Lifestyle choices and health care expenditure

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Abstract

It is widely accepted that lifestyle has substantial bearing on health and accordingly health care expenditure. However, providing empirical evidence on this is particularly challenging in universal public health care systems, when individuals do not know the cost of their health care consumption. In this study, we exploit the presence of linked survey and health administrative data in Australia, where all residents have public health insurance, to obtain individual health care expenditure. We then estimate a prospective model of health care expenditure and use the instrumental variable technique to deal with the potential endogeneity of lifestyle variables. Based on a sample of nearly 250,000 individuals, we find robust evidence that obesity increases both in- and out-of-hospital expenditure. Smoking increases in-hospital expenditure, whilst we find no evidence that risky drinking increases health care expenditure. Failure to account for endogeneity results in severe underestimation of the lifestyle effects on health care expenditure.

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JEL codes: C3, I1

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1. Introduction

The association between higher health expenditures and unhealthy lifestyles has been widely documented. At the aggregate level, rising obesity rates in the US are matched with rising expenditure on obesity-related conditions, These have been estimated to account for nearly 10% of the US health spending in 2008, up from 6.5% in 1998 (Finkelstein et al. 2009). The European Commission estimates that obesity accounts for up to 7% of EU health care costs in 2005 (European Commission, 2005). Smoking and risky consumption of alcohol also place a heavy burden on health care. Sassi and Hurst (2008) report that smoking alone is responsible for 22% of cardiovascular diseases, which is the leading cause of death in developed countries, while chronic illnesses due to alcohol abuse account for between 8% and 18% of the total burden of disease in men and between 2% and 4% in women. At the individual level, studies have found a positive link between health expenditure and obesity (Colaguiri et al. 2010; Finkelstein et al. 2009; Finkelstein et al. 2003), smoking (Sloan et al., 2004; Sturm, 2002) and alcohol consumption (Cook, 2007; Sturm, 2002). This literature suggests that changes in lifestyle may play a significant role in controlling current health care costs and limit their future growth. The aim of this paper is to investigate the effects of obesity, smoking and alcohol abuse on future health care costs. We make three significant contributions.

First we provide individual level evidence of the lifestyles-medical expenditure relationship under a universal public health care system. Cawley and Meyerhoefer, 2012 find that obesity-related cost in the US is the greatest among those who are eligible for public health insurance, but the study fails to establish statistical significance of this result. The presence of public insurance may lower the incentives for people to invest in health-promoting activities, which in turn may result in future health problems. There are several limitations to this study, most notably the

relatively small sample size involved in the analysis, which may explain the imprecise results. We might expect the moral hazard impacts of lifestyle choices on health expenditures to be exacerbated in universal public systems. We fill this gap in the literature.

In universal public health care systems, the primary challenge is measuring health care expenditure at the individual level. Relying on health expenditure from survey data, in which respondents report their health spending for a given period may not only be subject to significant recall error but is also restricted to out-of-pocket costs.[1] Individuals are unlikely to know the cost of publicly provided medical services or those health services that are covered through private health insurance.

In this study, we exploit a recently available large individual survey dataset from Australia that is representative of the population aged 45 and over (45+) and also linked to extensive administrative health expenditure and utilisation data, allowing the calculation of total health care cost for each survey respondent. We calculate each individual's total annual health expenditure, as well as disaggregated expenditure by type of health care (hospital admissions, emergency department presentations and out-of-hospital services and pharmaceuticals). Access to this kind of individual level health care expenditure data is rare; some examples may be found in European countries where data linkage to administrative records is possible, for example in Denmark (Hojgaard et al. 2008).

Our second contribution is the focus on a forward-looking or prospective model of health expenditure. The main advantage of a prospective model is that we avoid reverse causality problems of health expenditure affecting lifestyle choices that are difficult to deal with in the typical concurrent model.

[1] In contrast, in the US, linked survey and claims data are readily available; examples are the Medical Expenditure Panel Survey and Medicare Current Beneficiary Survey.

The third contribution is the estimation of the causal impact of several lifestyle factors (risky alcohol consumption, smoking and obesity) on health care expenditure. With the exception of Cawley and Meyerhoefer, 2012, the bulk of the literature to date has concerned correlation between lifestyles and medical costs, not causation. Correlation studies underestimate the effect of unhealthy lifestyles if those with an unhealthy lifestyle tend to have "distaste" for spending on health care that is unobserved by econometricians. For example, the literature on risky behaviours has modeled risky drinkers, obese individuals and smokers as individuals who tend to be myopic, have time-inconsistent preference and derive low utility from health investment (Cawley and Ruhm, 2011). To account for this potential unobservable heterogeneity bias, we use an instrumental variable technique. We propose that ancestry can be a valid instrument because it affects individual lifestyles but does not directly affect health care cost, conditional on other observed characteristics, which includes demographics, language and country of birth, region and socioeconomic factors, as well as the family's health history. Our controls include a large number of health conditions of the respondent's parents and siblings so that any genetic predisposition to health expenditure will be purged from the error term.

The argument for this instrument stems from the medical and neurology literature on the biological evolution of our ancestors that influences the way we crave for food, control thirst, react to alcoholic products and manage self-control today (Davidson et al., 2011; Cardell et al., 2011; Heath et al., 2001; Whitfield et al., 2004). For instance, as a result of very long cold winter and early life diary production in European countries, Europeans has evolved to favour diets that are high in dense and high-fat food. Individuals with European ancestors therefore may have higher health expenditure because they tend to be fatter than others. It is unlikely that those with European ancestors systematically have high discount rate and prefer to forgo health

treatment. In addition, this instrument is supported by Australia's historical immigration policies which have resulted in multicultural Australian population, providing us considerable variation in ancestral background.

For Australia, to the best of our knowledge, we are aware of only one previous study of total health care expenditure at the individual level by Colaguiri et al. (2010), which is based on self-reported use of health care services. In that study, each visit to a GP is assumed to incur the same fee, even though in Australia GPs are free to set their own fees. To price hospitalisation, a price schedule from the National Hospital Cost Data Collection is used, but it is not adjusted for length of stay or use of intensive care units and ventilators. In contrast, our study is based on administrative data which provide true variation in individuals' expenditure; for example, we know exactly how much each individual pays for a visit to a GP and we know detailed hospital scenario. In addition, the study only conducts a mean comparison analysis of health care costs according to body mass status which cannot be inferred as causal relationship between body mass and health care expenditure.

2. Background and Data

Every Australian resident is eligible for Medicare. Medicare fully covers public in-hospital treatment and subsidises a large proportion of private inpatient medical and pharmaceutical costs, as well out-of-hospital medical services and pharmaceuticals. Medical services are charged on a fee for service basis with no regulation of the level of fee charged. Pharmaceuticals covered by the PBS have an agreed price paid to the pharmaceutical company with a schedule of three regulated prices charged to consumers. The public share of total health expenditure has been stable around 70% in the past decade; it was 69% in 1999/2000 and 70% in 2008/2009 (Australian Institute of Health and Welfare, 2010a). The remaining share is funded

mainly by supplementary (duplicate) private health insurance which covers inpatient treatment. About half of the population has private health insurance, driven mainly by a 30% premium subsidy and income-based tax penalties, rather than motivated by individual's health care need (Fiebig et al., 2006; Johar et al., 2011). The Australian private health insurance market has strong community rating: meaning insurers cannot price discriminate based on any health indicators, and increases in premium must have government approval. Given this it is surprising that a number of studies have found that this market is characterised by a favourable selection i.e., those with private health insurance tend to be healthier than those without private health insurance (Doiron, Jones and Savage, 2008; Buchmueller et al., 2008). It is important to stress that even with the presence of private health insurance market, all Australians may elect to for public treatment. This is the sense of 'universality' that is referred to in this paper.

