BMJ Open

Effects of chronic disease diagnoses on alcohol consumption among elderly individuals: longitudinal evidence from China

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To cite: Hu Y, Chen Q, Zhang B. Effects of chronic disease diagnoses on alcohol consumption among elderly individuals: longitudinal evidence from China. *BMJ Open* 2022;**12**:e062920. doi:10.1136/bmjopen-2022-062920

► Prepublication history for this paper is available online. To view these files, please visit the journal online (http://dx.doi.org/10.1136/bmjopen-2022-062920).

Received 17 March 2022
Accepted 27 September 2022

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ABSTRACT

Objectives This study estimates the effect of chronic disease diagnoses (CDDs) on elderly Chinese individuals' alcohol consumption behaviour.

Subjects and participants Our analysis was applied to a publicly available dataset that covers 5724 individuals aged 50 or above and spans 15 years (2000–2015: six waves) from the China Health and Nutrition Survey.

Design The outcome variables are elderly individuals' weekly consumption of alcoholic beverages: beer, red wine, Chinese spirits and total alcohol intake. The explanatory variable of primary interest is the number of chronic diseases diagnosed (including hypertension, diabetes, stroke and myocardial infarction). Other covariates concern sample individuals' sociodemographic and health-related characteristics. A Chamberlain-Mundlak correlated random-effect Tobit model is adopted to simultaneously account for the clustering of 'zeros' in the outcome variable and endogeneity issues such as omitted variables and reverse causality.

Results Our estimation suggests that, on average, an additional chronic disease diagnosed by medical doctors reduced an elderly Chinese individual's weekly consumption of beer, red wine and Chinese spirits, respectively, by 1.49 (95% CI −2.85 to −0.13), 0.93 (95% CI −1.63 to −0.23) and 0.89 (95% CI −1.23 to −0.54) ounces. These effects translate into a reduction of 0.95 (95% CI −1.29 to −0.60) ounces in total weekly alcohol consumption and a reduction of 24% (95% CI −0.35 to −0.14) in the incidence of excessive drinking. Further explorations suggest that elderly Chinese individuals' alcohol consumption is most responsive to diabetes and stroke diagnoses, but the effects vary across different beverages. Moreover, males, rural residents, smokers and those living with non-drinkers respond to CDDs more strongly than their respective counterparts.

Conclusion While CDDs reduced alcohol consumption among elderly Chinese individuals, they failed to stop all heavy drinkers from excessive drinking. Relevant policies and measures are thus needed to urge heavy drinking patients to quit excessive drinking.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Compared with other observational studies, our application of the Chamberlain-Mundlak correlated random-effect Tobit model greatly helps address unobserved confounding.
⇒ We performed a series of robustness checks to support our main findings.
⇒ We performed informative subgroup analyses.
⇒ One key limitation of our study is that our data only contain information on four chronic conditions: myocardial infarction, diabetes, hypertension and stroke.

INTRODUCTION

Whereas alcohol consumption has important social and cultural functions, heavy drinking is considered one of the world's largest public health threats.[1] Not only may heavy drinking lead to devastating incidents such as car crashes and domestic violence,[2–6] but it is also strongly associated with the incidences of many chronic diseases, including diabetes, hypertension, myocardial infarction, stroke, among others.[7–11] Globally, alcohol consumption contributes to more than 3 million deaths each year and accounts for over 5% of the global burden of diseases and injuries.[1]

However, despite the increasing awareness of the adverse effects of heavy drinking and the considerable effort devoted by governments worldwide to curbing heavy drinking, alcohol consumption has been rising in many countries. China is no exception. Chinese residents' per capita alcohol consumption increased rapidly from 4.1 L in 2005 to 7.2 L in 2016,[1] leading many scholars to call for actions to control excessive drinking in China.[12] In such a context, an adequate understanding of who consumes alcohol (heavily) in China, what affects Chinese residents' alcohol consumption, and, more importantly, what may reduce (excessive) drinking, will undoubtedly help inform China's health policy.

One important factor is consumer health. Unfortunately, while there have been numerous studies on the impacts of alcohol consumption on one's health,[2 3 7–11] studies

that examine the effects of health on alcohol consumption have been limited, especially in China. The present study attempts to fill this gap by examining how chronic disease diagnoses (CDDs) affect elderly Chinese individuals' alcohol consumption. The role CDDs play is of particular interest, given the rapidly increasing incidences of chronic diseases in China[13 14]—for example, from 2015 to 2020, the prevalence of hypertension among Chinese adults increased from 25.2% to 28%, and that of diabetes from 9.75% to 12.2%.[15] If CDDs fail to serve as a wake-up call for drinkers to stop (heavy) drinking, effective early interventions may need to be carefully designed (to stop heavy drinking before one develops severe chronic conditions). One might argue that early interventions should always be implemented. Yet the fact that there are still many heavy drinkers in China and the widespread chronic diseases there suggest that early interventions targeting the general population are either very costly or not very effective. The addictive nature of alcohol consumption adds a layer of complexity to this issue. If drinkers keep on drinking even after developing chronic diseases, additional medicine and treatments may be needed to reduce the adverse effects of alcohol consumption on their health, which will certainly increase the costs of healthcare.

Our analysis draws on data from the China Health and Nutrition Survey (CHNS), a longitudinal household survey covering more than 10 provinces in China. The analytical sample covers 5724 individuals aged 50 or above and spans 15 years (2000–2015: six waves). To account for the fact that many respondents do not consume alcohol, which may lead to a 'zero-inflation' issue, and to allow for the correlation between unobserved individual fixed factors (eg, genetic traits) and observed explanatory variables, we adopt a Chamberlain-Mundlak correlated random-effect (CRE) Tobit model in the analysis.[16] The longitudinal structure of the CHNS data also helped address identification issues such as reverse causality and omitted variables to a large extent. We found that while CDDs reduced alcohol consumption among elderly Chinese individuals, they failed to stop all heavy drinkers from excessive drinking.

METHODS

Patient and public involvement

No patient was involved in this study.

Data

Our analysis draws on data from the CHNS (https://www.cpc.unc.edu/projects/china),[17] a longitudinal survey project jointly designed, implemented and managed by the Carolina Population Center at the University of North Carolina at Chapel Hill and the National Institute of Nutrition and Health at the Chinese Center for Disease Control and Prevention. Based on a stratified random sampling framework, the first wave of the CHNS, conducted in 1989, selected a sample of over 16 000 individuals from approximately 3800 households in nine provinces (Liaoning, Heilongjiang, Jiangsu, Shandong, Henan, Hubei, Hunan, Guangxi and Guizhou) that vary substantially in geographical features, economic conditions, public resources and health indicators. Follow-up waves were conducted in 1991, 1993, 1997, 2000, 2004, 2006, 2009, 2011 and 2015. Three municipal cities (Beijing, Shanghai and Chongqing) joined the project in 2011.

The CHNS collected rich information on sample individuals' sociodemographic characteristics, food consumption, nutrition intakes, health status, livelihoods and healthcare conditions of their residing communities (ie, rural villages and urban districts).

Most important for our study, the CHNS started to collect information on respondents' alcohol consumption in 1993 and that on CDDs in 1997. (The CHNS started to collect information on alcohol consumption in 1991, but only on individuals' drinking frequency. Information on the amounts of beer, red wine and spirits consumption was not collected until 1993. The project started to collect information on hypertension diagnoses in 1993 and on diabetes, stroke and myocardial infarction diagnoses in 1997.)

Sample restrictions

Given the purpose of our study, we applied several restrictions to form the analytical sample. First, we excluded data collected before 2000 to ensure that all observations have information on alcohol consumption and CDDs lagged for one time period (—lagged values are used for robustness checks, as detailed below). Second, to exploit as much longitudinal information as possible, we kept only observations from the nine original project provinces. Finally, we excluded respondents under the age of 50 in 2000 because the incidences of chronic diseases were very low for individuals under 50 in the data. The above procedures yielded an unbalanced panel of 21 013 individual-year observations for 5724 individuals residing in 228 communities, appearing at least in one wave (out of six in total) between 2000 and 2015.

Variables

Outcome variables

The outcome variables of primary interest are measures of elderly individuals' weekly consumption of alcoholic beverages in the previous year: beer, red wine, Chinese spirits ('baijiu') and total alcohol intake. The first three, reported directly by the respondents, are readily available in the CHNS data but measured in different units. Beer and red wine intakes were measured in bottles and Chinese spirits in *liang* (—1 *liang*=1/20 kg). For ease of comparison, we converted all three alcohol intake variables into multiples of (international standard) ounces (—one international ounce=30 mL). The last variable, total weekly alcohol consumption, is constructed based on the first three: beer and red wine consumption were

first converted into their spirits equivalents; then, all three sum to yield total alcohol intake.

