒绝回答 9 **进行到*SU8**

***SU4.** 请看手册的 **14 页**，在过去 12 个月您喝酒的日子中，您通常每天喝多少杯酒？

_____ 每天喝酒的杯数
不知道 998
拒绝回答 999

***SU5.** 有生以来，您是否曾经有一年的饮酒数量多于您过去 12 个月的饮酒量？

是的 1 **进行到*SU8**
不是 5
不知道 8
拒绝回答 9

***SU6.采访员检验点 (请看*SU3)**

***SU3 答案的编码为"4"..... 1**
所有其他 2 进行到*SU12

***SU7.采访员检验点 (请看 SU4)**

***SU4 答案的编码为"3"或者"3"以上数字..... 1 进行到*SU12**
所有其他 2 进行到*SU39

***SU8.** 回想一下有生以来您饮酒最多的那些年。在那些年中，您通常是几乎每天至少喝一次、每周 **3**-4 次、每周 1-2 次，每月 1-3 次，还是每月少于一次？

几乎每天一次 1
每周 **3**-4 次 2
每周 1-2 次 3
每月 1-3 次 4
每月少于一次 5 **进行到*SU39**

不知道 8 **进行到*SU39**

第 97 页

拒绝回答 9 **进行到*SU39**

第 97 页

***SU9.** 在饮酒最多的那些年您喝酒的日子里，您通常每天喝多少杯酒？

_____ 每天饮酒的杯数
不知道 998
拒绝回答 999

***SU10. 采访员检验点 (请看*SU8)**

***SU8 答案的编码为"4"** 1

所有其他 .....2 **进 行 到 *SU12**

*SU11. **采访员检验点** (请看*SU9)

***SU9 答案为"3"**.....1

所有其他 .....2 **进行到 *SU39 第 97 页**

采访员指示：如果受访人对问及的两个问题表示反感或者拒绝，就将*SU12 系列中没有回答的所有其他问题编码为"9"，并进行到*SU13	**是 (1)**	**不是 (5)**	**不知道 (8)**	**拒答 (9)**
***SU12**、下面要了解喝酒给您带来的问题，首先，您是否曾经一度由于经常喝酒或喝多了而影响了您在学校、家庭或工作岗位上的工作或责任？ **关键词**：影响了您的工作	1	5	8	9
***SU12a**、您曾经因为喝酒与家人、朋友、邻居或同事发生争执或出现其他严重的、反复发生的问题 **关键词**：与家人、朋友争执或发生其他的问题。	1	5 **到** SU12c	8 **到** SU12c	9 **到** SU12c
***SU12b**、尽管由于喝酒已经造成了您和亲友之间的一些问题，您仍然喝酒	1	5	8	9

***SU12c**、若干次出于喝酒而使自己处于可能受到伤害的情况之下（如骑自行车、开汽车、操纵机器等） **关键词**：因为喝酒而危及自身安全	1	5	8	9
***SU12d**、不止一次因为酒后驾车和醉酒的行为而被警察抓着了或者扣留 **关键词**：被警察抓着了或扣留	1	5	8	9

***SU13、采访员检验点、(请看*SU12 问题系列)**

没有编码为"1" 的答案..... 1 **进行到*SU39 第 97 页**

有一个编码为"1"的答案..... 2 **记录参照卡*SU12，然后进行到*SU15 INTRO1**

所有其他..... 3 **记录参照卡*SU12，然后进行到*SU15 INTRO2**

***SU15 INTRO1**	***SU15 INTRO2**
您的饮酒行为(关键词是在 SU12 问题系列中答案为"是"的)，您能够回忆起您第一次发生上述问题的确切年龄吗？ 可以..... 1 不可以..... 5 **到** SU15b 不知道 ..... 8 **到** SU15b 拒绝回答 ..... 9 **到** SU15b	您的饮酒行为(**关键词**是在 SU12 问题系列中答案为"是"的)。您能够回忆起您第一次发生这些问题的确切年龄吗？ 可以..... 1 不可以 ..... 5 **到** SU15b 不知道 ..... 8 **到** SU15b 拒绝回答 ..... 9 **到** SU15b

