EMPIRICAL ESSAYS IN HEALTH ECONOMICS

Van-Hai Nguyen

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Abstract

Empirical Essays in Health Economics

Van-Hai Nguyen, Ph.D.

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This thesis comprises four essays on important public health issues. The first essay studies how social interactions can spread petty corruption in the health sector. Using a Vietnam dataset, I find that social interactions measured by advice on hospital choice increase the propensity of patients to give bribes to hospital staff as well as raises the bribe amount. There is also evidence on the information-transmitting role of social networks.

The second essay evaluates long term health impacts of Agent Orange exposure in the VietnamWar on the Vietnamese population. I use a unique dataset that includes both self reported hypertension and objectively measured blood pressure. The results indicate that exposure to Agent Orange significantly increases the risk of having hypertension and reduce height, with the largest burden falling on the cohort born during the spraying period and on the most heavily sprayed areas. I also show that using self-reported hypertension data may lead to upward bias in the estimate of the effects of Agent Orange on hypertension. There is also evidence that

exposure to Agent Orange and herbicides during the Vietnam War increases risk of cancer and mental illness.

The third essay investigates a possible link between hypertension and happiness by examining possible impacts of neighborhood wealth on individuals' hypertension. Using *both* self-reported and objective hypertension data to proxy for happiness, I find that self-reported hypertension rate is much lower than objectively measured hypertension rate which lead to a large discrepancy between results obtained from self-reported and objective hypertension data. Moreover, I find that high neighborhood wealth raises hypertension risks for people aged 55-65 and not for younger or older age groups.

The fourth essay provides a theoretical rationale for smoking bans by proposing a theoretical model of maximizing behaviour on the part of smokers. It also empirically evaluates effects of smoking bans imposed at home and in workplace. Both calibrated model simulations and empirical results suggest that, with the exception of heavy smokers, workplace bans have relatively minor impacts on smokers while restrictions on smoking in the home are found to be of an order of importance greater.

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I dedicate this dissertation to my parents, my wife, my brothers and sisters for their love and support.

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Chapter 1

Introduction

This thesis investigates four public health issues: petty corruption in the health sector, the long term health impacts of Agent Orange exposure in the Vietnam War, the relationship between happiness and hypertension, and the effects of smoking bans on smoking behavior. The main approach is empirical, involving micro data and regression analyses.

To outline the methodology and empirical findings in this thesis, the first essay studies how social interactions can spread petty corruption in the health sector. To deal with difficulties with the identification of social networks, I use a unique measure of social interactions, i.e. social advice on hospital choice. I also address omitted variables by including a rich set of controls and deal with reverse causality by removing ex-ante bribery and gratitude motives. Finally, I use instrumental variable techniques. I find that advice on hospital choice increases the propensity of patients to give bribes to hospital staff as well as raises the bribe amount. I also find evidence on the information-transmitting role of social networks.

The second essay evaluates long term health impacts of Agent Orange exposure in the Vietnam War. This study has three main characteristics. First, I study the impacts on the Vietnamese population, rather than on a small sample of Vietnam veterans. Second, I investigate not only hypertension but also the height. Third, I employ a unique dataset that enables me to use both self reported hypertension and objectively measured

blood pressure. The results indicate that exposure to Agent Orange significantly increases the risk of having hypertension and reduce height, with the largest burden falling on the cohort born during the spraying period and on the most heavily sprayed areas. I also show that using self-reported hypertension data may lead to upward bias in the estimate of the effects of Agent Orange on hypertension. Finally, there is evidence that exposure to Agent Orange and herbicides during the Vietnam War increases risk of cancer and mental illness.

The third essay investigates a possible link between hypertension and happiness by examining possible impacts of neighborhood wealth on individuals' hypertension. Using *both* self-reported and objective hypertension data from a unique Vietnam health survey to proxy for happiness, I find that self-reported hypertension rate is much lower than objectively measured hypertension rate which lead to a large discrepancy between results obtained from self-reported and objective hypertension data. Moreover, I find that high neighborhood wealth raises hypertension risks for people aged 55-65 and not for younger or older age groups. One possible explanation for this result is that people care about relative wealth only when they reach ages around retirement.

The fourth essay, coauthored with Ian Irvine, provides a theoretical rationale for smoking bans by proposing a theoretical model of maximizing behaviour on the part of smokers. It also empirically evaluates effects of smoking bans imposed at home and in workplace. Both calibrated model simulations and empirical results suggest that, with the exception of heavy smokers, workplace bans have relatively minor impacts on smokers while restrictions on smoking in the home are found to be of an order of importance greater.

To the best of my knowledge, the thesis makes the following contributions to the literatures. The first essay is the first to investigate the role of social interactions in the context of corruption. It also represents a methodological break from the traditional literature on social network effects. Instead of using the average behavior of neighbor as an independent variable to detect the total social network effects which embed both the information and social norms channels, I use social advice to capture actual, direct social interactions. This goes a long way toward eliminating several difficulties with the identification of social networks effects and, at the same time, establishing the information transmitting role of social network through the workings of the word-of-mouth channel. In addition to these main contributions, this study also contributes to the literature on bribery in a health sector by studying a rich list of determinants of bribery behaviour using Vietnam data and sheds new light on the spreading mechanism of petty corruption in Vietnam.

Meanwhile, the second essay is the first population-based study to investigate long term health impacts of Agent Orange used in Vietnam War. Further, I use both self-reported hypertension and objectively-measured blood pressure data and show that the use of self-reported hypertension information might lead to upward bias in the estimate of the effects of Agent Orange exposure on hypertension. Lastly, this is the first study to investigate consequences of Agent Orange exposure on hypertension and height.

The contribution is the third essay is two-fold. First, I employ a unique dataset that enables us to document a large discrepancy in the results using self-reported hypertension and measured hypertension. Second, to the best of my knowledge this is the first to study the effects of relative wealth on hypertension, thus contributing to the literature on utility of relational goods as well as the literature on inequality and health.

Chapter 2

Social Interactions and the Spread of Corruption: Evidence from the Health Sector of Vietnam

2.1 Introduction

Social interactions[1] have been shown to have important effects on several social and economic phenomena. In some cases, social interactions can magnify good outcomes. One example is that those who live in a community with high health care use the benefit from information in their networks on available health care services (Deri, 2005). In the context of technology adoption, new technology is tried by some persons and spread around through social networks (Bandiera, 2006). In some other cases, however, social interactions can create undesirable outcomes. For example, in a poor area, where the disadvantaged interact mainly with the disadvantaged, networks can inhibit upward mobility and reinforce the poverty trap. This can happen because contacts in the network supply more information about welfare eligibility than job availability (Bertrand *et al*, 2000). In the drug context, social networks may provide negative peer pressure, leading to higher drug use among youth (Clark, 2007).

[1] Another frequently-used term is social networks. In this paper, I use these two terms interchangeably.

In this chapter, I consider the role of social interactions in spreading corruption. Specifically, in the context of medical treatment in hospitals in Vietnam, I ask the question: 'Do patients whose choice of hospital is driven by advice from social contacts, such as friends or medical staffs, have a higher tendency to bribe and/or bribe a larger amount than patients who choose a hospital by themselves?'

On the face of it, advice on hospital choice from friends or from medical institutions (advice for short, hereafter) may be a good thing for patients because it supplies information about making the best choice of hospital. But it may go as far as offering information on bribery to obtain better care from medical staff for better treatment outcome. As a result, well-meaning advice that is seemingly optimal for both the advice giver and the advice receiver has the externality of increasing bribery behaviour, and consequently, spreading petty corruption in the Vietnam health sector.

The motivation for this hypothesis is my observation that in Vietnam, people often 'teach' each other how to get around the red-tape or administrative rules and rigidities[2]. Understanding this mechanism of spreading corruption behavior will provide new insights for policymakers in designing strategies against petty corruption which is widespread in Vietnam. This is an important issue since petty corruption causes serious damage to the image of a country and also creates an atmosphere that is conducive to other forms of corruption.

To establish the causal effects of advice on bribery behaviour of patients, I carefully address the issue of endogeneity of advice using a control function approach and dealing with the reverse causality issue. Further, I use instrumental variable

[2] This can be seen in several contexts in Vietnam. For example, giving bribes to policemen when being caught for violating traffic rules is a common knowledge.

techniques. I also explore the mechanism through which advice leads to bribery behaviour using an interaction term that captures the idea that those advice receivers in areas of higher average bribery are more likely to bribe than those in areas of a lower average rate of bribery.

To preview the results, I find evidence that advice on hospital choice makes people more likely to bribe medical staff and to bribe more. One channel of causal effect is through word of mouth. That is, advice contains information on bribery to medical staff for better treatment. In addition to establishing the causal effect of advice and its mechanism, my analysis delivers other interesting findings. First, those who are insured are subject to less bribery pressure. This is probably because they are given a different route to registration and payment in hospital. Second, loan givers may influence the bribery behaviour of loan takers. Third, bribery is a regressive tax.

This study makes two important contributions. First, to the best of my knowledge, this is the first study that investigates the role of social interactions in the context of corruption. Second, this work represents a methodological break from the traditional literature on social network effects. Instead of using the average behavior of neighbour as an independent variable to detect the total social network effects which embed both the information and social norms channels, I use social advice to capture actual, direct social interactions. This goes a long way toward eliminating several difficulties with the identification of social networks effects and, at the same time, establishing the information transmitting role of social network through the workings of the word-of- mouth channel. In addition to these main contributions, this study also contributes to the literature on bribery in a health sector by studying a rich list of determinants of bribery behaviour using Vietnam data and sheds new light on

the spreading mechanism of petty corruption in Vietnam.

The rest of the chapter is organized as follows. Section 2.2 offers a brief review of related literature on corruption in the health sector and of social network effects, and also highlights differences of my approach. Empirical strategy and data are discussed in Sections 2.3 and 2.4. Regression results are presented in Section 2.5. Section 2.6 studies a mechanism through which social advice leads to bribery behaviour. Section 2.7 offers a discussion of the results. The conclusions are summarized in Section 2.8.

2.2 Literature review

2.2.1 Corruption and bribery in the health sector

Corruption has been extensively studied at the macro level. It has been shown to cause lower growth (Mauro, 1995) and lower foreign direct investment (Wei, 2000). In terms of causes, corruption across countries has been linked to flaws in the legal, political and fiscal systems. One main feature of these studies is that they largely rely on perceptions of corruption, rather than actual, measured corruption[3].

Recent empirical studies on corruption use individual-level data (either from micro data surveys or from experiments) to shed light on several aspects of corruption. Niehaus and Sukhtankar (2008) study dynamic incentives for corruption in one of the world's largest public transfer programs, India's National Rural Employment Guarantee Act. They measure the corruption as the discrepancy between the official data on

[3] This is understandable because data on corruption are either unobtainable or unreliable, and corruption in general does not lend itself to straightforward data collection.

payments to the workers and the data from surveying the workers themselves. They find evidence of "golden goose" effect. That is, when expected future opportunities for rent extraction are high, officials extract less rent today in order to preserve tomorrow's opportunities. As to experimental studies, Bertrand *et al* (2007) set up a field experiment to understand the structure of corruption in process of obtaining a driving license in Delhi. They randomly assign applicants who are aiming to get a driving license into one of three groups: one which receives a bonus for obtaining a driving license fast, one that gets free driving lessons, and a control group. They find that those in the "bonus" group get their licenses faster, but those who get the free driving lessons do not. Alatas *et al* (2006) conduct laboratory experiments in Australia, India, Indonesia, and Singapore to investigate if there exist gender differences in the acceptability of corruption, and to see if they differ between countries. They find that gender differences in attitudes towards corruption are not universal and may be more culture-specific. They also find that there are larger variations in women's attitudes towards corruption than in men's. This study share a common feature with these experimental studies in that I also looks at 'victim' side of corruption (in contrast to 'public official' side). Understanding the behavior or attitudes of people regarding corruption will contribute to our understanding of the severity, the persistence as well as the spread of the corruption.

In the health context, the issue of petty bribery made by patients has attracted a lot of attention[4]. As noted in Lewis's (2006) survey paper, patients' bribery is increasingly common and is directly related to welfare of patients. Using micro

[4] In Transparency International's Global Corruption Barometer (2004) most respondents considered "petty or administrative" corruption almost as serious a problem as "grand or political corruption".

data in the health sector from Peru and Uganda, Hunt (2007a, 2007c) investigates whether bribery is a progressive tax and whether the presence of a private sector affects bribery behaviour. She finds that rich patients are more likely to bribe in a public health care system. However, she does not find evidence that competition from the private health sector reduces bribery in the public sector. In a broader setting, Hunt (2007b) examines another equity aspect of bribery by looking at how corruption hits people when they are vulnerable. She shows that victims of misfortune, particularly crime victims, have a higher demand for public services, thus increasing victims' propensity to bribe officials, because victims are desperate and vulnerable, or demand services that are particularly prone to corruption.

This study contributes to the literature on bribery in a health sector by investigating the mechanism through which bribery behavior is spread. Specifically, I consider whether advice on hospital choice makes people more likely to bribe medical staff and bribe more. In addition to studying the role of social advice on spreading corruption, I also examine an expanded list of determinants of bribery behavior, including length of treatment, whether the patient takes a loan or not, whether the patient is insured or not, and the size of hospitals. I also consider the issue of progressivity of bribery and whether the presence of private health service providers affects bribery behavior.

2.2.2 Social network effects

Social networks have been studied in various contexts including: job search (Montgomery, 1991); education (Coleman *et al.* 1966); consumption (Abel, 1990); unemployment (Akerlof, 1980), technology adoption (Bandiera 2006); home

computer use (Goolsbee et al. 1998); retirement plan participation (Duflo and Saez, 2003); welfare use (Bertrand *et al.* 2000); health care use (Deri, 2005); prenatal care use (Aizer Anna and Janet Currie, 2004); and cigarette smoking (Cutler and Glaeser, 2007).

There are two important issues that concern the empirical literature on social networks. The first issue is to establish the causal effects of social networks on individual behavior. The second issue, which has received less attention, is to identify the channels through which social networks have an effect.

In studying the effects of social networks on individual behavior, one may use as an independent variable the average behavior calculated for the neighborhood in which individual resides and individual behavior as the dependent variable. A positive estimate of the coefficient on the average neighbourhood behaviour, however, cannot be interpreted as evidence of the causal effect of a social network on its individual members. Manski (1993) pointed out two main issues with this regression that make identification of social network effects difficult. First, this regression suffers the so-called reflection problem. That is, it is difficult to distinguish the *endogenous social interactions* (the effects of interest) from the *contextual effects* (the impacts of exogenous characteristics of the reference group on the individual behavior). For example, a person's school performance may be affected not just by his peer group's average academic performance but also by the exogenous characteristics of the peer group's parents. Second, there is self-selection based on unobserved personal characteristics and unobserved neighbourhood characteristics that drive both the average behaviour of the neighbourhood and the individual outcome. In this case, what I obtain is *correlated effects*, not endogenous social network effects. As an

example, in the context of welfare use, people with unobserved characteristics that increase welfare participation may disproportionately choose to live in high welfare participation areas. Hence, the observation that neighbourhood welfare participation rates are correlated with individual welfare participation may simply reflect omitted personal or neighbourhood characteristics, rather than a causal relationship.

Several empirical papers attempt to address these above identification issues using different approaches[5], including instrumental variable analysis, the use of panel data or some creative identification strategies. For example, by instrumenting for the average ownership of an individual's community with lagged average ownership of the states in which one's non-native neighbors were born, Brown *et al* (2007) establish a causal relation between an individual's decision on whether to own stocks and average stock market participation of the individual's community. Clark *et al* (2007) employ a panel data which enables them to use lagged peer-group behaviour as a right-hand side variable to examine risky behaviour (the consumption of tobacco, alcohol and marijuana) by American adolescents. As such, they avoid one aspect of the identification problem: while individual behaviour may depend on what his peers did in the past, their past behaviour cannot depend on what individuals are currently doing. Bertrand *et al* (2000) interact areas with language groups, and exploit the differential effects of increased contact availability across language groups within an area to identify the causal effects of social networks on welfare use. Hoxby (2000) exploits variation in gender composition between adjacent cohorts within a grade within a school to identify gender peer effects. This use of idiosyncratic variation

[5] Blume and Durlauf (2005) provide an excellent review of the recent literature on identification of social interactions effects.

convincingly overcomes the problem of unobserved variables associated with self selection or selection by other forces into a particular class, within a grade within a school, which plagued previous studies in school peer effects.

In terms of the channels of network effects, Bertrand *et al.* (2000) describe two channels through which social networks affect individual behaviour: information and norms. The informational channel emphasizes how a person's behaviour depends on the behaviour of others, operating mainly through information sharing. The social norms channel operates through peer pressure, stigma, or social approval. Almost all studies focus on identifying social network effects and rarely deal with the issue of separating these two channels although these two channels clearly have different policy implications.

A unique feature of my approach to studying social network effects in this chapter is the use of social advice which captures actual and direct social interactions (that is, advice one receives from direct social interactions with others). This is to be contrasted with the traditional use of average neighbourhood behaviour which represents expected, indirectly observable behaviour[6]. To see the difference between this study's approach and the traditional approach in our context of health bribery, the latter would involve using the average rate of bribery in a person's residence area and regress his bribery behavior on this average bribery rate. In my set-up, the independent variable of interest is not the average rate of bribery but the advice arising from social interactions, and I regress individual bribery behavior on this social advice variable.

[6] As emphasized in Manski (2000), social interactions can take place not only through extracting information from observation of actions or outcomes experienced of others (normally proxied by average group behaviour) but also through obtaining information directly from one another. It is this class of actual and direct social interactions that our variable social advice seeks to capture.

The use of advice yields several advantages. First, by capturing the actual and direct interactions, the use of social advice is less likely to suffer from the reflection problem than the use of average behaviour of the whole neighbourhood. In other words, the advice seekers are more likely to be influenced by the advice itself than by the exogenous characteristics of advice givers.

Second, it helps to eliminate many concerns about unobserved neighborhood characteristics which are at the core of the correlated effects. In principle, I can deal with the issue of unobserved neighborhood characteristics by including area fixed effects, which account for differences between areas. However, this is not feasible when one works with the traditional measure of average behaviour in the area. The reason is that when one adds area dummies into the regressions, there would be no variation in average behaviour of the neighbourhood at the same area level to exploit for identification of social network effects. With the use of advice, I essentially move from an area-level measure of social interactions to individual-level of social interactions which allows us to include area dummies to control for area fixed effects and, at the same time, exploit within-area variation in the dummy for advice for identification purpose.

Third, the use of advice enables us to focus on the informational channel of a social network which has been overlooked in the literature. This channel seems to be very relevant in the context of a developing country where lack of information is a major problem. This focus on the information channel may also help alleviate identification concerns. Unlike the norms channel (in which a person's behaviour is influenced by preferences of a large social group), the informational channel (which is based on interpersonal interactions and information flows) is less likely to

suffer from the self selecting into the neighbourhood.

Fourth, unlike the traditional measure of average behavior of a neighborhood, the use of social advice allows us to capture social interactions of several different types which include not just face-to-face interactions but also contacts over the phone or Internet. In the age of the Internet, neighbourhood effects based on geographical proximity might not be sufficient to capture all social interactions. Further, the use of advice allows us to take into account the social influence from those who never bribe. That is, there are people who never bribe medical staff but heard about it and are keen to give advice that has this information. If I use the traditional measure, i.e. the average rate of bribery in the area, then I cannot capture this aspect of social interactions, which seems quite popular in a developing country like Vietnam.

Fifth, the use of social advice on hospital choice (not social advice on bribing) is also interesting in the sense that I can explore the negative externality of a well-meaning social phenomenon, i.e. asking and giving advice on hospital choice.

While the use of social advice helps tackle several problems associated with the use of average group actions such as the reflection problem and self selection based on unobserved neighbourhood characteristics, there is still the issue of self-selection associated with social advice itself. In our current context, it is the patients' self selection into advice seeking. I will explain how I deal with this self selection issue in the next section.

One study that is most related to ours is Harrison *et al* (2004). This study shows that stock-market participation is influenced by social interaction captured by

the indicator of whether a household is social or not. However, they do not make a distinction between the informational channel and the norms channel. More importantly, they do not control for two types of endogeneity in their study. First, they don't address the issue of reverse causality, i.e. those who participated in the stock market might want to socialize more to obtain information that will help them make their investment decisions. Second, as pointed out in their study, there might be a possibility that social households are better listeners which in turn makes them more likely to participate in the stock market.

2.3 Empirical strategy

To study the impacts of advice on an individual's bribery behavior, I run two regressions: one regression studies the bribery participation decision and the other investigates the bribery amount for those who have participated in bribery. Formally:

$$Bribery_i = \tau + \alpha * Advice_i + \boldsymbol{X} * \beta + e_i \qquad (2.1)$$

$$log(BribeAmount_i) = \mu + \lambda * Advice_i + \boldsymbol{X} * \delta + \eta_i \qquad (2.2)$$

where $Bribery_i$ is indicator of whether patient i bribed the medical staff. $Advice_i$ is 1 if patient i chose hospital on the advice from others, and 0 if he chose the hospital by himself, $BribeAmount_i$ is bribe amount given by patient i, $\boldsymbol{X}$ is a vector of control variables which I will discuss in detail later.

The principal variable of interest in regressions (2.1) and (2.2) is the dummy *advice*. A positive coefficient on this dummy in (2.1) would mean that those who choose a hospital based on the advice from others are more likely to bribe medical staff than those who choose the hospital of treatment themselves. A positive coefficient in (2.2) means that among individuals who engage in bribing, those with advice tend to bribe more. Both regressions are estimated by OLS. There are two main reasons why I estimate the first regression, a binary choice model, by OLS. First, in presence of a large number of area dummies and categorical variables, it is computationally simpler. Second, since my model includes interaction terms, the marginal effects of advice and other variables are easier to interpret in the linear probability model than the probit model[7]. In the second regression, I use bribe amount in log form to account for skewness in the data.

2.3.1 Selection Issue and Endogeneity of Advice

The main econometric challenge in estimating the causal effect of advice on bribery behavior is patients' self-selection into seeking advice. That is, there might be some characteristics of patients that both drive them into seeking advice as well as bribing behavior. If this is the case, the identified relationship between advice and bribery behavior would be spurious.

[7] Ai, Norton and Wang (2004) point out that the marginal effects in probit model with interaction terms may be of opposite sign to that of the coefficient on the interaction terms. That is, marginal effects depend on the values of the covariates and the associated coefficients are often misleading.

There are several factors specific to patients that can drive self-selection. For example, rich patients tend to value health more and thus seek advice on hospital choice. They are also those who have higher ability to bribe than poor patients. Failing to account for this will overestimate coefficient on the variable *advice*. Or patients who are risk averse tend to ask for advice before making their choice of hospital. They are also careful enough to bribe medical staff in the hope of obtaining better attention from them for better treatment. Leaving out risk aversion will overestimate effect of hospital choice advice on bribery. Finally, the more severe the illness of the patient, the more likely it is that he seeks advice on hospital choice and pays bribery to medical staff. Failing to take into account severity of illness therefore will overestimate the advice coefficient.

Omission of other variables specific to hospital or geographical areas also makes variable *advice* endogenous. For example, large hospitals often get recommended. At the same time, medical staff in these big hospitals may require larger bribes than smaller hospitals.

These two things can lead to the spurious finding of positive effect of advice on bribery even though the advice itself has no causal effect on bribery behavior. It is also likely that different areas have different cultures regarding preference for modern versus traditional treatment, which drive both advice seeking and bribery behavior. Or some areas may have a high socialization and more kindness in paying the doctors than others.

2.3.2 Dealing with Endogeneity of Advice

In principle, one can deal with the selection issue and establish the causal effect of social advice by working with a sample that contains those who don't self-select into asking for advice. However, such random samples are difficult to obtain.