Key explanatory variables

The explanatory variable of primary interest is the number of chronic diseases diagnosed. Given data availability, we focus on four chronic diseases: hypertension, diabetes, stroke and myocardial infarction. It would be desirable to include other chronic conditions, such as gastritis, hepatitis and non-alcoholic fatty liver disease (NAFLD), in the analysis. Unfortunately, information on other chronic diseases is not available in the CHNS data (except that on cancer, but it was not available until 2011). In some analyses below, separate indicators of the diagnoses of specific chronic conditions are used to gain more insights into the CDDs–alcohol consumption relationship.

Other covariates

Other covariates in our empirical models developed below were chosen based on standard (health economic) theory and previous empirical findings. The first set of covariates concerns sample individuals' demographic characteristics. A male dummy is included to capture potential gender differences in alcohol consumption behaviour. Also included are age and age-squared to capture life-cycle patterns. Since marital status is found to affect alcohol consumption,[18–20] we also include a dummy for having a spouse in the model. The second set concerns sample individuals' socioeconomic status. Since both standard economic theory and previous empirical studies[21–23] suggest that income is an essential determinant of alcohol consumption, we include individuals' annual household income per capita in the models. Also included are years of education,[24–26] work status,[20 27] and a dummy for rural residents,[28] to capture potential differences in lifestyles associated with these factors. The final set of covariates is health-related. Height is included since individuals with larger body sizes naturally consume more food and beverages. We also include sample individuals' self-reported health status, a dummy for feeling 'good' or 'very good' (—other values include 'very poor', 'poor' and 'fair'), to measure their subjective health.[29 30] Since health insurance coverage has been found to affect the insured's health behaviour and chronic disease outcomes,[31–33] we also include a dummy for health insurance coverage in the analysis. Moreover, many studies have found that drinking and smoking are closely related[21 29 34]; we follow this strand of literature and include a dummy for smokers in the analysis. Finally, we include a dummy for whether other household members drink to capture family drinking culture.

Missing information (eg, on education or disease diagnosis) was filled in with available information provided in other waves first. For example, if a respondent was diagnosed with hypertension in 2004, we can infer that the missing hypertension diagnosis status for this respondent in 2006 should be imputed as 'had been diagnosed with hypertension'. The remaining missing values were filled in with sample group means within province-community-gender-age cells. The missing values of other variables (eg, education and height) were handled similarly.

Statistical method

To guide empirical modelling, consider first a standard statistical model that links one's alcohol consumption and its determinants:

$$A_{it} = \beta_0 + \beta_1 D_{it} + \boldsymbol{z}_{it}\boldsymbol{\beta}_2 + u_{it} \quad (1)$$

where the outcome variable, A_{it} ('alcohol'), is the amount of alcoholic content individual i consumes per week in year t (—in some analyses below, A_{it} is replaced with weekly intakes of specific alcoholic beverages). The explanatory variable of primary interest, D_{it} ('diagnosis'), is the total number of chronic conditions (diabetes, hypertension, myocardial infarction and stroke combined; as noted above, the choice of chronic diseases to study is constrained by data availability) that an individual i had ever been diagnosed with by medical professionals by time t, that is, $D_{it} = \sum_{j=1}^{4} d_{jit}$, where d_{jit} is a dummy for the diagnosis of the jth chronic disease mentioned above (—in some analyses below, D_{it} is replaced with d_{jit}). The vector $\boldsymbol{z}_{it}$ represents a set of other observed determinants of one's drinking behaviour (discussed in the Variables section). The error term u_{it} captures the influence of unobserved factors.

If equation (1) is correctly specified, the effect of CDDs, captured by β_1, can be consistently estimated by ordinary least-squares (OLS) techniques. However, several potential estimation issues may lead to misleading estimates of β_1. First, many sample individuals do not consume alcoholic beverages, which applies to 68% of all individual-wave cases in the analytical sample. Failing to account for the clustering of 'zeros' in the outcome variable may bias the estimates of the β's. A natural approach to handling this issue is to recast the above model into a Type-I Tobit (corner-solution) framework[35]:

$$A_{it} = \max\left(0, \boldsymbol{x}_{it}\boldsymbol{\beta} + \varepsilon_{it}\right), \; t = 1, \; 2, \ldots, T \quad (2)$$

where $\boldsymbol{x}_{it} = \left(1, D_{it}, \boldsymbol{z}_{it}\right)$ denotes the set of observed variables in equation (1), $\boldsymbol{\beta}$ is the corresponding coefficient vector and $\varepsilon_{it}|\boldsymbol{x}_{it} \sim \text{Normal}\left(0, \sigma_{\varepsilon}^2\right)$. The parameters in equation (2), $\boldsymbol{\beta}$ and σ_{ε}, can be estimated by maximum likelihood estimation (MLE). Given our main aim to test whether CDDs urge elderly individuals to reduce alcohol intake, our interest centres on estimating the marginal effect for the *uncensored* population (ie, those with $A_{it} > 0$):

$$\partial E\left(A_{it}|A_{it} > 0, \boldsymbol{x}_{it}\right) / \partial D_{it} = \beta_1 \left[1 + \frac{d\lambda}{dc}\left(\frac{\boldsymbol{x}_{it}\boldsymbol{\beta}}{\sigma_{\varepsilon}^2}\right)\right] \quad (3)$$

where $\lambda = \frac{\phi(c)}{\Phi(c)}$, $c = \frac{\boldsymbol{x}_{it}\boldsymbol{\beta}}{\sigma_{\varepsilon}^2}$ and ϕ and Φ are the probability density function and cumulative distribution of the standard normal distribution, respectively. Since the marginal effect of interest (3) is a function of $\boldsymbol{\beta}$ and σ_{ε}, consistent estimates of these parameters are needed to recover this effect.

The second issue is the potential existence of unobserved individual-specific fixed factors (eg, genetic fitness) that simultaneously affect one's alcohol consumption and health status, causing a spurious correlation between A_{it} and D_{it}. With the presence of such fixed factors (denoted f_i), equation (2) becomes:

$$A_{it} = \max\left(0, \boldsymbol{x}_{it}\boldsymbol{\beta} + f_i + \varepsilon_{it}\right),\ t = 1,\ 2, \ldots, T \qquad (4)$$

where $\varepsilon_{it}|\boldsymbol{x}_i, f_i \sim \text{Normal}\ (0, \sigma_\varepsilon^2)$, with $\boldsymbol{x}_i$ containing $\boldsymbol{x}_{it}$ for all t. The possible correlation between f_i and D_{it} (part of $\boldsymbol{x}_{it}$) may generate biased estimates of $\boldsymbol{\beta}$ and (thus) the marginal effects of interest (3).

In the linear case (equation 1), the individual-specific fixed effects f_i can be easily controlled by applying the standard fixed-effects (FE) approach. In a Tobit setup, however, the standard FE approach will yield inconsistent MLE of σ_ε^2, even if it can consistently estimate $\boldsymbol{\beta}$.[16] This is unfortunate because a consistent estimate of σ_ε^2 is needed to compute the marginal effects of interest.[3] As an alternative, we adopt a Chamberlain-Mundlak CRE specification,[35] which allows f_i and $\boldsymbol{x}_i$ to be correlated and seeks to capture the former explicitly in the model. Formally, the Chamberlain-Mundlak CRE Tobit model assumes that

$$f_i|\boldsymbol{x}_i \sim \text{Normal}\left(\psi + \bar{\boldsymbol{x}}_i\boldsymbol{\eta}, \sigma_r^2\right) \qquad (5)$$

where $\bar{\boldsymbol{x}}_i$ is the average of $\boldsymbol{x}_{it}$ across all T periods, and σ_r^2 is the variance of r_i in the equation $f_i = \psi + \bar{\boldsymbol{x}}_i\boldsymbol{\eta} + r_i$ (— $\boldsymbol{x}_{it}$ may include time-invariant variables, which play the role of $\bar{\boldsymbol{x}}_i$ in the CRE specification).