***SU15a**、您那时多大年纪？

_____ 年龄 **进行到*SU16**

不知道.....998 **进行到*SU16**

拒绝回答.....999 **进行到*SU16**

***SU15b**、您第一次出于饮酒产生这些问题中的一个或者几个时，多大岁数？

（如果受访人回答“生下来就这样”或者“很长时间了”，追问题：那么是在十三岁以前吗？

如果受访人回答是“不”或者“不知道”，追问：是在二十多岁以前吗？）

_____ 年龄

十三岁以前 12
二十岁以前 19
二十岁以后 20
不知道 998
拒绝回答 999

***SU16**、由于饮酒，您最近一次产生这些问题中的一个或者几个是在什么时候？过去一个月内、前两到六个月、前七到十二个月、还是超过十二个月以前？

过去一个月内 1 **进行到*SU18**
前两到六个月 2 **进行到*SU18**
前七到十二个月 3 **进行到*SU18**
超过十二个月以前 4
不知道 8 **进行到*SU18**
拒绝回答 9 **进行到*SU18**

*SU17、您最后一次由于饮酒产生这些问题中的一个或者几个时，您有多大岁数？

_____ 年龄

不知道 998
拒绝回答 999

***SU18**、有生以来，您有大约多少个年头出现过这个问题或者这些问题？

_____ 年份数

不知道 998
拒绝回答 999

采访员指示：如果受访人对两个问题表示反感或者拒绝，就将*SU19系列中没有回答的所有其他问题编码为"9"，并进行到*SU20	是 1	不是 5	不知道 8	拒答 9
（下列问题是关于由于饮酒您可能存在的其他一些问题。） ***SU19**、您是否曾经有过喝酒欲望特别强烈，不喝不行，不能想其他事情的时候。 **关键词**：您有强烈的不可抑制的饮酒欲望	1	5	8	9
*SU19a、您是否需要喝更多的酒才能得到愉快和舒服的感觉，或者说您是否发现按以前通常的数量喝酒，您不再有愉快和舒服的感觉？ **关键词**：您需要喝更多的酒才能得到愉快和舒服的感觉	1	5	8	9
*SU19b、在一段时间内持续喝酒的人，减量喝酒或戒酒会有不适感。这些感觉比醉酒后的不适感更强烈，而且持续时间更长。您是否有过在减量喝酒或戒酒后，出现疲乏、泻肚、头疼等症状或情绪问题？ **关键词**：不喝酒时有不适感	1 **到** *SU19d	5	8	9
*SU19c、您是否有时喝酒是为了避免出现上述这类问题？ **关键词**：喝酒以避免出现身体或精神不适感	1	5	8	9
***SU19d**、是否尽管有时您下决心不喝酒，还是喝了酒，或者，有时您喝酒的量要比您打算喝的量多很多？ **关键词**：不打算喝酒时喝酒，或喝酒的量大于打算喝的量	1 **到** *SU19g	5	8	9
*SU19e、是否有时您喝酒的次数或连续喝酒的天数比您原先打算的要多？ **关键词**：喝酒的次数多于原先的打算	1 **到** *SU19g	5	8	9
*SU19f、您是否有时在不想喝酒时喝了酒而且喝醉了？ **关键词**：不想喝酒时喝醉了	1	5	8	9
***SU19g**、是否有时您试图不喝酒或减量喝酒，但发现自己做不到？ **关键词**：试图不喝酒或减量喝酒但做不到	1	5	8	9

*SU19h、是否有过持续数天或更长的一段时间，您用于喝酒或从醉酒状态中恢复的时间太长，使您没有多少时间做别的事情？ **关键词：**做其他事情的时间少于喝酒或从醉酒状态中恢复的时间	1	5	8	9
*SU19I、是否有持续一个月或更长的一段时间，您由于喝酒而放弃或极大地缩减了重要的活动，比如体育锻炼、工作、探望朋友和家人？ **关键词：**由于喝酒而放弃或减少了重要活动	1	5	8	9
*SU19j、当您知道喝酒可能会给您造成严重的身体或精神问题，或者使您的身体或精神问题恶化时，您是否还继续喝酒？ **关键词：**即使喝酒会造成或恶化身体或精神问题，您仍然会喝酒	1	5	8	9

***SU20、采访员检验点、(请看*SU19 问题系列)**

编码为"1"的答案有 0-2 个.....1 进行到*SU39 第 97 页

所有其他答案 2

***SU25**、您说过您有很多由于饮酒产生的问题，那么您是否可以回忆起您第一次产生这些问题时的确切年龄吗？

可以 1

不可以.....5 **进行到*SU25b**

不知道.....8 **进行到*SU25b**

拒绝回答 9 **进行到*SU25b**

***SU25a、**您那时多大岁数？

_____岁 **进行到*SU26**

不知道 998 **进行到*SU26**

拒绝回答.....999 **进行到*SU26**

***SU25b**、第一次出现这些由于饮酒产生的问题时，您大约多大岁数？

（如果受访人回答"生下来就这样"或者"很长时间了"，追问、在您十三岁以前吗？

如果受访人回答是"不是"或者"不知道"，追问、在您二十岁前吗？）

_____ 年龄

十三岁前.....12
二十岁前.....19
二十岁后.....20
不知道.....998
拒绝回答.....999

***SU26**、您最近一次产生这样的问题是在什么时候？是过去一个月内、前两至六个月、前七到十二个月，还是超过十二个月以前？

过去一个月内	1	**进行到*SU28**
前两至六个月	2	**进行到*SU28**
前七到十二个月	3	**进行到*SU28**
超过十二个月以前	4	
不知道	8	**进行到*SU28**
拒绝回答	9	**进行到*SU28**