The data are derived from the 45 and Up Study of 267,188 New South Wales (NSW) residents aged 45 and over (45+) (45 and Up Study Collaborators, 2007). Although it is a sub-population study, it is noteworthy that NSW is the most populous state of Australia (about 7 million residents), and the 45+ population incurs 62% of Australia's health expenditure (AIHW, 2010b).[2] The 45 and Up survey was self-completed by respondents between 2006 and 2010. However, most (80%) completed the survey in 2008 or later. Of the original sample, 265,468 respondents (99.4%) are included in our analysis; the excluded respondents are those who volunteered to participate in the survey or who had an invalid age.

The survey data is linked at the individual level to the following administrative data from 2006 to 2009: (1) private and public hospital admissions (NSW Admitted Patient Data Collection), (2) emergency department (ED) presentation registers, (3)

[2] Excluding expenditure on non-admitted patients, high-level residential aged care, over-the-counter pharmaceuticals and other health practitioner services

out-of-hospital medical services such as GP and specialist consultations (Medical Benefits Schedule, MBS), and (4) subsidised prescription pharmaceuticals (Pharmaceutical Benefits System, PBS).

To impute the cost of hospitalisation, we use the cost weight attached to each Australian-Refined Diagnostic Related Groups (AR-DRG) in the *Costs of Care Standards 2009/10* guidelines released by NSW Department of Health. The individual cost weights are adjusted according to the characteristics of each admission: hospital type, type of care (overnight, same day, transfer, in mental health unit, non- or sub-acute care units such as rehabilitation), length of stay, ICU hours and the use of ventilation machine.[3] For ED presentations, the *Costs of Care Standards 2009/10* provides a cost schedule by hospital type, triage category (a more urgent category is more expensive) and whether the patient is subsequently admitted. The individual costs incurred on medical and subsidised pharmaceutical items are contained in the MBS and PBS, requiring no imputation.[4] All expenditures are annual and indexed to constant $2009. Individual annual total health expenditure is calculated as the sum of the four components: (1) hospital admission, (2) ED presentations, (3) charges for out-of-hospital MBS items, and (4) prices of out-of-hospital PBS drugs in any given year. We validate these expenditures by comparison with Australian Institute of Health and Welfare statistics.

Given that the administrative data end in 2009 and the survey responses are concentrated in 2008, we can only estimate a one-year prospective model without a

[3] Since data from private health insurers are unavailable we impute hospital costs for private inpatient care as well. Consistent with AIHW (2010), estimates of expenditure on medical services for private patients in hospitals and pharmaceuticals dispensed in hospital are included in admitted patient hospital costs. Details of the costing rules and data construction are provided in Appendix.

[4] For medical items we use the full fee charged by the provider and for PBS items we use the agreed price paid to the supplier.

great loss in sample size.[5] We lose 4,279 respondents (1.6%) who completed the survey in 2009 and 2010. We exclude a further 28,507 respondents (10.7%) due to missing lifestyle information. The final sample consists of 236,961 respondents.

Table 1 reports the summary statistics of health expenditure variables. Less than 3% of the sample has zero total annual expenditure. Of those with positive expenditure, the mean annual total health expenditure is $5,107 (7.5 in log scale). Hospital admissions, which are incurred by 28% of individuals, are the most expensive component. Nearly 14% of the sample presents at an ED. Expenditure on out-of-hospital medical services and prescription pharmaceuticals are incurred by almost the whole sample.

[Table 1 here]

Those reporting risky drinking (more than 14 standard drinks per week) comprise 14% of the sample. Somewhat unexpectedly, these risky drinkers have similar, if not, slightly lower utilisation rate and expenditure than the overall sample. Among the non drinkers and moderate drinkers, we find that the non drinkers tend to have higher health care expenditure than others, including relative to the very risk drinkers. It may be that alcohol abstinence is required by an individual with some diagnosed health problems. The much publicised alcohol-related problems such as alcohol-related accidents peak in younger cohorts (Australian Bureau Statistics, 2009) and burden of disease studies which often rely on aggregate data may be masking

[5] Variation in survey year across individuals is driven by the sampling process rather than individual choice. The sample is surveyed only once so all information from the survey is constant across all years. The respondent's age is updated from his/her age in the survey year. Respondents who died during 2006-2009 are excluded in the prospective model. Not all deaths however are identifiable. However, we argue that any possible bias from failing to identify deaths at home is minimal since most respondents completed the survey in 2008-2009. A support for this can be provided by the comparable share of zero health expenditure in pre- and post-survey years. Had there been many (unobserved) deaths at home we would observe higher share of zero expenditure in the post-survey year.

variation in alcohol-related health costs across age groups. We currently have limited knowledge about alcohol-related health costs among older population.

Figure 1 shows the relationship between alcohol consumption and average health expenditure by gender. While gender differentials in drinking are a feature of the literature (e.g., Cawley and Meyerhoefer, 2012), in our data, females and males have the same pattern of health expenditure by drinking intensity: those who never drink have the highest health expenditure and those who are regarded as risky drinkers have the lowest health expenditure. Males are much more likely to drink, and drink excessively, than females who tend to be low risk drinkers. In the empirical analysis below, we group 'Never' to 'Medium' risk together and compare relative to the 'High' risk drinkers.

[Figure 1 here]

Nearly a quarter of the sample is obese (22%). Obese individuals have generally higher health care utilisation rate and health expenditure than the overall sample. Figure 2 presents the distribution of BMI and average health expenditure by gender. Overweight is more prevalent among males, but morbidly obese individuals are mostly females. Throughout the BMI distribution, males have higher health expenditure than females, but the relationship between BMI and health expenditure among females and males exhibit the same trend. Except for a small share of people who are borderline underweight, health expenditures of the normal weight and overweight individuals are not very different, especially for males. Obese individuals have higher expenditure. Note that the average 45+ Australian is overweight (BMI of 26). We group the non obese individuals together as a comparison group relative to obese individuals.

[Figure 2 here]

Past and current smokers (ever smoke), who make up 43% of the sample, also have higher health care utilisation rate and health expenditure than the overall sample, and higher expenditure on hospital services than obese individuals. Figure 3 presents smoking history and average health expenditure by gender. This relationship is the same for females and males: past smokers have the highest expenditure while current smokers have the lowest. This pattern may reflect accumulation and permanent health consequences of smoking; cessation may be caused by health problems. In the sampling population, the 45+, current smokers are a minority, accounting for less than 10%. For this reason, as well as to capture the permanent or long term health damage due to smoking, we group past and current smokers together.