Instead, I deal with endogeneity of advice in a number of ways. First, I adopt the control function approach which aims to control for all possible factors that are likely to drive patients' self-selection into advice. This approach is likely to be effective in this case because our data are rich with information on areas, hospitals, and individuals and thus allows us to control for area, hospital and individual level characteristics that may affect both advice and bribery behavior, as discussed above. Specifically, at the individual level, I include 'education' and 'consumption' to capture wealth of patients.

To capture patient's risk aversion, I add an indicator of whether a patient is insured[8]. I also add 'length of staying in the hospital' to capture a patient's illness severity and a dummy indicating whether a person takes a loan to pay for the treatment to take into account the possibility that those who ask others for advice on hospital choice also ask for a loan. This loan in turn will ease the budget constraint of the patient thereby enabling the patient to make a bribe.

At the hospital level, I include a dummy for hospital size (large or small). By large, I mean a hospital at the provincial or regional level. Hospitals and medical centers at a lower administrative level are classified as small. As noted,

[8] Another reason for including insurance status is because insurance holders follow a different route of registration, which make incentives for them to pay bribe different from those without insurance.

size of hospital is included because large hospitals may be more likely to get recommended and their staff more likely to demand larger bribes, because of its advantages in technology or quality of treatment. At the area level, I add provincial dummies to capture area fixed differences. As noted, some provinces may have the culture of high socialization and more kindness in paying the doctors than others.

Going beyond adding controls that are likely to drive patients' self-selection into advice, I also deal with two other causes of endogeneity of advice, namely reverse causality and remove the gratitude motive in giving money to doctors. Lastly, I address the endogeneity issue using the instrumental variable techniques. Conditional on finding good instruments for the indicator *advice* that are sufficiently correlated with advice and at the same time do not affect bribery behaviour (that is, are not correlated with the error terms), this method will deliver an estimate of the causal effects of advice. I will discuss my choice of the instruments and tests of their validity later on.

2.4 Data and Summary Statistics

The data used in this study are from the Vietnam National Health Survey (VNHS) 2001-2002. This nationally representative survey consists of 36,000 households with 158,000 individuals. It has information on demographics as well as on health status and health behavior of individuals and their households. It also covers characteristics of respondents' living areas and of health care system at community, district and provincial levels.

Of our special interest is the section that covers hospitalized patients in the last 12 months[9]. For this group of respondents, I am interested in two types of information: first, if they make a bribe to medical staff and second, if they chose their hospital for treatment by themselves or through advice from others. Bribery is defined as payments to medical staff outside official payment. I construct an indicator of bribery from responses to the question: 'Did you make an outside payment to doctors and nurses?' I note that this payment may include voluntary payment, probably out of gratitude, and payment in response to requests from medical staff. Later on, I explicitly distinguish between payment with a gratitude motive and payment with no such motive. The indicator of advice is constructed from patients' responses to the question: 'Who recommended this hospital to you?' It takes value 1 if the response is that the patient chose the hospital on advice from friends, relatives or medical institutions, and 0 if they chose the hospital by themselves. I then combine the data on inpatients with data on their socio-economic characteristics, their experience with the health care system and information on the hospitals they used. Our final sample contains 9,861 observations covering patients from 1,200 communes (the smallest administrative area in Vietnam) belonging to 61 provinces from 8 regions in Vietnam.

[9] Hunt (2007a) uses a sample (of size 12,262 from 2002 and 2003 waves of the Peruvian household survey) that includes both hospitalized patients and outpatients, probably because her sample would be too small if she only used hospitalized patients. Our dataset also has a sample of outpatients of the size 29000, but this group of patients has little bribery activity (around 2%). Another reason for our focus on inpatient sample only is that the issue of social interaction for advice on hospital choice does not arise for outpatients who are not seriously ill.

The first two tables present summary statistics regarding the decision to bribe or not and the amount of bribery for those who engage in bribing, respectively. Table 2.1 shows that 23% of inpatients pay bribes to medical staff. The group of bribers has a higher rate of receiving advice on hospital choice, has a higher income and education, and stays in the hospital longer.

Table 2.2 breaks down bribery amount by different categories of bribers. Those who receive advice bribe, on average, more than those who don't. By expenditure groups, those in the highest quantile bribe the most. Urban people pay more than those who live in rural area. More educated people bribe more, except for those with elementary schooling who pay less than those who are illiterate. One interesting result is that those who bribe after treatment bribe much less than those who bribe during the treatment who in turn pay less than those bribing before treatment. I will exploit this information to strengthen my identification strategy later. Table 2.3 sheds some initial light about the regressivity of bribery: although the rich pay more than the poor, bribery accounts for a larger proportion of the poor's income than the rich's. This implies that bribery is regressive.

Table 2.1 Means of key variables for bribery participation decision

Variables	All	Bribery	No bribery
Bribery	0.23	1	0
Advice	0.31	.36 (.48)	.29 (.45)
Length	8.48	10.31 (13.19)	7.94 (9.84)
Insurance	0.236	.24 (.43)	.23 (.42)
Distance	25.1	31.74 (88.30)	23.12 (67.64)
Large hospital	0.78	.83 (.37)	.77 (.42)
Education (5 levels)	3.32	3.73 (1.39)	3.21 (1.41)
Loan	0.32	.35 (.47)	.32 (.46)
Urban	0.34	.37 (.48)	.33 (.47)
Age	36.15	35.78 (21.28)	36.26 (23.35)
Married	0.69	.76 (.42)	.67 (.47)
Gender	0.43	.37 (.48)	.45 (.49)
Expenditure (1000 VND) (per capita in the household)	3816.79	4326.93 (3963.34)	3667.45 (3788.23)
Observations	9861	2233	7628

Note: Standard deviations are in parentheses.

Table 2.2 Means of key variables for bribery amount decision

	Bribery amount (1,000 VND)
Consumption quintile	
First	105.47
	(275.87)
2nd	99.11
	(143.08)
3rd	108.96
	(159.81)
4th	139.92
	(236.60)
5th	198.27
	(436.26)
Advice	
Yes	164.28
	(383.57)
No	120.92
	(207.85)
Urban	
Yes	168.98
	(354.20)
No	117.27
	(232.09)
Education	
Less than elementary	112.91
	(184.78)
Elementary	99.04
	(154.85)
Secondary	143.08
	(394.24)
High school	182.57
	(321.56)
More than high school	183.91
	(266.06)
Timing of bribery	
Before treatment	213.19
	(326.42)
During	139.43
	(377.15)
After	96.43
	(149.73)

Note: Standard deviations are in parentheses.

Table 2.3 Regressivity of bribery

Expenditure quintile	Income	Bribery amount	Bribery /Income
First	1794.28	105.47	0.059
		(275.87)	
2nd	2437.0493	99.11	0.04
		(143.08)	
3rd	3061.4504	108.96	0.035
		(159.81)	
4th	3981.3939	139.92	0.035
		(236.60)	
5th	8232.0605	198.27	0.024
		(436.26)	

Notes: Standard deviations in parentheses

2.5 Empirical results

2.5.1 Baseline Regressions

The econometric analysis starts with a basic specification that includes only an indicator for advice and the demographic variables such as age, gender, urban and education. I then add explanatory variables to address the endogeneity of the dummy *advice* discussed above. Sample weights are incorporated to provide nationally representative estimates and the standard errors are clustered at provincial level. I discuss results from bribery participation and bribery amount regressions in turn.

2.5.1.1 Bribery Participation

Table 2.4 presents the results on marginal effects of covariates in the bribery participation regression. Column 1 of Table 2.4 presents results from the baseline regression. Advice has a positive and significant impact on bribery participation. Specifically, those who receive advice are more likely to bribe by 7.6 percentage points. Except for urban, other demographic variables are significant. Women are more likely to bribe than men. This may reflect female's higher concern about health. Age has a negative effect on bribery propensity, although this effect is small. Education enters as dummies relative to the category of illiterate. Education has increasing nonlinear impact on bribery. The higher education you have, the more likely you are to bribe. In column 2, I include provincial dummies into the regression. This aims to control for fixed differences across provinces that can cause part of the variation in bribery behavior of patients. The magnitude of the advice coefficient drops (from

0.076 to 0.067) but it is still positive and significant. I notice a large drop in effect of education, more than 50% at each level of education. There is little change in age, marital status and gender. *Urban* has now become significant.

I next include log of expenditure by capita in the family which is a proxy for individual income[10]. The positive and significant coefficient on this variable indicates that the higher is income the greater the propensity to bribe. This is expected because people with higher income value their health more and are willing to pay money to obtain better treatment. This result is consistent with Hunt (2007) that higher expenditure levels increase the likelihood of bribing. Education continues to decrease in its impact. The indicator *urban* is again insignificant. Importantly, the advice coefficient is still significant and positive with little change in magnitude.

Column 4 includes length of treatment. Column 4 shows that the coefficient on length of treatment is positive and statistically significant, meaning that the longer one stays in hospital, the more likely the person is to bribe. The advice coefficient drops to 0.057 but is still significant. This suggests that a patient decides to bribe not just because of the influence of the advice but also because of severity of illness. There is little change in coefficients on the other variables.

Columns 5-7 include size of hospital of treatment, whether patient has been covered by insurance or not, and whether patient has obtained a loan. These characteristics of the patients might drive self-selection of patients into asking for advice and thus might confound the effects of advice. Hospital is classified as large if it is a hospital at provincial or regional level. As expected, large hospitals are associated

[10] Self-reported income is likely to be severely under-reported

with a higher propensity to bribe, as shown in column 5. This may be due to a higher demand for service from large hospitals which results in higher bribery as patients compete for better care and services. Importantly, the variable *advice* continues to be significant and positive, although the difference in the propensity to bribe (between those patients with advice and those without) is reduced to 4.4 percent. Demographic variables remain unchanged from the previous specification. Column 6 shows that those who have insurance are less likely to pay the medical staff. This is surprising, because if insurance only captures the risk aversion of the patient, I would expect this coefficient to be positive. I would also expect a positive coefficient because insurance holders are more likely to be richer than those who do not hold insurance. However, having insurance also has the effect of reducing red tape in hospital for patients since they go through a separate route of registration and other paperwork. This reduced red tape for the insured may lessen the need to bribe the medical staff. Again, the advice coefficient is significant and positive. In the last column, those who obtain a loan are more likely to bribe[11]. This is expected since the loan may ease financial constraint imposed on making bribe. More importantly, although the magnitude of variable *advice* decreases, it is still statistically significant and positive.

In summary, the baseline results indicate that adding variables accounting for confounding effects decreases the magnitude of advice. But more importantly, in all specifications advice has a positive and statistically significant effect. To the extent that the endogeneity of advice is addressed through controlling these variables that

[11] I also explore the impact of the presence of private health care services in the area. If this presence is strong, it might compete with public health providers and reduce incentives to bribe. However, the coefficient on private competition is very small and not significant.

drive self-selection into asking for advice, this positive coefficient suggests that advice has a positive *causal* effect on the propensity to bribe.

2.5.1.2 Bribery Amount

For those who choose to bribe medical staffs, the question is what determines their bribery amount. I estimate the bribery amount regressions by OLS using the sample of those who engage in bribing. As with the bribery participation regression, I start with a basic specification and include additional variables to control for confounding effects.

The results are presented in Table 2.5 which has the same format as Table 2.4. Two variables of interest are *advice* and *expenditure*. The coefficient on *advice* decreases in magnitude as the controls are added but, in the final specification, it is still positive and statistically significant. To the extent that our controls capture the confounding effects by unobserved variables, this means that the bribers under the influence of advice will bribe more than the bribers without advice.

Another important result concerns impacts of income proxied by expenditure per capita. We see from the expenditure row that the coefficient on expenditure is positive and statistically significant. This means that people with higher income give a higher bribe, which is consistent with Hunt (2007). Further, its magnitude is less than one indicating that the burden of bribery (i.e. the share of bribery in income) is larger for the poor than the rich. This implies that bribery is regressive.

Table 2.4. Bribery participation regression results

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Advice	0.076***	0.067***	0.068***	0.057***	0.044***	0.043***	0.040***
	(0.011)	(0.016)	(0.016)	(0.015)	(0.014)	(0.014)	(0.014)
Gender	-0.048***	-	-	-	-	-	-
		0.041***	0.038***	0.043***	0.044***	0.043***	0.045***
	(0.010)	(0.009)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)
Age	-0.002***	-	-	-	-	-	-
		0.003***	0.003***	0.004***	0.004***	0.004***	0.004***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Married	0.139***	0.159***	0.164***	0.176***	0.188***	0.182***	0.181***
	(0.016)	(0.025)	(0.024)	(0.023)	(0.024)	(0.023)	(0.023)
Urban	0.012	0.023**	0.010	0.008	-0.002	0.002	0.005
	(0.011)	(0.010)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
Educ2	0.066***	0.036**	0.021	0.016	0.016	0.020	0.019
	(0.014)	(0.016)	(0.016)	(0.016)	(0.016)	(0.015)	(0.015)
Educ3	0.127***	0.039**	0.010	0.003	0.000	0.009	0.009
	(0.016)	(0.018)	(0.019)	(0.020)	(0.019)	(0.019)	(0.019)
Educ4	0.146***	0.051*	0.012	0.003	-0.001	0.011	0.011
	(0.021)	(0.027)	(0.027)	(0.028)	(0.028)	(0.028)	(0.027)
Educ5	0.226***	0.131***	0.079***	0.064**	0.057**	0.081***	0.082***
	(0.023)	(0.026)	(0.029)	(0.028)	(0.028)	(0.027)	(0.027)
Income			0.072***	0.074***	0.071***	0.072***	0.083***
			(0.016)	(0.016)	(0.016)	(0.016)	(0.017)
Length				0.004***	0.004***	0.004***	0.004***
				(0.001)	(0.001)	(0.001)	(0.001)
Large					0.087***	0.090***	0.085***
					(0.018)	(0.018)	(0.017)
Insurance						-	-
						0.052***	0.046***
						(0.016)	(0.017)
Loan							0.044***
							(0.012)
Constant	0.116***	0.377***	-0.186	-0.223*	-0.248*	-0.255*	-0.353**
	(0.016)	(0.018)	(0.127)	(0.129)	(0.131)	(0.133)	(0.136)
Province dummies	No	Yes	Yes	Yes	Yes	Yes	Yes
Obs	9861	9861	9861	9861	9861	9861	9861
R-Squared	0.042	0.111	0.115	0.125	0.131	0.133	0.135

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, ** 5 percent, ***1 percent.

Table 2.5. Bribery amount regression results

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Advice	0.247***	0.258***	0.276***	0.241***	0.184***	0.184***	0.141**
	(0.057)	(0.058)	(0.069)	(0.063)	(0.059)	(0.059)	(0.056)
Gender	0.030	0.064	0.072	0.030	0.022	0.021	0.002
	(0.064)	(0.063)	(0.067)	(0.065)	(0.064)	(0.064)	(0.066)
Age	0.006***	0.004**	0.004*	0.000	-0.001	0.001	0.001
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Married	0.110	0.145	0.149	0.233**	0.294**	0.270**	0.283***
	(0.091)	(0.091)	(0.120)	(0.114)	(0.116)	(0.107)	(0.104)
Urban	0.378***	0.252***	0.184***	0.182***	0.151**	0.161***	0.201***
	(0.065)	(0.063)	(0.062)	(0.061)	(0.061)	(0.059)	(0.060)
Educ2	0.124	0.100	0.064	0.018	0.024	0.041	0.023
	(0.115)	(0.113)	(0.100)	(0.099)	(0.103)	(0.101)	(0.103)
Educ3	0.335***	0.265**	0.170*	0.107	0.099	0.139	0.115
	(0.119)	(0.118)	(0.099)	(0.097)	(0.106)	(0.102)	(0.103)
Educ4	0.513***	0.406***	0.259*	0.192	0.167	0.229	0.203
	(0.138)	(0.136)	(0.133)	(0.138)	(0.145)	(0.144)	(0.150)
Educ5	0.576***	0.489***	0.277**	0.204*	0.174	0.309***	0.292**
	(0.141)	(0.137)	(0.127)	(0.117)	(0.122)	(0.113)	(0.118)
Income			0.332***	0.347***	0.339***	0.343***	0.446***
			(0.069)	(0.065)	(0.064)	(0.064)	(0.071)
Length				0.017***	0.017***	0.018***	0.016***
				(0.004)	(0.004)	(0.004)	(0.004)
Large					0.423***	0.428***	0.380***
					(0.072)	(0.071)	(0.070)
Insurance						-	-
						0.274***	0.225***
						(0.067)	(0.062)
Loan							0.394***
							(0.072)
Constant	3.362***	3.710***	1.052*	0.842	0.608	0.553	-0.340
	(0.124)	(0.163)	(0.564)	(0.540)	(0.542)	(0.539)	(0.597)
Province dummies	No	Yes	Yes	Yes	Yes	Yes	Yes
Obs	2233	2233	2233	2233	2233	2233	2233
R-squared	0.077	0.145	0.156	0.186	0.202	0.209	0.229

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent.

The effects of other controlling variables can be seen in the lower rows. The coefficient on the variable *large* is positive and significant throughout, indicating that large hospitals require larger bribes. As to the impact of the length of treatment, the longer a patient stays in hospital, the larger is the bribe. For those who bribe, insurance decreases bribes. This provides further evidence of the advantage on the insurance holder's part. I also see that those who have access to a loan bribe a higher amount. This is expected because the loan helps ease the budget constraint of the patient. Demographic variables have the expected effects. For example, urban area people bribe more than rural area people. More educated people bribe more.

2.5.2 Reverse Causality

In the above analysis, I assume that endogeneity of the advice variable is caused by omitted variables that drive selection into seeking advice on hospital choice (and affect bribery behavior). However, endogeneity can also be caused by reverse causality from bribery behavior to advice on choice of hospital. This can happen when patients who want to bribe come to ask for advice including information on which hospital to choose.

Given that those who bribe before or during treatment bribe more than those who bribe after treatment, it would be plausible to argue that if patients have a bribery motives in the first place, which lead them to consult people on choice of hospital, then they are likely to give a bribe in advance of the treatment. Thus, I construct a sample of patients who are likely to be free from the bribery motive by excluding those patients who pay bribes before the treatment. The estimation results

using this refined sample are presented in column 2 (for the bribery participation decision) and column 5 (for the amount of the bribe) of Table 2.6. Compared with the coefficient obtained with the full sample (reproduced in column 1), the coefficients on the dummy *advice* are still statistically significant and positive.

2.5.3. Gratitude Motive in Informal Payment

Patients who pay the doctors outside official payments may aim to jump the queue, receive better or more care, obtain drugs, or just simply to gain any care at all. But they can also give money out of gratitude to the medical staff, a common practice in Vietnam. To rule out this gratitude element and further strengthen the causal interpretation of the impact of advice, I exclude individuals with this gratitude motive from our sample. Given that those who pay after treatment pay least, compared to those who pay before or during the treatment, I argue that those who have gratitude as a motive are most likely to be those who pay after the treatment. After excluding these patients from the refined sample, I am left with a sample that contains only those who bribe at some point during treatment and those who simply do not bribe. The results in Table 2.6 show that advice on hospital choice still leads to higher propensity to bribe (column 5) and higher bribery amount (column 6).

Table 2.6 Bribery & Gratitude Motives

	Bribery Participation regression			Bribery amount regression		
Variables	Full sample	Sample 1	Sample 2	Full sample	Sample 1	Sample 2
	(1)	(2)	(3)	(4)	(5)	(6)
Advice	0.040***	0.038***	0.026**	0.141**	0.182***	0.288***
	(0.014)	(0.013)	(0.013)	(0.056)	(0.061)	(0.094)
Income	0.083***	0.067***	0.034***	0.446***	0.375***	0.394***
	(0.017)	(0.017)	(0.012)	(0.071)	(0.074)	(0.132)
Length	0.004***	0.004***	0.003***	0.016***	0.013***	0.021***
	(0.001)	(0.001)	(0.001)	(0.004)	(0.004)	(0.006)
Large	0.085***	0.057***	0.056***	0.380***	0.371***	0.393***
	(0.017)	(0.016)	(0.010)	(0.070)	(0.073)	(0.105)
Insurance	-0.046***	-0.030*	-0.027*	-0.225***	-0.219***	-0.186
	(0.017)	(0.016)	(0.016)	(0.062)	(0.071)	(0.115)
Loan	0.044***	0.022*	0.028***	0.394***	0.341***	0.300***
	(0.012)	(0.012)	(0.010)	(0.072)	(0.084)	(0.103)
Constant	-0.353**	-0.249*	-0.165	-0.340	0.283	0.170
	(0.136)	(0.145)	(0.099)	(0.597)	(0.631)	(1.089)
Observations	9861	9350	8397	2233	1722	769
R-Squared	0.135	0.115	0.076	0.229	0.234	0.314

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in table 2.4.

2.5.4 Instrumental Variable Analysis

I have tried to deal with self- selection into advice seeking by controlling for province fixed effects, hospital level and individual level characteristics and by ruling out reverse causality and the gratitude element that might confound the causal effect of advice. However, as with any control function exercise, it would be too ambitious to say that I have controlled for all elements that drive self- selection into advice seeking. In addition, the endogeneity test (not reported) indicates that advice is endogenous. In this section, I will use instrumental variable techniques to causally estimate effects of advice on bribery behaviors.

I employ three instruments for advice. The first instrument is at the individual level, measuring the distance between a patient's home and the treatment place. This is the main instrument. The other two instruments are at commune level: (i) the availability of the dog vaccination program in the commune where the patient resides, and (ii) the availability of car roads that lead to the local government house. I expect that the farther a patient is away from the hospital, the more likely it is that he asks for advice. Meanwhile, those who reside in the commune with dog vaccination are expected to be less likely to ask for information on hospital because these communes should have better public health information available to its residents. Those who live in areas with a car road leading to local government house should be more keen to ask for advice because of easy transportation to the commune center (i.e. local government house). At the same time, there are no reasons to expect these variables to affect the bribery behaviors, except through the channel of affecting

the advice seeking. Thus, these instruments are likely to be valid exclusions.

The results of these IV regressions are reported in Table 2.7. The first stage regression results in column 1 confirm my expectation about the signs of these instruments. Those who live further from the hospital or in the area with car road leading to local government building are more likely to ask for advice while those whose area of residence has dog vaccination program are less likely to ask for advice. The coefficients on these three instruments are statistically significant. The F-statistic for the weak instrument test is 17.58, comfortably exceeding the conventional benchmark of 10, which indicates that the instruments are unlikely to be weak.

Columns 2 show 2 SLS results. The coefficient on advice is of larger magnitude than the OLS result and statistically significant. This suggests that the OLS underestimate the advice effect. The third column presents GMM estimation results. The advice coefficient is of similar magnitude to the 2 SLS estimate while other variable coefficients have expected signs. Since the 2 SLS and GMM results are likely to be biased in finite sample, I also estimate the regression by LIML which has been shown to be less biased than 2 SLS and GMM. The results in column 4 show that the LIML estimate is almost identical to the 2 SLS and GMM. This suggests that our IV estimates are unlikely to be biased. Further, all the over-identifying tests indicate that the instruments are valid. In column 5-7 in Table 2.7, I also show the results of the regressions that employ less than three instruments. We see that using one or two instruments change the results very little.