*SU27、您最后一次出现这些因为饮酒引起的问题时是多大年纪？

（如果受访人回答"生下来就这样"或者"很长时间了"，追问：在您十三岁以前吗？

如果受访人回答是"不是"或者"不知道"，追问：在您二十岁前吗？）

_____ 年龄

十三岁以前.....12
二十岁以前..... 19
二十岁以后..... 20
不知道..... 998
拒绝回答..... 999

***SU28**、有生以来，您有多少个年头曾经每年至少出现一个这样的问题？

_____ 年数

不知道.....998
拒绝回答.....999

*SU29、在同一年中，您是否曾经出现三个或者更多的这些问题？

是.....	1	
不是.....	2	**进行到 SU32**
不知道.....	8	**进行到 SU32**
拒绝回答.....	9	**进行到 SU32**

SU29.1.** 采访员检验点：在参照卡（第二面）的长/短组中圈出字母"N"，然后进行到SU30**

*SU30、 第一次发生同一年中出现这些问题中的三个或者更多问题时，您多大岁数？

（如果受访人回答“生下来就这样”或者“很长时间了”，追问：在您十三岁以前吗？

如果受访人回答是“不是”或者“不知道”，追问：在您二十岁前吗？）

_____ 年龄
十三岁以前.....12
二十岁以前 19
二十岁以后 20
不知道 998
拒绝回答 999

*SU31、有生以来，大约有多少个年头您出现这些问题中的三个或者更多的问题？

_____ 年数
不知道.....998
拒绝回答.....999

***SU32**、从您开始出现这些问题时起，您曾经多少次认真地尝试过戒酒？

_____ 次数
不知道.....998
拒绝回答.....999

***SU33、采访员检验点： (请看*SU32)**

***SU32 答案的编码为“1”或者更多.....1**

所有其他 2 进行到*SU37

***SU34**、从那以后，您不喝酒的时间最长持续了多久？

_____ （数字） 请记录时间单位： 天…1 星期…2
月…3 年…4
不知道.....998
拒绝回答.....999

***SU35、采访员检验点：(请看*SU32 *SU34)**

***SU32 答案的编码为“1”.....1 进行到*SU37**

*SU30、 第一次发生同一年中出现这些问题中的三个或者更多问题时，您多大岁数？

（如果受访人回答"生下来就这样"或者"很长时间了"，追问：在您十三岁以前吗？

如果受访人回答是"不是"或者"不知道"，追问：在您二十岁前吗？）

_____ 年龄
十三岁以前.....12
二十岁以前 19
二十岁以后 20
不知道 998
拒绝回答 999

*SU31、有生以来，大约有多少个年头您出现这些问题中的三个或者更多的问题？

_____ 年数
不知道.....998
拒绝回答.....999

***SU32**、从您开始出现这些问题时起，您曾经多少次认真地尝试过戒酒？

_____ 次数
不知道.....998
拒绝回答.....999

***SU33、采访员检验点： (请看*SU32)**

***SU32 答案的编码为"1"或者更多.....1**

所有其他 2 进行到*SU37

***SU34**、从那以后，您不喝酒的时间最长持续了多久？

_____ （数字） 请记录时间单位： 天…1 星期…2
月…3 年…4
不知道.....998
拒绝回答.....999

***SU35、采访员检验点：(请看*SU32 *SU34)**

***SU32 答案的编码为"1".....1 进行到*SU37**

***SU32 答案的编码为"2"或者更多并且*SU34 答案的编码为"3"或者更多**....2

所有其他 3 **进行到*SU37**

***SU36**、您有多少次不喝酒的时间持续3个月或更长时间？

_____ 次数
不知道.....998
拒绝回答.....999

***SU37、采访员检验点(请看 SU26 第 96 页)**

***SU26 答案的编码为 1、2 或 3**.....1

所有其他.....2 **进行到*SU38e.1**

***SU39、采访员检验点(请看参照卡，长组或者短组)**

在长组或者短组中记录过任何字母.....1 **进行到*SU40**

所有其他的.....2

***SU39.1、采访员检验点、(请看受访人的随机号)**

受访人随机号以 01-25 结尾.....1

所有其他编号 .....2 **短组** **进行到*DM1 部分 第 260 页**

***SU87**、不管您是否喝过酒或吸毒，下面的问题与您第一次有喝酒或吸毒的机会有关。"有机会"是指：别人给您酒或毒品；别人喝酒或吸毒时您在场，而且您想喝或吸就可以喝或吸。医务人员向您提供免费样品的情况不包括在内。 （请回想一下有生以来的情况）当您第一次有机会饮酒或者吸毒时，您大约有多大岁数？ **采访员提示：询问*SU87 关于酒精和毒品，然后问*SU88**	***SU88**、在您（喝酒/吸毒）以前，您有多少次饮酒或者吸毒的机会？ **采访员：如果受访人从来没有饮酒或者吸毒，追问：那么有生以来您有多少次喝酒/吸毒的机会？**

酒类	***SU87a**_____ 年龄 十三岁以前.....12　从未饮过酒...997 二十岁以前.....19　不知道.....998 二十岁以后.....20　拒绝回答.....999	***SU88a**_____次数 不知道.....998 拒绝回答...999
毒品	***SU87b**_____年龄 十三岁以前.....12　从未吸过毒...997 二十岁以前.....19　不知道.....998 二十岁以后.....20　拒绝回答.....999	***SU88b**_____次数 不知道.....998 拒绝回答...999

Questions about childhood experiences

*CH1. The next questions are about your childhood background. Did you live with both of your biological parents up until you were sixteen?