Given the Chamberlain-Mundlak formulation (5), equation (4) becomes

$$A_{it} = \max\left(0, \psi + \boldsymbol{x}_{it}\boldsymbol{\beta} + \bar{\boldsymbol{x}}_i\boldsymbol{\eta} + r_i + \varepsilon_{it}\right), t = 1,\ 2, \ldots, T \ (6)$$

where $\varepsilon_{it}|\boldsymbol{x}_i, r_i \sim \text{Normal}\ (0, \sigma_\varepsilon^2)$ and $r_i|\boldsymbol{x}_i \sim \text{Normal}\ (0, \sigma_r^2)$. The 'trick' here is that the equation $f_i = \psi + \bar{\boldsymbol{x}}_i\boldsymbol{\eta} + r_i$ translates the individual-level *fixed effect* (f_i) into a *random effect* (r_i). Now, equation (6), combined with the two normality assumptions immediately below it, is a standard random-effects (RE) Tobit model with a set of observed covariates ($\bar{\boldsymbol{x}}_i$) added. As such, the parameters in equation (6), ψ, $\boldsymbol{\beta}$, $\boldsymbol{\eta}$, σ_ε^2 and σ_r^2, as well as the marginal effects defined based on these parameters (3), can be consistently estimated by MLE. Note also that conditional on $\bar{\boldsymbol{x}}_i$, the variation in $\boldsymbol{x}_{it}$ exploited to identify $\boldsymbol{\beta}$ is the time-varying part of it. Since $\bar{\boldsymbol{x}}_i$ is held fixed, the time-varying variation in $\boldsymbol{x}_{it}$ plays a role analogous to that of 'deviations from means' resulting from a 'within transformation' in linear FE models.

The third issue concerns the potential reverse causality from alcohol consumption (A_{it}) to chronic disease diagnosis (D_{it}). As has been widely documented in the literature, excessive drinking can cause serious harm to one's health. With such reverse causality, the statistical relationship between individuals' alcohol consumption and their diagnosed chronic conditions observed in the same period t may reflect this reverse causality rather than the actual effect of CDDs on alcohol consumption. To address this issue, we replace D_{it} with its lagged values observed in the previous period, $D_{i,t-1}$, in some analyses. Since whether a person drinks in a given period should not affect his/her health status in the previous period, this strategy effectively circumvents the reverse-causality problem (—of course, if the duration from t–1 to t is long, this approach may lead to some notable changes in the estimates and the related interpretation). Since the reverse-causality issue may also apply to one's subjective health status, we also replace this variable with its lagged values in the model as a robustness check.

The final issue is the potential existence of other omitted determinants of alcohol consumption. While the Chamberlain-Mundlak CRE specification helps take care of unobserved fixed factors (f_i), there is no guarantee that it captures the influence of all unobserved confounders. One (partial) solution is to add lagged values of the outcome variable, $A_{i,t-1}$, as an additional control variable in the model. Since $A_{i,t-1}$ is a function of all relevant factors, observed or unobserved, in period t–1, it should reasonably capture the influence of all omitted factors. It is worth noting that $A_{i,t-1}$ is also a 'corner-solution' outcome at t–1, which suggests that the censored and uncensored parts of it may have different effects on A_{it}. Thus, following Wooldridge's recommendation,[35] we include a dummy for censored observations at t–1, $c_{i,t-1}$ ('censored') and a term $(1-c_{i,t-1})\times A_{i,t-1}$ in the model to capture both effects—a more general approach, detailed in Wooldridge,[35] is to include both $A_{i,t-1}$ and A_{i0}, the initial value of A_{it}, in the model (which yields very similar results—not reported in this article but made available on request). Note also that conditional on $A_{i,t-1}$, which summarises one's disease-diagnosis and drinking histories up to t–1, identification of the CDD effect comes from the variation in D_{it} between periods t–1 and t. Admittedly, this identification mechanism is not immune to confounding factors varying between t–1 and t. Yet unlike acute diseases, which are usually driven by short-time shocks, chronic diseases are more likely to be caused by factors accumulated for an extended period (—this feature also alleviates concerns about reserve causality running from alcohol consumption to chronic disease diagnosis). Thus, it seems safe to assume that D_{it} is uncorrelated with unobserved confounding factors varying between t–1 and t.

Finally, the above framework can be easily adapted to estimate the effect of CDDs on the likelihood of (stopping) excessive drinking (E_{it})—E_{it}=1 if the daily intake of pure alcohol exceeded 25 g for an adult male or 15 g for an adult female and 0 otherwise—and the likelihood of (quitting) alcohol drinking altogether. A Probit model with the Chamberlain-Mundlak CRE specification can be applied to the analytical sample:

$$\text{Prob}\left(E_{it} = 1|\boldsymbol{x}_i\right) = \Phi\left(\psi^0 + \boldsymbol{x}_{it}\boldsymbol{\beta}^0 + \bar{\boldsymbol{x}}_i\boldsymbol{\eta}^0\right) \qquad (7)$$

Again, we are interested in estimating the marginal effect of an additional CDD on the probability of heaving drinking: $\partial \text{Prob}\left(E_{it}=1|\boldsymbol{x}_i, r_i\right)/\partial D_{it}=\beta_1\phi\left(\frac{\psi^0+\boldsymbol{x}_{it}\boldsymbol{\beta}^0+\bar{\boldsymbol{x}}_i\boldsymbol{\eta}^0}{1+\sigma_r^2}\right)$.

All estimations discussed above were performed in Stata SE V.14.0.

RESULTS

Descriptive statistics

Table 1 presents the profile of the individuals in the analytical sample by their drinking status (drinkers vs non-drinkers) at the time of the survey—a survey

Table 1 Summary statistics

	Full sample		Drinkers		Non-drinkers	
Variable	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Whether drinking (dummy, =1 if yes)	0.28	0.45	1	0	0	0
Weekly beer intake (oz) (current)	4.93	29.69	18.43	55.51	0	0
Weekly red wine intake (oz) (current)	0.77	7.88	2.86	15.09	0	0
Weekly Chinese spirits intake (oz) (current)	4.70	14.74	17.32	24.44	0	0
Total alcohol consumption (oz) (current)	5.54	15.88	20.29	25.29	0	0
Total alcohol consumption (oz) (lagged)	6.27	16.99	18.34	25.74	1.82	8.80
Excessive drinking (dummy, =1 if consuming ≥25 g (15 g) pure alcohol daily for males (females))	0.15	0.36	0.44	0.50	0	0
Hypertension (current)	0.34	0.48	0.29	0.45	0.36	0.48
Diabetes (current)	0.08	0.27	0.05	0.22	0.09	0.29
Stroke (current)	0.06	0.24	0.04	0.19	0.06	0.24
Myocardial infarction (current)	0.04	0.19	0.02	0.15	0.04	0.19
Hypertension diagnosed (lagged)	0.26	0.44	0.21	0.41	0.28	0.45
Diabetes diagnosed (lagged)	0.06	0.23	0.04	0.19	0.06	0.24
Stroke diagnosed (lagged)	0.04	0.19	0.02	0.15	0.04	0.20
Myocardial infarction (lagged)	0.02	0.15	0.01	0.12	0.03	0.16
Number of chronic diseases diagnosed (current)	0.44	0.69	0.36	0.60	0.49	0.73
Number of chronic diseases diagnosed (lagged)	0.33	0.61	0.26	0.53	0.37	0.65
Male (dummy, =1 if yes)	0.48	0.50	0.84	0.36	0.34	0.47
Self-reported health status (current) (dummy, =1 if self-reported health is 'good' or 'very good')	0.45	0.50	0.52	0.50	0.41	0.49
Self-reported health status (lagged)	0.49	0.50	0.56	0.50	0.46	0.50
Whether smoking (dummy, =1 if yes)	0.26	0.44	0.53	0.50	0.16	0.37
Other drinkers in the household (dummy, =1 if yes)	0.46	0.50	0.44	0.50	0.46	0.50
Age (years)	67.75	9.31	65.18	8.42	68.04	8.96
$Age^2/100$	46.77	13.08	43.19	11.34	47.09	12.51
Education (years)	4.82	4.35	6.04	4.34	4.43	4.32
Annual income (yuan)	26 626	41 910	26 238	41 378	26 496	41 116
Spouse (dummy, =1 if having a spouse)	0.75	0.43	0.84	0.36	0.72	0.45
Health insurance (dummy, =1 if having medical insurance coverage	0.71	0.45	0.70	0.46	0.71	0.45
Working (dummy, =1 if currently working)	0.31	0.46	0.44	0.50	0.25	0.44
Rural (dummy, =1 if the respondent resides in a rural area)	0.64	0.48	0.63	0.48	0.64	0.48
Minority (dummy, =1 if the respondent is of ethnic minority)	0.13	0.33	0.14	0.35	0.12	0.33
Height (cm)	158.00	9.12	162.49	7.99	156.26	8.93
Number of individuals	5724		2249		4591	
Number of individual-wave observations	21 012		5510		14 364	

Source: Author's calculation using China Health and Nutrition Survey data.

wave-individual observation is defined as a 'drinker' if positive alcohol consumption is reported for this observation. Consistent with the spatial distribution of survey sites, about two-thirds of the observations came from rural areas. Despite the average age of 68, nearly one-third of the observations reported 'currently working' during the survey period. Presumably because of their relatively old age, only 75% of the observations reported 'having a spouse'. The average respondent had about 5 years of education and was living in a household with an annual income of slightly less than 27 000 yuan (≈US$3913 at 2000 prices). As a result of the rapid expansion of China's health insurance sector since the early 2000,[31 33 36] 71% of the observations had health insurance coverage at the time of the survey.