Table 2.7 IV regression results

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	1st stage	2 SLS	GMM	LIML	2 SLS	2 SLS	2 SLS
Advice		0.213**	0.214**	0.214**	0.206*	0.212*	0.204*
		(0.104)	(0.104)	(0.105)	(0.106)	(0.117)	(0.118)
Logexpen	-0.016	0.084***	0.084***	0.084***	0.084***	0.085***	0.085***
	(0.017)	(0.016)	(0.017)	(0.016)	(0.016)	(0.017)	(0.017)
Length	0.003***	0.003***	0.003***	0.003***	0.003***	0.003***	0.003***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Large	0.178***	0.051**	0.051**	0.051**	0.053**	0.052*	0.053**
	(0.021)	(0.025)	(0.025)	(0.025)	(0.025)	(0.027)	(0.027)
Insurance	0.007	-0.04***	-0.04***	-0.04***	-0.04***	-0.04***	-0.04***
	(0.016)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)
Loan	0.065***	0.029**	0.029**	0.029**	0.030**	0.029**	0.029**
	(0.012)	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)
Constant	0.225	-0.37***	-0.37***	-0.37***	-0.37***	-0.37***	-0.38***
	(0.136)	(0.135)	(0.135)	(0.135)	(0.135)	(0.136)	(0.136)
Distance	0.001***	√	√	√	√	√	√
	(0.000)						
Dog	-0.049**	√	√	√	√		
	(0.020)						
Road	0.048*	√	√	√		√	
	(0.028)						
Weak instrument test	F(3,60)= 17.58				F(2,60)= 23.55	F(2,60)= 23.81	F(1,60)= 41.23
Over-identifying restrictions test		S=0.49 (p=0.78)	J=0.49 (p=0.78)	AR=0.49 (p=0.78)	S=0.052 (p=0.81)	S=0.43 (p=0.50)	
Obs.	9831	9831	9831	9831	9831	9839	9839
R-squared	0.116	0.103	0.103	0.103	0.106	0.103	0.106

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in Table 2.4.

2.6 Differential impacts and word-of-mouth effects

2.6.1 Differential impacts by income, urban and loan status

Having established the causal effects of advice on bribery, I consider how advice affects different groups of patients. I do so by adding into my models, one by one, interaction terms between advice with a number of covariates of interest such as expenditure, urban, loan and insurance. A priori, the direction of the impact of the interaction terms involving expenditure, urban and loan is ambiguous. Urban people with higher expenditure might have the means to bribe when receiving such advice. But they may have more information or have a higher moral standard which make them less influenced by advice. The coefficient on the term involving loan is anticipated to be positive because of the observation that loan takers are more influenced by advice of loan givers than those who are not loan takers.

The results from the specification which includes three interaction terms with expenditure, urban and loan are reported in Table 2.8. For comparison, column 1 of Table 2.8 repeats the result of previous regression in column 8 of Table 2.4. Columns 2-3 indicate that coefficients on terms involving expenditure and urban are negative. This means that the rich and those who live in urban areas are less influenced by the advice in terms of engaging in bribery. In column 4 *loan* enters positively in the interaction term meaning that advice has a larger bribery effect on the patients who take a loan than those who do not take a loan. This interesting result provides evidence that financial constraints can translate into behavioral constraints. I also see a dramatic increase in the magnitude of the coefficient on the indicator *advice* going from column

1 to column 2-4. This is not a surprise, however, since this coefficient now captures the effect of *Advice* when the variables that interact with advice (i.e. expenditure, urban and loan) are equal to zero and thus, are of little interest here.

The results of adding interactions in the bribery amount regression are presented in Table 2.9. The expenditure term first enters as negative and statistically significant but when I add the term involving urban into the regression, both terms lose significance. When I include all three interaction terms simultaneously, only the interaction term between advice and loan is significant and positive. Again, this can be taken as evidence that those who take out loans are more vulnerable than those who do not, in terms of being subject to advice on bribery.

Table 2.8 Bribery participation regression with interaction terms

Variables	(1)	(2)	(3)	(4)
Advice	0.040***	0.486***	0.385**	0.270
	(0.014)	(0.177)	(0.167)	(0.173)
Adv_Income		-0.055**	-0.041**	-0.030
		(0.022)	(0.021)	(0.021)
Adv_Urban			-0.048*	-0.045*
			(0.025)	(0.025)
Adv_Loan				0.050**
				(0.024)
Income	0.083***	0.100***	0.096***	0.092***
	(0.017)	(0.014)	(0.015)	(0.014)
Urban	0.005	0.005	0.018	0.017
	(0.011)	(0.011)	(0.014)	(0.014)
Large	0.085***	0.082***	0.081***	0.082***
	(0.017)	(0.017)	(0.017)	(0.017)
Length	0.004***	0.004***	0.004***	0.004***
	(0.001)	(0.001)	(0.001)	(0.001)
Insurance	-0.046***	-0.046***	-0.045***	-0.045***
	(0.017)	(0.017)	(0.017)	(0.017)
Loan	0.044***	0.043***	0.043***	0.026*
	(0.012)	(0.012)	(0.012)	(0.014)
Constant	-0.353**	-0.492***	-0.462***	-0.426***
	(0.136)	(0.114)	(0.121)	(0.117)
Observations	9861	9861	9861	9861
R-Squared	0.135	0.136	0.136	0.137

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in Table 2.4.

Table 2.9 Bribery amount regression with interaction terms

Variables	(1)	(2)	(3)	(4)
Advice	0.141**	1.851*	1.936*	1.272
	(0.0561)	(0.939)	(1.032)	(1.002)
Adv_Income		-0.210*	-0.221*	-0.153
		(0.115)	(0.128)	(0.125)
Adv_Urban			0.0347	0.0556
			(0.139)	(0.141)
Adv_Loan				0.236**
				(0.105)
Income	0.446***	0.509***	0.512***	0.490***
	(0.0706)	(0.0769)	(0.0739)	(0.0740)
Urban	0.201***	0.201***	0.191***	0.183***
	(0.0604)	(0.0608)	(0.0517)	(0.0521)
Large	0.380***	0.373***	0.374***	0.378***
	(0.0700)	(0.0706)	(0.0708)	(0.0692)
Length	0.0155***	0.0156***	0.0156***	0.0155***
	(0.00379)	(0.00383)	(0.00383)	(0.00381)
Insurance	-0.225***	-0.221***	-0.221***	-0.223***
	(0.0624)	(0.0619)	(0.0619)	(0.0620)
Loan	0.394***	0.389***	0.389***	0.290***
	(0.0717)	(0.0708)	(0.0708)	(0.0765)
Observations	2233	2233	2233	2233
R-squared	0.229	0.230	0.230	0.232

Notes: Robust standard errors clustered at province level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in Table 2.4.

2.6.2 'Word-of-mouth' channel

Given the impact of advice on bribery behavior, I would like to know the channels of its effects. There are possibly two channels in which advice leads to bribery behavior. In the first channel, advice on hospital choice may be as specific as which doctor from which hospital to visit. In this case, the patient feels the need to make unofficial payment to the recommended doctor in return for better service and also not to disappoint the recommender. While this is consistent with our interpretation of the causal effect of advice on bribery, this is more about the exploitation of personal connections, rather than the second channel in which someone keenly or habitually passes on information about bribery when giving advice.

This 'word of mouth' channel – the transfer of information on bribery when giving advice – is what I am interested in. There are at least three reasons why the advice givers want to embed the information on bribery in their advice to advice seekers. First, advice givers feel the need to follow the social norms. Second, advice givers want to make their advice effective and practical, especially in this case where advice carries some responsibility. Third, those who are approached for advice may be those who have connections with the hospital or doctors and hence, there are relational or financial incentives for them to advise the advice seekers to give extra payment to the medical staff.

To explore this 'word of mouth' channel, I include a variable that captures the average rate of bribery in the region defined at commune level and its interaction with advice. My hypothesis is that if advice contains information on bribery that leads

to bribery behavior, then those advice receivers who live in the area of higher level of bribery activity will be more likely to bribe than those advice receivers who live in areas of lower bribery activities[12]. In other words, if advice contains information on bribery, which makes patients who receive advice bribe, then the marginal effect of advice ought to be more pronounced in areas where there is a higher average rate of bribery.

My goal in testing this hypothesis is twofold. First, I want to further strengthen the causal interpretation of the effect of advice on bribery behavior. Second, it sheds light on one channel by which advice affects bribery behavior, i.e. the information on bribery flow from advice giver to advice receiver.

I define area at the level of communes. As noted before, there are 1,200 communes in total in Vietnam. These geographical areas are meaningful for social interactions, like asking for advice. I calculate the average rate of bribery in each commune.

To begin with, I add the variable *average rate of bribery* alone and find that its coefficient is significant and positive as expected. This result shown in column 2 of Table 2.10 suggests that a higher average area rate of bribery raises the probability of a patient to bribe. This, in traditional way of identifying social network, is the social network effect of bribery. That is, the more people in the community bribe, the more likely each individual is affected and hence is likely to bribe.

Next, I add the interaction term between advice and the average rate of bribery in the area. Results in columns 3 of Table 2.10 indicate that those who live in

[12] This is in spirit of Bertrand *et al* (2000)

areas of higher bribery are more likely to bribe if receiving advice than those who live in areas of lower bribery. To make sure this result is not driven by other confounding factors, I also run the regression using the sample which excludes those with a bribery motive and a gratitude motive (i.e. the refined sample I used in robustness checks). I still obtain positive and significant coefficients on the interaction term between advice and bribery, as can be seen in columns 4-5 of Table 2.10. This indicates that advice on hospital indeed contains information on bribery and higher average bribery level will spread bribery more intensively through this word-of-mouth channel[13].

The word-of-mouth effect, however, does not exist for the amount of bribe. As can be seen in Table 2.11, which has the same format as Table 2.11, the coefficient on the interaction term between advice and the average rate of bribery is not significant in the three samples. To provide an explanation for why the word-of-mouth channel could affect the propensity to bribe and the bribe amount differently, I speculate that bribes in situations like hospital treatment tend to be a set menu, i.e. a certain privilege (of jumping the queue, getting a high-quality X-ray, etc.) costs a certain amount. In this case the word-of-mouth information would affect the propensity to bribe but not the bribe amount, which is set by the market and passed on from one patient to another.

[13] I also use alternative choice of areas such as province to calculate the average rate of bribery. The coefficient on the interaction term always remains positive and significant.

2.7 Discussion of Policy Implications

The role of social interaction in spreading petty corruption suggests another dimension of corruption that policymakers should consider when designing anti-corruption practices. Until now, efforts to tackle corruption focus on incentives for public officials and administrative measures. If one asks for advice on hospital choice because of lack of information and becomes vulnerable to advice on bribery, it may be important to improve the availability and quality of health care and health service information accessible to the public.

Our establishment of the word-of-mouth channel suggests that it is likely that social interaction can not only spread *actual* corruption but also fuel *perception* about corruption. This happens when one observes or hears about such petty corruption existing in a sector and conveys his perception to others. As a result, perception of corruption may be worse than the actual level of corruption. If this is the case, this helps explain why there is a gap between the actual level of corruption and perception about corruption noted in the *Transparency International Global Corruption Barometer 2004* report[14] report and investigated in Olken (2007).

More importantly, to the extent that perception of corruption influences individual behavior, this will create more actual corruption. In some areas, this word-of-mouth effect creates the expectation in the public that graft is necessary to obtain the services. Consequently, corruption in the system becomes a self-fulfilling prophecy,

[14] The Transparency International Global Corruption Barometer 2004 indicates that while those who admit bribing remain a relatively small percentage of all those surveyed (only 10 per cent of the general public admitted that a member of the household had paid a bribe over the last 12 months), many of those surveyed express grave concern about petty and, especially, political corruption.

as people pay where they assume it is necessary. This has implications for policymakers. It is important that the government carries out information campaigns which provide the public with accurate and transparent information to help alleviate myths about corruption.

The existence of the word-of-mouth effect may also help provide an explanation why developing countries have higher petty corruption than developed countries. In developing countries where gossip and informal consultancy are popular, this word-of-mouth channel operates more actively. At the same time, this word-of-mouth effect also raises the question whether corruption in developing countries may be exaggerated in corruption surveys if those surveys just focus on corruption perception rather than actual corruption.

Other policy related issues also follow from our econometric analysis. First, taking a loan from friends or relatives is popular in developing countries, especially for the poor. Because advice has a larger impact on bribery behavior for those who obtain a loan than for those who did not, loans in developing countries may carry not only financial interest but also behavioral constraints to which borrowers are subject. Second, those who are covered by insurance have an advantage over those who are not. Specifically, insurance holders are less likely to engage in bribing behavior. To the extent that this happens because they are processed in a different window, changes in registration and administrative procedure at hospitals may lead to lower bribery.

Table 2.10 Word-of-mouth effect in bribery participation

Variables	(1)	(2)	(3)	(4)	(5)
Advice	0.270	0.247	0.263	0.156	0.109
	(0.173)	(0.167)	(0.165)	(0.159)	(0.127)
Advice*Income	-0.030	-0.027	-0.034	-0.021	-0.018
	(0.021)	(0.021)	(0.021)	(0.020)	(0.016)
Advice*Urban	-0.045*	-0.035*	-0.037*	-0.032	-0.031*
	(0.025)	(0.021)	(0.021)	(0.020)	(0.018)
Advice*Loan	0.050**	0.036*	0.036*	0.037*	0.045***
	(0.024)	(0.020)	(0.020)	(0.020)	(0.017)
Income	0.092***	0.067***	0.070***	0.055***	0.028**
	(0.014)	(0.012)	(0.012)	(0.012)	(0.011)
Educ2	0.019	0.010	0.009	0.012	0.007
	(0.015)	(0.014)	(0.014)	(0.014)	(0.012)
Educ3	0.008	0.003	0.001	-0.003	0.003
	(0.019)	(0.017)	(0.017)	(0.017)	(0.015)
Educ4	0.010	0.005	0.003	0.005	-0.003
	(0.027)	(0.022)	(0.022)	(0.021)	(0.018)
Educ5	0.081***	0.057**	0.056**	0.062***	0.009
	(0.027)	(0.023)	(0.023)	(0.023)	(0.021)
Large	0.082***	0.065***	0.064***	0.044***	0.049***
	(0.017)	(0.011)	(0.011)	(0.011)	(0.009)
Length	0.004***	0.003***	0.003***	0.003***	0.003***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Insurance	-0.045***	-0.046***	-0.046***	-0.035***	-0.027**
	(0.017)	(0.013)	(0.013)	(0.013)	(0.011)
Loan	0.026*	0.031***	0.030**	0.011	0.014
	(0.014)	(0.012)	(0.012)	(0.012)	(0.010)
Average bribery rate		0.949***	0.900***	0.822***	0.509***
		(0.008)	(0.018)	(0.022)	(0.033)
Advice*Average bribery			0.152***	0.161***	0.222***
			(0.043)	(0.047)	(0.055)
Constant	-0.426***	-0.610***	-0.615***	-0.483***	-0.308***
	(0.117)	(0.097)	(0.097)	(0.097)	(0.089)
Observations	9861	9861	9861	9350	8397
R-squared	0.137	0.286	0.287	0.257	0.177

Notes: Robust standard errors clustered at commune level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in Table 2.4.

Table 2.11 Word of mouth effect in bribery amount

Variables	(1)	(2)	(3)	(4)	(5)
Advice	1.272	1.270	1.214	0.940	-1.906
	(1.002)	(1.097)	(1.099)	(1.195)	(1.647)
Advice*Income	-0.153	-0.152	-0.151	-0.117	0.244
	(0.125)	(0.136)	(0.136)	(0.148)	(0.200)
Advice*Urban	0.056	0.056	0.054	0.119	0.181
	(0.141)	(0.140)	(0.139)	(0.157)	(0.237)
Advice*Loan	0.236**	0.237**	0.238**	0.167	0.260
	(0.105)	(0.115)	(0.115)	(0.134)	(0.203)
Income	0.490***	0.489***	0.490***	0.407***	0.310**
	(0.074)	(0.081)	(0.081)	(0.083)	(0.136)
Educ2	0.022	0.023	0.023	-0.095	-0.076
	(0.102)	(0.119)	(0.119)	(0.126)	(0.176)
Educ3	0.111	0.112	0.112	0.026	0.151
	(0.101)	(0.124)	(0.124)	(0.131)	(0.177)
Educ4	0.199	0.199	0.199	0.093	0.136
	(0.149)	(0.149)	(0.149)	(0.157)	(0.251)
Educ5	0.294**	0.294**	0.294**	0.219	0.165
	(0.118)	(0.148)	(0.148)	(0.157)	(0.245)
Large	0.378***	0.378***	0.378***	0.373***	0.399***
	(0.069)	(0.069)	(0.069)	(0.074)	(0.125)
Length	0.015***	0.015***	0.015***	0.013***	0.020***
	(0.004)	(0.003)	(0.003)	(0.003)	(0.005)
Insurance	-0.223***	-0.222***	-0.223***	-0.217***	-0.189
	(0.062)	(0.073)	(0.073)	(0.081)	(0.125)
Loan	0.290***	0.290***	0.289***	0.267***	0.194
	(0.076)	(0.082)	(0.082)	(0.091)	(0.137)
Average bribery rate		-0.030	-0.066	-0.243	0.056
		(0.158)	(0.195)	(0.199)	(0.315)
Advice*Average bribery			0.098	0.235	0.144
			(0.274)	(0.290)	(0.422)
Constant	-0.670	-0.652	-0.635	0.162	0.880
	(0.640)	(0.715)	(0.718)	(0.706)	(1.154)
Observations	2233	2233	2233	1722	769
R-squared	0.232	0.232	0.232	0.237	0.319

Notes: Robust standard errors clustered at commune level are in parentheses. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent. All specifications include the demographic variables and province dummies entered in Table 2.4.

2.8 Concluding Remarks

A growing empirical literature has examined effects of social networks on individual behavior in a wide variety of settings. In this chapter, I examined the role of social interactions in spreading petty corruption in health sector in Vietnam. Instead of establishing the complete effects of a social network which embed both norms and informational channels, I focus on the latter channel.

This chapter provides evidence on the causal effect of social advice on bribery behavior. I found that social interaction in the form of advice on hospital choice leads to an increase in the propensity to bribe and the amount of bribery. Moreover, social advice has a higher impact for those who live in areas of higher average level of bribery. This means that advice contains information on bribery to medical staff for better attention. As a result, well-meaning advice can lead to a spread of petty corruption in society. Based on the findings, I have discussed several implications for policymakers in designing strategies against petty corruption in actuality and in perception.

Chapter 3

Long-Term Health Effects of Exposure to Agent Orange and Other Herbicides in the Vietnam War: A Population-Based Study

3.1 Introduction

During the period 1961-1971 in the Vietnam War[15], the US military used herbicides to clear dense forests where opposition forces were hiding and to destroy crops that those forces relied on. Consequently, civilians and members of both US and Vietnamese military forces were exposed to the herbicides. Among the chemicals used are the highly toxic Agent Orange and other herbicides including Agents Purple, Pink and Green, all of which were contaminated with varying levels of tetrachlorodibenzo-para-dioxin (TCDD) during the manufacturing process.

After the Vietnam War, the long-term health effects of herbicide exposure have been a subject of debate and controversy. There have been many lawsuits brought by both US and Vietnamese veterans against the manufacturers of these

[15] Vietnam War is between the communist Northern Vietnam and American-supported Southern Vietnam. It began in 1959 and ended in 30 April, 1975.

chemicals. The importance of the issue is also reflected in the Act by the US congress that required the Institute of Medicine to review of scientific evidences and publish its conclusions on the health effects of Agent Orange every two years.

Despite numerous studies on the effects of Agent Orange, there is not yet a population- based study on the long-term health impacts of exposure to Agent Orange and other herbicides (Agent Orange exposure, for short) on the Vietnamese population. This is unfortunate for a number of reasons. First, until now all Vietnamese veterans' legal campaigns to claim compensation from US chemical companies that produced the toxic herbicides have not achieved any success. Analysts observed that, political reasons aside, the lawsuit failed because there is a lack of convincing proof that the chemicals caused people's disabilities and deadly diseases. Second, Stellman *et al* (2003) substantially revised up the amount of dioxin sprayed, which is almost doubled as the previous estimates, and suggested that millions of Vietnamese were likely to have been sprayed upon directly. This raises the urgent need to investigate the long term health impacts of dioxin on the whole population.

In this chapter, I exploit a unique, nationally representative health survey to provide the first population-based evidence on the effects of herbicide exposure on the health of Vietnamese population. I focus on the effects of Agent Orange exposure on hypertension (i.e. high blood pressure) and height. I also investigate the effects on other health problems including cancer, mental illness, and epilepsy.

To preview the main findings, I find that people who live in the South of Vietnam that was directly exposed to Agent Orange and other herbicides during the

spraying period have a higher probability of having hypertension. I also find that Agent Orange exposure decreases height. In addition, there is evidence that exposure to Agent Orange and herbicides increases risks of cancer and mental illness.

This study makes three important contributions. First, this is the first population-based study to investigate long term health impacts of Agent Orange used in Vietnam War. Second, I use both self-reported hypertension and objectively-measured blood pressure data and show that the use of self-reported hypertension information might lead to upward bias in the estimate of the effects of Agent Orange exposure on hypertension. Third, to my best knowledge this is the first study to investigate consequences of Agent Orange exposure on height.

The structure of this chapter is organized as follows. In the next section, I review the literature on long term health effects of Agent Orange exposure. Section 3.3 describes the data used in this study. The empirical strategy and results are presented and discussed in sections 3.4 and 3.5 respectively. Section 3.6 concludes.

3.2 Literature review

Given the medical nature of the topic, it is perhaps not surprising that there is no study from economic literature on the Agent Orange issue. However, this study is related to a recently emerged economic literature that evaluate the long term effects of several past important events on health and educational outcomes using microdata surveys. Examples include effects of Chernobyl nuclear accident (Almond et al. 2007a)), of malaria eradication in India (David Cutler, et al. 2007), the 1959-1961 famine in China (Almond et al. 2007(b)), and income shocks (Banerjee et al. 2007).

Meanwhile, there are numerous medical and epidemiological studies on health effects of Agent Orange. The list of diseases linked with the Agent Orange exposure is long. To name just a few, Agent Orange is linked with various types of cancer (lung, prostate, larynx, and prostate), type 2 diabetes (IOM, 2006), skin diseases (IOM, 2002), cardiovascular disease mortality (Humblet, *et al* (2008) and references therein). The disease burden falls not only on the War veterans and civilians who were directly exposed and also on the children who were born after the spraying period. It has been found that the children of veterans exposed to Agent Orange during the Vietnam War may have a higher risk of having a certain type of leukemia (Institute of Medicine, 2000). There is also higher risk of birth defects (Anh, D. *et al.* 2006).

Until now, there is no study on the link between Agent Orange exposure and height. On the effects of Agent Orange on hypertension, there are few studies, and the results are mixed. Calvert *et al.* (1998) investigated cardiovascular outcomes in a cohort of herbicide factory workers exposed to TCDD. The study does not find a significant association between measured serum TCDD and hypertension after controlling for hypertension risk factors. However, this study is limited by self-reported diagnosis of hypertension and possibly by selection bias due to low response rates (28 percent for neighborhood referents, 70 percent for living and located cohort members, and 48 percent for the original cohort).

Bertazzi *et al* (2000) study health effects of the dioxin accident that took place in 1976 in Seveso, Italy. The authors use follow-up data on people from a number of areas. They find that, fifteen years after the accident, mortality among

men in high-exposure zones A (804 inhabitants) and B (5,941 inhabitants) increased from all cancers, rectal cancer, and lung cancer. Hypertension risk is found to be elevated, but not significantly, for the zone A, the most heavily contaminated area. This result is also consistent with Chen *et al.* (2006) which show that serum concentrations of polychlorinated dibenzodioxins (PCDDs) are not associated with an increased incidence of hypertension when major risk factors are adjusted for. This study is however limited by lack of information on the criteria for diagnosing hypertension and by a very narrow range of serum total toxic equivalency (TEQ) concentrations.

The most cited study on the potential effects of Agent Orange on hypertension is by Kang *et al.* (2006)[16]. The authors conduct a health survey of a group of 1,499 Vietnam veterans and 1,428 non-Vietnam veterans who were assigned to chemical operations jobs. They look at a number of diseases (diabetes, heart disease, hypertension, and chronic respiratory disease) and measure exposure to herbicides by analyzing serum specimens from a sample of 897 veterans for dioxin. After controlling for a number of individual characteristics and well established hypertension risk factors such as age, weight, BMI, diabetes, they find that the US army veterans who were occupationally exposed to herbicides in Vietnam experienced significantly higher risks of diabetes, heart disease, hypertension, and non-malignant lung diseases than other veterans who were not exposed.