[Figure 3 here]

Table 2 reports the summary statistics of covariates and the instruments for the full sample; as there is lack of gender differentials in health expenditure patterns across lifestyle indicators, the main empirical analysis will be based on the full sample. About 40% of the sample is over 65 and 12% is over 80. The majority of respondents has at least a diploma or equivalent educational attainment, is still in the labour force, has a reported annual income of at least $40,000, was born in Australia and has a fair skin. The inclusion of country of birth and language as control variables aims to capture individual heterogeneity in initial health endowment (birth environment), attitudes towards medical care, familiarity with the health system, and language barrier in obtaining health services.

[Table 2 here]

The medical literature has placed considerable emphasis the role of genetic factors on the development of many diseases. To control for genetic disease predisposition parents' and siblings' medical conditions are included as covariates.

The siblings' information may capture hereditary carriers better than the parents' health, which may be confounded by age. Moreover, sibling's condition may reflect any genetic trait that skips a generation (i.e. the respondent's grandparents' conditions). We have quite rich information on a wide spectrum of diseases.

Our instruments for lifestyle choice comprise six dummy variables for ancestry that are not mutually exclusive. For example, an individual can have both Australian and Asian ancestors. In the sample, the majority report English (including Scottish and Irish) ancestry. The ancestry variables are not coincident with country of birth. Nearly three quarters of the sample was born in Australia, but only half of the sample has an Australian ancestor. The pairwise correlations between matched ancestry and country of birth (e.g., Australia and Australia) are high but far from perfect; they range between 0.18 and 0.5.

3. Methodology

We estimate a multi-equation model of health expenditure (y) and lifestyle factors. We consider three lifestyle variables: (i) risky alcohol consumption, as defined by the National Health and Medical Research Council (NHMRC, 2009) guideline as consumption of more than 2 standard drinks per day or 14 standard drinks per week (*alcohol*); (ii) ever smoke (*smoking*); and (iii) obese, as indicated by having a body mass index of 30 or greater (*obese*). Since some individuals do not spend on health, there are two equations for health expenditure capturing positive of health expenditure and the level of the health expenditure, conditional on positive health expenditure. The two common models for this two-stage process are the Heckman selection model and the two-part model where the first stage consists of a binary model of positive expenditure and the second stage consists of a regression model for only those with positive health expenditure. The selection model assumes non-zero correlations between the two

stages whilst in the two-part model, the correlation between the two stages are non-nested (Dow and Norton, 2003; Deb et al., 2011). The underlying assumptions behind the two models differ in that the latter assumes that the observed zero expenditure is a true zero, not missing. Generally the two-part model is preferred to the selection model when there is no exclusion restriction to identify the first stage equation, as it is the case here (Dow and Norton, 2003; Deb et al., 2011).

Consider latent process governing positive health expenditure of individual *i* in the following year:

$$y_i^* = \beta_{10} + \beta_{11} D_i + \beta_{12} E_i + \beta_{13} G_i + \beta_{14} I_i + \alpha_1 Lifestyle_i + \varepsilon_{1i}$$

related to the observed health expenditure by

$$y_i = 1 \;\; if \;\; y_i^* > 0; \; y_i = 0 \;\; otherwise.$$

D_i, E_i, G_i and I_i denote a vector of demographic, economic, geographic, and intergenerational health conditions, respectively, in the survey year. $\{\beta_{11}, \beta_{12}, \beta_{13}, \beta_{14}\}$ are their associated parameters. $Lifestyle_i$ indicates the lifestyle measure of interest: *alcohol*, *smoking* or *obese*. The effect of a lifestyle factor on the probability of incurring positive health expenditure is measured by α_1 and ε_{1i} is a random error term that captures the unobserved determinants of positive health expenditure. This is the selection stage of the two-part models and can be estimated by probit models. In the second stage, conditional on positive health expenditure, we assume health care expenditure to follow:

$$y_i \mid y_i > 0 = \beta_{20} + \beta_{21} D_i + \beta_{22} E_i + \beta_{23} G_i + \beta_{24} I_i + \alpha_2 Lifestyle_i + \varepsilon_{2i}$$

We estimate separate models for total health expenditure and each component of total expenditure. Following the literature, to deal with the high skewness of the expenditure series, we use log transformation.

We also adopt the latent variable framework for the lifestyle choices. The latent process reflecting net utility from risky drinking is assumed to follow:

$$alcohol_i^* = \beta_{30} + \beta_{31} D_i + \beta_{32} E_i + \beta_{33} G_i + \beta_{34} I_i + \delta_1 Z_i + \varepsilon_{3i}$$

The latent alc_i^* is related to the observed drinking pattern alc_i by:

$alcohol_i = 1$ if $alcohol_i^* > 0$; $alcohol_i = 0$ otherwise.

We can similarly define a latent process for the other lifestyle variables;

$obese_i^* = \beta_{40} + \beta_{41} D_i + \beta_{42} E_i + \beta_{43} G_i + \beta_{44} I_i + \delta_2 Z_i + \varepsilon_{4i}$; $obese_{it} = 1$ if $obese_i^* > 0$

$smoking_i^* = \beta_{50} + \beta_{51} D_i + \beta_{52} E_i + \beta_{53} G_i + \beta_{54} I_i + \delta_3 Z_i + \varepsilon_{5i}$; $smoking_i = 1$ if $smoking_{it}^* > 0$.

Z_i is a vector of instruments which affect lifestyle but do not directly influence health expenditure, and $\{\delta_1, \delta_2, \delta_3\}$ are their associated parameters in each lifestyle equation. The Z_i appear only in the lifestyle equations and not in the expenditure equations, satisfying the exclusion restriction identifying the lifestyle effects on health cost.

We do not include individual health conditions as covariates. This is the standard practice is the literature because lifestyles affect health conditions, which makes health conditions endogenous (Finkelstein et al., 2009; Cawley and Meyerhoefer 2012). Moreover, it is also likely that the relationship between lifestyles, health conditions and health expenditure is a non-linear one. For instance, the marginal effect of a heart condition may be larger for obese individuals due to surgical complications. This means that a "full" model which deals with all of these issues has to be non-linear, with the number of equations equal to the number of health conditions and lifestyle factors plus the health care expenditure equations. It is highly likely that such a model would prove intractable. There are some ways to reduce the dimension of health conditions, for example through grouping rules of homogenous conditions like 'hierarchical health condition' or 'aggregate health condition' groups (Zhao et al., 2005), which take into account co-morbidities and severity of conditions, but still, they are at least 30 of them. In addition to this problem of tractability, one also needs to find satisfactory instruments that influence health conditions but not directly affecting lifestyles and health care expenditure. We do not believe our data

contains such instruments. Therefore, following the literature, we interpret the coefficient on *alcohol* as "alcohol-related" effect, the coefficient on *obese* as "obesity-related" effect and the coefficient on *smoking* as "smoking-related" effect.

A challenge here is that the dependent variables are of a different nature: the expenditure variable is continuous and the lifestyle variables are discrete. One possible estimation option would be to discretise the expenditure variable and estimate a four equation multivariate probit model. For example, we may define a latent process governing high expenditure which relate to observations in the top 25% of the expenditure distribution. The computation of the likelihood function for the multivariate probit model involves multidimensional integration and simulation methods have been used to approximate such a function, with the GHK simulator being the popular choice.