Regarding alcohol consumption, 28% of the wave-individual observations reported drinking. The average respondent consumed 5 ounces of beer, 0.8 ounces of red wine and 4.7 ounces of Chinese spirits per week, which amounted to a liquor content equivalent to 5.5 international unit ounces. Nearly 44% of the drinkers were heavy drinkers—heavy drinking is defined as consuming more than 25 g of pure alcohol per day for adult males and 15 g per day for adult females.[37] The average number of chronic diseases diagnosed (hypertension, diabetes, myocardial infarction and stroke combined) is 0.44 among sample individuals. Hypertension has the highest prevalence (34%), followed by diabetes (8%), stroke (6%) and myocardial infarction (4%).

A quick comparison between drinkers and non-drinkers suggests that the incidences of chronic diseases are, in general, higher among non-drinkers, which seems counterintuitive at first glance. However, this observation may indeed suggest that CDDs cause the patients to reduce alcohol consumption. To sort out the direction of causality, we use the Chamberlain-Mundlak CRE Tobit model discussed above to control for potential confounding factors in the analysis.

Main estimation results

Table 2, panel B, presents the main results of estimating the Chamberlain-Mundlak CRE Tobit model (equation 6) for elderly Chinese individuals' weekly consumption of three alcoholic beverages (beer, red wine and Chinese spirits) and weekly total alcohol intake, based on all available observations (an unbalanced panel). For comparison purposes, the results of traditional RE Tobit models are presented in panel A. All columns report the marginal effects for the uncensored population $(\partial \hat{E}\left(A_{it}|A_{it}>0, \boldsymbol{z}_{it}, \bar{\boldsymbol{x}}_i\right)/\partial x_k$, for a given variable $x_k)$.

Three notable findings emerge from the table. First, many $\bar{\boldsymbol{x}}_i$ variables (ie, within-individual means) in the Chamberlain-Mundlak CRE Tobit specification (table 2, panel B) have statistically significant predictive power (at the 5% level) for elderly Chinese individuals' alcohol consumption, suggesting that the individual-specific unobserved effects (f_i) are indeed correlated with observed variables $(\boldsymbol{x}_{it})$, at least through some $\bar{\boldsymbol{x}}_i$'s. As such, the Chamberlain-Mundlak CRE Tobit specification represents a more suitable modelling device than the traditional RE Tobit (table 2, panel A). (The traditional RE Tobit models reported in table 2 control for province dummy variables. Traditional RE Tobit models that control for community dummy variables yield quite similar estimates of the effects of CDDs, but are less significant. Results are not reported here but are available on request.) Comparisons between panels A and B suggest that the traditional RE Tobit tends to underestimate the effects of CDDs.

Second, all columns in table 2 suggest that the number of chronic conditions diagnosed has a statistically significant and negative effect on elderly individuals' alcohol consumption, but the effect size is modest. CRE Tobit estimates (panel B) suggest that, on average, an additional chronic condition diagnosed reduces a drinker's weekly beer intake, red wine intake and spirits intake, respectively, by 1.49 (95% CI −2.85 to −0.13), 0.93 (95% CI −1.63 to −0.23) and 0.89 ounces (95% CI −1.23 to −0.54). The reduction in red wine intake appears to be modest in absolute terms. But given the small amount of red wine intake (2.86 ounces) among drinkers (table 1), the estimate in column (2) implies a sizeable reduction in red wine intake in proportional terms (32.5%), much larger than those in beer (8.1%) and spirits intakes (5.1%). Yet, in any case, the estimated effect of CDDs is small in practical terms. Since the average drinker consumes more than 20 ounces of alcoholic beverages per week (table 1), the reduction in total weekly alcohol consumption (0.95 ounces (95% CI −1.29 to −0.60)) induced by one additional CDD amounts to a mere 4.7% reduction.

Nevertheless, CDDs exert a sizeable effect on the likelihood of stopping excessive drinking and quitting drinking. Table 3 reports Chamberlain-Mundlak CRE Probit estimates of the effects of CDDs (equation 7). The results indicate that an additional CDD is associated with a reduction in the likelihood of quitting excessive drinking by 24.3% (95% CI −0.35 to −0.14) (column 2) and a reduction in the likelihood of quitting drinking by 19.7% (95% CI −0.28 to −0.11) (column 4). Again, the traditional RE Probit specification (columns 1 and 3) yields estimates of smaller sizes.

Finally, the effects of many other explanatory variables differ greatly across alcoholic beverages, depicting different demands for different products. Specifically, CRE Tobit estimates (table 2, panel B) show that males consume substantially more of all three beverages than females, but the gender gap in beer intake (15.8 ounces (95% CI 13.62 to 17.95)) is much wider than those in red wine (2.6 ounces (95% CI 1.48 to 3.70)) and spirits (8.1 ounces (95% CI 7.46 to 8.80)) intakes. For all three beverages, the age profile has an inverted-U shape, but that for beer is the steepest among the three. Smokers drink significantly more beer (by 4.4 ounces (95% CI 2.74 to 6.14)) and spirits (by 2.3 ounces (95% CI 1.92 to 2.76)), but not red wine, than non-smokers. The presence of other drinkers in the household induces more consumption of

Table 2 Tobit estimates of associations between the number of chronic diseases diagnosed and weekly alcohol consumption (age≥50, unbalanced panel)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Outcome variables (unit: ounces/week)	**Beer**	**Red wine**	**Chinese spirits**	**Total**	**Beer**	**Red wine**	**Chinese spirits**	**Total**
Estimator	**(A) Standard random-effects Tobit**				**(B) Chamberlain-Mundlak Correlated random-effects Tobit**			
Number of chronic diseases diagnosed	−1.273***	−0.639***	−0.765***	−0.754***	−1.488**	−0.927***	−0.886***	−0.946***
	(0.465)	(0.243)	(0.134)	(0.134)	(0.694)	(0.357)	(0.174)	(0.177)
Male (=1 if yes)	17.409***	2.969***	8.668***	8.701***	15.784***	2.592***	8.133***	8.094***
	(1.049)	(0.530)	(0.313)	(0.309)	(1.106)	(0.566)	(0.341)	(0.338)
Self-reported health status	1.237**	0.359	0.713***	0.779***	0.496	−0.242	0.471***	0.501***
	(0.523)	(0.267)	(0.134)	(0.137)	(0.584)	(0.296)	(0.144)	(0.147)
Smoking (=1 if yes)	4.216***	0.173	2.903***	2.984***	4.440***	0.113	2.342***	2.524***
	(0.629)	(0.354)	(0.174)	(0.180)	(0.867)	(0.492)	(0.213)	(0.222)
Other drinkers in the household (=1 if yes)	1.531***	0.931***	1.011***	1.186***	1.895***	0.991***	0.787***	0.992***
	(0.586)	(0.301)	(0.156)	(0.159)	(0.731)	(0.374)	(0.179)	(0.183)
Age (years)	0.318	0.415*	0.254**	0.242**	0.979*	0.553**	0.299**	0.365***
	(0.484)	(0.224)	(0.118)	(0.119)	(0.591)	(0.274)	(0.139)	(0.141)
$Age^2/100$	−0.555	−0.317*	−0.243***	−0.238***	−0.940**	−0.421**	−0.298***	−0.339***
	(0.359)	(0.163)	(0.086)	(0.087)	(0.442)	(0.199)	(0.101)	(0.102)
Years of education (years)	0.122	0.128***	−0.024	0.013	0.096	0.099**	−0.016	0.014
	(0.083)	(0.043)	(0.025)	(0.025)	(0.086)	(0.044)	(0.025)	(0.026)
Annual income (yuan) in log	0.458*	0.307**	0.018	0.045	0.377	0.262	0.009	0.023
	(0.264)	(0.141)	(0.068)	(0.069)	(0.311)	(0.165)	(0.075)	(0.077)
Has spouse (=1 if yes)	−1.104	−0.334	−0.426*	−0.250	−2.320*	−0.585	0.075	0.063
	(0.863)	(0.427)	(0.234)	(0.237)	(1.351)	(0.630)	(0.328)	(0.333)
Insurance coverage (=1 if yes)	3.174***	1.245***	−0.651***	−0.449**	2.732***	0.509	−0.471**	−0.436*
	(0.766)	(0.419)	(0.195)	(0.200)	(0.944)	(0.524)	(0.228)	(0.234)
Working (=1 if yes)	2.029***	0.146	0.459***	0.627***	−0.466	−0.337	0.133	0.150
	(0.651)	(0.357)	(0.170)	(0.175)	(0.765)	(0.420)	(0.189)	(0.195)
Rural (=1 if yes)	−2.053***	−2.696***	0.740***	0.111	−3.103***	−2.784***	0.493**	−0.186
	(0.738)	(0.389)	(0.234)	(0.234)	(0.771)	(0.409)	(0.246)	(0.246)
Minority (=1 if yes)	−1.704	0.476	0.876**	0.781**	−1.897	0.460	0.681*	0.593
	(1.326)	(0.728)	(0.374)	(0.385)	(1.316)	(0.727)	(0.374)	(0.384)
Height (cm)	0.091*	0.027	0.006	0.017	0.104**	0.023	0.012	0.022
	(0.052)	(0.028)	(0.015)	(0.015)	(0.052)	(0.028)	(0.015)	(0.015)
Within-individual means ($\bar{x}_i$)								