This study of Kang team has limitations too. First, there is concern about

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[16] Based on findings in this study, IOM 2006 concludes that there is "limited or suggestive evidence of an association" between exposure to Agent Orange and hypertension (blood pressure > 140/90 mm Hg)

the use of self reported data on hypertension and Agent Orange exposure. In particular, self reported hypertension can be underreported while Agent Orange exposure may be also underreported or over- reported. Second, sample selection bias could arise from the cross-sectional nature of the study. The study only looks at disease prevalence among people in the originally deployed and non-deployed cohorts who were still alive and participated. These two issues remain a threat to reliability of their findings, even though the authors show that there are relatively high participation rates in both the Vietnam and non-Vietnam veteran groups in their study, and the prevalence rates of some of these medical conditions among non-Vietnam veterans are comparable to general populations (IOM, 2006).

If one is interested in evaluating the health effects of Agent Orange on the whole Vietnamese population, these above studies and several other studies on other Agent Orange-related diseases share some other limitations. First, most of the medical studies utilize small samples of Vietnam War veterans (Kang *et al*, 2006), chemical workers (Ott *et al* (1996), Fingerhut *et al* (1991), Manz *et al* (1991)), pesticide manufacturers and applicators (Hooiveld *et al* (1998), Saracci *et al* (1991)). This means that the results may be not representative. As a result, wide-scale health impacts of Agent Orange on Vietnamese population are difficult to obtain. Another consequence is that this might underestimate effects of Agent Orange since US veterans are relatively less exposed to Agent Orange than Northern Vietnamese veterans who in turn are probably less exposed than the civilians in sprayed areas. Further, several medical studies confine to detecting traces of dioxin (an ingredient in Agent Orange) in blood of affected people in Vietnam, rather than show a causal link

between Agent Orange exposure during Vietnam War and the incidence of a number of diseases such as cancer, mental underdevelopment, etc. In addition, potential confounders such as environmental pollution that are correlated with people's health anomalies are not accounted for in several studies.

Last but not least, while the treatment group is well defined in all medical studies, it is not clear who constitute the appropriate control group, especially when this control group is selected from exposed areas. This raises questions about the valid comparability between the treatment and control groups.

The present study overcomes these limitations and offers several advantages. First, by using a nationally representative and large micro-data survey, I can avoid the sample selection problem. Also, by exploiting the fact that the Northern part of Vietnam is exempted from Agent Orange spraying, I can provide a valid comparison between the treatment group (those who reside in the South) with the control group (those who reside in the North). Second, this study will shed light on the Agent Orange effects on the whole Vietnamese population rather than just a group of Vietnam veterans or exposed workers, which was the focus in most other studies. Further, the coverage of the whole population enables us to study Agent Orange impacts on not only the people who were directly sprayed during the Vietnam War but also on the children who were born during and after the spraying period.

Regarding the mechanisms of Agent Orange effect on high blood pressure, Kopf *et al* (2008) demonstrates that sustained Aryl hydrocarbon receptor (AhR) activation by TCDD increases blood pressure and induces cardiac hypertrophy,

which may be mediated, in part, by increased superoxide. In this study, adult male mice were dosed with 300 mg TCDD/kg three times per week for 60 days. They observed significantly increased arterial pressure and also increase in superoxide production in the kidney, heart, and aorta of TCDD-exposed mice. However, uncertainty exists about whether the extremely potent toxicity of TCDD in experimental animals also applies to humans (Johnson, 1993). In addition, the possible risk, if any, to human health of widespread, low-level dioxin contamination of the environment has still to be assessed.

3.3 Empirical strategies

3.3.1 Regional and Cohort Comparisons

To identify the effects of Agent Orange exposure, I first exploit the fact that only the Southern part of Vietnam was sprayed herbicides while the Northern part was exempted. Therefore, people who reside in the North will form our control group while those who reside in the South are considered as treatment group. Our first regression compares health outcomes of people in the two regions.

$$HealthOutcomes_i = \alpha * South + \Phi * Controls + YOB\text{-}fixed\text{-}effects + e \quad (3.1)$$

where $HealthOutcomes_i$ is a dummy, equal 1 if person i has a specific disease that is linked to Agent Orange such as cancer, mental illness, epilepsy and

hypertension or a continuous variable measuring a person's height. Dummy *South* is my variable of interest, equal 1 if the person resides in the South and 0 otherwise. *Controls* is a vector of control variables that control for several individual characteristics (such as education, gender, marital status, being employed or not, whether the respondent migrated over the past 3 years, etc) as well as well-established risk factors for diseases and blood pressure (such as health-related behaviors and weight). In addition, I also include as a control variable the number of pharmacies available in the individual's residence area. This aims to capture some aspects such as the general health and economic conditions of the area in which the person lives. *YOB fixed effects* are year-of-birth dummies that control for cohort-specific factors that affect both the North and the South. These year-of-birth dummies also capture the effects of age[17].

I next exploit the fact that the Agent Orange was only sprayed for a specific period in the War, i.e. 1961-1971. Specifically I run the regression (3.1) for three different cohorts. The first cohort consists of those who were born before the Agent Orange and other herbicides were sprayed (i.e., before 1961) and thus, were exposed directly to the chemical. That is, I look at Agent Orange effects on the first generation. The second cohort consists of those who were born during the period of Agent Orange spraying (1961-1971). For this cohort, I am interested in the '*in utero*" effects, i.e. effects that take place while the child is *in utero* stage[18]. The

[17] For cancer, mental illness and epilepsy, I use variable *Age* instead of *Year of Birth* dummies. This is because the rates of these diseases are very low, resulting loss of a large number of observations due to no variation in disease status within several yearly cohorts.

[18] This '*in utero*' stage has been shown to have large influence on later developments of the child. See, for example, Douglas Almond et al (2007a), Douglas Almond et al (2007b)

third cohort is composed of those who were born after the spraying period. This cohort enables us to study whether Agent Orange effects are passed from one generation to another. I note that in addition to enabling us to study Agent Orange effects on different age groups, this age division can minimize the confounding issues through eliminating some factors that differentially affect health outcomes of these age groups.

The variation in the amount of Agent Orange and herbicides used across areas within the South also likely leads to differential health impacts across the Southern areas. I explore this possibility by replacing the dummy South in the regression (3.1) by a set of dummies for four areas within the South, namely the Central, the Mekong, the Southeast, and the Highland. The reference area is still unchanged, namely the North that is exempted from Agent Orange spraying.

Formally,

$$HealthOutcomes_i = a*Central + b*Highland + c*Mekong + d*Southeast + \\ + \beta*Control\_Variables + YOB\text{-}fixed\text{-}effects + e_i \quad (3.2)$$

Although most of the health outcomes I am considering are binary, I estimate all the regressions using the linear probability model. In presence of a large number of dummies and categorical variables, the OLS estimation is more computationally feasible than probit or logit model. Further, because I will also estimate the difference-in-difference regressions (discussed in the next section) which include several interactions terms, the OLS estimates have the advantage of yielding a straightforward interpretation.

3.3.2 Difference-in-Difference Analysis

The above regression specifications identify the Agent Orange exposure effects by comparing health outcomes of people residing in the North with those of people staying in the South for the whole population as well as across three different cohorts. One possible concern is that the use of the North and the South as areas is too general. Consequently, the observed difference between the South and the North in health outcomes might confound the effect of some factors other than Agent Orange. For example, if I find that the cohort born in the period 1961-1971 in the South has higher rate of disease than the same cohort residing in the North, this difference may be due to some events or shocks other than the Agent Orange that affect all cohorts and regions.

To deal with this confounding factor issue, I conduct a difference-in-difference (DID) analysis. This method involves comparing the outcomes for the same cohort between the regions or areas, and then comparing this difference among various cohorts to remove the common shocks or trends affecting all cohorts and regions. Formally, I estimate the following DID regression:

$$HealthOutcomes_i = South + I(cohort\ 1961\text{-}1971) * South + I(cohort\ 1960) * South + Control\ Variables + YOB\text{-}fixed\text{-}effects + e \quad (3.4)$$

where *I(cohort 1961-1971)* and *I(cohort 1960)* are dummies, equal 1 if the person was born during 1961-1971 and in or before 1960 respectively. This specification

implies that common shocks or trends will be captured by the South-North difference observed for the unexposed cohort (i.e. born after the spraying period 1961-1971)[19]. As a result, the interaction term between the South dummy with the cohort dummy born after 1971 is omitted from the DID regression.

3.3.3. Domestic Migration and Identification of Agent Orange effects

The main threat to my identification strategies is the migration of the people within Vietnam during and after spraying period. If the migration is large and correlates with health outcomes, then my estimates of Agent Orange effects would be biased. For example, Agent Orange-exposed people migrating from the South to the North will lead to under-estimating the effects of Agent Orange exposure. Ideally, I should deal with this issue by using birthplace information to assign people to where they should be. However, this information is not available in the survey.

Although controlling for the birth place of individuals is not possible, the migration issue is not serious and can be alleviated in a number of ways. First, a great deal of migration occurred *within* the South and *within* the North. For example, those who were born in the poor central provinces of Vietnam that were sprayed Agent Orange mainly migrate to the Southern cities where the weather is more kind and jobs

[19] I assume that the unexposed cohort born after 1971 was not affected by the Agent Orange and other herbicides. But if they were (that is, if the Agent Orange effects were transmitted from parents to the children), the health effects of Agent Orange exposure obtained from this DID method would be *under-estimated.*

more abundant. There were also scores of people in the Central area moving to the Highland area. My definition of regions as large as the South (consisting of the Central, the Central Highland, the Mekong Delta, and the Southeast) and the North enables us to internalize this *within* migration[20].

The second pattern of migration that might threaten my identification is the *between* the North and the South. Regarding the direction from the South (including the Central and the Central Highland) to the North, the number of migrants is small and negligible, as shown in Phan and Coxhead (2008). Furthermore, I have information on who fought in the South and returned to the North after the War, and can control for that in the regressions.

Meanwhile, there are a large number of people moving from the unexposed North (including Red River delta, North East, and Northern Central Coast) to the Highland[21]. However this migration is not likely to threaten my choice of the South as treatment group and the North as control group. First, I have information on whether a person moved to their current residence area recently (within the past 3 years of the survey year 2001). This information is quite useful for my purpose, because most of the migration from Northern poor provinces to the South occurred after the economy and its transportation developed from the second half of 1990s. Second, for those who migrated earlier, I believe they should be considered as exposed people and thus validly included in the treatment group,

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[20] This consideration also suggests that if one wants to exploit variation in Agent Orange exposure at more detailed, lower area levels such as province, district or commune, one might risk obtaining the biases caused by this migration process

[21] This occurred following government policies during that period that encouraged people to move to the Central Highland to uncover this region.

because Agent Orange and other herbicides are found to take too long to disappear.

By conducting South versus North comparison, I can also take into account the external effects of Agent Orange spraying. That is, it is possible that dioxin went into soil and found its way to the rivers that connect several areas and consequently, contaminated areas that were not sprayed. As a result, Agent Orange sprayed on one area could affect the health of people in the neighboring areas. Using data at a lower area level might underestimate this.

3.4 Data and Descriptive Statistics

3.4.1. The Vietnam National Health Survey and descriptive statistics.

The data used in my analysis is from the Vietnam National Health Survey (VNHS) conducted in 2001-2002. This is a multi-stage, complex survey that covers nearly 160,000 individuals from 36,000 households. The survey has self reported individual- level information on various types of diseases said to be related to Agent Orange exposure, including cancer, mental illness, disabilities and hypertension. More importantly, in addition to self-reported hypertension it also contains valuable objective information on blood pressure of all respondents aged 16 and older[22].

The survey also has information on whether a person's family members fought in the War. Especially, there is valuable information on geographical

[22] During the survey, medical doctors interviewed each household and took blood pressure measurements for three times according to a standard procedure which required measuring blood pressure after the person had rested at least 15 minutes.

residence of respondents which enables us to assign a person to be in the control group (the North) or the treatment group (the South).

Information on cancer, mental illness and epilepsy is from respondents' answers while respondents' height is objectively measured. For hypertension, I report both the self reported hypertension as well as hypertension based on measured blood pressures. I follow the medical literature to classify a person as having hypertension if his or her systolic blood pressure readings consistently equal or exceed 140 mm Hg in all three checks. There is some non-overlap between objectively measured hypertension and self-reported hypertension. This may be due to misreporting or unawareness of having hypertension. It is also possible that those with hypertension were under medication at the time of blood pressure measuring, then three consecutive blood pressure tests administered on them might not detect their hypertension. I therefore coded someone as having hypertension if he scored >=140 mm Hg in all three tests or self-reported having hypertension and score >=40 in one of the three blood tests.

Table 3.1 presents descriptive statistics of a number of health outcomes. The cancer rate in the South is higher than that of the North, with the average rate for the whole country is very low. This suggests that the self-reported cancer rate is likely to be under-reported. There is a large difference between self reported hypertension rate and measured hypertension rate. Based on the blood pressure results, 13.8 per cent of population aged 16 or older has hypertension, while only 7.6% of the population reported having hypertension. The average height of the population is 156.55 cm, and Southern people are slightly higher than Northern people. The South has higher rate of

mental illness but a litlle less epilepsy problem than the North. The rates of these two medical conditions reported by survey respondents are very low, however.

Table 3.1 Summary statistics for health outcomes

	Vietnam	South	North
Measured hypertension	0.138	0.132	0.146
	(0.0011)	(0.0015)	(0.0018)
Self-reported hypertension	0.076	0.084	0.067
	(0.0009)	(0.0013)	(0.0019)
Height	156.55	156.90	156.16
	(0.028)	(0.038)	(0.042)
Cancer	0.000965	0.00104	0.000885
	(8.93e-05)	(0.000127)	(0.000126)
Mental illness	0.00333	0.00357	0.00307
	(0.000164)	(0.000229)	(0.000234)
Epilepsy	0.0028	0.00267	0.003
	(0.0001)	(0.00019)	(0.0002)
Observations	158019	71705	86314
(Hypertension)	(100442)	(45833)	(54609)

Notes: Weight-adjusted standard errors in parentheses.

Table 3.2 shows hypertension rates and height for different cohorts between the North and the South. I only consider people aged 70 or less because these two outcomes are likely to be naturally affected by older ages. For the height, I only consider those aged 18 or over, because at this age a person's height is fully developed. We observe higher hypertension rates for the cohorts born before 1961 and during 1961-1971 in the South than in the North. In contrast, the cohort born after spraying period, i.e. after 1971, in the South has lower hypertension rate. For the height, the Southern people born before 1961 are taller than Northern people of the same cohort, while there is little difference for people in the cohort 61-71 between two regions. Meanwhile, the cohort born after the spraying period is taller in the South than in the North.

Table 3.2. Summary of hypertension condition and height, by cohorts

Cohort	High blood			Height		
	Vietnam	North	South	Vietnam	North	South
Before 1961 (and <=70 years old)	0.236 (0.00248)	0.232 (0.0035)	0.240 (0.00349)	155.8 (0.0455)	155.4 (0.0654)	156.2 (0.0631)
During 1961-1971	0.052 (0.0015)	0.0495 (0.0022)	0.0541 (0.00209)	157.71 (0.051)	157.69 (0.0761)	157.72 (0.0706)
After 1971 (and >=16 years old)	0.016 (0.00073)	0.0187 (0.0011)	0.0138 (0.00088)	157.86 (0.050)	157.7 (0.0774)	157.97 (0.0666)

Notes: Weight-adjusted standard errors in parentheses.

3.4.2 Geographical and Time Distribution of Herbicide Sprays

The geographical distribution of Agent Orange and other herbicides is shown in Table 3.3. The Southeast (the military region III) received the highest spraying, with Agent Orange the main chemical used. The next heavily sprayed areas are the Highland (military region II) and the Central (military region I). The Mekong Delta area (military region IV) is the least sprayed area.

Table 3.3 Herbicide Use by Military Region, 1965-1971 (Million Gallons)

Military Region	Agent Orange	Agent White	Agent Blue	TOTAL (%)
I	2.25	0.36	0.30	2.91 (16.5)
II	2.52	0.73	0.47	3.72 (21.0)
III	5.31	3.72	0.29	9.32 (52.7)
IV	1.23	0.44	0.06	1.73 (9.8)
Total	11.31	5.25	1.13	17.68
(%)	63.9	29.7	6.4	100

(Source: Tschirley, 1992)

Table 3.4 shows the amount of herbicides sprayed in each year over the period. The Agent Orange was mainly used between 1965 and 1970. Other herbicides including Agents Pink, Green and Purple which were also contaminated with varying levels of TCDD (Stellman *et al*, 2003) were sprayed throughout the 1961-1971 period. I also note that, as the amount of herbicides used was revised to be almost doubled in Stellman's work, these geographical and time distributions of herbicide amounts are likely under- estimated.

Table 3.4. Quantity of Herbicides Recorded on the Services HERBS Tape (gallons)

Year	Agent	Agent White	Agent Blue	Other/	TOTAL
1962				3,700	3,700
1963				4,885	4,885
1964				14,560	14,560
1965	34,025			244,725	278,750
1966	242,800	45,900		182,161	470,861
1967	167,085	33,835	25,401	23,795	250,116
1968	77,259	80,245	36,846	72,977	267,327
1969	79,922	29,745	17,917	71,460	199,044
1970	27,805	10,655	26,623	28,495	93,578
1971			11,063	2,400	13,463
Total	628896	200380	117850	649158	1596284

(Source: U.S. Army and Joint Services Environmental Support Group, 1986)

3.5 Estimation Results

3.5.1 Self-reported Health Outcomes

Regression results using self-reported data on various diseases are reported in Table 3.5. The first column concerns cancer. The positive and statistically significant coefficient on my variable of interest, the South dummy, means that people in the South have higher risk of cancer than the North. Other coefficients have expected signs. Cancer risk increases with age, indicated by the positive coefficient on *Age*. Women and people in urban areas are more likely to have cancer, while larger family leads to higher rate of cancer, though statistically significant. The positive coefficients on education and income variables suggest that cancer is likely to be under-reported. That is, I only get reporting of cancer from educated and high

income people who have resources or awareness to have their health problems checked and detected. Meanwhile, the negative coefficient on smoking variable should not be interpreted as evidence that smoking reduces cancer risks. Rather, it means those who were diagnosed with cancer chose to quit smoking.

Column 2 presents the results from mental illness regression. People in the South also have higher risks of having mental illness. Larger family is associated with less mental issue. Mental issue is more popular with male than female and develops with age. Not surprisingly, those who report no mental issue have higher education, higher income and have family. Meanwhile, the South has less epilepsy problem than the North, as indicated in column 3.

Turning to the hypertension regression, the results in column 4 suggest that people in the South are more likely to suffer from hypertension than the North. Education is found to increase hypertension risk while health-related behaviours such as smoking and drinking are associated with less hypertension risk. These counter-intuitive results, however, are indicative of the selection issue. That is, only those who have higher education can detect their hypertension problem and those who were diagnosed with hypertension chose to quit smoking and drinking. This also implies that self-reported hypertension rate is likely to be under-reported.

Table 3.5 Self-reported results for various diseases, South-North Comparison

Variables	Cancer	Mental	Epilepsy	Hypertension
	(1)	(2)	(3)	(4)
South	0.115*	0.0579	-0.0636*	0.100***
	(0.0632)	(0.0358)	(0.0366)	(0.0181)
Income	0.0169	-0.208***	-0.0565	0.0328*
	(0.0628)	(0.0435)	(0.0459)	(0.0175)
Male	-0.0815	0.169***	0.125***	-0.200***
	(0.0687)	(0.0352)	(0.0358)	(0.0249)
Age	0.0147***	0.0202***	0.00682***	
	(0.00194)	(0.00129)	(0.00122)	
Family size	0.000471	-0.0187**	-0.0331***	-0.0345***
	(0.0160)	(0.00942)	(0.00944)	(0.00435)
Educ_2	0.236***	-0.202***	-0.197***	0.0143
	(0.0827)	(0.0463)	(0.0438)	(0.0226)
Educ_3	0.271***	-0.0444	-0.329***	0.0946***
	(0.0944)	(0.0493)	(0.0570)	(0.0246)
Educ_4	0.168	-0.165**	-0.551***	0.125***
	(0.121)	(0.0733)	(0.0960)	(0.0289)
Married	0.163	-0.822***	-0.396***	0.268***
	(0.116)	(0.0770)	(0.0693)	(0.0394)
Urban	0.194***	0.0345	0.0888*	-0.0285
	(0.0730)	(0.0440)	(0.0461)	(0.0193)
Smoking	-0.303***			-0.123***
	(0.0961)			(0.0242)
Drinking				-0.166***
				(0.0239)
Employed				-0.249***
				(0.0237)
Weight				0.0193***
				(0.00102)
Migrated	-0.0426	0.0257	0.137*	0.0521
	(0.144)	(0.0753)	(0.0774)	(0.0352)
Constant	-4.080***	-1.329***	-2.047***	-1.607***
	(0.526)	(0.3630	(0.38)	(0.592)
Observations	157459	157463	157463	93255
R-squared	0.09	0.07	0.034	0.1662

Notes: Regressions control for War veteran status and number of pharmacies in commune. Column (4) control for year of birth dummies. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table 3.6 Self-reported Results for various Diseases, Southern areas *versus* North

Variables	Cancer	Mental	Epilepsy	Hypertension
	(1)	(2)	(3)	(4)
Central	-0.0839	-0.0341	-0.107*	0.0685***
	(0.105)	(0.0574)	(0.0574)	(0.0263)
Highland	0.0589	-0.118	-0.173**	0.0109
	(0.126)	(0.0741)	(0.0751)	(0.0366)
Mekong	0.119	0.0909**	-0.0233	0.192***
	(0.0809)	(0.0448)	(0.0461)	(0.0220)
Southeast	0.275***	0.196***	-0.0312	0.0127
	(0.0884)	(0.0545)	(0.0574)	(0.0275)
Income	-0.0425	-0.266***	-0.0787	0.0417**
	(0.0695)	(0.0478)	(0.0479)	(0.0186)
Age	0.0148***	0.0202***	0.00684***	
	(0.00193)	(0.00128)	(0.00122)	
Educ_2	0.240***	-0.197***	-0.195***	0.0239
	(0.0831)	(0.0463)	(0.0438)	(0.0227)
Educ_3	0.284***	-0.0314	-0.322***	0.113***
	(0.0952)	(0.0498)	(0.0570)	(0.0249)
Educ_4	0.197	-0.144*	-0.541***	0.141***
	(0.121)	(0.0740)	(0.0962)	(0.0294)
Married	0.164	-0.818***	-0.399***	0.263***
	(0.115)	(0.0761)	(0.0690)	(0.0396)
Urban	0.185**	0.0357	0.103**	-0.000696
	(0.0727)	(0.0437)	(0.0460)	(0.0194)
Smoking	-0.308***			-0.119***
	(0.0966)			(0.0243)
Drinking				-0.161***
				(0.0240)
Employed				-0.254***
				(0.0237)
Weight				0.0185***
				(0.00103)
Migrated	-0.0875	-0.00786	0.133*	0.0931***
	(0.147)	(0.0786)	(0.0787)	(0.0357)
Constant	-3.597***	-0.852**	-1.866***	-1.680***
	0.574	0.395	0.397	0.595
Obs	157459	157463	157463	93255
R-squared	0.10	0.07	0.03	0.17

Notes: Regressions control for male, family size, War veteran status and number of pharmacies in commune. Column (4) control for year of birth dummies. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

I break down the disease burden in the South by smaller areas and compare them with the North. The results in Table 3.6 show that all the areas but the Central have higher rate of cancer than the North. In particular, the most heavily sprayed area, the Southeast, actually suffered the most. The Southeast also have highest rate of mental illness followed by Mekong area. Meanwhile, all areas have lower epilepsy burden compared to the North. Regarding self-reported hypertension, positive coefficients on all the area dummies in column 4 suggest that these areas have higher risk of hypertension than the North, although the Southeast and Highland dummies are not statistically significant.