YES.....1 **GO TO *CH6**
NO.....5
DON'T KNOW .....8 **GO TO *CH6**
REFUSED .....9 **GO TO *CH6**

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*CH2. Why didn't you live with your biological parents?
INTERVIEWER: CIRCLE ALL THAT APPLY. DO NOT READ LIST.
(IF NEC: Did your biological mother or father die, were they separated or divorced, or was there some other reason?)

	*CH2a. How old were you when (you/ your) (EVENT)? IF VOL "LESS THAN ONE YEAR OLD," CODE "1."
MOTHER DIED 1	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
FATHER DIED..... 2	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
PARENTS SEPARATED/ DIVORCED 3	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
PARENTS NEVER LIVED TOGETHER 4	
ADOPTED 5	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
WENT TO BOARDING SCHOOL.. 6	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
FOSTER CARE 7	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
LEFT HOME BEFORE AGE SIXTEEN	

..... 8	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
OTHER (SPECIFY)..... 9 _____ _____	_____ YEARS OLD DON'T KNOW ..... 998 REFUSED 999
DON'T KNOW..... 98	
REFUSED..... 99	

***CH6.** Up until you were sixteen, were <u>you</u> ever away from home for six months or longer – either in foster care, with other relatives, in a boarding school, hospital, juvenile detention center, or elsewhere?
INTERVIEWER: CODE "NO" IF R VOLUNTEERS "RETURNED HOME ON

WEEKENDS" OR OTHER OCCASIONS DURING SIX-MONTH PERIOD.

YES.....1
NO .....5 **GO TO *CH8**
DON'T KNOW.....8 **GO TO *CH8**
REFUSED.....9 **GO TO *CH8**

*CH6a. Where did you go?

INTERVIEWER: CIRCLE ALL THAT APPLY.

LIVING WITH OTHER RELATIVES 1
BOARDING SCHOOL 2
HOSPITAL 3
JUVENILE DETENTION CENTER 4
FOSTER HOME.....5
OTHER (SPECIFY).....6

DON'T KNOW.....8
REFUSED.....9

*CH6b. How old were you the first time you went away?

_____.....YEARS OLD

DON'T KNOW.....998
REFUSED.....999

.....

*CH6c. Altogether, how many months or years were you away from home up until you were sixteen?

_____ DURATION NUMBER

CIRCLE UNIT OF TIME: MONTHS.1 YEARS 2

DON'T KNOW.....98
REFUSED.....99

*CH8. Who was the head of your household for most of your childhood?

INTERVIEWER: IF R SAYS "FATHER", PROBE: Was that your biological father, step-father, adoptive father, or someone else?

INTERVIEWER: IF R SAYS "MOTHER", PROBE. : Was that your biological mother, step-mother, adoptive mother, or something else?

INTERVIEWER: IF R SAYS IT CHANGED, PROBE: Who was the male head of your household for most of the time before you turned seventeen?