Continued

Table 2 Continued

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Outcome variables (unit: ounces/week)	**Beer**	**Red wine**	**Chinese spirits**	**Total**	**Beer**	**Red wine**	**Chinese spirits**	**Total**
Estimator	**(A) Standard random-effects Tobit**				**(B) Chamberlain-Mundlak Correlated random-effects Tobit**			
Number of chronic diseases diagnosed					0.930	0.793	0.605**	0.795***
					(0.969)	(0.487)	(0.278)	(0.278)
Self-reported health status					3.582***	2.913***	1.831***	2.002***
					(1.298)	(0.684)	(0.393)	(0.395)
Smoking					−0.540	0.178	1.535***	1.257***
					(1.271)	(0.715)	(0.367)	(0.378)
Other drinkers in the household					−1.343	−0.253	0.767**	0.590
					(1.238)	(0.643)	(0.367)	(0.370)
Age					−0.902	−0.178	−0.089	−0.230
					(0.842)	(0.397)	(0.228)	(0.228)
$Age^2/100$					0.684	0.158	0.113	0.216
					(0.645)	(0.297)	(0.171)	(0.171)
Annual income (yuan) in log					0.411	0.007	0.031	0.085
					(0.593)	(0.310)	(0.171)	(0.173)
Has spouse					1.997	0.417	−0.930*	−0.596
					(1.813)	(0.881)	(0.478)	(0.483)
Has insurance coverage					1.160	1.844**	−0.579	0.045
					(1.556)	(0.849)	(0.437)	(0.444)
Working					8.756***	1.790**	1.625***	2.434***
					(1.471)	(0.815)	(0.444)	(0.450)
Province fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of individual-wave observations	18 707	18 705	18 707	18 707	18 707	18 705	18 707	18 707
Log pseudolikelihood	−13160.66	−4693.10	−24091.19	−27933.96	−13133.40	−4677.08	−24054.45	−27890.94

Marginal effects for the non-censored population $\left(\partial \hat{E}\left(A_{it} | A_{it} > 0,\ \boldsymbol{x}_{it}\right) / \partial x_k\right)$ are reported.
SEs, adjusted for intra-individual clustering, are reported in parentheses.
*p<0.1, **p<0.05, ***p<0.01.

all three beverages, but the effect is larger for beer than for red wine and spirits. Rural residents consume more spirits but less beer and red wine than urban residents. Interestingly, health insurance coverage encourages beer consumption but discourages spirits consumption.

Robustness checks

The above findings are convincing only to the extent that the Chamberlain-Mundlak CRE specifications are valid. This section performs several checks to assess the performance of CRE Tobit in addressing the estimation issues

Table 3 Probit estimates of associations between the number of chronic diseases diagnosed and incidences of drinking or excessive drinking (age≥50, unbalanced panel)

Outcome variables	(1) Drinking	(2)	(3) Excessive drinking	(4)
Estimator	Probit	Chamberlain-Mundlak CRE Probit	Probit	Chamberlain-Mundlak CRE Probit
Number of chronic diseases diagnosed	−0.162***	−0.197***	−0.132***	−0.243***
	(0.031)	(0.043)	(0.039)	(0.053)
Male (=1 if yes)	1.930***	1.810***	1.604***	1.471***
	(0.077)	(0.083)	(0.093)	(0.098)
Self-reported health status (=1 if 'good/very good')	0.107***	0.020	0.184***	0.108**
	(0.033)	(0.036)	(0.039)	(0.043)
Smoking (=1 if yes)	0.802***	0.757***	0.680***	0.553***
	(0.044)	(0.055)	(0.050)	(0.064)
Other drinkers in the household (=1 if yes)	0.374***	0.361***	0.153***	0.124**
	(0.039)	(0.045)	(0.045)	(0.054)
Age (years)	0.045	0.099***	0.069**	0.125***
	(0.028)	(0.034)	(0.035)	(0.043)
Age (years) squared/100	−0.047**	−0.082***	−0.060**	−0.095***
	(0.021)	(0.024)	(0.025)	(0.031)
Years of education (years)	0.007	0.003	−0.010	−0.011
	(0.006)	(0.006)	(0.007)	(0.007)
Annual income (yuan) in log	0.009	−0.003	0.018	0.007
	(0.016)	(0.019)	(0.020)	(0.023)
Has spouse (=1 if yes)	−0.088	−0.053	−0.074	−0.013
	(0.055)	(0.079)	(0.067)	(0.098)
Insurance coverage (=1 if yes)	−0.023	−0.066	−0.065	−0.045
	(0.048)	(0.057)	(0.056)	(0.068)
Working (=1 if yes)	0.157***	0.047	0.144***	0.046
	(0.042)	(0.047)	(0.049)	(0.057)
Rural (=1 if yes)	−0.146***	−0.203***	0.129**	0.088
	(0.055)	(0.058)	(0.064)	(0.068)
Minority (=1 if yes)	0.144	0.117	0.225**	0.190*
	(0.092)	(0.092)	(0.100)	(0.101)
Height (cm)	0.005	0.006*	0.001	0.002
	(0.004)	(0.004)	(0.004)	(0.004)
Within-individual means ($\bar{x}_i$)				
Number of chronic diseases diagnosed		0.156**		0.314***
		(0.066)		(0.078)
Self-reported health status		0.588***		0.522***
		(0.094)		(0.110)
Smoking		0.139		0.345***
		(0.092)		(0.104)
Other drinkers in the household		0.001		0.067
		(0.089)		(0.103)
Age		−0.100*		−0.103

Continued

Table 3 Continued

Outcome variables	(1) Drinking	(2)	(3) Excessive drinking	(4)
Estimator	Probit	Chamberlain-Mundlak CRE Probit	Probit	Chamberlain-Mundlak CRE Probit
		(0.054)		(0.064)
Age^2/100		0.077*		0.075
		(0.040)		(0.048)
Annual income (yuan) in log		0.044		0.047
		(0.041)		(0.048)
Has spouse		−0.070		−0.104
		(0.114)		(0.138)
Insurance coverage		0.129		−0.096
		(0.106)		(0.124)
Working		0.570***		0.443***
		(0.108)		(0.124)
Province fixed effects	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes
No. individual-wave observations	18 703	18 703	18 707	18 707
Log pseudolikelihood	−7230.34	−7185.58	−4815.00	−4779.93

Marginal effects are reported. SEs, adjusted for intra-individual clustering, are reported in parentheses.
*p<0.1, **p<0.05, ***p<0.01.

discussed above. To keep the discussion focused, we only report results based on the model for total weekly alcohol consumption (—results for the three specific alcoholic beverages are available on request).