An alternative estimation approach is the conditional (recursive) mixed process (CMP) estimator as described in Roodman (2009). This estimator fits a wide range of seemingly unrelated regression systems and can accommodate the different nature of the dependent variables; in our case 2 binary equations and 1 continuous equation.[6] For problems involving higher dimensional integration like ours, simulated maximum likelihood is used to estimate the parameters in the model. We use the GHK simulator with 1,000 Halton draws and impose no restriction in the error correlation structure.[7] So, in addition to the covariate parameters, there are an additional 9 unique pairwise error correlation coefficients to be estimated and one variance parameter for the

[6] Roodman (2009) explains that the CMP estimator can provide consistent estimates for systems that are (i) recursive, with clearly defined stages, and (ii) fully observed meaning that the endogenous variables appear as covariates only as observed variables (as opposed to latent). For our problem, given prospective modelling, health expenditure equations are clearly the final stage equation.

[7] Instead of taking independent random draws, simulation can potentially be improved (in terms of having smaller expected approximation error) by selecting evaluation points more systematically. Greater precision in the simulated probabilities in turn translates into greater precision in parameter estimates. Halton draws have been found to provide far more accuracy than a comparable number of independent draws (e.g. Train 2003 in the context of mixed logit).

expenditure variable. The two-part model does not explicitly try to estimate the correlation between the first and second parts of the model, which are assumed to be independent of one another. The implementation of the mixed process estimator is done in STATA using the *cmp* routine supplied by Roodman (2009). The standard errors are computed using bootstrap method.

We allow the health expenditure equations and the lifestyle equation to be correlated, but estimate the impact of each lifestyle factor separately. We have jointly estimated the impact of all three lifestyle factors on health expenditures and found that the results are similar to the independent models while the computation of the joint model was very time intensive. The computation of the bootstrapped standard errors proved computationally infeasible. There is a significant positive correlation between the error terms of alcohol and smoking, but allowing for this correlation does not meaningfully alter the size of the alcohol and smoking effects from those estimated by the respective two independent models. Thus, in what follows, we focus on the results from the independent model.

4. Results

4.1. Instruments

To establish the endogeneity of lifestyle factors, we need exogeneity to be rejected in one or both of the propensity of positive expenditure or the level of expenditure equations. To test for this, we perform Wu-Hausman exogeneity tests based on the residuals from the lifestyle equations. Because this test is based on a linear model, we estimate linear probability models for lifestyle factors as well as the indicator variables for positive expenditures. We find that exogeneity is rejected at the 1% significance level for all lifestyle factors.

Establishing the validity of our instruments is a more demanding task. First, they must be strong predictors of every lifestyle factor. We find ample evidence that they are. The likelihood ratio statistics from independent probit models for alc, bmi and smk with and without the instruments are a large 250, 200 and 276 respectively ($\chi^2_{5,0.01} = 15.09$). If we estimate linear probability models and perform partial F tests, which is equivalent to the Cragg-Donald's weak identification test, we obtain corresponding F-statistics of 30, 28 and 39, which are greater than the minimum standard power value of F=10 suggested by Stock and Yogo (2002). These results indicate that our instruments are not weak instruments. Furthermore, the coefficients on the instruments have signs that are consistent with the instrument's intuition, that is, ancestry affects health care because evolutionary processes that our ancestors went through (e.g., long winter, agricultural economy, memories of hunger and starvation, brewery development etc) shape our lifestyles today. All else equal, those with European and Australian ancestors are more likely to be risky drinkers, be obese and take up smoking, whilst those with Asian ancestor tend to have completely opposite characteristics.

Second, the instruments must have no explanatory power over each of the health care expenditure variables. Because we have three endogenous variables and six instruments, we perform overidentification tests. The standard overidentification test is the Sargan's test which is based on linear models. So, again, we estimate linear probability models for lifestyle factors and the indicators for positive expenditures. The Sargan's test statistics range between 2.9 and 31.4, with an average of 12. These statistics are reported in Table 3. Because Sargan's test statistic increases linearly with sample size, and our sample is very large (about 10 fold the sample size of most empirical studies), using the standard 1% significance level may lead to over rejection of the test statistics. Thus it is sensible to use a higher critical value of $\chi^2_{5,0.001} = 20.515$,

above which we reject the null hypothesis of valid instrument; one may view the use of this significance criterion as the trade-off between type I and type II errors in the classical hypothesis test where we do not wish to keep the former fixed while the latter goes to zero as the sample size increases. Four cases fail the test, three of them related to *alcohol*. We verify that this rejection is driven by large sample size by selecting 10% random sample from our data, and re-perform the overidentification test. The resulting Sargan's statistics range between 0.8 and 11.4, way under the critical value at 1% level $\chi^2_{5,0.01} = 15.09$.

[Table 3 here]

While passing or not passing statistical tests is a useful tool to judge the validity of our results, the more substantive question is how much bias we introduce by using "almost valid" instruments. In appraising the empirical literature on the use of instrumental variables, Murray (2006) puts a heavy weight on weak instruments, and finds that bias from having strong and almost valid instruments may be small. As in most studies, unfortunately, alternative instruments are not available in our data. The strategy to use exogenous area variation is limited given the Australian setting. Many studies from the US on the other hand have taken advantage of a large number of states and cities implementing different taxes and regulations (Cawley and Ruhm, 2011). In Australia, there are only eight states and territories, and within state, taxes and policies are generally uniform across cities. We experimented with postcode variations in demographics, housing, access to internet and mode of transport as instruments but this strategy leads to a worsening of the problem of weak instruments.

4.2.Lifestyle effects

Table 4 shows the results from lifestyle models, which estimate each of the lifestyle factors one at a time. The results in the first three columns ignore the

endogeneity of the lifestyle factor and the results in the second three columns are derived using the instruments. We report the marginal effects of each lifestyle factor on the next year's health expenditure holding the effects of other covariates constant at their sample means. We present the marginal effects (ME) on the: probability of positive expenditure $\Pr(y>0|\mathbf{X})$, where X denotes all covariates in the model, and on the expected expenditure $E(y|\mathbf{X})$.[8]

[Table 4 here]

Ignoring endogeneity, obesity and smoking this year are associated respectively with $1,373 and $647 higher total annual health expenditure next year. These large marginal costs may be explained by the link between obesity and smoking and many chronic conditions (e.g., diabetes, cancer, etc). The higher total expenditure is the result of increases in all expenditure subcategories: admission, ED and out-of-hospital. The marginal effects on the probability of having positive total and out-of-hospital expenditures are very small because almost everyone uses medical services in a given year. Obesity and smoking are associated with higher use of hospital services. Obesity is also associated with higher use of out-of-hospital services. On the other hand, risky drinkers tend to have a lower probability of ED presentation and lower ED costs than non drinkers and moderate drinkers. This negative effect of risky drinking may be explained by non drinkers comprising those who abstain from alcohol due to high-cost health problems.