3.5.2 Agent Orange and Hypertension using Measured Blood Pressure

3.5.2.1 South *versus* North comparison

I examine the relationship between Agent Orange and hypertension using the measured blood pressure. I estimate the same regression (1) for the whole sample (aged 16-70) and for various cohorts. The results are reported in Table 3.7. For the whole sample, the South coefficient is positive and statistically significant. Its magnitude, however, is only one-third of the estimate using the self reported data. This suggests that using self reported hypertension data is likely to over-estimate the effects of Agent Orange.

Other covariates included to control for hypertension risks have expected signs. For example, hypertension risks increase with weight and drinking. Marriage

and being employed are associated with lower hypertension risks. Meanwhile, higher income helps reduce hypertension burden.

3.5.2.2 Cohort comparison

Column 2-4 in Table 3.7 show the results for various cohorts. The cohort 1961-71 (born during the spraying period 1961-1971) in the South has higher rate of hypertension than that in the North, indicated by the positive and statistically significant coefficient on the 1961-1971 dummy. This suggests that exposing to Agent Orange during *utero* stage is harmful to blood pressure later in life. The cohort born before 1961 has higher hypertension risk, but not statistically significant. Meanwhile, the cohort born after the spraying period 1961-1971 has lower hypertension risk compared to the North.

Table 3.7 Effects of Agent Orange on Hypertension, South-North Comparison

Variables	Full sample, Age 16-70 (born in 1931-1986)	Born before 1961	Born during 1961-1971	Born after 1971
South	0.00383*	0.00667	0.00885**	-0.00428**
	(0.00232)	(0.00561)	(0.00354)	(0.00173)
Income	-0.0205***	-0.0358***	-0.0218***	-0.00709***
	(0.00240)	(0.00584)	(0.00375)	(0.00156)
Urban	0.00629**	0.0125**	0.000561	0.000121
	(0.00263)	(0.00614)	(0.00384)	(0.00183)
Male	-0.0183***	-0.0439***	0.000805	0.00296
	(0.00299)	(0.00789)	(0.00556)	(0.00193)
Family size	-0.00328***	-0.00712***	-0.00398***	0.000336
	(0.000569)	(0.00131)	(0.000965)	(0.000416)
Educ2	-0.00246	0.00297	-0.00734	-0.00818***
	(0.00317)	(0.00689)	(0.00485)	(0.00247)
Educ3	-0.00482	0.00114	-0.00844*	-0.00749***
	(0.00331)	(0.00743)	(0.00501)	(0.00266)
Educ4	-0.00856**	0.000688	-0.0159***	-0.00916***
	(0.00398)	(0.00926)	(0.00577)	(0.00328)
Married	-0.0211***	-0.0749***	-0.0268***	-0.00689***
	(0.00308)	(0.0148)	(0.00663)	(0.00234)
Employed	-0.0321***	-0.0652***	-0.00973	-0.00190
	(0.00395)	(0.00847)	(0.00849)	(0.00187)
Weight	0.00454***	0.00731***	0.00293***	0.00152***
	(0.000172)	(0.000333)	(0.000262)	(0.000187)
Drinking	0.0253***	0.0388***	0.0218***	0.0144***
	(0.00324)	(0.00690)	(0.00474)	(0.00338)
Smoking	-0.00242	0.00497	-0.00293	-0.00284
	(0.00325)	(0.00706)	(0.00541)	(0.00299)
Migrated	0.00641	0.00118	0.00965	0.00903**
	(0.00483)	(0.0117)	(0.00760)	(0.00411)
Constant	0.560***	0.635***	0.126***	-0.00210
	(0.0436)	(0.062)	(0.034)	(0.015)
Observations	92053	33828	24563	33662
R-squared	0.175	0.107	0.024	0.018

Notes: Regressions control for year of birth dummies, War veteran status, timing of blood pressure test, number of pharmacies in commune. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.5.2.3 Southern Areas *versus* North comparison

I also investigate the distribution of Agent Orange effects across Southern affected areas by estimating the regression (2). The first column of Table 3.8 reports results for full sample. All areas but the Central have positive sign. This suggests that these areas have higher risk of hypertension, although only the Highland coefficient is statistically significant. The next three columns compare the regional impacts for three different cohorts. For the cohort born before 1961, Highland's people have highest risk of hypertension, followed by the Mekong's and the Southeast's. For the cohort 1961-1971, the risks are highest for the Highland and the Southeast, while the Mekong coefficient is positive but not statistically significant. These results are consistent with the fact that the two former areas are the most heavily sprayed ones and the Mekong the least exposed. Meanwhile, people belonging to the cohort born after 1971 in all areas but the Highland do not show higher risks of hypertension. This suggests that the hypertension burden of Agent Orange and other herbicides does not extend to the generations born after the spraying period.

Table 3.8 Effects of Agent Orange on Hypertension, Southern areas *versus* North

Variables	Full sample, Age 16-70 (born in 1931-1986)	Born before 1961	Born during 1961-1971	Born after 1971
Central	-0.0152***	-0.0260***	-0.00449	-0.00827***
	(0.00319)	(0.00754)	(0.00451)	(0.00218)
Highland	0.0343***	0.0544***	0.0365***	0.0151***
	(0.00458)	(0.0112)	(0.00760)	(0.00431)
Mekong	0.00448	0.0117	0.00477	-0.00687***
	(0.00290)	(0.00719)	(0.00453)	(0.00202)
Southeast	0.00455	0.00874	0.0113**	-0.00793***
	(0.00356)	(0.00851)	(0.00535)	(0.00236)
Income	-0.0199***	-0.0353***	-0.0211***	-0.00489***
	(0.00256)	(0.00612)	(0.00398)	(0.00164)
Urban	0.00525**	0.0121**	-0.00134	-0.00107
	(0.00262)	(0.00613)	(0.00383)	(0.00184)
Male	-0.0183***	-0.0439***	0.000518	0.00307
	(0.00298)	(0.00789)	(0.00556)	(0.00192)
Family size	-0.00346***	-0.00754***	-0.00420***	0.000366
	(0.000571)	(0.00131)	(0.000967)	(0.000417)
Educ_2	-0.00208	0.00318	-0.00733	-0.00850***
	(0.00319)	(0.00690)	(0.00489)	(0.00250)
Educ_3	-0.00481	0.000629	-0.00963*	-0.00849***
	(0.00337)	(0.00752)	(0.00514)	(0.00272)
Educ_4	-0.00836**	0.000984	-0.0170***	-0.0105***
	(0.00407)	(0.00939)	(0.00593)	(0.00333)
Married	-0.0221***	-0.0760***	-0.0272***	-0.00733***
	(0.00309)	(0.0148)	(0.00664)	(0.00235)
Employed	-0.0322***	-0.0637***	-0.00987	-0.00252
	(0.00395)	(0.00849)	(0.00851)	(0.00188)
Weight	0.00450***	0.00722***	0.00293***	0.00151***
	(0.000172)	(0.000334)	(0.000263)	(0.000187)
Drinking	0.0254***	0.0390***	0.0219***	0.0143***
	(0.00324)	(0.00691)	(0.00474)	(0.00338)
Smoking	-0.00237	0.00526	-0.00278	-0.00277
	(0.00325)	(0.00706)	(0.00542)	(0.00299)
Migrated	0.00746	0.00268	0.00946	0.0102**
	(0.00490)	(0.0118)	(0.00768)	(0.00417)
Constant	0.560***	0.639***	0.122***	-0.0188
	(0.0442)	(0.0638)	(0.0349)	(0.0154)
Obs	92053	33828	24563	33662
R-squared	0.176	0.109	0.026	0.020

Notes: Regressions control for year of birth dummies, War veteran status, timing of blood pressure test, number of pharmacies in commune. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.5.2.4 Difference-in-Difference Analysis

So far I have studied effects of Agent Orange by comparing health outcomes between the South and the North, between Southern areas and the North, and across age cohorts. I now combine the area and cohort comparisons to conduct a DID analysis. As mentioned, this method will allow us to control for the common trends or shocks that affect both areas and cohorts during the period of study. The results are reported in Table 3.9. I can see that the coefficients on the interaction terms between the cohorts born during 1961-1971 and born before 1961 with the South are positive and statistically significant, indicating that these cohorts in the South have higher hypertension risks than their counterparts in the North.

I also carry out the DID analysis for Southern areas. The results are presented in column 2 of Table 3.9. Compared with the same cohort in the North, the cohort 1961-1971 in all Southern areas have higher hypertension burden. Similarly, the cohort born before 1961 in all Southern areas but the Central also have higher rate of hypertension than the North. I also see that the coefficients on the interactions terms involving the Southeast and the Highland are of largest magnitudes, which is consistent with their status as the most heavily sprayed areas.

Table 3.9 Effects of Agent Orange on Hypertension, Difference in Difference Analysis

Variables	South - North	Southern areas - North
South	-0.00243	
	(0.00192)	
South_Cohort6171	0.00733**	
	(0.00338)	
South_Cohort60	0.0119**	
	(0.00500)	
Central		-0.00872***
		(0.00228)
Highland		0.0157***
		(0.00436)
Mekong		-0.00285
		(0.00223)
Southeast		-0.00572**
		(0.00288)
Central_Cohort6171		0.00400
		(0.00484)
Central_Cohort60		-0.0195***
		(0.00750)
Highland_Cohort6171		0.0213**
		(0.00864)
Highland_Cohort60		0.0379***
		(0.0118)
Mekong_Cohort6171		0.00527
		(0.00435)
Mekong_Cohort60		0.0165**
		(0.00642)
Southeast_Cohort6171		0.00781
		(0.00506)
Southeast_Cohort60		0.0223***
		(0.00791)
Logpcexp	-0.0209***	-0.0196***
	(0.00241)	(0.00256)
Migrated	0.00590	0.00733
	(0.00482)	(0.00490)
Educ2	-0.00169	-0.00215
	(0.00318)	(0.00320)
Educ3	-0.00363	-0.00486
	(0.00334)	(0.00340)
Educ4	-0.00805**	-0.00870**
	(0.00400)	(0.00408)
Observations	93118	92053
R-Squared	0.177	0.177

Notes: Regressions control for year of birth dummies, War veteran status, timing of blood pressure test, family size, marital status, urban, gender, employment status, weight, drinking, smoking, number of pharmacies in commune. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.5.3 Agent Orange and Height

I now evaluate impacts of Agent Orange on height. As with hypertension, I start by comparing height of people in the South with that of people in the North. I then explore this difference by age cohorts and by Southern areas before combining these two comparisons to conduct a difference-in-difference analysis. The height regression specification differs from the hypertension regression in that I drop hypertension-risk factors such as weight, smoking and drinking.

3.5.3.1 South *versus* North and Cohort comparisons

The results are presented in Table 3.10. Although the results using the whole sample indicate that Southern people have lower height than the Northern people, comparison by cohorts gives us a rather different picture. Those who were born before 1960 are actually taller than those in the North. Strikingly, those who were born during the spraying period 1961-1971 have lower height than those in the North. Meanwhile, those who were born after the spraying period are a bit shorter than the Northern, but this is not statistically significant.

3.5.3.2 Southern Areas *versus* North comparison

As with hypertension, I also compare 4 Southern areas with the North. The results are given in Table 3.11. I first look at height of various cohorts within each area. For the Southeast area, people who were born before 1961 are taller. Those born during the most heavily sprayed period 1961-1971 see their height reduced most,

followed by those born after the spraying period. Similar results are observed for the Central and Highland areas, except that those born before spraying period in the Central area have lower height than the North. Again, those who born during spraying period suffer the largest decrease in height compared to other cohorts. For the Mekong area, even if its people in all cohorts are taller than those in the North, there is a drop in the height of people born during the spraying period compared with the before-spraying cohort. There is some recovery for the after spraying cohort. This cohort is taller than that of the during-spraying cohort, although they are still shorter than the before spraying cohort.

If we compare each cohort's height across areas, we see that for the cohort 1961-1971, those in the Southeast area suffer the most damage, followed by people in the Central and the Highland areas. The Central in 1961-1971 suffer less than the Southeast, but still more than the Highland. Meanwhile, the Mekong area, the least affected by Agent Orange, has higher height than that of the North. This pattern is consistent with the intensity of Agent Orange exposure data in which the Southeast are most heavily affected, followed by the Central and the Highland.

Table 3.10 Effects of Agent Orange on Height, South-North Comparison

Variables	Full sample (18-70)	Born in 1960 or before	Born in 1961- 1971	Born after 1971 and >=18 years
South	0.143***	0.682***	-0.428***	-0.0731
	(0.0468)	(0.0756)	(0.0880)	(0.0817)
Income	1.373***	1.276***	1.509***	1.394***
	(0.0474)	(0.0739)	(0.0920)	(0.0839)
Urban	-0.000831	-0.000897	0.123	-0.0453
	(0.0513)	(0.0814)	(0.0948)	(0.0916)
Migrated	-0.328***	-0.300*	-0.187	-0.501***
	(0.0985)	(0.161)	(0.176)	(0.175)
Male	10.24***	10.26***	10.12***	10.39***
	(0.0418)	(0.0696)	(0.0761)	(0.0727)
Hhsize	0.0245**	0.0869***	0.0258	-0.0611***
	(0.0112)	(0.0174)	(0.0240)	(0.0184)
Educ2	0.212***	0.102	0.374***	0.465***
	(0.0598)	(0.0923)	(0.116)	(0.110)
Educ3	0.429***	0.275***	0.469***	0.776***
	(0.0646)	(0.103)	(0.119)	(0.121)
Educ4	0.833***	0.513***	0.752***	1.322***
	(0.0766)	(0.124)	(0.140)	(0.141)
# Pharmacies	0.00157	0.00616	-0.00742	0.00400
	(0.00273)	(0.00451)	(0.00488)	(0.00478)
Constant	137.0***	137.3***	140.3***	141.2***
	(0.616)	(0.774)	(0.742)	(0.668)
Obs.	85252	33821	24634	26797
R-squared	0.498	0.500	0.476	0.490

Notes: Year-of-birth fixed effects are included. Robust standard errors in parentheses:
*** p<0.01, ** p<0.05, * p<0.1

Table 3.11 Effects of Agent Orange on Height, Southern areas versus North

Variables	Full sample (18-70)	Born in 1960 or before	Born in 1961-1971	Born after 1961 and >=18 years old
	(1)	(2)	(3)	(4)
Central	-0.628***	-0.308***	-0.970***	-0.840***
	(0.0660)	(0.106)	(0.121)	(0.119)
Highland	-0.368***	0.0856	-0.803***	-0.551***
	(0.0868)	(0.146)	(0.155)	(0.150)
Mekong	1.014***	1.745***	0.361***	0.677***
	(0.0586)	(0.0945)	(0.112)	(0.101)
Southeast	-0.272***	0.324***	-0.994***	-0.452***
	(0.0711)	(0.114)	(0.136)	(0.122)
Income	1.287***	1.149***	1.505***	1.293***
	(0.0501)	(0.0775)	(0.0980)	(0.0890)
Urban	0.234***	0.256***	0.327***	0.194**
	(0.0509)	(0.0808)	(0.0941)	(0.0914)
Migrated	-0.0572	-0.0372	0.109	-0.239
	(0.100)	(0.163)	(0.178)	(0.179)
Male	10.22***	10.22***	10.10***	10.38***
	(0.0416)	(0.0692)	(0.0759)	(0.0725)
Family size	0.0301***	0.0853***	0.0496**	-0.0603***
	(0.0112)	(0.0174)	(0.0240)	(0.0184)
Educ2	0.309***	0.214**	0.488***	0.541***
	(0.0597)	(0.0919)	(0.116)	(0.110)
Educ3	0.616***	0.481***	0.653***	0.950***
	(0.0653)	(0.103)	(0.122)	(0.123)
Educ4	1.041***	0.737***	0.921***	1.544***
	(0.0777)	(0.125)	(0.143)	(0.144)
# Pharmacies	-0.00203	0.00139	-0.00943*	0.000422
	(0.00275)	(0.00455)	(0.00493)	(0.00483)
Constant	137.7***	138.3***	140.1***	141.8***
	(0.628)	(0.796)	(0.786)	(0.705)
Observations	85252	33821	24634	26797
R-squared	0.502	0.506	0.480	0.494

Notes: Year-of-birth fixed effects are included. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

3.5.3.3 Difference-in-Difference Analysis

The DID results are presented in Table 3.12. The first column gives results of North South comparison. The coefficient on the interaction term between cohort 1961-1971 and the South is negative and statistically significant. This means that those who were born in this period from the South are shorter than that in the North. This is striking, especially when I notice that the South actually is taller than the North before 1961 indicated by the positive coefficient on the interaction between the South and before-1961 cohort.

The results for DID analysis involving the Southern areas presented in column 2 are similar. In all the Southern areas, the cohort 1961-1971 has higher height than the North. Meanwhile, those born before 1961 in all Southern areas are taller. This result is again consistent with the data on areas' exposure levels.

3.5.3.4 Robustness check

One potential confounder is that the lack of nutrition during the war years might affect the height of southern people more than the northern people. Although this is unlikely because the North was poorer than the south during the war period, I still address this possibility formally. If herbicides affect height during *utero* period, it is likely that herbicides also affect those at very young ages during the spraying period when their height have not fully developed. I explore this by dividing the cohort born before 1961 into two cohorts: born before 1950 and born during 1950-

1960. The war began in 1959, so those who were born 1950-1960 could be considered as before the war, which mean they were less likely to suffer from poor nutritional conditions. If they have lower height than those born before 1950, then this is evidence that herbicides affected the height. The results given in Table 3.13 indicate that the cohort 1950-1960 are still higher than that in the North, but its average height is 1 cm lower compared to its previous generation.

3.6 Concluding Remarks

In this chapter, I investigated the long-term effects of Agent Orange on Vietnamese population health. I especially focused on the effects of Agent Orange on hypertension and height using objectively measured data. I found that Agent Orange exposure significantly raises the risk of having hypertension and reduce height, especially for the cohort born during the spraying period. In addition, I find that using self reported data lead to upward bias in the estimate of the effects of Agent Orange on hypertension. My study also suggested that Agent Orange raised cancer and mental illness risks.

Table 3.12 Effects of Agent Orange on Height, Difference in Difference Analysis

Variables	South-North comparison	Various regions *versus* North
South	-0.183**	
	(0.0761)	
South_Cohort6171	-0.199*	
	(0.105)	
South_Cohort60	0.946***	
	(0.0982)	
Central		-0.910***
		(0.117)
Highland		-0.713***
		(0.149)
Mekong		0.534***
		(0.0935)
Southeast		-0.560***
		(0.114)
Central_Cohort6171		-0.0130
		(0.165)
Central_Cohort60		0.679***
		(0.155)
Highland_Cohort6171		-0.0674
		(0.212)
Highland_Cohort60		0.944***
		(0.206)
Mekong_Cohort6171		-0.114
		(0.132)
Mekong_Cohort60		1.302***
		(0.122)
Southeast_Cohort6171		-0.278*
		(0.160)
Southeast_Cohort60		0.871***
		(0.150)
Income	1.387***	1.305***
	(0.0472)	(0.0499)
Migrated	-0.326***	-0.0560
	(0.0981)	(0.0997)
Educ2	0.261***	0.360***
	(0.0596)	(0.0596)
Educ3	0.460***	0.647***
	(0.0644)	(0.0652)
Educ4	0.839***	1.041***
	(0.0761)	(0.0772)
Observations	86302	86302
R-Squared	0.499	0.504

Notes: Regressions control for year of birth dummies, War veteran status, family size, urban, gender, number of pharmacies in commune. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Table 3.13 Height and Agent Orange, Robustness Check

Variables	Whole sample	Before 1950	1950-60	1961-71	After 1971
South	0.143***	1.207***	0.211**	-0.428***	-0.0731
	(0.0468)	(0.109)	(0.101)	(0.0880)	(0.0817)
Income	1.373***	1.201***	1.387***	1.509***	1.394***
	(0.0474)	(0.110)	(0.0962)	(0.0920)	(0.0839)
Urban	-0.000831	-0.211*	0.0944	0.123	-0.0453
	(0.0513)	(0.124)	(0.104)	(0.0948)	(0.0916)
Male	10.24***	10.35***	10.22***	10.12***	10.39***
	(0.0418)	(0.109)	(0.0880)	(0.0761)	(0.0727)
Family size	0.0245**	0.102***	0.0690***	0.0258	-0.0611***
	(0.0112)	(0.0239)	(0.0242)	(0.0240)	(0.0184)
Educ2	0.212***	0.0942	0.0919	0.374***	0.465***
	(0.0598)	(0.132)	(0.125)	(0.116)	(0.110)
Educ3	0.429***	0.355**	0.0541	0.469***	0.776***
	(0.0646)	(0.162)	(0.131)	(0.119)	(0.121)
Educ4	0.833***	0.735***	0.297*	0.752***	1.322***
	(0.0766)	(0.198)	(0.157)	(0.140)	(0.141)
Migrated	-0.328***	-0.152	-0.385*	-0.187	-0.501***
	(0.0985)	(0.254)	(0.200)	(0.176)	(0.175)
Constant	137.0***	137.5***	140.2***	140.3***	141.2***
	(0.616)	(1.027)	(0.792)	(0.742)	(0.668)
Obs.	85252	15505	19767	24634	26797
R-squared	0.498	0.490	0.481	0.476	0.490

Notes: Regressions control for year of birth dummies, War veteran status, and number of pharmacies in commune. Robust standard errors in parentheses: *** p<0.01, ** p<0.05, * p<0.1

Chapter 4

Neighborhood Wealth, Hypertension, and Happiness

4.1 Introduction

Traditional measures of national well-being such as Gross Domestic Product (GDP) have long been recognized as insufficient to capture several important aspects of human well- being and development. Efforts have been made towards constructing a national well- being index that measures not only material aspects but also emotional happiness of people.

One such candidate measure is subjective happiness reported by individuals in response to survey questions on life satisfaction and happiness. However, there have been several criticisms of the use of this reported happiness to measure human well-being (see Kahneman *et al* (2004), Argyle (2001), and Kahneman and Riis (2005)). In particular, Kahneman *et al* (2004) note two main puzzling results using this standard measure of happiness. First, there are surprisingly small effects of circumstances on well-being (e.g., income, marital status, etc.). For example, data from the German Socio-Economic Panel indicate that effects of both marriage and widowhood on life satisfaction largely dissipate

within three years of the event. Second, there are implausibly large differences in the level of life satisfaction in various countries. Further, the reported life satisfaction is influenced by various factors including current mood and the immediate context, by comparisons with other people and with past experiences. Also, people might report the same experience of pleasure or displeasure differently, depending on the context and the standard to which they are comparing.

Recent efforts to find a better measure of well-being focus on hypertension as either a proxy for happiness or in conjunction with happiness in constructing national well-being index. Blanchflower and Oswald (2008) document a negative cross-country relationship between self-reported happiness and self-reported hypertension. This result has two important implications. First, if the negative cross-country relationship between happiness and hypertension exists, then the reported happiness variation across nations is validated (because high blood pressures are assumed to be reported more objectively than happiness) and it may be valid to use happiness data. Second, given its negative relationship with happiness, hypertension may be used as a good measure of the well- being of a country's population. This will facilitate more accurate evaluations of policy impacts on people's well-being. Indeed, Blanchflower and Oswald (2008) suggest that in constructing new kinds of economic and social policies in the future, where well-being rather than real income is likely to be a prime concern, there are grounds for economists to study people's blood pressure.

Although Blanchflower and Oswald (2008) carefully deal with several issues in their analysis, two problems remain with this study. First, the well-

known problems with cross-country analysis make it difficult to establish convincing evidence of a causal, direct link between happiness and hypertension. Second, the validity of their finding crucially depends on the assumption that self-reported hypertension is objectively and accurately reported. I therefore believe that further studies on this relationship are warranted, especially given the importance of their finding and its implications.