Column 1 of table 4 reproduces column 8 of table 2, providing the basis for comparisons. To address potential reverse causality from alcohol consumption to health, column 2 of table 4 replaces the number of chronic diseases diagnosed and (self-reported) health status with their respective one-period lagged measures. The estimate remains quite comparable, greatly alleviating the concern about reverse causality.

To address the potential concern of omitted variables, column (3) includes the lagged outcome measure ($A_{i,t-1}$) in the model, allowing the censored ($A_{i,t-1} = 0$) and uncensored ($A_{i,t-1} > 0$) parts to have different effects. The estimate becomes slightly smaller but still indicates a significantly negative impact of CDDs on alcohol consumption. The similarity in the estimates across these columns suggests that the benchmark CRE Tobit model (table 3, column 1) depicts the CDDs–alcohol consumption relationship reasonably well.

The third concern is that some of the control variables, such as smoking and working status, might be endogenous. To see how the inclusion of such variables affects our estimation results, we drop all potentially endogenous explanatory variables from the model in column (4). Reassuringly, the estimated effect of CDDs in this column remains very similar to the benchmark estimate in column (1).

The final concern is over the use of an unbalanced panel. Identification of parameters using an unbalanced panel exploits partly cross-individual variations in CDDs, which may be contaminated with unobserved individual-specific factors if the Chamberlain-Mundlak CRE specification fails to capture such factors. As a test, panel B restricts the sample to be a *balanced* panel, ensuring that identification relies entirely on time-varying variations. Reassuringly, the results are comparable to their counterparts reported in panel A, again suggesting that the benchmark Chamberlain-Mundlak CRE Tobit model (table 4, column 1) performs reasonably well. Therefore, we base most of the analyses below on this specification.

Further exploration: effects of the diagnoses of specific chronic diseases

Diagnoses of different chronic diseases may have different effects on alcohol consumption; thus, pooling different chronic conditions together in the analysis may mask important patterns. This section explores how the effects of the diagnoses of different chronic conditions might differ. Based on the benchmark Chamberlain-Mundlak CRE Tobit model, we replace the number of chronic diseases diagnosed (D_{it}) with dummies for the diagnoses of specific chronic conditions (d_{it}) and re-estimate the models.

Table 4 Chamberlain-Mundlak correlated random-effects Tobit estimates of associations between the number of chronic diseases diagnosed and weekly total alcohol consumption (aged 50 or above)

Outcome variable=total weekly alcohol intake (ounce)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(A) Unbalanced panel				(B) Balanced panel			
Number of chronic diseases diagnosed	−0.946***		−0.761***	−0.979***	−0.957***		−0.816***	−1.013***
	(0.177)		(0.215)	(0.176)	(0.248)		(0.276)	(0.254)
Number of chronic diseases diagnosed; 1-period lagged		−0.820***				−1.016***		
		(0.238)				(0.297)		
Total alcohol consumption, 1-period lagged ($A_{i,t-1}$)			0.078***				0.073***	
			(0.005)				(0.007)	
Did not consume alcohol, 1-period lagged (dummy, $A_{i,t-1}$=0)			−5.170***				−4.943***	
			(0.213)				(0.303)	
Male (dummy)	8.094***	7.407***	3.755***	10.118***	8.248***	7.928***	4.166***	11.031***
	(0.338)	(0.391)	(0.262)	(0.309)	(0.559)	(0.543)	(0.367)	(0.494)
Self-reported health status 'good/very good' (dummy)	0.501***		0.358**		0.554***		0.428**	
	(0.147)		(0.174)		(0.202)		(0.215)	
Self-reported health status, 1-period lagged		−0.000				0.008		
		(0.173)				(0.216)		
Smoking (dummy)	2.524***	2.717***	2.594***		2.314***	2.769***	2.621***	
	(0.222)	(0.270)	(0.268)		(0.309)	(0.340)	(0.335)	
Other drinkers in the household (dummy)	0.992***	1.029***	0.888***		1.115***	1.015***	0.854***	
	(0.183)	(0.224)	(0.219)		(0.247)	(0.274)	(0.267)	
Age (years)	0.365***	0.495**	0.213	0.283**	−0.590	−0.307	−0.299	−0.567
	(0.141)	(0.218)	(0.216)	(0.139)	(0.384)	(0.320)	(0.279)	(0.399)
Age (years) squared/100	−0.339***	−0.480***	−0.238	−0.339***	−0.204	−0.365*	−0.105	−0.163
	(0.102)	(0.156)	(0.157)	(0.102)	(0.138)	(0.188)	(0.189)	(0.141)
Years of education (years)	0.014	0.010	0.009	−0.016	0.010	−0.030	−0.015	−0.026
	(0.026)	(0.029)	(0.021)	(0.026)	(0.040)	(0.040)	(0.029)	(0.041)
Annual income (yuan) in log	0.023	−0.048	0.027	0.071	−0.043	−0.108	−0.073	0.007

Continued

Table 4 Continued

Outcome variable=total weekly alcohol intake (ounce)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(A) Unbalanced panel				(B) Balanced panel			
	(0.077)	(0.092)	(0.092)	(0.077)	(0.106)	(0.115)	(0.114)	(0.108)
Has spouse (dummy)	0.063	−0.397	−0.419	0.168	−0.439	−0.699	−0.685	−0.358
	(0.333)	(0.431)	(0.412)	(0.336)	(0.478)	(0.554)	(0.529)	(0.489)
Has insurance coverage (dummy)	−0.436*	−0.271	−0.080		−0.186	0.045	0.160	
	(0.234)	(0.281)	(0.279)		(0.314)	(0.346)	(0.340)	
Working (dummy)	0.150	0.239	0.312		−0.095	0.089	0.201	
	(0.195)	(0.230)	(0.229)		(0.260)	(0.280)	(0.277)	
Rural (dummy)	−0.186	−0.268	−0.075	0.372	−0.886**	−1.072**	−0.405	−0.293
	(0.246)	(0.292)	(0.175)	(0.245)	(0.433)	(0.422)	(0.247)	(0.440)
Minority (dummy)	0.593	0.546	0.239	1.059***	0.432	0.290	0.288	1.060*
	(0.384)	(0.436)	(0.254)	(0.399)	(0.595)	(0.573)	(0.331)	(0.640)
Height (cm)	0.022	0.035**	0.025**	0.012	0.025	0.019	0.022	0.020
	(0.015)	(0.018)	(0.012)	(0.016)	(0.024)	(0.023)	(0.016)	(0.024)
Within-individual means ($\bar{x}_i$)								
Number of chronic diseases diagnosed	0.795***		0.572**	0.112	0.957**		0.764**	0.020
	(0.278)		(0.290)	(0.282)	(0.451)		(0.386)	(0.470)
Number of chronic diseases diagnosed; 1-period lagged		0.615				1.194**		
		(0.423)				(0.554)		
Self-reported health status	2.002***		1.222***		2.803***		1.584***	
	(0.395)		(0.337)		(0.795)		(0.477)	
Self-reported health status, 1-period lagged		2.437***				2.933***		
		(0.458)				(0.723)		
Smoking	1.257***	1.251***	−0.779**		2.098***	1.377**	−0.565	
	(0.378)	(0.449)	(0.351)		(0.619)	(0.621)	(0.464)	
Other drinkers in household	0.590	0.435	−0.266		1.611**	1.312**	0.084	
	(0.370)	(0.446)	(0.322)		(0.638)	(0.632)	(0.430)	
Age	−0.230	−0.330	−0.025	−0.647***	0.252	0.318	0.360	−0.363
	(0.228)	(0.306)	(0.242)	(0.227)	(0.556)	(0.478)	(0.335)	(0.586)
$Age^2/100$	0.216	0.361	0.097	0.510***	0.454	0.361	0.056	0.756**
	(0.171)	(0.229)	(0.184)	(0.172)	(0.328)	(0.343)	(0.248)	(0.352)

Continued

Table 4 Continued

Outcome variable=total weekly alcohol intake (ounce)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(A) Unbalanced panel				(B) Balanced panel			
Annual income (yuan) in log	0.085	0.141	0.015	0.164	0.032	0.135	0.137	0.301
	(0.173)	(0.212)	(0.152)	(0.168)	(0.315)	(0.310)	(0.209)	(0.308)
Has spouse	−0.596	−0.179	0.044	−0.851*	−0.344	−0.031	0.298	−0.408
	(0.483)	(0.613)	(0.506)	(0.494)	(0.792)	(0.850)	(0.675)	(0.834)
Has insurance coverage	0.045	0.294	0.094		1.321	1.027	0.556	
	(0.444)	(0.549)	(0.387)		(0.901)	(0.887)	(0.557)	
Working	2.434***	2.852***	1.409***		4.317***	4.197***	2.174***	
	(0.450)	(0.536)	(0.374)		(0.777)	(0.753)	(0.505)	
Province fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of individual-wave observations	18 707	12 925	13 554	19 107	9242	7900	8318	9265
Log pseudolikelihood	−27890.94	−18407.45	−18999.74	−28651.79	−14475.23	−11885.32	−12430.07	−14643.35

Marginal effects for the non-censored population $\left(\partial \hat{E}\left(A_{it} | A_{it} > 0,\ \boldsymbol{x}_{it}\right) / \partial x_k\right)$ reported.
SEs adjusted for intra-individual clustering are in parentheses.
*p<0.1, **p<0.05, ***p<0.01.