BIOLOGICAL FATHER.....1
ADOPTIVE FATHER.....2
STEP FATHER (SPOUSE/ PARTNER OF MOTHER) 3
OTHER MALE (SPECIFY).....4

BIOLOGICAL MOTHER.....5
ADOPTIVE MOTHER.....6
STEP MOTHER (SPOUSE/ PARTNER OF FATHER) 7
OTHER FEMALE (SPECIFY).....8

DON'T KNOW.....98 **GO TO *CH22**
REFUSED.....99 **GO TO *CH22**

*CH8a. How many years of school did (he/ she) complete?

_____ YEARS

DON'T KNOW 98
REFUSED 99

*CH9. How much of your childhood did (male head of household / female head of household) either work for pay or work in a family business? Would you say all of the time, most, some, a little or not at all?

INTERVIEWER: IF NEC CLARIFY: Work for pay includes self-employment.

INTERVIEWER: IF R SAYS FATHER WAS A FARMER, CLARIFY: Farming counts as working in a family business.

ALL.....1 **GO TO *CH11**
MOST .....2 **GO TO *CH11**
SOME 3
A LITTLE.....4
NOT AT ALL 5
DON'T KNOW.....8
REFUSED.....9

*CH9a. .....What was the main reason he was not working for pay during most of your childhood years?

INTERVIEWER: IF R SAYS: "He/She was self employed," CLARIFY: Work for pay includes self-employment. REREAD ***CH9.**

INTERVIEWER: IF R SAYS, "He/She ways a farmer," CLARIFY: Farming counts as working for pay. REREAD ***CH9**.

INTERVIEWER: CIRCLE ALL THAT APPLY.

PHYSICAL DISABILITY OR INJURY.....1
ALCOHOL OR DRUG ABUSE 2
MENTAL OR EMOTIONAL DISABILITY 3
TO STAY AT HOME TO RAISE CHILDREN.....4
UNABLE TO FIND JOB.....5
OTHER (SPECIFY).....7

DON'T KNOW.....8
REFUSED.....9

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*CH10. INTERVIEWER CHECKPOINT (SEE ***CH9**):

***CH9** EQUALS'3' OR '4' 1
ALL OTHERS .....2 **GO TO *CH22**

***CH22.** Overall, how would you rate (your parents' relationship/ the relationship of the people who raised you) while you were growing up – excellent, good, fair, or poor?

EXCELLENT 1
GOOD 2
FAIR 3
POOR 4
NO COUPLE (IF VOLUNTEERED) 5
DON'T KNOW 8
REFUSED 9

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*CH23. How much conflict and tension was there in your household while you were growing up – a lot, some, a little, or none?

A LOT 1
SOME 2
A LITTLE 3
NONE 4
DON'T KNOW 8
REFUSED 9

(RB, PG 38) LIST FOR QUESTIONS *CH28 - *CH29

- PUSHED, GRABBED OR SHOVED
- THREW SOMETHING
- SLAPPED, HIT, OR PUNCHED

*CH28. (RB, PG 38) When you were growing up, how often did someone in your household do any of the things (on the list on page 38 in your booklet) to you – often, sometimes, rarely, or never?

OFTEN 1
SOMETIMES 2
RARELY 3
NEVER .....4 **GO TO *CH29**
DON'T KNOW .....8 **GO TO *CH29**
REFUSED .....9 **GO TO *CH29**

*CH28a. Who did this to you?

(PROBE: Anyone else?)

INTERVIEWER: CIRCLE ALL THAT APPLY.

BIOLOGICAL FATHER.....1
ADOPTIVE FATHER.....2
STEP FATHER.....3
BIOLOGICAL MOTHER.....4
ADOPTIVE MOTHER.....5
STEP MOTHER.....6
BROTHER/ SISTER.....7
OTHER PERSON.....8
DON'T KNOW.....98
REFUSED.....99

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*CH41. During the years you were growing up, did (WOMAN WHO RAISED R) ever have periods lasting 2 weeks or more where she was sad or depressed most of the time?

YES.....1
NO.....5 **GO TO *CH46**
DON'T KNOW.....8 **GO TO *CH46**
REFUSED.....9 **GO TO *CH46**

*CH41a. Was this during all, most, some, or only a little of your childhood?

ALL.....1
MOST.....2
SOME.....3
A LITTLE.....4
DON'T KNOW.....8
REFUSED.....9

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*CH42. During the time her depression was at its worst, did she also have other symptoms like low energy, changes in sleep or appetite, and problems with concentration?

YES.....1
NO.....5 **GO TO *CH46**

DON'T KNOW.....8 **GO TO *CH46**
REFUSED.....9 **GO TO *CH46**

***CH46**.....During the time you were growing up, did (WOMAN WHO RAISED R) ever have periods of a month or more when she was constantly nervous, edgy, or anxious?