In this chapter, I provide an empirical investigation of this negative relationship. My premise is that, if hypertension and happiness are negatively correlated, then the results in previous studies that employed happiness as outcome can be replicated using hypertension as a proxy for happiness. One such important study is Luttmer (2005) who studies the negative externality of neighborhood's earnings. After controlling for an individual's own income, he finds that higher earnings of neighbors are associated with lower levels of self-reported happiness. I revisit this paper by using hypertension as a proxy for happiness. In the context of the negative relationship between hypertension and happiness, the use of hypertension should imply that neighborhood earnings have positive impacts on individual's hypertension.

Using a large health dataset from Vietnam that contains rich information on both self reported and objectively measured blood pressure, I obtain two main results. First, self reported hypertension substantially under-reports the true rate of hypertension in the context of developing country. Second, there is evidence that neighborhood wealth raises hypertension risk, but this effect is only restricted to people aged between 55 and 65. I also find weak evidence that higher

neighbors' wealth is associated with higher blood pressure of people aged 45 or older.

The results I obtain have two important implications. First, self-reported hypertension data from developing countries might be much less accurate than that in developed countries, due to poor access to blood pressure check. Second, this study lends some support to the results in Luttmer (2005) and suggests that one channel through which neighborhood's earnings has negative effect on individual happiness is its effects on hypertension.

The contribution of our study is two-fold. First, I employ a unique dataset that enables us to document a large discrepancy in the results between the use of self-reported hypertension and of measured hypertension. Second, to the best of our knowledge I am the first to study the effects of relative wealth on hypertension, thus contributing to the literature on utility of relational goods as well as the literature on inequality and health.

The structure of the chapter is organized as follows. The next section reviews the literature on happiness and hypertension as well as studies on the impacts of relative income on happiness and utility. Section 4.3 and 4.4 describes the data and the empirical strategy used in this study. The findings are presented in section 4.5 followed by a discussion in section 4.6. Section 4.7 concludes.

4.2 Literature review

4.2.1 Hypertension and Happiness

Blanchflower and Oswald (2008) use Eurobarometer data that randomly select about 15,000 individuals from 16 countries to study the relationship between hypertension and happiness. They estimate two cross-sectional regressions with hypertension and happiness as outcomes of interest. In each regression they focus on the set of country dummies while controlling for individual socio-demographic variables. The estimated coefficients on the set of country dummies in each regression are collected and plotted against each other. They find a negative relationship between these two sets of country dummies, implying that the happier countries reported less blood pressure problems. More specifically, Denmark, Sweden and some other countries rank lowest on blood pressure and highest on happiness, while some eastern European countries rank highest on hypertension and lowest in happiness. The authors then conclude that self-reported high blood pressure across individuals and countries is negatively correlated with self- reported happiness.

Their result was later confirmed by Mojon-Azzi and Sousa-Poza (2007) which shows that even with more objective measures of hypertension a negative relationship between high blood pressure problems and life satisfaction can be observed. These authors studied the relationship between life satisfaction (scored from 1-4) and self-reported blood pressure (information on whether the

respondent took blood pressure medication) for a sample aged 50+ from the Survey on Health, Ageing and Retirement in Europe. They find that happy countries seem to have fewer blood-pressure problems.

However, there are reasons to treat the results of these studies with caution. First, there are typical shortcomings associated with the cross-country analysis. Countries differ in several aspects including culture, optimism, access to doctors, physical activeness, etc. Thus, the country dummy coefficients in the regressions in Blanchflower and Oswald, 2008) will capture any country-specific factors that affect both hypertension and happiness of individuals. It is therefore not possible to rule out completely spurious correlation, even though their study controls for age, education and gender as well as obesity. As admitted by the authors, the paper's conclusion might be 'a product of the fact that an inherently cheery nation will be optimistic about everything.

Second, Blanchflower and Oswald (2008) use self-reported rather than objectively measured hypertension. They cited a number of medical studies (Giles et al., 1995; Muhajarine et al., 1997; Vargas et al., 1997; Martin et al., 2000; Liman-Costa et al., 2004; Alonso et al., 2005; Yoon and Zhang, 2006) that find a high agreement between reported and real check hypertension. However, it should be noted that these medical studies often rely on small samples. Further, they are all conducted using data from developed countries. In developing countries where access to physicians and health care are much less readily available, the situation may be different. Consequently, self- reported hypertension may be severely under-reported, making its use problematic.

Even if all hypertension cases are tracked and objectively reported, there are still the problem with omitted variables that drive both hypertension and happiness. For example, there might be environmental pollution in the country that makes people feel unhappy and at the same time, causes hypertension. If there is such a systematic relationship between pollution, hypertension and happiness across countries, then there is no direct link between happiness and hypertension and therefore, weakening the case for using hypertension as a proxy for happiness.

Last, I note that these studies only use the data from developed countries. To generalize and exploit the relationship between hypertension and happiness in constructing national well-being index across countries, one has to see whether the inverse relationship still holds in developing countries. Also, as mentioned in Blanchflower and Oswald (2008) the individual-level association between well-being and blood pressure is imperfectly understood. Thus, it is useful to further examine the relationship between hypertension and happiness at individual level.

4.2.2 Utility as A Function Of Relative Income

The idea that utility may be a function of relational goods has long been recognized and studied in several contexts. For example, Daly, Wilson and Johnson (2007) show that suicide decisions appear to be affected by comparisons. Clark (2003) and Powdthavee (2007) argue that it is psychologically preferable to be unemployed in an area where there are many other jobless people. Blanchflower *et al* (2008) shows that utility is determined by relative weight and obesity may be subject to comparison.

Regarding the effects of relative income, using US micro data Luttmer (2005) provides evidence that utility depends in part on relative position. In particular, he shows that an increase in neighbors' earnings keeping one's own income constant will lead to a reduction in his happiness. In studying the effects of neighborhood wealth on hypertension, our study complements Luttmer (2005) in a number of ways. First, even though Luttmer (2005) identified the link between neighborhood earnings and happiness, his identification strategy suggests that it cannot be strictly interpreted as causal relationship. Thus, any further evidence that suggests this relationship would add strength to Luttmer's findings. Our study can be considered as an empirical test of the relationship between happiness and neighbors' earnings. Second, Luttmer (2005) uses self-reported happiness which, as I reviewed above, is problematic. Although he tested his results by using other measures such as frequency of marital disagreements and depressions, these measures are self-reported by nature. Our use of objective hypertension in this study would allow us to deal with possible biases caused by the self-reported measure. Third, Luttmer (2005) tested whether the effect of neighbors' earnings on happiness varies by different demographic groups including age. He however could not reject the hypothesis that the effect is the same across subgroups. I find this result a bit surprising. In the context of hypertension, I examine whether effects of neighbors' earnings on one's hypertension vary across different age groups.

Another potential problem with Luttmer's study is the use of a very

large area as benchmark to calculate neighbors' earnings[2322]. Our study uses smaller and probably more reasonable areas, namely communes. Further, our study will shed light on another mechanism through which neighbors' incomes affect individual's happiness. Luttmer (2005) offers evidence of the social mechanism when he finds that socialized people are more likely to feel unhappy when living in high income neighborhood. By studying effects of neighbors' income on hypertension, I am exploring the biological pathway of the effects. Finally, unlike Luttmer (2005) that focuses on neighborhood's income, I use neighborhood's predicted expenditure. The household predicted expenditure captures the wealth of the household better than reported income because it takes into account not only income but also expenditure as well as household assets such as houses, durable goods. In the context of developing countries where bequests and unofficial income are high, I believe that wealth is probably a more appropriate benchmark for comparison among the households rather than income.

4.2.3 Impacts of social factors on hypertension

Studying the impacts of social factors such as neighbor's wealth on hypertension is interesting in itself. As pointed out in Steptoe (2000), the causes of hypertension may involve not just genetic factors, dysfunction in a number of biochemical and physiological regulatory processes but also life style factors such as diet, physical exercise, as well as psychosocial factors such as anxiety, social inhibition,

[23] I am grateful to Anindya Sen for pointing out this to me.

and job stress.

Several studies have investigated the relationships between blood pressure and socioeconomic status. For example, Gaudemaris *et al* (2002) show that prevalence of hypertension was higher among lower occupational categories. As summarized in Colhoun *et al* (1998), the relationship between socioeconomic status and hypertension vary between rich and poor countries. In developed countries, most studies find that higher socioeconomic status was associated with a lower prevalence of hypertension. However, this relationship is not clear for middle and low-income countries with findings of both positive and negative associations.

A number of studies that are more closely related to our study look at the effect of social environment on hypertension and health. Unden *et al* (1991) study the impact of social isolation or low social interactions on blood pressure. They find that daytime heart rates tend to be lower in working people who report high social support. Using combined datasets from Germany and the Czech Republic, Dragano *et al* (2007) measure area-level socio-economic status by local unemployment rate and degrees of overcrowding and examine their effects on health. They find a weak association between deprived neighborhoods and hypertension in Germany, but not in the Czech Republic. However, none of these studies look at the impacts of neighborhood wealth on its residents' hypertension.

In a broader context, Deaton (2003) reviews the literature on the relationship between inequality and health. He notes that evidence from this

literature is mixed. He further argues that there is no direct link to ill health from income inequality per se; all else equal, individuals are no more likely to be sick or to die if they live in places or in periods where income inequality is higher. Our study contributes evidence to this literature by studying the impacts of neighbors' wealth on health mediated through hypertension.

4.3 Empirical strategy

To study the impact of neighborhood's wealth on one's hypertension, I regress one's hypertension status on his or her neighborhood wealth. The regression specification is as follows.

$$Hypertension = Neighborhood\ wealth + Individual\ controls + commune\ controls + Province\ fixed\ effect + e \quad (4.1)$$

where *hypertension* is a dummy variable, taking value 1 if the person has hypertension and 0 if he does not. *Neighborhood wealth* is the average predicted expenditure of the people that live in the same area with the individuals. Neighborhood is defined at community level.

Given a large number of dummies and categorical covariates, I will estimate the model using the linear probability method. Later, in our robustness checks I will replace the dummy *hypertension* by a continuous *measured blood pressure* and estimate the same specification using both Ordinary Least Square (OLS) and quantile regression methods.

The use of measured hypertension offers several advantages. Not only does it help to deal with under-reporting due to people's unawareness of having hypertension, it also addresses endogeneity of neighborhood wealth when using self-reported measures of hypertension and happiness. For example, an optimistic person might choose to live in a rich neighborhood and at the same time, has the tendency to overlook his health problems such as hypertension. This type of misreporting is eliminated when I use measured hypertension.

There are still possibilities, however, that neighbors' wealth is endogenous due to individuals' self-selection into the area and also due to unobserved area characteristics. For example, those who have unhealthy lifestyles want to stay in rich areas to enjoy activities such as smoking and drinking which lead to hypertension. I deal with this self-selection issue by controlling for a whole range of health behaviors such as smoking, drinking, and exercising. I also include variables of weight and height of individuals that have been shown to affect hypertension likelihood[24].

It is also possible that area characteristics correlate with both neighborhood wealth and hypertension. For example, rich areas attract people who have poor health including hypertension to come to benefit from better medical care and access. It is also possible that high housing prices in the area correlate with high neighborhood wealth and at the same time put a pressure on poor people

[24] In principle, panel data would provide a better way to control for unobserved individual heterogeneity that drives self selection. However, for panel data methods to work, one needs to have a long panel so that there is sufficient variation in neighborhood wealth. Such a long panel would be a luxury in a developing country.

and raise their hypertension risk. Or rich area might attract criminals who make people more likely to have hypertension. I deal with this by including province fixed effects as well as some variables at commune level such as commune size, population, urban status, and number of pharmacies in each commune. I expect these commune level variables to capture the liveliness as well as medical access of the area which can drive one's self selection into the area. Lastly, I include season dummies to control for the differences in area temperatures which according to the medical literature also affect blood pressure.

4.4 Data and Descriptive statistics

Our data is from the Vietnam National Health Survey (VNHS) 2001-2002. This survey is nationally representative, covering more than 158,000 individuals from 36,000 households. The survey has rich information on individuals' health status and health behaviors as well as socio-economic characteristics. It also has information about respondents' living areas and health care systems at various area levels.

For our focus on studying the effects of neighbors' wealth on hypertension, this dataset offers some unique advantages. First, in addition to self-reported hypertension, it also contains valuable objective information on blood pressure of all respondents aged 16 and older. During the survey, medical doctors interviewed each household and took blood pressure measurements for three times according to a standard procedure which required measuring blood pressure after the person had rested at least 15 minutes. I follow the same procedure in chapter 3

in constructing the hypertension status for the survey respondents.

The second advantage is the survey's rich information on living standards of households and individuals. I know that reported income by individuals in developing countries is not accurate due to their diverse income sources and the severe under-reporting. Therefore, in addition to collecting reported income, the survey also designs a strategy to predict households' expenditure that uses all information on income, consumption and several other items belonging to households such as houses, land, durable goods[25]. As a result, this predicted expenditure can be considered to be more accurate than reported income and more importantly, has the advantage of representing more "permanent income" compared to income based indicators which fluctuate significantly over time.

I will use this predicted expenditure to construct the wealth of a respondent's neighborhood which is defined at commune level[26]. For each respondent, his or her corresponding neighborhood wealth is the mean predicted expenditure of all his neighbors in the commune[27]. Our working sample containing respondents of age 16 and older has 100,442 observations

[25] The strategy consists of two steps. First, real total household expenditures in 1997-98 are regressed on a large list of independent variables including consumption, owned assets, household size, etc. The coefficients calculated from this regression were then multiplied by the values of the same variables from the VNHS 2001-02. The predicted real total household expenditures for 2001-02 were then calculated. The exponential of this value was taken and divided by household size to give 2001-02 real per capita expenditures. There are a total of 35918 cases with information on the independent variables for which total household expenditures could be predicted.

[26] Ideally, I would compute neighborhood earnings using Census data as done in Luttmer (2005) because of its large sample size. However this is not possible because the data is not available. Also, since I am using wealth rather than income, collecting detailed information on assets and detailed consumption for each individual household is impossible for the Census.

[27] With 36000 households in 1200 communes, each commune has 30 households.

from 1,200 communes belonging to 61 provinces in Vietnam. Table 4.1 presents descriptive statistics of the variables used in our regression analysis. There is a large discrepancy between self-reported hypertension and measured hypertension rate: the measured hypertension rate is nearly double the self-reported hypertension rate. There may be many reasons for this large discrepancy, but the main one is probably that most Vietnamese people do not have blood pressure tests and thus do not know that they have hypertension.

Table 4.1 Summary statistics (Age 16+)

Variables	Observations	Mean	Standard deviation
Measured hypertension	100442	.1380308	.3374358
Self reported hypertension	100442	.0755162	.2642238
Neighbor's wealth	100308	8.210073	.4135382
Own wealth	100308	8.130873	.554996
Urban	100442	.3480417	.4763517
Commune population	100340	11060.73	7073.69
Commune size	100442	2913.078	5738.857
Number of pharmacies	100287	8.380508	8.552766
Male	100442	.4617889	.4985403
Age	100442	39.39381	17.10692
Family size	100442	4.982617	2.009747
Education	100442	1.360576	1.07474
Employed	100442	.8256805	.3793859
Weight	99742	49.52847	8.275507
Height	99316	156.6347	7.866478
Drinking	100195	.2338939	.4233076
Smoking	100440	.2792413	.4486286

Other characteristics of our sample are as follows. The average age in our sample is nearly 40. The average expenditures at individual and community levels are similar, as I expected. On average, families have 5 members including

grand parents. 82% of respondents reported having some work to do over the last 12 months. The average height is 1m57 while average weight is 49 kg. 23% of the sample engaged in drinking while 26% in smoking.

Table 4.2 breaks down the hypertension incidence by age groups, using both self reported data and measured hypertension. For both data, we see that hypertension increases with age. The measured hypertension indicates that age group 55-65 is almost two (five) times more likely to have hypertension than age group 45-54 (30-44). The group aged older than 65 have almost 50% probability of having hypertension rate. We see that when using self reported hypertension data, hypertension rate is under-reported across age groups, most severely for age groups older 45. This suggests that using self reported hypertension would seriously bias the results.

Table 4.2 Self-reported and measured hypertension rates by age groups

Age groups	Measured hypertension		Self reported		Obs.
	Mean	Std. dev.	Mean	Std. dev.	
16 - 29	.0160555	.125691	.00911423	.09503385	33135
30 - 44	.066769	.2496252	.04746443	.21263324	33878
45 - 54	.1870723	.3899828	.11227048	.31570963	14759
55- 65	.3310546	.4706224	.18736408	.39022585	7899
66 - 75	.48834	.4998985	.24245678	.42859213	7247
75+	.5965483	.4906512	.1983871	.39910722	3998

Table 4.3 reports the differences between hypertension and no-hypertension groups of those aged 45 or older. For both types of hypertension data, the hypertension group has lower wealth, higher age, lower education, lower height, higher weight and is less likely to have a job. Surprisingly, the hypertension group has lower rates of drinking and smoking. This is likely due to self- selection issue: those who are diagnosed with hypertension are more likely to engage in healthy activities.

There is also some discrepancy between self-reported and measured hypertension. For self reported hypertension data, the difference in neighborhood wealth between hypertension and no-hypertension groups is larger than that in calculation based on measured hypertension data. The own income of hypertension group is higher than that of no- hypertension group for self reported data than for measured data. Males register a lower hypertension rate in self reported data than in measured data. Education for hypertension group using self reported data is higher than that in measured hypertension. This suggests that those who report hypertension are likely to do so because they are more educated and thus more likely to have blood pressure and aware of their hypertension problem. The self-reported data also indicate that those with blood pressure problem are even more likely to engage in healthy activities than those with blood pressure problem in measured hypertension data. This is a further evidence of the bias in the self-reported sample: those who report hypertensions are more educated and more likely to follow a healthier lifestyle.

Table 4.3 Characteristics of hypertension versus non-hypertension groups (Age 45+)

Variables	Using measured hypertension		Using self reported hypertension	
	No hypertension	Hypertension	No hypertension	Hypertension
Neighbors' wealth	8.220961 (0.3978806)	8.230888 (.4075177)	8.199047 (.4022981)	8.346812 (.370706)
Own wealth	8.172063 (5418589)	8.157269 (.529437)	8.139769 (.5383479)	8.302764 (.515399)
Urban	.3501333 (.4770219)	.3711145 (.4831259)	.339915 (.4736886)	.4391189 (.496323)
Male	.4431525 (.4967687)	.4219105 (.4938878)	.4495929 (.4974616)	.3721586 (.483423)
Age	56.7602 (10.38008)	64.83747 (11.76508)	58.55487 (11.33665)	63.00194 (11.39219)
Hhsize	4.693316 (2.087967)	4.511657 (2.205072)	4.672047 (2.113036)	4.459559 (2.187198)
Education	1.078551 (1.092821)	.8156748 (1.042482)	1.004828 (1.080938)	.9503084 (1.098272)
Employed	.7587096 (.4278754)	.5209439 (.4995848)	.7171579 (.4503885)	.5198238 (.4996509)
Weight	48.54221 (8.911103)	48.57256 (10.10349)	48.1768 (9.143272)	50.42203 (9.824783)
Height	155.0739 (7.904012)	153.3259 (8.305365)	154.7012 (8.138406)	153.7085 (7.665327)
Drinking	.2469125 (.4312248)	.2334793 (.4230647)	.2619168 (.4396855)	.1482991 (.3554277)
Smoking	.2968484 (.4568791)	.2498341 (.4329374)	.2998126 (.458184)	.1949242 (.3961773)
Commune population	10973.97 (6794.817)	11074.59 (6796.835)	10706.54 (6698.993)	12469.79 (7069.625)
Commune size	2683.279 (5641.282)	2553.119 (5394.64)	2707.415 (5670.336)	2323.225 (5005.4)
Number of pharmacies	8.518533 (8.577098)	8.633109 (8.246371)	8.265221 (8.308735)	9.968965 (9.109703)

Notes: Standard deviation in parentheses

4.5 Empirical Results

4.5.1 Self reported hypertension *versus* measured hypertension

I first report results obtained from self reported hypertension data. Results from using the whole sample (aged 16-75)[28] are reported in column 1 of Table 4.4. I find that neighborhood's wealth has positive impacts on one's hypertension risks. Meanwhile, one's own wealth reduces his probability of having hypertension, but this effect is not statistically significant. Those who stay in urban and populated areas are more likely to develop hypertension. Higher age is associated with higher propensity of hypertension. Male is less likely to have hypertension. So are persons with higher education. As expected, areas with higher number of pharmacies have less hypertension problem. Those who are short or have high weight are more likely to have hypertension. Employed people have less hypertension risk, probably because they have higher income and less depression. Surprisingly, those who smoke or drink are less likely to have hypertension while those who exercise more are associated with higher hypertension risk. These counter-intuitive estimates are probably due to the presence of many young people in the sample who smoke or drink but are still too young to develop hypertension. It is also possibly due to individual's self- selection into activities, that is, those diagnosed with hypertension conditions self select into exercising or choose to stop smoking and drinking. Another problem is the under- reporting of hypertension that

[28] I restrict the maximum age to be 75 because, as indicated in Table 4.2, people older than 75 have a very high likelihood of having hypertension. For these people, hypertension is mostly due to biological reasons rather than social reasons.

I have documented in our descriptive analysis.

To deal with this youth issue, I re-estimate the regression using only respondents older than 45 years old. The results are reported in column 2 of Table 4.4. The coefficient on neighbors' wealth now increases more than three-fold. This is not surprising, given that most of hypertension incidence happens after one reach 45 years old and hypertension risks increase with age. Meanwhile, the effect of one's own wealth reduces. Results for other coefficients do not change much. All coefficients retain significance or stay insignificant. For example, shorter people are more likely to have hypertension. And so are heavier people. Higher age is associated with higher propensity of hypertension. Being employed reduces stress and thus, hypertension risk. I still find some unexpected signs for some variables. Thus, self selection seems the issue here. I also see the consequences of under-reporting of hypertension on some of coefficient estimates. For example, hypertension risk increases when living in urban areas or having higher education. This is probably because poor people in rural area where awareness of hypertension and availability of blood pressure checks are limited are not aware of their hypertension problem and consequently, report no hypertension.

Now I turn to the results using measured hypertension data in columns 3 and 4 of Table 4.4. The coefficient on neighbors' wealth change a lot. Its magnitude decreases substantially and is no longer significant. This should not be surprising because I have noted early that people under-report their hypertension conditions. It is likely that underreporting of self reported hypertension happen in the manner that only people who live in rich area with better access to health care report

hypertension. As a result, I find statistically significant results for the impact of neighbors' wealth on one's hypertension when using self reported hypertension but not for measured hypertension.

We also note that the use of measured hypertension yield more reasonable results than self reported hypertension. For example, one's own wealth coefficient now becomes statistically significant and larger in magnitude. Urban areas are still not significant but its coefficient sign change from positive to negative, meaning that living in urban actually lowers hypertension risk. This suggests that self-reported hypertension mainly come from people in urban areas who have better access to blood pressure checks, and thus report hypertension which in turn lead to the positive, significant results when using self reported data. Another notable difference is that men have a higher probability of having hypertension than women. Also, I observe that smoking and drinking now do raise hypertension risk. However, the exercise coefficient is still positive which most likely reflect self-selection into this activity of those with hypertension.

The results obtained for other coefficients are similar with self-reported data. Community population is positively correlated with hypertension risk. Higher age and weight are positively associated with hypertension, while higher height, larger family, and being employed lower hypertension risks. Also, those who are more educated are recorded with less hypertension. However, when I use older age sample, the pattern are similar to that when using self reported hypertension: better educated people are more likely to record hypertension.