Table 5 reports the main results—to avoid cluttering the table, we omitted results on the control variables (which are available on request). Two findings are informative. First, in the aggregate, the diagnoses of all four specific chronic conditions help reduce elderly Chinese individuals' alcohol consumption, although not all effects are statistically significant (panel A). Second, elderly Chinese individuals' alcohol consumption is most responsive to diabetes and stroke diagnoses, but the effects vary across different beverages. More specifically, diabetes diagnosis has the largest (negative) effect on beer (panel B) and red wine consumption (panel C), while stroke diagnosis has the biggest effect on spirits consumption (panel D). In comparison, the diagnosis of myocardial infarction exerts a significant effect only on spirits consumption but not on the consumption of other alcoholic beverages (panel D). In fact, the effect of myocardial infarction diagnosis is very imprecisely estimated in general, presumably due to the very few cases with myocardial infarction diagnosis (4%) in the sample.

Further exploration: subgroup analyses

To further examine whether our findings are driven by certain subsets of respondents, this section performs a series of subgroup analyses. We divide the sample into subsamples defined based on characteristics that significantly influence one's drinking behaviour (table 2): gender, age, residential area (urban vs rural areas), whether one smokes, whether there are other drinkers in the household, health insurance coverage and subjective health status. The Chamberlain-Mundlak CRE Tobit model is then applied to each subsample.

The results, reported in table 6, again, reveal a robust and statistically significant link between CDDs and alcohol consumption, suggesting that our findings discussed above reflect a general pattern rather than a pattern driven by some specific subgroups. There appears to be some heterogeneity in the effects of CDDs across subgroups, although the differences are all statistically insignificant. For example, elderly males (panel A) respond to CDDs more strongly than elderly females (panel B). Since elderly males and elderly females have similar numbers of chronic conditions (0.42 for males and 0.46 for females), the difference in their responses is presumably due to the fact that males consume much more alcohol than females (tables 2 and 3), which leaves more room for reductions in alcohol consumption among males. Compared with urban residents (panel F), rural residents' alcohol consumption is more responsive to CDDs

Table 5 Chamberlain-Mundlak correlated random-effects Tobit estimates of effects of the diagnosis of specific chronic disease on alcoholic beverages consumption (aged 50 or above)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(A) Weekly total alcohol consumption (ounces)				**(B) Weekly beer consumption (ounces)**			
Hypertension diagnosis (=1 if yes)	−0.624**				0.292			
	(0.279)				(1.092)			
Diabetes diagnosis (=1 if yes)		−1.536***				−8.290***		
		(0.546)				(2.229)		
Strokes diagnosis (=1 if yes)			−3.334***				−6.628***	
			(0.513)				(1.977)	
Myocardial infarction diagnosis (=1 if yes)				−1.047				1.397
				(0.679)				(2.573)
Control variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Within-individual means of control variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of individual-wave observations	18 262	18 040	18 056	18 026	18 262	18 040	18 056	18 026
Log pseudolikelihood	−27250.703	−26963.902	−26956.218	−26975.486	−12817.085	−12679.990	−12713.691	−12713.433
	(C) Weekly red wine consumption (ounces)				**(D) Weekly Chinese spirits consumption (ounces)**			
Hypertension diagnosis (=1 if yes)	−1.096**				−0.592**			
	(0.547)				(0.273)			
Diabetes diagnosis (=1 if yes)		−2.028**				−1.129**		
		(0.980)				(0.541)		
Strokes diagnosis (=1 if yes)			−1.167				−3.154***	
			(1.099)				(0.504)	
Myocardial infarction diagnosis (=1 if yes)				−1.472				−1.393**
				(1.406)				(0.698)
Control variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Within-individual means of control variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of individual-wave observations	18 260	18 039	18 054	18 025	18 262	18 040	18 056	18 026
Log pseudolikelihood	−4557.902	−4525.322	−4501.226	−4520.112	−23519.825	−23255.391	−23249.379	−23261.866

'Control variables' include those reported in columns (8) in table 2. Marginal effects for the non-censored population $\left(\partial \hat{E}\left(A_{it}|A_{it}>0,\ \boldsymbol{x}_{it}\right)/\partial x_k\right)$ are reported.
SEs adjusted for intra-individual clustering are reported in parentheses.
*p<0.1, **p<0.05, ***p<0.01.

Table 6 Heterogenous impacts of chronic diseases diagnoses on total weekly alcohol consumption

	(1)	(2)	(3)	(4)
	(A) Male	(B) Female	(C) Age<median	(D) Age≥median
Number of chronic diseases diagnosed (current)	−1.653***	−0.302	−1.250***	−0.696***
	(0.317)	(0.192)	(0.318)	(0.213)
Control variables	Yes	Yes	Yes	Yes
Within-individual means of controls	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes
Number of individual-wave observations	8917	9790	9473	9234
Log pseudolikelihood	−23080.043	−4678.1720	−16460.664	−11566.023
	(E) Rural residents	**(F) Urban residents**	**(G) No health insurance**	**(H) With health insurance**
Number of chronic diseases diagnosed (current)	−1.223***	−0.502**	−1.045**	−0.689***
	(0.247)	(0.227)	(0.506)	(0.189)
Control variables	Yes	Yes	Yes	Yes
Within-individual means of controls	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes
Number of individual-wave observations	11 859	6848	5365	13 342
Log pseudolikelihood	−18006.406	−9735.883	−8331.663	−19601.007
	(I) Not smoking	**(J) Smoking**	**(K) No other drinkers in household**	**(L) Other drinkers in household**
Number of chronic diseases diagnosed (current)	−0.709***	−1.372***	−1.218***	−0.632**
	(0.179)	(0.494)	(0.256)	(0.257)
Control variables	Yes	Yes	Yes	Yes
Within-individual means of controls	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes
Number of individual-wave observations	13 803	4904	10 317	8390
Log pseudolikelihood	−13653.346	−14228.689	−16099.332	−11961.540
	(M) Health status='Fair' or below	**(N) Health status='good' or above**	**(O) <2 chronic diseases diagnosed**	**(P) ≥2 chronic diseases diagnosed**
Number of chronic diseases diagnosed (current)	0.750***	−0.960***	−0.437*	−1.461***
	(0.232)	(0.290)	(0.245)	(0.512)
Control variables	Yes	Yes	Yes	Yes
Within-individual means of controls	Yes	Yes	Yes	Yes
Province fixed effects	Yes	Yes	Yes	Yes
Survey-wave fixed effects	Yes	Yes	Yes	Yes

Continued

Table 6 Continued

	(1)	(2)	(3)	(4)
	(A) Male	(B) Female	(C) Age<median	(D) Age≥median
Number of individual-wave observations	10 290	8417	17 091	1616
Log pseudolikelihood	−13510.42	−14658.393	−26329.010	−1558.112

'Control variables' include those reported in columns (8) of table 2. Marginal effects for the non-censored population $\left(\partial \hat{E}\left(A_{it}|A_{it}>0,\ \boldsymbol{x}_{it}\right)/\partial x_k\right)$ reported.
SEs in parentheses, adjusted for intra-individual clustering.
*p<0.1, **p<0.05, ***p<0.01.

(panel E), which might reflect the higher level of vulnerability of rural residents' livelihood against health risks. This is also consistent with the pattern revealed in panels G and H—individuals' responses are stronger without health insurance. Smokers' alcohol consumption is also more responsive to CDDs, possibly reflecting the fact that smokers are more prone to chronic diseases. This finding is, in fact, consistent with the pattern shown in panels O and P, where individuals with different numbers of chronic diseases diagnosed are compared. Finally, those living with other drinkers are less likely to cut back on drinking when diagnosed with a chronic disease, possibly due to within-family peer pressure.