[8] Let $\bar{x}^1$ and $\bar{x}^0$ denotes level of all covariates at the sample mean except lifestyle factor x_k which is set at 1 and 0, respectively. Further let $c^1=\bar{\mathbf{x}}^{\mathbf{1}}\hat{\beta}_1$ and $c^0=\bar{\mathbf{x}}^{\mathbf{0}}\hat{\beta}_1$. To convert to dollar scale, we assume homoskedastic Duan's smearing factor $D=\overline{\exp(\hat{\varepsilon}_2)}$. The marginal effects are derived as follows. On the probability: $\hat{\Phi}(c^1)-\hat{\Phi}(c^0)$. On the overall expenditure:
$E(y|\mathbf{X}=\bar{\mathbf{x}}^{\mathbf{1}},x_k=1)-E(y|\mathbf{X}=\bar{\mathbf{x}}^{\mathbf{0}},x_k=0)=\hat{\Phi}(c^1)\bar{\mathbf{x}}^{\mathbf{1}}\hat{\beta}_2-\hat{\Phi}(c^0)\bar{\mathbf{x}}^{\mathbf{0}}\hat{\beta}_2$. In dollar scale, this is modified to $\left\{\hat{\Phi}(c^1)\exp(\bar{\mathbf{x}}^{\mathbf{1}}\hat{\beta}_2)-\hat{\Phi}(c^0)\exp(\bar{\mathbf{x}}^{\mathbf{0}}\hat{\beta}_2)\right\}D$

When we account for endogeneity, we find consistent results in terms of signs and statistical significance: both obesity and smoking are associated with higher health care costs while risky drinking has a much smaller correlation with health care cost, if any, it is a negative correlation. Ignoring endogeneity results in serious under estimation of the lifestyle effects, especially for obesity. It is estimated that obesity-related health problems increases annual total health care costs by about $4,400, more than three times the estimate from the exogenous model. This negative bias is reflected in the strong negative correlation between the error terms in the expenditure equations and in the obesity equation, suggesting that unobserved factors that promote obesity tend to discourage health care expenditure. These factors may be lower valuation of health investment, fear of medical treatment or lower mobility. Using US data, Cawley and Meyerhoefer (2012) finds a larger underestimation bias (about 4 times), which may be explained by supply constraint in the US according to patients' ability to pay. If obesity is more prevalent among low income households then the supply problem may also lead to underestimation of the obesity effect in their low income sample. Their estimate for obesity-related cost among those eligible for universal health care – using a completely different instrument – is comparable to our estimates, about US$3,800 in 2005.

The marginal effect of smoking on total expenditure is also underestimated, by about two-fold. Smoking has a big positive impact on hospital use in both admission and ED. Since the mean admission and ED presentation rates are 0.28 and 0.14, respectively, the marginal effects suggest that an average person who ever takes up smoking is almost twice as likely to use hospital services than an otherwise similar average person.

Risky drinking has no significant effect on total health care cost, but it has significant effect on ED use. There is a strong positive selection on the unobservables

influencing ED presentation, but there is an even stronger negative selection on the unobservables for the cost of ED presentation. This may suggests that those who are likely to be risky drinkers are also likely to develop health conditions that require ED services, but these services tend to be less urgent, are not likely to lead to subsequent admission, or tend to be treated in smaller hospitals, which are all cost less than their counterparts (urgent, subsequently admitted and large hospitals). The overall effect is that risky drinkers have $71 less in annual expenditure on ED.

We summarise the effects of the control variables on health care expenditure as follows.[9] We find a strong positive age gradient, predicting a rapid increase in use and expenditure for all health expenditure categories up to 80 years of age where the effect of age diminishes. Males are less likely to use out-of-hospital medical services and spend less on these services than females, but they are more likely to use in-hospital services and spend more on these services. Education and full-time employment tend to lower all types of healthcare costs. Income has a greater impact on health expenditure level than on the probability of health care use: high income individuals have lower total health expenditure. Health expenditure also varies across regions, with city dwellers having higher expenditure than those living in regional or remote areas. Almost all intergenerational conditions are associated with higher use and expenditure of health care services. Family history of heart disease, high blood pressure, cancer and depression has the strongest positive associations with use and expenditure on ED and out-of-hospital products. In contrast, family history of heart disease, bone disease, cancer and depression is positively linked to the probability of admission, but the history of cancer lowers admission costs. This result may reflect effectiveness of increased preventive measures on the part of those whose parent or sibling has had cancer.

[9] Full results available from authors.

We summarise the effects of factors that correlate with lifestyles as follows.[10] Risky drinking is negatively associated with very old age (70+). Married individuals, those born in Asia, Middle East and Africa are all less likely to be risky drinkers, whilst males, native speakers and those without university certificate are more likely to be risky drinkers. Income is positively related to risky drinking. Compared to full time workers, part time workers and retirees are likely to drink more. Obesity is negatively correlated with age, education, never married, separated and in partner relationship, born overseas, full-time employment, very low income and living in major cities. Lastly, those who have ever smoked tend to exhibit similar characteristics to risky drinkers in terms in gender, marital status, employment status and region. However, in contrast to risky drinkers, they tend to have low education and be low income.

4.3.Aggregate costs

In 2009, there are about 8 million Australians aged 45 and over, and about 22% are obese. With the marginal costs of obesity in Table 4, we estimate that obesity-related health care cost amounts to $7.74 billion ($4400 x 8 x 0.22) in 2009. If we are prepared to generalise to the whole 14 million Australians aged 25 and over, of whom 27% are obese, then the obesity-related health care cost is estimated to be $16.6 billion ($4400 x 14 x 0.27). This is double the 'obesity cost' reported by Colaguiri et al (2010) of $8.3 billion in 2005 using the AusDiab database comprising of some 11,000 adults aged 25 years and older. However, their 'obesity cost' estimate is based on the average heath care costs of obese individuals in their sample, thus cannot be interpreted as health care costs due to obesity. An earlier report for Diabetes Australia estimated obesity cost related to four chronic diseases (diabetes (type I and II), cardiovascular diseases, osteoarthritis and cancers (breast, colorectal, uterine and

[10] Full results available from authors.

kidney)) to be $873 million in 2002 (Access Economics, 2006). Again, this estimate cannot be interpreted as causal; the obesity-related cost is estimated as an exogenous proportion ("attributable fraction") of total costs of each of the diseases derived from various studies in the literature. Interestingly, our estimate is comparable to Cawley and Meyerhoefer (2010), which provides causal effect of obesity on American parents. They estimate that obesity costs US$0.68 million per one million parents in 2005. Our estimate is AUS$0.97 million per one million 45+ individuals in 2009, or US$0.64 million in 2005 (the inflation adjustment is 0.89 and the exchange rate is 0.743, which is the average in December 2005).

5. Conclusion

In this paper, we investigate the effects of several lifestyle factors on the health expenditure of the biggest consumer group of healthcare services in Australia, the 45+ population. Advancing the literature, we focus on a prospective model of health expenditure which avoids potential bias due to feedback effect of health expenditure on lifestyle, and estimate multi-equations system to allow for correlations between health expenditure and lifestyle, as well as correlation among the lifestyle factors themselves. Based on sample of nearly 300,000 individuals, we find that all else equal, obesity has a significant bearing on future health care cost, increasing the use of all types of health services, as well as the expenditure on these services. Having ever smoked also has health implications, especially on hospital services. Contrary to popular expectation, we find no significant evidence that risky drinking has substantial impact on health care cost among the 45+ population. As well, we find a significant negative selection on the unobservables related to obesity and smoking which leads to underestimation of their impact on future health care burden in correlation analyses.