Table 4.4 Regression results (Self-reported hypertension *versus* measured hypertension)

Variables	Self reported hypertension		Measured hypertension	
	Age 16 - 75	Age 45 -75	Age 16 - 75	Age 45 -75
	(1)	(2)	(3)	(4)
Neighbor's wealth	0.0111**	0.0367***	0.00759	0.0262
	(0.00473)	(0.0120)	(0.00657)	(0.0159)
Own wealth	-0.00250	-0.00179	-0.0182***	-0.0456***
	(0.00256)	(0.00648)	(0.00296)	(0.00882)
Urban	0.00538*	0.00711	-0.00138	-0.00594
	(0.00306)	(0.00764)	(0.00374)	(0.00912)
Male	-0.00900***	-0.0188**	0.00961***	0.00254
	(0.00284)	(0.00801)	(0.00360)	(0.0102)
Age	-0.000748*	0.0177***	-0.00701***	0.0158***
	(0.000430)	(0.00389)	(0.000529)	(0.00505)
Agesq	5.59e-05***	-0.000104***	0.000184***	-1.31e-05
	(5.41e-06)	(3.36e-05)	(6.45e-06)	(4.37e-05)
Hhsize	-0.00231***	-0.00350***	-0.00364***	-0.00767***
	(0.000511)	(0.00133)	(0.000657)	(0.00150)
Educ_2	0.00480*	0.0123**	-0.000218	0.00271
	(0.00280)	(0.00596)	(0.00328)	(0.00756)
Educ_3	0.0104***	0.0387***	-0.00510	0.00180
	(0.00310)	(0.00774)	(0.00364)	(0.00922)
Educ_4	0.0107***	0.0463***	-0.00845**	0.0103
	(0.00381)	(0.0102)	(0.00407)	(0.0109)
Married	0.0106***	0.0349***	-0.0246***	-0.0806***
	(0.00234)	(0.0129)	(0.00330)	(0.0188)
Employed	-0.0372***	-0.0709***	-0.0316***	-0.0605***
	(0.00351)	(0.00706)	(0.00396)	(0.00785)
Weight	0.00395***	0.00667***	0.00618***	0.0107***
	(0.000171)	(0.000355)	(0.000211)	(0.000421)
Height	-0.00201***	-0.00365***	-0.00399***	-0.00764***
	(0.000189)	(0.000464)	(0.000238)	(0.000580)
Drinking	-0.0226***	-0.0398***	0.0265***	0.0417***
	(0.00256)	(0.00614)	(0.00359)	(0.00855)
Smoking	-0.0128***	-0.0156**	-0.000588	0.0115
	(0.00255)	(0.00632)	(0.00327)	(0.00769)
Constant	0.112**	-0.497***	0.530***	0.365*
	(0.0496)	(0.166)	(0.0630)	(0.212)
Observations	95782	29351	95782	29351
R-squared	0.099	0.086	0.209	0.128

Notes: Robust standard errors clustered at commune level are in parentheses. The regression controls for province fixed effects, seasonal dummies, and communes' number of pharmacies, population and area. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent.

4.5.2 Effects by Age Groups

I have shown that using self reported hypertension will over-estimate the effects of neighbors' wealth on hypertension. Also, the effect of neighbors' wealth on hypertension becomes insignificant. To explore the relationship between neighbors' wealth and hypertension further, I estimate the regression for separate age groups. Table 4.5 presents the results. The major difference between the results across various age groups lies in the coefficients on neighbors' wealth. While the neighborhood wealth's coefficients in regressions for young age groups are not statistically significant and small, I obtain statistically significant and positive coefficient for the age group 55-65. This interesting result means that people aged 55-65 are more likely to develop hypertension if they live with neighbors whose wealth is higher than them.

One may argue that this result occurs simply because the age group 55-65 has higher average hypertension rate than the younger age groups. To check this, I run the same regression using data for older group aged 65-75. As seen in Table 4.3, this age group clearly has higher rate of hypertension. If the result I obtain for age group 55-65 is driven by its high rate of hypertension, the coefficient on neighbors' wealth for age group 65-75 would be even of larger magnitude and more statistically significant. Yet, what I obtain reported in the last column of Table 4.5 is that the magnitude of this coefficient reduces and is insignificant. This result is also unlikely to be driven by selection into areas because I have controlled for province-fixed effects, several commune level characteristics, as well as individual's health related behavior. More importantly, all age groups have the same neighborhood with the same characteristics. Any biases therefore should apply to all age groups.

Table 4.5 Regression results (Measured hypertension; by age groups)

Variables	Age 16-30	Age 30-45	Age 45-55	Age 55-65	Age 65-75
Neighbors' wealth	-0.00215	0.000785	0.00611	0.0608**	0.0370
	(0.00392)	(0.00834)	(0.0199)	(0.0294)	(0.0357)
Own wealth	-0.00433**	-0.0136***	-0.0344***	-0.0729***	-0.0398**
	(0.00179)	(0.00498)	(0.0126)	(0.0152)	(0.0196)
Urban	-0.00208	-0.000977	-0.00962	0.00246	-0.0114
	(0.00198)	(0.00450)	(0.0107)	(0.0156)	(0.0193)
Male	0.00798***	0.0176***	-0.0214*	0.0217	-0.00103
	(0.00220)	(0.00571)	(0.0120)	(0.0186)	(0.0237)
Age	0.00288	-0.00938	0.0690*	0.0323	0.0880
	(0.00196)	(0.00605)	(0.0385)	(0.0731)	(0.133)
Agesq	-3.23e-05	0.000211**	-0.000563	-0.000144	-0.000563
	(4.29e-05)	(8.25e-05)	(0.000388)	(0.000610)	(0.000941)
Hsize	0.000202	-0.0042***	-0.0063***	-0.0124***	-0.00427
	(0.000421)	(0.000932)	(0.00206)	(0.00279)	(0.00350)
Educ_2	-0.00751***	-0.00237	0.00497	-0.0192	0.0255
	(0.00247)	(0.00468)	(0.00886)	(0.0145)	(0.0201)
Educ_3	-0.00681**	-0.0113**	0.000913	0.0110	-0.00311
	(0.00271)	(0.00494)	(0.0111)	(0.0184)	(0.0287)
Educ_4	-0.0101***	-0.0208***	0.0151	-0.0119	0.0426
	(0.00340)	(0.00585)	(0.0131)	(0.0219)	(0.0362)
Married	-0.00619***	-0.0319***	-0.0657***	-0.0918**	-0.122*
	(0.00225)	(0.00710)	(0.0225)	(0.0434)	(0.0668)
Employed	-0.00237	-0.0117	-0.0538***	-0.0601***	-0.0624***
	(0.00196)	(0.00850)	(0.0147)	(0.0140)	(0.0144)
Weight	0.00171***	0.00442***	0.00929***	0.0110***	0.0130***
	(0.000224)	(0.000297)	(0.000529)	(0.000826)	(0.000986)
Height	-0.00061***	-0.0028***	-0.0066***	-0.0076***	-0.0089***
	(0.000183)	(0.000330)	(0.000708)	(0.00113)	(0.00134)
Drinking	0.0143***	0.0278***	0.0499***	0.0222	0.0477**
	(0.00314)	(0.00445)	(0.0103)	(0.0153)	(0.0195)
Smoking	-0.00245	-0.000482	0.0260***	0.00887	-0.00214
	(0.00306)	(0.00508)	(0.00954)	(0.0144)	(0.0191)
Constant	0.0599	0.474***	-0.946	-0.239	-2.011
	(0.0467)	(0.142)	(0.960)	(2.214)	(4.672)
Observations	35006	35574	15492	8600	6134
R-squared	0.026	0.044	0.061	0.072	0.078

Notes: Robust standard errors clustered at commune level are in parentheses. The regression controls for province fixed effects, seasonal dummies, and communes' number of pharmacies, population and area. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent.

4.5.3 Effects on Blood Pressure

While neighbors' wealth only has an impact on *hypertension* of a specific age group of 55-65, it might affect *blood pressure* for a broader group. To explore this possibility, I regress the blood pressure on the same set of independent variables using all respondents aged 45-75. The results in Table 4.6 indicate that the coefficient on neighbors' wealth is positive and almost significant at 10% level. This result strengthens our interpretation of effect of neighbors' wealth on hypertension. The positive coefficient suggests that neighbors' wealth is likely to raise blood pressure of all adult people, with effects becoming sufficiently strong to raise hypertension risk for the age group of 55-65.

4.6 Discussion

The results from self reported hypertension data that neighborhood earnings have positive impact on people's self reported hypertension is consistent with findings in Luttmer (2005) and Blanchflower and Oswald (2008). This is because when hypertension and happiness are inversely correlated (as shown in Blanchflower and Oswald's paper), the negative impacts of neighborhood earnings on happiness (as shown in Lutmer's paper) would imply that neighborhood earnings would have a positive impact on hypertension. Thus, had I only had and used self reported hypertension data, I would have been tempted to conclude that I find evidence in support of the inverse relationship between hypertension and happiness.

Table 4.6 OLS regression results, using continuous blood pressure

Variables	Age 45-75
Neighbors' wealth	0.966
	(0.733)
Own wealth	-2.029***
	(0.359)
Urban	-0.370
	(0.426)
Male	1.596***
	(0.446)
Age	0.990***
	(0.224)
Agesq	-0.00172
	(0.00195)
Hsize	-0.506***
	(0.0673)
Educ_2	0.393
	(0.339)
Educ_3	-0.376
	(0.388)
Educ_4	-0.0783
	(0.459)
Married	-4.860***
	(0.828)
Employed	-3.200***
	(0.369)
Weight	0.663***
	(0.0195)
Height	-0.474***
	(0.0267)
Drinking	2.750***
	(0.362)
Smoking	0.701*
	(0.361)
Constant	134.6***
	(9.612)
Observations	29347
R-squared	0.174

Notes: Robust standard errors clustered at commune level are in parentheses. The regression controls for province fixed effects, seasonal dummies, and communes' number of pharmacies, population and area. Asterisks indicate significance levels: *10 percent, **5 percent, ***1 percent.

However, the results from measured hypertension data indicate a different story. I have shown that the magnitude of coefficients on neighborhood wealth drops substantially and become insignificant. In addition, the regressions using measured hypertension data generate more reasonable results for other potential determinants of hypertension than using self-reported hypertension. This suggests that one has to be cautious with the use of self reported hypertension. This is especially the case when one uses data from a developing country, where I have shown that measured hypertension rate is nearly twice the self-reported hypertension rate. I believe that if hypertension is used in constructing national well-being index, it should be measured hypertension rather than self reported hypertension.

To provide a reason why this effect only happens to the age group 55-65, I speculate that when people approach the age of retirement[29], they have time to reflect and tend to take stock of what they have achieved and what they have not and they do this in comparison with their neighbors. If they fall short of their neighbors' achievement, then they might become unhappy, depressed and more likely to get hypertension. One might argue the same happens for the older age group, but it is possible that the older age groups (i.e. 65-75) are not affected by neighborhood wealth because they have adapted to their situation. This adaptation of happiness to higher income is documented in Di Tella *et al.* (2007).

Despite the discrepancy between the self reported and actual hypertension, our results provide some support to Luttmer's findings. The effects on hypertension found for people aged 55-65 suggest that neighborhood wealth has negative impacts

[29] In Vietnam, retirement age is 55 for women and 60 for men.

on his residents' hypertension and thus happiness. Further, our result also points out that a possible channel through which neighborhood earning affect happiness is through its effects on hypertension.

Our results suggest another potential channel through which retirement affect people's health. Dave *et al* (2006) document that retirement has negative impacts on health and physical activities of people. They show that this happens through the changes in lifestyles and a lack of social interactions. An implication of their results is that retirement age should be extended, not just to reduce pressure on retirement benefits but also to alleviate health burden facing retired people. Our finding of bad impact of neighbors' wealth on one's hypertension for age group 55-65 gives yet another possible reason for extended retirement age: those who retire will take stock and compare themselves with other people and will feel unhappy if they achieve less than their neighbors.

Given our findings of the effects of neighbors' wealth in raising the *hypertension* risks for age group 55-65 only and in raising *blood pressure* for a wider age group, a possible story is that neighbors' wealth is damaging to blood pressure of adults, up to the point where it has as strong as an effect of raising hypertension risk for people around their retirement age. After that, due to people's adaption to the situation, people are more relaxed and become less concerned about the difference in wealth between themselves and the rest of the community.

Regarding the policy implications of our results, it seems important to devise policies to support people at the age of retirement. It probably makes sense to encourage saving before the retirement age, so that when people have retired they

still can maintain their consumption.

Also, the findings should not be considered as evidence against the use of hypertension as a potential proxy for happiness and its use in constructing national well-being index. But I think that measured rather than self reported data should be used. Further, given our findings of neighbors' wealth in raising the *hypertension* risks for age group 55-65 only and in raising *blood pressure* for a wider age group, a distribution of blood pressure for all age groups should be more useful in characterizing the happiness of the nation than just a simple profile of hypertension.

4.7 Concluding Remarks

In this chapter I provided an empirical test for the negative relationship between happiness and hypertension identified recently in the literature. I examined a previous study that investigated the effects of neighbors' earnings on happiness. I argued that if hypertension and happiness are inversely related, then I expect to see the positive effects of neighbors' earnings on hypertension.

The study delivers two main results. First, I found that the measured hypertension rate is nearly twice as high as the self-reported hypertension rate. This leads to large discrepancy between the results using self reported hypertension and measured hypertension. The implication is that one has to use self reported hypertension data with caution, especially in the context of a developing country. Second, using measured hypertension, I found that neighbors' wealth affect one's

measured hypertension for the age group 55-65. This finding lends some support to the idea that people's utility depends on relative income and wealth. I speculate that the effect only occurs to people around their retirement age when they have time to watch each other and are more concerned about their life achievement relative to their neighbors.

Chapter 5

Toxic Choices: The Theory and Impact of Smoking Bans

5.1 Introduction

The purpose of this chapter[30] is to formalize theoretically and evaluate empirically the effectiveness of smoking bans or restrictions both in the workplace and the home. A substantive empirical literature now documents the quantitative impact of workplace smoking bans, and many empirical papers that estimate the impact of tax/price measures attempt to control for the impact of bans, broadly defined. Evans, Farrelly and Montgomery (1998) has been particularly influential because it controlled for the possible endogeneity of the choice of work place. While there is a concensus at the present time that workplace bans reduce smoking, there has been very little by way of theoretical support for such findings. In particular, why do smokers not substitute heavily in their smoking to periods of the day where smoking is not restricted?

Furthermore, if smokers do reduce the number of cigarettes they smoke as a result of restrictions on their behavior, are they likely to smoke in a more intensive manner? Higher intensity means that smokers take longer, deeper and more frequent puffs. It has long been recognized in the toxicology literature (e.g. Jarvis et

[30] This chapter is joint work with Ian Irvine.

al, 2001a) that the quantity of cotinine in a smoker's saliva or bloodstream is only loosely correlated with the number of cigarettes smoked or indeed the strength of cigarettes smoked; 'strength' denoting where in the spectrum between 'light' and 'regular' that a particular cigarette brand is located. Regular strength cigarettes have the potential to deliver more nicotine and other pleasure yielding toxins than lighter brands. Evans and Farrelly (1998) proposed that higher per unit taxes induce smokers to switch from light to regular, and Harris (1980) recommended a tax based upon nicotine content. More recently, Adda and Cornaglia (2006) have observed that the amount of cotinine in a smoker's body increases only weakly with the number of cigarettes smoked; indicating a strong degree of intensity substitution in response to changes in the number of cigarettes smoked, that might in turn be induced by policy measures designed to restrain smoking.

The first objective of this chapter is to develop a theoretical model of choice on the part of a smoker who faces three choices: how many cigarettes to smoke, at what intensity to smoke them, and at what intervals during the day. Having developed a model that involves these trade-offs we impose time restrictions on smokers that limit when they can smoke. In order to maximize their utility, smokers must choose a new triple. We solve this problem using numerical methods, having parameterized the model in such a way that it mimics observed behaviors. In essence this is a type of rationing problem. But while the theory underlying the rationing of 'goods' is well developed (Tobin and Houthakker, 1950-51, and Neary and Roberts, 1980), less energy has been devoted to understanding how the rationing of 'bads' might work, in a world where virtually all rations are directed to such products. For examples: most

drugs require a prescription from a physician and are sold in limited quantities; bars and betting establishments are limited in their hours of operation; and many toxic products cannot legally be sold to minors.

The theory and simulations we develop suggest that a workplace ban should have an imperceptible impact on low-number of cigarette smokers, that substitution into adjoining periods should be strong for medium-number smokers, and that a ban should only really bite for heavy smokers. To test this prediction we estimate quantile regressions of the log of number of cigarettes smoked on a range of covariates that includes a variable denoting whether the individual is subject to a workplace ban or not. The data are individual-level from the Canadian Community Health Survey of 2003. The theoretical conjecture is confirmed, and the data further indicate that restrictions on smoking in the home are an order of magnitude stronger than workplace bans, even after instrumenting. Our policy conclusion is that smoking bans in the workplace are important because, even though they should incite most smokers to substitute heavily into adjoining periods, the presence of a complementary restriction on smoking in the home can limit this option.

This chapter is developed as follows. Section 5.2 describes the public policy and toxicological backgrounds to the issue at hand. Section 5.3 develops a quantity-intensity-timing model of smoking during a typical working day. It contains parameterizations and a solution algorithm. Section 5.4 assesses the impact of a workplace ban within the context of the theory. Section 5.5 describes the data used in the estimation section. Section 5.6 contains the main econometric results. Conclusions are offered in the final section along with some caveats on what remains to be learned.

5.2 Background

5.2.1 Public Policy

While tax increases were once almost the sole policy instrument aimed at reducing tobacco use, currently governments and municipalities worldwide are relying progressively on smoking bans in public places, the workplace, and even the, once considered sacred, five Bs: bars, billiard halls, betting shops, bingo halls and bowling alleys. Some of the earliest municipal ordinances were enacted in California around 1990 (see Moskowitz et al, 2000). In part bans have been introduced out of the recognition that the effectiveness of ever higher taxes is limited, on account of the incentive they provide for illegal production and trans-border shipment[31] , and in part because bans are seen as an additional and distinct measure in the fight against tobacco use. They have become part of what is now termed the public health move to 'denormalize' smoking. In this vein, Cutler and Glaeser (2007) have recently proposed that smoking reductions achieved through bans may have a social multiplier. As a measure of public policy, smoking bans have two objectives: to induce smokers to smoke fewer cigarettes, or even quit smoking, in the interests of their own health; and to protect other individuals in the environs of smokers from the impact of environmental tobacco smoke (ETS), also known as second hand smoke (SHS). This chapter focuses primarily upon the first of these impacts.

[31] As of 2006, more than one quarter of cigarettes sold in Canada were supplied illegally (Gfk Research Dynamics, 2006, and ConvenienceCentral, 2006), while a figure of 22% is proposed in West et al (2008) for the UK.

While health groups universally support the implementation and extension of strictures on smoking in places shared with others, some research has been less than fully supportive. For example, Adams and Cotti (2008) propose that bans in bars have been found to encourage patrons to seek out bars in adjoining jurisdictions where smoking is not banned, with the consequence that road and vehicle accident rates increase as a result of driving further under the influence of some amount of alcohol.

The strength of bans (and the level of taxes) varies widely, depending upon the degree of anti-tobacco 'sentiment' in the jurisdiction in question (e.g. deCicca et al 2006). Sentiment against tobacco control is stronger in states or regions where tobacco is grown. For example, Kentucky, Virginia and the Carolinas have lower tax rates on cigarettes than Massachusetts, because tobacco furnishes a livelihood for many in the former states (Tobacco-free kids). While anti-tobacco sentiment may well translate into more widespread bans on public place use, in the present chapter we are less concerned with the source or motivation for bans than with their impact.

On the theoretical front, public policy interventions against smoking have received support from several recent developments that have addressed the implications of deviations from the assumptions of the traditional utility-maximizing model: Gruber and Koszegi (2006) and O'Donohue and Rabin (2001) have developed policy measures based on models of time inconsistent behaviour or projection bias, while Bernheim and Rangel (2004, 2005) have developed a framework in which environmental cues are capable of triggering mistakes on the part of the brain's decision mechanism. The former propose internality-correcting taxes, and the latter a correction to environments that may cue decision mistakes resulting in excessive drug consumption.

These models stand in contrast to the rational addiction (RA) model of Becker and Murphy (1988) and Becker, Grossman and Murphy (1994), where individuals are capable of consuming a toxic substance 'rationally'. The essential element in the RA models is that the consumer correctly recognizes the impact of current decisions on future states, and smoking may be rational if the future is sufficiently discounted or if current consumption has just a 'small' impact on the utility of future consumption. In this context, public policy measures designed to reduce smoking could be in the interests of individuals exposed to second hand smoke, but not in the interests of rational smokers.

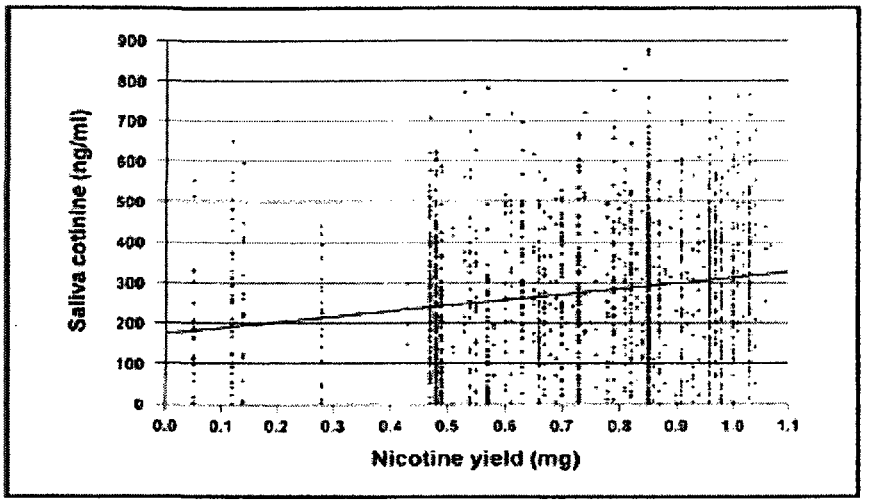
While the model that is developed in the present chapter focuses upon intra-day behavior, it is conditioned upon an individual's degree of addiction, and past experience. Furthermore, to the extent that bans or restrictions on smoking can alter the current/flow behavior of an individual, this in turn impacts the stock of accumulated experience with tobacco and hence impacts future smoking choices.

5.2.2 Toxicological Basics

An individual who smokes an average number of cigarettes per day at an average degree of intensity, ingests about one milligram of nicotine per cigarette (e.g. Perez-Stable et al, 1998). Very few smokers ingest less than 0.8 milligrams or more than 1.4 milligrams. African Americans tend to smoke more intensively, though whether this is due to a higher genetic disposition or their tendency to smoke mentholated cigarettes, which reduce the burning sensation, is still a somewhat open question (Benowitz et al, 2004). In contrast, Chinese Americans smoke many fewer cigarettes than occidentals, primarily because nicotine stays in their system for a longer time period and therefore satisfies the brain's need for the substance for a longer duration (Benowitz et al 2002).

As a starting point, Figure 5.1 below is instructive. It is taken from Jarvis et al (2001a), and maps the cotinine level (vertical axis) in the saliva samples of individuals who smoke cigarettes of varying strength (horizontal axis). Cotinine is a metabolite of nicotine and has a half life of about 20 hours, whereas nicotine has a half-life of one hour. Consequently, whatever nicotine content may be present in a blood or saliva sample, it is a poor indicator of the amount of nicotine actually ingested in a 24-hour period. Cotinine content is therefore a standard indicator in studies where such samples are used.