YES.....1
NO.....5 **GO TO *CH51**
DON'T KNOW.....8 **GO TO *CH51**
REFUSED.....9 **GO TO *CH51**

***CH46a**.....Was that during all, most, some, or only a little of your childhood?

ALL.....1
MOST.....2
SOME.....3
A LITTLE.....4
DON'T KNOW.....8
REFUSED.....9

***CH47**.....During the time her nervousness was at its worst, did she also have other symptoms like being restless, irritable, easily tired, and difficulty falling asleep?

YES.....1
NO.....5 **GO TO *CH51**
DON'T KNOW.....8 **GO TO *CH51**
REFUSED.....9 **GO TO *CH51**

***CH51.** Did (WOMAN WHO RAISED R) ever complain about anxiety attacks where all of a sudden she felt frightened, anxious, or panicky?

YES.....1
NO.....5 **GO TO *CH52**
DON'T KNOW.....8 **GO TO *CH52**
REFUSED.....9 **GO TO *CH52**

*CH51a. Did she ever comment that during these attacks that her heart was pounding, or that she was short of breath, felt ill, or was fearful that she would die?

YES 1
NO 5
DON'T KNOW 8
REFUSED 9

***CH52.** Did (WOMAN WHO RAISED R) ever have a problem with alcohol or drugs?

YES.....1
NO.....5 **GO TO *CH61**
DON'T KNOW.....8 **GO TO *CH61**
REFUSED.....9 **GO TO *CH61**

---

*CH67. Did (WOMAN WHO RAISED R) ever attempt to commit suicide?

YES.....1
NO.....5
DON'T KNOW.....8
REFUSED.....9

*CH71. During the years you were growing up, did (MAN WHO RAISED R) ever have periods lasting 2 weeks or more where he was sad or depressed most of the time?

YES.....1
NO.....5 **GO TO *CH76**
DON'T KNOW.....8 **GO TO *CH76**
REFUSED.....9 **GO TO *CH76**

*CH71a. Was this during all, most, some, or only a little of your childhood?

ALL.....1
MOST 2
SOME 3
A LITTLE 4
DON'T KNOW.....8
REFUSED.....9

---

*CH72. During the time his depression was at its worst, did he also have other symptoms like low energy, changes in sleep or appetite, and problems with concentration?

YES.....1
NO.....5 **GO TO *CH76**

DON'T KNOW.....8 **GO TO *CH76**
REFUSED.....9 **GO TO *CH76**

***CH76**.....During the time you were growing up, did (MAN WHO RAISED R) ever have periods of a month or more when he was constantly nervous, edgy, or anxious?

YES.....1
NO.....5 **GO TO *CH81**
DON'T KNOW.....8 **GO TO *CH81**
REFUSED.....9 **GO TO *CH81**

*CH76a. .....Was that during all, most, some, or only a little of your childhood?

ALL 1
MOST.....2
SOME.....3
A LITTLE 4
DON'T KNOW 8
REFUSED 9

***CH77**.....During the time his nervousness was at its worst, did he also have other symptoms like being restless, irritable, easily tired, and difficulty falling asleep?

YES.....1
NO.....5 **GO TO *CH81**
DON'T KNOW.....8 **GO TO *CH81**
REFUSED.....9 **GO TO *CH81**

***CH80**. Did his nervousness ever interfere a lot with his life or activities?

YES.....1
NO.....5
DON'T KNOW.....8
REFUSED.....9

***CH81.** Did (MAN WHO RAISED R) ever complain about anxiety attacks where all of a sudden he felt frightened, anxious, or panicky?

YES.....1

NO.....5 **GO TO *CH82**
DON'T KNOW.....8 **GO TO *CH82**
REFUSED.....9 **GO TO *CH82**

*CH81a. Did he ever comment that during these attacks that his heart was pounding, or that he was short of breath, felt ill, or was fearful that he would die?

YES 1
NO 5
DON'T KNOW 8
REFUSED 9

***CH82.** Did (MAN WHO RAISED R) ever have a problem with alcohol or drugs?

YES.....1
NO.....5 **GO TO *CH91**
DON'T KNOW.....8 **GO TO *CH91**
REFUSED.....9 **GO TO *CH91**

Questions about conduct problems

***CD1.** (RB, PG 42) The next questions are about things adults don't like children to do. We want to know if these are things you did during your childhood or teenage years.