Another contribution of this study is providing evidence in the presence of universal health insurance. The bulk of health care costs are borne by tax payers under this system hence the policy implications of our results are eminent. First, the finding that obesity and smoking have causal impacts on health care utilisations and expenditures suggests that effective prevention policies against obesity and smoking can help reduce the nation's future health care burden. Our result may serve as a base for future cost-effectiveness studies of alternative policies or measurement of social costs. Secondly, we find that these impacts are the largest on the hospital system. This finding may shed light on the mechanism behind the lifestyle impacts on health care use, for example along the story of moral hazard or over servicing. In contrast to public hospital services, out-of-hospital services like doctor fees and prescription drugs may involve significant co-payment to individuals; doctors are free to set fees, of which 85% are subsidised. As such, there are financial incentives for individuals to avoid doctor's appointment. Meanwhile, doctors may also increase precaution when dealing with obese patients or those with smoking history by ordering more medical tests/screening or extending their length of stay. These two pathways may explain relatively larger lifestyle impacts on hospital services than on out-of-hospital services. The first pathway relates to cost minimisation strategies targeted to individuals such as healthy lifestyle promotion campaigns, whilst the second pathway suggests some scope for "best practice" guidelines targeted to providers. Finally, our finding that unhealthy lifestyles are associated with distaste for health service use suggests that commonly enacted hospital-based interventions may not effectively reach their targeted population. Those who lead unhealthy lifestyles *and* use health services are selective part of the targeted population, who are, arguably, more likely to be positively affected by the intervention or voluntarily seek help regardless of

intervention. As a result, an evaluation study of the intervention would yield a biased estimate of its effect (overestimation) due to sample selection.

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Table 1: Summary statistics of health care use and expenditure

	y>$0 (%)	Mean ($)	Mean |y>$0 ($)	Mean |y>$0 (log)
Health expenditure				
Total	0.973	$4,971 ($11,371)	$5,107 ($11,496)	7.531 (1.471)
Admission	0.282	$2,473 ($9,866)	$8,758 ($17,021)	8.299 (1.188)
ED	0.144	$99 ($352)	$690 ($677)	6.287 (0.664)
Out-of-hospital	0.968	$2,399 ($3,760)	$2,479 ($3,796)	7.174 (1.246)
Health expenditure | *alcohol*=1 (N=34,618 (0.143))				
Total	0.967	$4,611 ($11,321)	$4,767 ($11,479)	7.439 (1.482)
Admission	0.280	$2,343 ($9,918)	$8,371 ($17,348)	8.246 (1.186)
ED	0.125	$82 ($334)	$652 ($721)	6.228 (0.660)
Out-of-hospital	0.961	$2,186 ($3,488)	$2,275 ($3,530)	7.174 (1.251)
Health expenditure | *obese*=1 (N=52,895 (0.220))				
Total	0.979	$5,629 ($11,778)	$5,749 ($11,874)	7.732 (1.424)
Admission	0.302	$2,730 ($10,081)	$9,032 ($16,712)	8.347 (1.175)
ED	0.163	$111 ($369)	$683 ($666)	6.274 (0.678)
Out-of-hospital	0.975	$2,788 ($4,164)	$2,859 ($4,193)	7.365 (1.211)
Health expenditure | *smoking*=1 (N=102,893 (0.428))				
Total	0.972	$5,503 ($12,264)	$5,663 ($12,404)	7.637 (1.482)
Admission	0.299	$2,777 ($10,750)	$9,292 ($18,060)	8.346 (1.203)
ED	0.160	$114 ($382)	$713 ($695)	6.311 (0.678)
Out-of-hospital	0.964	$2,612 ($3,840)	$2,708 ($3,877)	7.259 (1.260)

Note: The full sample size is 236,961 individuals. Sample weight is used which reflects the 45+ NSW population by region due to oversampling in regional areas.

Table 2: Summary statistics of covariates and instruments

	Mean		Mean		Mean
Demographic		COB: Middle East	0.003	**Intergenerational diseases**	
Age: 45-19	0.105	COB: New Zealand	0.020	Parent: Alzheimer	0.150
Age: 50-54	0.166	COB: South Pacific	0.010	Parent: Parkinson	0.038
Age: 55-59	0.173	COB: Australia	0.723	Parent: bone diseases	0.309
Age: 60-64	0.153	Skin: very fair	0.157	Parent: depression	0.082
Age: 65-69	0.125	Skin: fair	0.546	Parent: heart disease	0.414
Age: 70-74	0.092	Skin: light olive	0.250	Parent: high BP	0.451
Age: 75-79	0.067	Skin: dark olive	0.016	Parent: stroke	0.236
Age: 80+	0.119	Skin: brown	0.022	Parent: diabetes	0.178
Male	0.476	Skin: black	0.001	Parent: cancer	0.342
Married	0.689	Skin: missing	0.001	Sibling: Alzheimer	0.014
Never married	0.066	**Economic**		Sibling: Parkinson	0.010
Widowed	0.086	Income: <$5k	0.015	Sibling: bone diseases	0.067
Divorced	0.073	Income: $5-<10k	0.038	Sibling: depression	0.053
Separated	0.027	Income: $10-<20k	0.131	Sibling: heart disease	0.108
Unknown	0.006	Income: $20-<30k	0.088	Sibling : high BP	0.161
Partner	0.053	Income: $30-<40k	0.075	Sibling: stroke	0.034
Foreign language	0.116	Income: $40-<50k	0.071	Sibling: diabetes	0.082
Health card	0.274	Income: $50-<70k	0.104	Sibling: cancer	0.153
Educ: no post-school	0.123	Income: >=70k	0.265	**Instruments**	
Educ: certificate	0.313	Income: missing	0.212	Ancestry: UK	0.568
Educ: diploma	0.313	FT	0.360	Ancestry: Mediterranean	0.043
Educ: university	0.252	PT or other work	0.145	Ancestry: other Europe	0.083
COB: East Asia	0.017	Fully retire	0.362	Ancestry: Asia	0.039
COB: South East	0.019	Disabled	0.037	Ancestry: other	0.312
COB: South Asia	0.008	Not in Labour Force	0.097	Ancestry: Australia	0.496
COB: UK	0.101	**Region**			
COB: other Europe	0.039	Remote	0.092		
COB: South America	0.005	Outer region	0.079		
COB: Mediterranean	0.024	Inner region	0.157		
COB: Africa	0.011	Major city	0.672		

Note: The full sample size is 236,961 individuals. Because all covariates are binary, all figures in the table are sample proportions. Sample weight is used which reflects the 45+ NSW population by region due to oversampling in regional areas. Parent's (sibling) health conditions are based on the question whether your father or mother (brother or sister) has the stated condition. Cancer includes breast, ovarian, prostate, melanoma, bowel and lung. Bone diseases include hip fracture, osteoporosis and arthritis. Of the ancestry variables listed in the survey, they are grouped as follows. Ancestry UK includes English, Irish and Scottish. Ancestry Mediterranean includes Italian, Greek and Maltese. Ancestry other Europe includes German, Dutch, Polish and Croatian. Ancestry Asia includes Chinese, Filipino, Vietnamese and Indian. Ancestors that are not explicitly asked in the survey question are grouped as 'other'.