DISCUSSION

Contributions of this study

Many studies have examined the effect of alcohol consumption on health, but research on the impact of health shocks (especially CDDs) on drinking has been limited. Only a few studies have been conducted on this topic; even fewer have been done in developing countries like China. The present study fills the gap by estimating the impacts of CDDs on elderly individuals' alcohol consumption behaviour in China. Our study contributes to the literature in two ways. First, to the best of our knowledge, this study is among the few that attempt to estimate the effects of CDDs on elderly individuals' alcohol consumption in a developing-country setting. Second, it complements previous studies of the impact of CDDs on Chinese individuals' food intake behaviour,[38 39] helping to depict a fuller picture of CDDs' behavioural impacts.

Analysing an (unbalanced) panel dataset on 5724 Chinese individuals over age 50, we discovered that diagnoses of chronic diseases (among stroke, myocardial infarction, diabetes and hypertension) help curb elderly individuals' alcohol consumption in China. Specifically, an additional chronic disease diagnosed by medical professionals reduces one's weekly consumption of beer, red wine and Chinese spirits by 1.49 (95% CI −2.85 to −0.13), 0.93 (95% CI −1.63 to −0.23) and 0.89 (95% CI −1.23 to −0.54) ounces, respectively. These effects translate into a reduction of 0.95 (95% CI −1.29 to −0.60) ounces in total weekly alcohol consumption. More importantly, CDDs significantly reduced the likelihood of excessive drinking—defined as consuming more than 25 g/15 g of pure alcohol daily for an adult male/female—by 24% (95% CI −0.35 to −0.14). These results are robust to a series of specification checks.

The patterns we found are consistent with those in existing studies. For example, Kim and Sambou, also using panel data, found that the odds of drinking and heavy drinking decreased as the number of chronic diseases increased among South Korean individuals.[22] Yet, our study discovered more heterogeneity in the effects of CDDs. First, while the effect of CDDs on elderly Chinese individuals' total alcohol consumption is modest, they have a notable effect on the likelihood of stopping excessive drinking. Second, CDDs exert different effects on elderly individuals' consumption of different alcoholic beverages. Specifically, diabetes diagnosis has the largest impact on beer and red wine consumption, while stroke diagnosis has the largest impact on spirits consumption. Third, different subgroups of individuals responded differently to CDDs. Males, rural residents, smokers and those respondents living with non-drinkers respond to CDDs more strongly than their respective counterparts.

Policy implications

Identifying and understanding behaviour changes on medical diagnoses are important for patients, physicians and policymakers. First of all, unfortunately for patients, many chronic conditions, such as diabetes and heart diseases, cannot be cured entirely; thus, the adverse effects of these chronic conditions on patients' health may last for an extended period. Worse still, some chronic conditions, such as hypertension, are asymptomatic before the patient is seriously ill and thus might undermine his/her health for a long time without being noticed. CDDs provide chronic disease patients with an up-to-date and accurate assessment of their health status, facilitating them to consider lifestyle adjustments that may improve their health status. Our findings suggest that many elderly Chinese individuals cut back on their alcohol consumption on CDDs, implying that regular health checks have the potential to improve patients' health—or prevent their health from deteriorating—by changing their lifestyles.

It is also essential for physicians to understand their patients' drinking responses to CDDs, which enables the former to provide specific medical advice tailored to the latter's behavioural changes. There are different ways of providing medical advice, but the effectiveness of medical advice provided depends on patients' responses to CDDs to a large extent. If, for example, the patients stopped or reduced drinking on CDDs, suggesting that they are open to lifestyle adjustments, then physicians can provide them with detailed advice on how to maintain a healthier lifestyle. In contrast, if CDDs had little influence on a patient's drinking behaviour, physicians may then need to emphasise the potential harms that will be caused by continuing drinking and adopt compulsory medical measures to alleviate the adverse effects of continuing drinking on the patient's health.

Finally, understanding behaviour changes on medical diagnoses is of great policy relevance. Our findings suggest that many elderly Chinese individuals continued to drink (heavily) even after being diagnosed with certain chronic diseases. As such, policymakers may need to formulate relevant policies and measures to urge chronic disease patients to stop excessive drinking. A heavier tax on alcoholic beverage consumption is one possible option.

Limitations

Although we have performed a series of robustness checks to verify our main findings, we note several limitations in the study. The first concerns recall bias. During the CHNS, respondents reported their weekly alcohol intake over the last 12 months rather than the past week or month. As such, the amount of alcohol consumption reported may not be perfectly accurate. Yet on a more positive note, such a survey strategy helped smooth out fluctuations in alcohol consumption due to short-time shocks such as drinking at social events. Also, recall errors are likely to be random errors (with zero mean), which may not introduce serious biases in our estimates. In the Tobit setup, such random errors become part of the disturbance term ε and thus increase its variance (σ_{ε}^2). But since σ_{ε}^2 are jointly estimated with the coefficient parameters, the influence of these errors will be accounted for in our estimates of marginal effects.

Second, due to data limitations, we could not include the diagnoses of other chronic diseases such as hepatitis and NAFLD in the analysis. A natural concern is that this lack of information will influence our estimates. For example, NAFLD has been found to be the most common cause of chronic liver disease worldwide (with a prevalence of 25%–30%) and an important contributor to the incidences of many chronic diseases, including stroke and myocardial infarction, type-II diabetes and hypertension.[40–43] As such, those respondents diagnosed with the chronic conditions examined in this study may also suffer from NAFLD. To the extent that alcohol consumption contributes to the development of NAFLD,[41] the lack of control for NAFLD might bias our estimates of the effects of CDDs somewhat due to the associations between the presence of NAFLD and other major chronic conditions.

Yet, given our data structure and estimation strategy, this problem may not be as serious as it appears. First, controlling for individual-level fixed factors ($\bar{\boldsymbol{x}}_i$), our Chamberlain-Mundlak CRE Tobit specification exploited *changes* in the diagnoses of chronic conditions rather than the *presence* of these conditions for identification. As long as NAFLD was not diagnosed *along with* the chronic conditions examined in this study (—in fact, NAFLD is likely to be diagnosed *before* chronic conditions such as stroke and myocardial infarction, given the former's much higher prevalence), the coexistence of NAFLD and other chronic conditions would not overturn our findings. Second, to the extent that CDDs serve to reduce elderly individuals' alcohol consumption in general, our findings (obtained without including the diagnoses of chronic diseases such as NAFLD) still provide lower-bound estimates of the effects of CDDs. Of course, future studies with more information on other chronic diseases are desirable, which may help depict a more accurate picture of the CDDs–alcohol consumption relationship.

Despite these limitations, we believe that our analysis has added new and valuable information to the literature, thereby deepening our understanding of Chinese individuals' health behaviour and helping to inform China's healthcare policy.

Acknowledgements The authors thank Yubin Wang, Laping Wu, Qiran Zhao, Zhihao Zheng, Chen Zhu, Junfeng Zhu and two reviewers for their helpful comments on earlier versions of this paper. This work was supported by the 2115 Talent Development Programme of China Agricultural University. Financial support from the National Science Foundation of China (grant number: 71973134) is also gratefully acknowledged. This research was conducted based on the data from the China Health and Nutrition Survey (CHNS). We thank the National Institute of Nutrition and Food Safety, China Center for Disease Control and Prevention; the Carolina Population Center, the University of North Carolina at Chapel Hill; the National Institutes of Health (NIH; R01-HD30880, DK056350 and R01-HD38700); and the Fogarty International Center, NIH, for financial support for the CHNS data collection and analysis files since 1989.

Contributors YH, QC and BZ contributed substantially to the conception and design of the study, the choice of analytical methods, and the interpretation of results; YH and QC performed data analysis; YH drafted the article; YH, QC and BZ contributed substantially to critical revision and approved the final version. QC submitted the manuscript.

Funding This study was supported financially by the National Natural Science Foundation of China (No. 71973134) and the 2115 Talent Development Programme at China Agricultural University.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval Since the present study was conducted based on a deidentified, publicly available secondary dataset (CHNS: https://www.cpc.unc.edu/projects/china/data/datasets), it does not constitute human-subject research. There was no interaction with any individual, and no identifiable private information was used: its institutional review board review was thus waived.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available in a public, open access repository. This research was conducted based on the (publicly available) data from the China Health and Nutrition Survey (CHNS). The data can be found at: https://www.cpc.unc.edu/projects/china/data/datasets.

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