(IF NEC: As a child or teenager,…)	YES (1)	NO (2)	DK (8)	RF (9)
*CD1a. As a child or teenager, did you often tell lies to trick people into giving you things or doing what you wanted them to do? (KEY PHRASE: telling lies to trick people)	1 **GO TO *CD1c**	5	8	9
*CD1b. …did you often get out of doing things you were supposed to do by fooling people or lying to them? (KEY PHRASE: getting out of doing things by fooling people or lying)	1	5	8	9
***CD1c**. … did you often stay out much later at night than your parents wanted? (KEY PHRASE: staying out later than your parents wanted)	1	5	8	9
*CD1d. … did you often skip school without permission? (KEY PHRASE: skipping school)	1	5	8	9
*CD1e. … did you ever shoplift or steal something worth at least $10? (KEY PHRASE: shoplifting)	1	5	8	9

*CD1f. … did you ever steal money or other things from your parents or the other people you lived with? (KEY PHRASE: stealing from people you lived with)	1	5	8	9
*CD1g. … did you ever break into someone's locked car, or a locked home or building? (KEY PHRASE: breaking into a car, home, or building)	1	5	8	9
*CD1h. … did you ever set a fire to try to cause serious damage? (KEY PHRASE: setting a fire to try to cause damage)	1	5	8	9
*CD1i. (Other than by setting fires,) …did you ever deliberately damage someone's property by doing something like breaking windows, slashing tires, vandalizing, or writing graffiti on buildings? (KEY PHRASE: damaging property)	1	5	8	9
*CD1j. … did you ever run away from home and stay away for at least four days? (KEY PHRASE: running away from home)	1 **GO TO *CD2**	5	8	9
*CD1k. … did you run away from home overnight more than once? (KEY PHRASE: running away from home)	1	5	8	9

(IF NEC: As a child or teenager, …)	YES (1)	NO (5)	DK (8)	RF (9)
*CD16b. …did you often get involved in physical fights?	1	5	8	9
*CD16c. …did you ever use a weapon on another person, like a baseball bat, glass bottle, knife, gun, or brick?	1	5	8	9
*CD16d. … were you ever physically cruel to an animal and hurt it on purpose? (IF NEC: This does not include hunting or getting rid of pests like rodents or insects.)	1	5	8	9
*CD16e. …were you ever physically cruel to a person and hurt them on purpose?	1	5	8	9
*CD16f. …did you ever force someone to give you something like money, jewelry, or clothing by threatening them or causing them injury?	1 **GO TO *CD16h**	5	8	9
*CD16g. …did you ever steal someone's purse, wallet, luggage, package or bag by grabbing it from them? (IF NEC: This does not include stealing from someone who wasn't aware of the theft, such as stealing a piece of luggage when the owner wasn't watching.)	1	5	8	9
***CD16h**. …did you ever make anyone do something sexual by either forcing, intimidating, or threatening them?	1	5	8	9

Appendix tables

Table A4.3.1.7. p values from Wald tests for endogeneity in males. Data from WMH-mC, 2001-2002.

	p values of the test of H_0: atanhρ=0					
	parental drinking/drug problems alone	parental mental disturbances alone	Being beat badly alone	Conduct problem alone	Parental drinking/drug problems+ mental problems	Parental variables +beat+conduct
early try (<=12 yrs)	**0.01**	**0.03**	0.37	0.99	**<0.01**	0.05
early drinking (<=19 yrs)	0.10	0.49	0.61	0.98	0.42	0.30
early socially maladaptive problem (<=23 yrs)	0.51	0.48	0.98	0.14	0.74	**0.03**
early dependence problem (<=23 yrs)	**<0.01**	0.23	0.15	0.12	0.10	0.58
early any problem (<=23 yrs)	0.52	0.47	0.99	0.14	0.75	**0.03**
ever heavier drinking	0.20	0.91	0.89	**0.04**	0.29	**0.04**
ever socially maladaptive problems	0.38	0.74	0.93	**0.02**	0.32	0.15
ever dependence	**0.02**	**0.02**	0.09	0.44	**0.03**	0.56
any dependence clinical feature	0.13	0.07	0.62	0.19	**0.03**	0.60
onset of problem after 16	0.58	0.69	0.92	**0.03**	0.30	0.18

1. Method 1 is probit regression with continuous endogenous variable; method 2 is linear regression with binary endogenous variable; model 3 is recursive probit regression.

Table A4.3.1.8 Estimates for CPP from recursive probit models after taking endogeneity into account in males. Data from WMH-mC, 2001-2002.