Table 3: Overidentification test results

	All sample (N=236,961)			10% random sample (N=23,762)		
	alcohol	*obese*	*smoking*	*alcohol*	*obese*	*smoking*
Total						
Prob(y>0)	8.39	8.46	8.39	4.13	3.85	4.14
E(y|y>0)	23.80	8.33	19.28	9.15	5.60	8.38
Admission						
Prob(y>0)	31.37	8.15	13.82	6.76	5.99	6.98
E(y|y>0)	5.08	6.10	8.03	3.48	4.15	2.65
ED						
Prob(y>0)	14.52	5.62	4.72	9.20	7.45	8.62
E(y|y>0)	2.94	5.21	4.33	4.97	8.82	7.82
Out-of-hospital						
Prob(y>0)	9.57	10.43	10.43	0.84	0.88	1.42
E(y|y>0)	26.37	17.05	25.89	11.39	11.01	10.16
Critical value						
$\chi^2_{5,0.001}$	20.52			$\chi^2_{5,0.01}$	15.09	

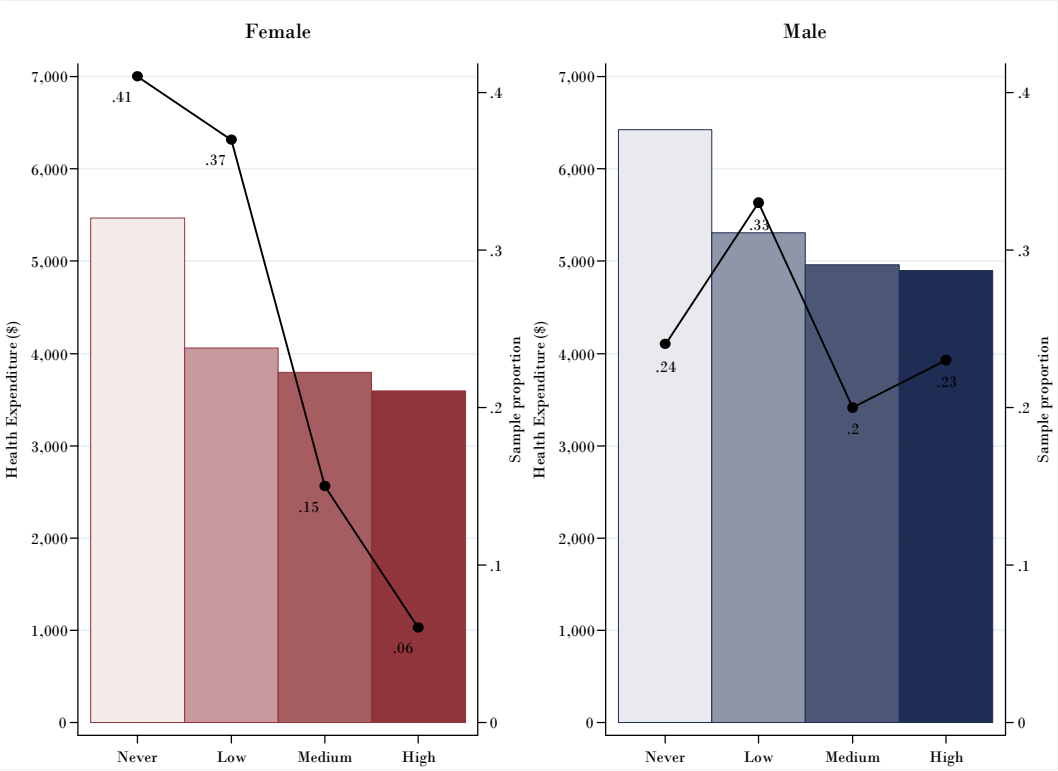
Note: linear probability model is assumed for lifestyle factors and indicator variables for positive expenditure. The overidentification test is based on N*R-sq where N is the sample size and R-sq is obtained from the regression where the dependent variable is the residual from the instrumented regression, and the regressors are all covariates and the instruments. In large sample, this test follows a Chi-squared distribution with df=k–1 where k is the number of instruments. These statistics are obtained from 48 independent regressions.

Table 4: Results from models of health care expenditure by type

	Exogenous (Non IV)			Endogenous (IV)		
	alcohol	*obese*	*smoking*	*alcohol*	*obese*	*smoking*
Total						
ME on Prob(y>0 | X)	-0.0000	0.0063	0.0002	-0.0091	0.0344	0.0056
	(0.0008)	(0.0006)**	(0.0006)	(0.0086)	(0.0085)**	(0.0180)
ME on E(y | X)	-2.0	1,373.5	647.1	-112.5	4,428.3	1,518.7
	(33.8)	(32.5)**	(24.4)**	(165.6)	(885.4)**	(956.4)
Corr($\varepsilon_1, \varepsilon_l$)				0.088	-0.413**	-0.066
Corr($\varepsilon_2, \varepsilon_l$)				0.008	-0.217**	-0.092
Admission						
ME on Prob(y>0 | X)	0.0024	0.0413	0.0258	-0.0458	0.0713	0.1970
	(0.0027)	(0.0023)**	(0.0019)**	(0.0225)	(0.0024)**	(0.0416)**
ME on E(y | X)	-14.6	564.4	303.2	-184.9	3,783.1	1,702.0
	(34.6)	(28.4)**	(24.3)**	(413.2)	(137.5)**	(1,005.5)
Corr($\varepsilon_1, \varepsilon_l$)				0.084	-0.050^	-0.319**
Corr($\varepsilon_2, \varepsilon_l$)				-0.050	0.422^	-0.030
ED						
ME on Prob(y>0 | X)	-0.0141	0.0262	0.0161	-0.163	0.0891	0.1184
	(0.0020)**	(0.0018)**	(0.0015)**	(0.004)**	(0.0248)**	(0.0273)**
ME on E(y | X)	-9.4	19.2	13.5	-71.3	53.0	75.9
	(1.5)**	(1.6)**	(1.10)**	**(14.0)****	(45.0)	(36.5)
Corr($\varepsilon_1, \varepsilon_l$)				0.580**	-0.143*	-0.268**
Corr($\varepsilon_2, \varepsilon_l$)				-0.675**	0.0363	-0.006
Out-of-hospital						
ME on Prob(y>0 | X)	-0.0003	0.0069	-0.0017	0.0056	0.0396	0.0013
	(0.0009)	(0.0007)**	(0.0007)*	(0.0084)	(0.0089)**	(0.0303)
ME on E(y | X)	4.6	614.0	267.6	11.3	1,378.0	489.8
	(14.0)	(13.8)**	(10.6)**	(74.8)	(221.7)**	(373.9)
Corr($\varepsilon_1, \varepsilon_l$)				-0.059	-0.408**	-0.031
Corr($\varepsilon_2, \varepsilon_l$)				0.002	-0.135**	-0.056

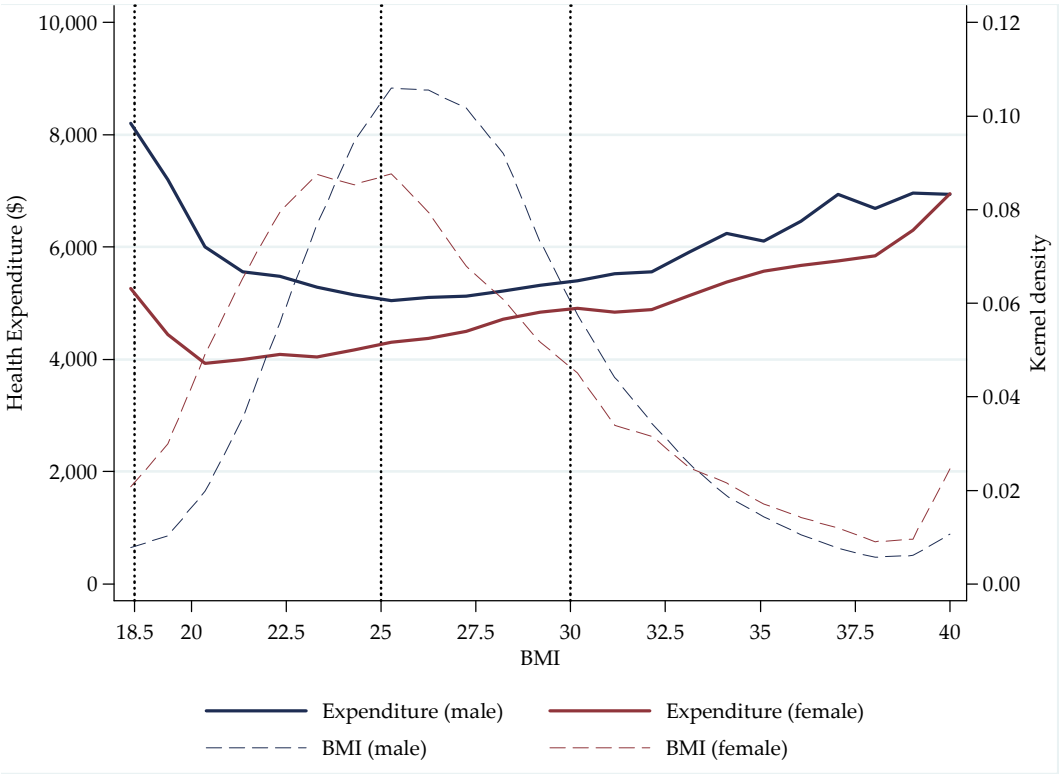
Note: ME stands for marginal effects. * and ** indicates significance at 1% and 0.1%, respectively. ^ these coefficients are constrained due to the presence of multiple maxima, some of which are associated with unexpected correlation coefficients. This model has statistically identical log likelihood value level than the unconstrained model at the 0.1% level; the log likelihood value from the restricted model is tested using an LR test with 2 degrees of freedom. Standard errors in parentheses. Bootstrapped standard errors with 100 reps are used for the marginal effects on E(y| X) in the exogenous model. Because of computation burden, bootstrapped standard errors with 100 reps on a random 25% of the sample are used for the marginal effects on E(y| X) in the endogenous model. Corr($\varepsilon_1, \varepsilon_l$) is the correlation coefficient between the error term in the positive expenditure equation and the error term in the lifestyle l equation. Corr($\varepsilon_2, \varepsilon_l$) is the correlation coefficient between the error term in the expenditure level equation and the error term in the lifestyle l equation.

Figure 1: Alcohol consumption and average health expenditure



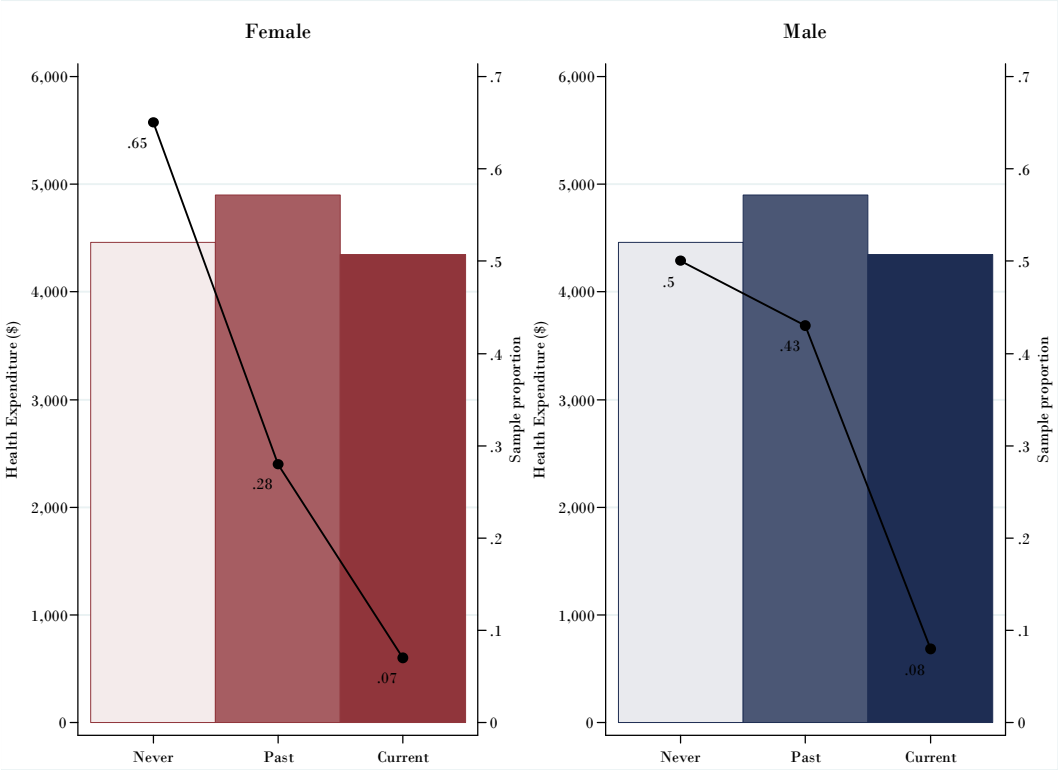
Note: the full sample size is 111,818 males and 125,143 females. Sample weight is used which reflects the 45+ NSW population by region due to oversampling in regional areas. The length of the bar graphs indicate weighted average of health expenditure (left axis) and the connected lines indicate prevalence of each group (right axis). 'Low' risk means 1-7 standard drinks per week, 'Medium' risk means more than 7 to 14 drinks per week and 'High' risk means more than 14 drinks per week.

Figure 2: BMI prevalence and average health expenditure



Note: the full sample size is 111,818 males and 125,143 females. Sample weight is used which reflects the 45+ NSW population by region due to oversampling in regional areas. The three dotted vertical lines mark 4 regions: underweight (<18.5), normal (18.5 – <25), overweight (25 – <30) and obese (≥30). The four lines reflect smoothed weighted sample averages of health expenditure across BMI distribution (solid lines, left axis) and BMI prevalence (dashed lines, right axis) using Gaussian weights.

Figure 3: Smoking behaviour and average health expenditure



Note: the full sample size is 111,818 males and 125,143 females. Sample weight is used which reflects the 45+ NSW population by region due to oversampling in regional areas. The length of the bar graphs indicate weighted average of health expenditure (left axis) and the connected lines indicate prevalence of each group (right axis).