	Parental drinking/drug problems alone		Parental mental disturbances alone		Parental variables together		Conduct problem along		Parental variables +beat+conduct problems	
	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.	coef.	95% C.I.
early try (<=12 yrs)	**1.7**	**0.8, 2.5**	**1.3**	**0.5, 2.2**	**1.4**	**0.7, 2.1**			**1.2**	**0.3, 2.2**
early drinking (<=19 yrs)										
early socio-maladaptive problem (<=23 yrs)									**1.9**	**0.9, 2.9**
early dependence problem (<=23 yrs)	**3.1**	**2.1, 4.1**								
early any problem (<=23 yrs)									**1.9**	**0.9, 2.9**
ever heavier drinking							**2.4**	**2.1, 2.7**	**1.2**	**0.3, 2.2**
ever socio-maladaptive problems							**2.5**	**2.0, 3.0**		
ever dependence	**2.6**	**1.4, 3.7**	**2.4**	**1.3, 3.6**	**2.2**	**1.0, 3.4**				
any dependence clinical feature			**1.9**	**0.5, 3.3**	**1.7**	**0.5, 2.9**				
onset of problem after 16										

Table 4.3.2.3. Variations of the association between childhood physical punishment and drinking outcomes among people who initiated drinking after 16 (with weight). Data from WMH-mC, 2001-2002.

if drk16=1, baddrk= bch28 indic bch28*indic age sexm paradprolm

	ALR with sex and age				GEE with sex and age				GEE with no covariates			
	OR	95% CI		*p* [1]	OR	95% CI		*p* [1]	OR	95% CI		*p* [1]
early try (<=12 yrs)	0.8	0.3	2.2		0.8	0.3	2.1		0.8	0.3	2.1	
early MTM drinking (<=19 yrs)	1.5	0.8	2.6	0.36	1.5	0.9	2.6	0.39	1.5	0.8	2.7	0.36
early socio-maladaptive problem (<=23 yrs)	**12.4**	**4.5**	**34.2**	**<.001**	**12.5**	**4.0**	**38.5**	**<.001**	**11.9**	**4.4**	**32.5**	**<.001**
early dependence problem (<=23 yrs)	**5.7**	**1.7**	**19.1**	**0.01**	**5.4**	**1.3**	**22.9**	**0.04**	**5.4**	**1.4**	**20.9**	**0.03**
early any problem (<=23 yrs)	**11.0**	**4.1**	**29.7**	**<.001**	**11.0**	**3.6**	**33.1**	**<.001**	**10.5**	**4.0**	**27.9**	**<.001**
ever heavier drinking	1.6	0.8	3.7	0.24	1.7	0.8	3.4	0.33	1.7	0.8	3.3	0.31
ever socio-maladaptive problems	**2.8**	**1.3**	**6.3**	**0.04**	3.0	1.3	6.7	0.05	**3.1**	**1.2**	**7.8**	**0.05**
ever dependence	**3.2**	**1.3**	**7.9**	0.06	3.1	1.1	8.6	0.05	**2.9**	**1.4**	**5.9**	**0.05**
any dependence clinical feature	2.9	1.0	8.9	0.11	2.9	1.1	7.8	0.07	2.8	1.1	7.1	0.07

[1]-p value of the product term of CPP•item indicator

Table 4.3.3.1. The association between childhood physical punishment and stages of drinking involvement without weight. Data from the WMH-mC, 2001-2002

	CPP	n	wt %	model 1			model 2: sex+age+paradprolm			model 3: model2+wmd+mmd		
				uOR	95% CI		aOR	95% CI		aOR	95% CI	
alcohol opportunity	no	1393	78.3									
	yes	235	88.5	**1.13**	**1.05**	**1.18**	**1.10**	**1.00**	**1.16**	1.08	0.98	1.15
trying given opportunity	no	1091	85.3									
	yes	208	91.4	1.07	0.99	1.12	1.05	0.95	1.11	1.05	0.95	1.10
drinking given tried	no	931	59.2									
	yes	190	72.1	**1.22**	**1.07**	**1.34**	1.13	0.96	1.28	1.10	0.92	1.26
ever heavy drinking among drinkers	no	537	21.3									
	yes	135	30.3	**1.41**	**1.03**	**1.86**	1.18	0.83	1.61	1.12	0.78	1.56
ever socio-maladaptive problems in drinkers	no	547	15.6									
	yes	137	28.4	**1.82**	**1.29**	**2.47**	**1.63**	**1.14**	**2.25**	**1.66**	**1.16**	**2.29**
ever dependence problems in drinkers	no	550	8.3									
	yes	137	17.9	**2.16**	**1.22**	**3.59**	**1.93**	**1.06**	**3.30**	**1.91**	**1.06**	**3.22**

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