Figure 5.1. Cotinine levels as a function of cigarette strength (Jarvis et al, 2001)



The strength of cigarettes is traditionally determined by smoking machines (Benowitz et al 2005, Kozlowski et al 1998, US DHHS, 2000): cigarettes are inserted into a machine receptacle; the machines then puff on the cigarettes and a measure is taken of the milligrams of nicotine (and other toxins) inhaled by the machine for many different cigarette brands. Each brand therefore has a nicotine 'standard', and it is this standard that is measured on the horizontal axis.

Apart from the high degree of variability in cotinine levels of individuals who smoke a given strength of cigarette, a stark feature of Figure 5.1 is the very moderate increase in cotinine registered as the strength of cigarette increases. A similar figure is to be found in Adda and Cornaglia (2006), indicating that the amount of cotinine in saliva increases equally moderately in response to increases in the number of cigarettes smoked .

In sum, individuals seem to compensate strongly in their nicotine intake in response to different strength cigarettes and different numbers of cigarettes smoked.

The reason that individuals do not smoke each cigarette to its maximum possible nicotine yield is that, while smoking cigarettes more intensively results in additional nicotine and other ingredients that give greater pleasure to the brain's receptors, more intensive smoking also yields more carbon monoxide that can induce dizziness or mild nausea. These two effects form a trade-off for the individual smoker, and together they determine an internal solution for intensity: whereas nicotine provides pleasure for some time after being inhaled, during the time of smoking inhalation also provides disutility on account of the carbon monoxide. Consequently, an optimal degree of intensity (conditional on a given number of cigarettes) is where the marginal disutility from greater intensity during the inhalation phase equals the marginal utility from the additional nicotine for the period during which it remains in the body. The time dimension of this trade-offs, and the time-impact of nicotine are critical to understanding the compensatory behaviours that smokers may adopt in response to the imposition of bans that declare certain extended periods of the day to be off-limits to smoking.

5.3 A Quantity-Intensity-Timing Model of Nicotine Intake

5.3.1 A model of individual behavior

To formalize the foregoing, suppose a smoker ingests N units of nicotine[32] at time t_1. Then, the amount $Ne^{-\delta(t-t_1)}$ of nicotine resides in the system at any time/instant t thereafter, where α is the known decay rate - that is, the decay rate yielding a half life of one hour. A smoker gets positive utility U_p from this nicotine and let us suppose that this is of the form $U_p = N^{\alpha}$ where $\alpha < 1$[33]. It follows that, in the interval {t1; t2}, utility is the integral

$$\int_{t_1}^{t_2} N_{t_1}^{\alpha} e^{-\alpha\delta(t-t_1)} dt \qquad (5.1)$$

If an individual smokes c cigarettes per day, and inhales N units of nicotine from each, starting at instant t1 and ending at T , then utility is the sum of utility in each of the c sub-periods:

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[32] The word 'nicotine' should be interpreted broadly in this context. Cigarettes generate utility as a consequence of inhaling a variety of substances. Toxicologoists believe that nicotine is the most important of these. Thus we do not view nicotine gum or a nicotine 'patch' as being identical to cigarettes.

[33] This condition implies that the marginal utility of nicotine intensity approaches infinity as intensity tends towards zero. Accordingly, this specification guarantees that an individual will always choose some positive amount - higher prices may induce reduced consumption but not quitting. Quitting can be incorporated by assuming that there exists a fixed cost to smoking - perhaps a stigma cost. In a world of indexed tastes, smokers are those individuals whose preferences are such that they obtain a surplus above this value. Since a workplace ban reduces utility, those individuals just on the smoking margin may quit if a ban results in less surplus than the fixed cost

$$U_p = \sum_{i=1..T-1} \left(\int_{t_i}^{t_{i+1}} N_{t_i}^{\alpha} e^{-\alpha\delta(t-t_i)} dt \right), \tag{5.2}$$

where N_{ti} is the amount of nicotine in the system at the start of each interval. The c intervals are bounded by the c + 1 points or instants t1 ...tT .

The choice of intensity N is determined both by the amount of pleasure it yields throughout the day through nicotine, and by the short-term disutility it generates on account of the associated nausea that, in turn, is determined by the rate of inhalation. For the moment this disutility is instantaneous; it will have a discrete time dimension in the numerical optimization. Accordingly, defining the disutility U_d associated with this latter impact by $U_d = N^{\Phi}$, the net utility U from daily smoking is

$$U = U_p - U_d = \sum_{i=1..T-1} \left(\int_{t_i}^{t_{i+1}} N_{t_i}^{\alpha} e^{-\alpha\delta(t-t_i)} dt \right) - cN^{\Phi}, \tag{5.3}$$

In intuitive terms, the above states that if, for example, a smoker were to smoke one cigarette each hour, the resulting stock of nicotine in the body yields utility throughout the day, but that there is some disutility in the initial phase of each hour on

account of the nauseous impact of the carbon monoxide associated with inhalation. It is this negative utility potential of high-intensity smoking that limits the intake of nicotine to a level below its potential maximum per cigarette.

5.3.2 Optimization and solution algorithm

For a given set of relative prices between cigarettes and other goods, the consumer must choose the optimal number of cigarettes, the optimal spacing during the day of such cigarettes, and the optimal intensity with which to smoke them. The solution strategy is sequential: we optimize on the timing of each cigarette, conditional upon a given number of cigarettes purchased; then the optimal intensity can be chosen; Finally, relative prices determine the quantity of cigarettes purchased. The timing of the smoking decision is obviously critical in a model incorporating bans on smoking during particular phases of the day. Bans will impact the quantity purchased, distort the timing and increase the intensity.

Formally, in terms of equation (2) above, the smoker first chooses the set $\{t1\ ;\ t2\ ...tT\text{-}1\}$, conditional upon the number of cigarettes smoked. Denoting the vector of time choices by ti, the choice of timing can be separated from the choice of intensity, since the maximand can be written as:

$$Max_{\{t_i,N;\varepsilon\}}\ U = N^{\alpha}\sum_{i=1..T-1}\left(\int_{t_i}^{t_{i+1}} e^{-\alpha\delta(t-t_i)}dt\right) - cN^{\beta} = N^{\alpha}V - cN^{\beta},$$

where V is the positive utility that accrues during the day to smoking each cigarette at unit intensity $N = 1$. Thus, total positive utility can be written as the product of the level of nicotine intake raised to the power of α, and V. It is clear immediately that the program defined by equation (5.4) is separable in the choice of timing and intensity.

This program can be integrated with respect to t, and then a set of choices for the c time period boundaries t_i may be obtained from the gradient vector $\partial U_s / \partial t_i = 0, \forall i$. Integrating yields

$$U = N^{\alpha} \sum_{i=1..T-1} \frac{e^{-\alpha\delta(t_{i+1}-t_i)} - 1}{-\alpha\delta} - cN^{\phi}. \tag{5.5}$$

Differentiating this with respect to each t_i yields conditions that are difficult to work with. To see this, suppose an individual smokes 30 cigarettes per day. The choice of when to smoke the second or third cigarette will have consequences on the utility obtained from every subsequent cigarette - because nicotine decay is incomplete from interval to interval. Postponing the time of the next cigarette means that more nicotine is carried to all subsequent time intervals. Consequently, the choice of, say, t_2 influences the utility obtained in all 30 time intervals. Accordingly, to reduce the dimensionality of the problem to manageable proportions, we adopt a search algorithm that is based on an approximate set of first order conditions in making the timing choices.

Since the decay rate for nicotine is moderate, in practice a very good numerical approximation to the underlying first order conditions can be obtained by limiting attention to the impact of the choice of any t_i on a small number of intervals. In particular, focusing on the utility obtained in the intervals on either side of any t_i, and two further future periods, means that an approximate first order condition can be obtained by differentiating

$$Z = N^{a}_{t_{i-2}} \frac{e^{-a\delta(t_i - t_{i-1})} - 1}{-a\delta} + N^{a}_{t_i} \frac{e^{-a\delta(t_{i+1} - t_i)} - 1}{-a\delta} + N^{a}_{t_{i+1}} \frac{e^{-a\delta(t_{i+1} - t_{i+1})} - 1}{-a\delta} + N^{a}_{t_{i+1}} \frac{e^{-a\delta(t_{i+2} - t_{i+2})} - 1}{-a\delta} \tag{5.6}$$

with respect to t_i, using the relations

$$N_{t_i} = N_{t_{i-1}} e^{-\delta(t_i - t_{i-1})} + N; \quad \frac{\partial N_{t_i}}{\partial t_i} = N_{t_{i-1}}(-\delta) e^{-\delta(t_i - t_{i-1})}; \quad \frac{\partial N_{t_{i+1}}}{\partial N_{t_i}} = e^{-\delta(t_{i+1} - t_i)} \tag{5.7}$$

This yields, after some rearranging of terms:

$$\frac{\partial Z}{\partial t_i} = N_{t_{i-1}}^{\alpha} e^{-\alpha\delta(t_i - t_{i-1})} - N_{t_i}^{\alpha} e^{-\alpha\delta(t_{i+1} - t_i)} + \left(e^{-\alpha\delta(t_{i+1} - t_i)} - 1\right) N_{t_i}^{\alpha} N_{t_{i-1}} e^{-\delta(t_i - t_{i-1})} - \left(e^{-\alpha\delta(t_{i+2} - t_{i+1})} - 1\right) N_{t_{i+1}}^{\alpha-1} N e^{-\delta(t_{i+1} - t_i)} - \left(e^{-\alpha\delta(t_{i+3} - t_{i+2})} - 1\right) N_{t_{i+2}}^{\alpha-1} N e^{-\delta(t_{i+2} - t_i)} \quad (5.8)$$

The solution algorithm starts by allocating the cigarettes evenly over the whole smoking day, thus determining a starting set of t_i values. We then compute $\partial Z/\partial t_i$ at each such value of t_i, and adjust the t_i that corresponds to the largest gradient. If that gradient is negative its t_i value is reduced, if positive, the value is increased. Each time a value of t_i is adjusted the new value of U_p is calculated, a new gradient vector is calculated and some t_i is again adjusted.

The routine stops when $dU_p < 0{:}001$. Since the numerical value of utility typically falls in the range {50; 150}, this criterion means that the value of the objective function is changing by less than one in one hundred thousand at the final iteration.[34]

[34] While a sufficient condition for this mechanism to attain a maximum is that the function b e negative semi definite, we cannot demonstrate that it has this property because of the complexity of the associated Hessian. The function will attain a maximum if it has a unique optimum and positive first derivatives everywhere in the *ti* space. While the order of the problem makes it dicult to establish this in the in the general case, we have explored exact solutions to the maximand where there are a small number of intervals. In such cases the numerical solutions obtained from the solution algorithm match the analytical solutions, and the 3D images of the function indicate that it has a unique maximum.

The smoking day is specified to lie between 7:30 am and 10:00 pm. This is broken into 145 units of 6 minutes each, on the grounds that it takes about 6 minutes to smoke a cigarette (a frequent pattern is one where the smoker inhales perhaps ten times, with 35 second breaks between puffs- see Hammond et al, 2006). So the solution algorithm yields integer values for the t_i vector in the range {1...145}.

5.3.3 Optimizing on intensity

An optimal value of intensity N is obtained from equation (5.4) above:

$$\begin{aligned} \partial U/\partial N &= \alpha N^{\alpha-1} V - c\phi N^{\phi-1} = 0 \\ N^* &= V^{1/(\phi-\alpha)} \left(\frac{\alpha}{c\phi}\right)^{1/(\phi-\alpha)} . \end{aligned} \tag{5.9}$$

For intensity to be decreasing in the number of cigarettes (and thus match the evidence), the parameters in the model must satisfy the relation implied by the condition $\frac{\partial N^*}{\partial c}$ <0. Experimentation suggests that a range of values satisfy this requirement. But the parameter values must also be able to generate intensity outcomes that fall in the range of 0.8 mg to 1.4 mg of nicotine per cigarette, in order to conform to observed magnitudes. We find that pairs in the neighbourhood of {α=0:3; Φ= 2:5} satisfy both of these requirements. The intuition on the relative magnitudes of and is straightforward: the smoking of the cigarette lasts for a much shorter period than the utility-yielding nicotine stays in the body. And to obtain the required intensity

trade-off, the immediate disutility from the high intensity must exceed the immediate positive utility from the nicotine, since the latter is longer lasting.

5.3.4 Prices quantities and demand functions

To this point, the optimal timing and intensity rules are conditioned upon a given quantity consumed. The link between a chosen quantity and a given price can be established easily by invoking a quasi-linear utility structure:

$$W = U(c) + \theta y, \tag{5.10}$$

where y represents other goods. Normalizing the price of y at one and defining p as the price of cigarettes the optimality condition is

$$\frac{U_c'}{p} = \theta. \tag{5.11}$$

In this quasi-linear framework a change in price requires a new quantity of cigarettes such that marginal utility divided by price is restored to the initial value. Numerically, the value of utility is obtainable for any quantity of cigarettes purchased (maximizing simultaneously on timing and intensity), and a marginal utility schedule drops out of this.[35]

[35] For numerical purposes, in order to get a continuous and differentiable marginal utility schedule, we regress the utility values obtained in the optimization on a low-order polynomial in c.

5.4 Assessing the Impact of Smoking Bans

5.4.1 Modeling workplace bans

Smoking bans come in different forms. The most common one, and one which would be anticipated to have the greatest impact on behaviour, is a ban on smoking in the workplace. Workplace bans effectively make smoking more difficult and costly for about one half of the effective day, and therefore may be expected to have a substantial impact on behaviour.

Within the context of a utility maximizing agent, subject to a budget constraint, such bans are best envisaged as increasing the cost of a cigarette smoked during these periods: if individuals choose to smoke a cigarette during their working day, it must be outside the confines of their office or workshop. This involves a time cost that changes radically the price of a cigarette. During unrestricted segments of the day a single cigarette may cost in the range of 20 - 40 cents, depending upon whether it is purchased in Europe or the US; but during the restricted segments of the day an individual must incur the time costs of smoking. Approximately one sixth of an hour is required to smoke one cigarette (ten minutes – six to smoke and four to commute out doors), and so the effective cost to a smoker with a $21 per hour job of one such cigarette approaches $4:00 – a tenfold increase in price during the working day in this instance.

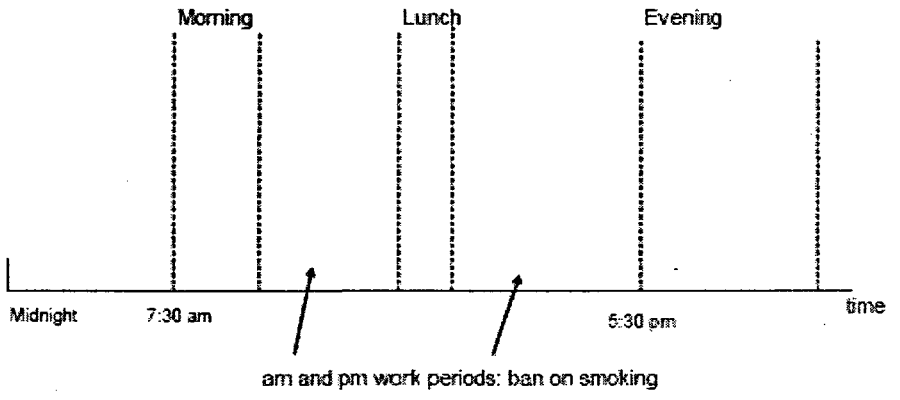
Conceptually the solution to the problem of choosing the optimal number of cigarettes to purchase, when to smoke them and how intensively to smoke them is not difficult: the optimality condition is that the marginal utility per dollar must be the same

for a cigarette smoked during the working day as one smoked during the unrestricted segments of the day. And each of these must equal the marginal utility of consumption on other goods, which, by assumption of quasi-linear utility, is constant and ascertainable from a base parameterization of the model.

To understand the impact of a workplace ban, consider figure 5.2 below. The day runs from 7:30 am to 10:00 pm at night, and the working day from 9:00 am to 12:30 and from 13:30 to 17:30. If the price during the working/restricted day, p_r , is ten times the price during the unrestricted period, p_u , then the marginal utilities must bear the same tenfold relationship in equilibrium:

$$\frac{MU_u}{p_u} = \frac{MU_r}{p_r} = \theta. \qquad (5.12)$$

Figure 5.2 Daily restricted and unrestricted periods



A requirement that marginal utility during the working day increase by a factor of ten will require a substantial reduction in quantity consumed during that period. As a consequence of such a quantity reduction, the marginal utility of cigarettes smoked

during the unrestricted periods must rise. The mechanism by which a new equilibrium is attained depends upon the fact that cigarettes smoked in any phase of the day contribute to the stock of nicotine in the body beyond the smoking period.

In the first place, cigarettes smoked in the initial unrestricted period of the day (morning) have a carry-over utility value: each morning cigarette produces a stock of nicotine that has lasting utility value through the morning work period. These early morning cigarettes produce a greater marginal utility in the absence of smoking during the morning work period: the nicotine stock they produce is not augmented further by work-time cigarettes, and therefore their marginal utility increases. We term this the knock-on effect.

Consider now the unrestricted evening period. A reduction in afternoon smoking means that the stock of nicotine in the body is depleted when the evening period arrives. In turn this implies that the marginal utility of cigarettes smoked in the early phase of the evening period is high and therefore it becomes optimal to smoke more cigarettes during this early evening phase than in the absence of an afternoon smoking ban. This impact we term the nicotine deficit impact.

It is clear that the mid-day response to a ban on morning and afternoon work time smoking will likewise demand an increase in the number of cigarettes smoked, because both the nicotine deficit effect and the knock-on effect are in play. This then is the intuition underlying the results for the computable model. While the following section of this chapter estimates some quantile regressions, it is instructive to examine how much smoking substitution is implied by the calibrated model. To get a sense of this we model the optimal response behaviour of a heavier than average smoker - one

who smokes 18 cigarettes per day in a 'no restricions' workplace. The price of a cigarette is assumed to be 40 cents (corresponding to a Euro price of about e5:50 per pack or eight dollars – somewhat higher than the current US price).

Optimality requires a smoking strategy that satisfies eq. (5.12) above and in addition that allocates a given daily total of cigarettes across all five periods such that utility is maximized. That is, defining the intervals I as I1...I5, and the number of cigarettes smoked in each interval by i; j; k; l; m, a utility maximum for any total c requires that:

$$U(I1_i, I2_j, I3_k, I4_l, I5_m) > U(I1_{i'}, I2_{j'}, I3_{k'}, I4_{l'}, I5_{m'}) \quad \forall \; i', j', k', l', m'. \tag{5.13}$$

where:

$$i + j + k + l + m = c = i' + j' + k' + l' + m'. \tag{5.14}$$

The dimensions of the optimization are reduced by noting first that the initial cigarette of the day should be smoked at the first possible moment. This is because postponing that cigarette would essentially waste a small amount of nicotine at the end of the day. Second, it is straightforward to show that, with a sufficient difference between the full price of a cigarette in the unrestricted and restricted intervals, the last cigarette to be smoked in intervals I1 and I3 should be at the latest possible moment in those intervals (a cigarette in the following instant costs ten times as much but is a close substitute). By the same reasoning, the first cigarette smoked in intervals I3 and

I5 should be at the first possible instant in those intervals on account of the nicotine-deficit effect.

5.4.2 Numerical results and behaviours

The results for this particular experiment are contained in table 5.1. At a price of \$0:4 per cigarette in the unrestricted interval, and \$4:0 in the restricted intervals, it is optimal to reduce total purchases from 18 to 16, to smoke none in the restricted intervals and to distribute the cigarettes in a {I1 = 6; I2 = 0; I3 = 5; I4 = 0; I5 = 5} pattern, as indicated in column (ii).

There are several notable aspects of this experiment. First is the allocation within the day: lunch time smoking increases due to a combination of the nicotine-deficit effect and the knock-on effect, each described above, operating in the mid-day interval. An optimal plan involves a quick nicotine catch-up when the lunch interval arrives, and simultaneously a stocking up for the afternoon period. In contrast, the evening allocation should not be so great as to loose the utility value of nicotine in the body when the end of the day arrives – it is optimal to have a low stock of nicotine at the end of the day, and therefore to avoid consuming too large a number in the evening interval.

The second notable aspect of the constrained decision making is that condition (5.13) is satisfied at a value of c that is surprisingly close to its unconstrained value (16 rather than 18). This result is due to the stock-flow nature of the model. A reduction

in smoking during the restricted intervals increases the marginal utility of cigarettes in the unrestricted periods.

Third, the optimal value of intensity increases - see the final row in table 5.1. This occurs on account of the increase in the marginal utility that the reduced number of cigarettes entails, in turn requiring an increase in the disutility of intensity - which occurs at a higher level of intensity. Consequently the reduction in nicotine ingested is even less than the amount suggested by the reduction in quantity consumed.

Fourth, the switch from smoking during the working day to the unrestricted intervals sees a jump in morning smoking, despite the reduction in the total number of cigarettes smoked. Evening smoking is affected little, even though it has a substantially greater duration, for the reason that the utility value of cigarettes smoked at the end of the day is not as great as at the start of the day. The model suggests that virtually all of the impact of the workday smoking ban is transferred to the morning and mid-day periods, and very little to the evening period. This predicted increase in morning smoking could increase exposure to SHS on the part of other family members. Jarvis et al (2000, 2001b) report that cotinine concentrations among children in the UK have fallen over time as a result of lower exposure levels globally; they also report that cotinine levels among non smoking partners increase with the number of cigarettes smoked by a smoking partner. And while the cotinine concentrations among non-smokers are typically no more than one percent of a smoker, Hackshaw et al (1997) report that the difference in cotinine levels between partners of non smokers and smokers is sufficiently large to be significant in the sense of inducing higher morbidity risk.

Markowitz (2006) proposes that both bans and taxes reduce the incidence of sudden infant death syndrome. The econometric results we present in the next section suggest that such declines may be better explained by restrictions in the home than in the workplace.

Fifth, this model suggests that high-income individuals should respond more to a workplace ban than lower-income individuals because their opportunity cost if time is greater. Gruber and Koszegi (2004) propose that high-income groups have less elastic responses than low-income groups to changes in the purchase prices of cigarettes. If they are correct, then the impact of di□erent reduction measures (taxes versus bans) varies by income groups. Our econometric results below provide strong support for this observation. For illustrative purposes, the optimal nicotine patterns for a restricted and unrestricted day are represented in figure 5.3, and the corresponding utility flow in figure 5.4.

Sixth, demographic and peer impacts should be important: if A becomes subject to a workplace smoking ban and wishes to substitute his smoking towards the home in the morning, the ban may be more effective if he has a non-smoking partner. However, if he has a smoking partner B, she too may wish to smoke more in the morning at home, and A and B may together facilitate this substitution. We investigate this empirically below by using information on the home demographic environment of the smoker.

Finally, we observe in practice that individuals do smoke during the working day - frequently congregating at the workplace entrance at mid morning or mid afternoon. Such observations are consistent with the model we have developed and with the simulations reported above. It may be optimal for low wage smokers to incur the

higher price during work hours; or it may be the case simply that the employer is bearing the cost of the workbreak. It follows that the number of cigarettes smoked in this regime must be at least as great as in the regime where no smoking is permitted during work.

Table 5.1 Optimal smoking patterns with and without smoking bans (7:30 am - 10 pm)

	(i) C = 18 Unrestricted	(ii) C = 16 Restricted	(iii) C = 16 Unrestricted
Interval 1 (morning pre work)	1	1	1
t = 1 .. 15	2	2	2
	9	4	11
		12	
		14	
		15	
Interval 2(morning work)	18		21
t=16....50	26		30
	35		40
	43		49
Interval 3(lunch)	51	51	58
t=51....60	60	52	
		56	
		59	
		60	
Interval 4 (afternoon work)	68		68
t=61....100	77		77
	8		87
	94		96
Interval 5 (evening)	102	101	106
t=101....145	110	102	114
	118	108	125
	127	116	131
	133	124	
Optimal intensity	1.054	1.081	1.094

Note: There are 145 six-minute intervals in this smoking day.

Figure 5.3 Optimal nicotine patterns for 16 cigarettes

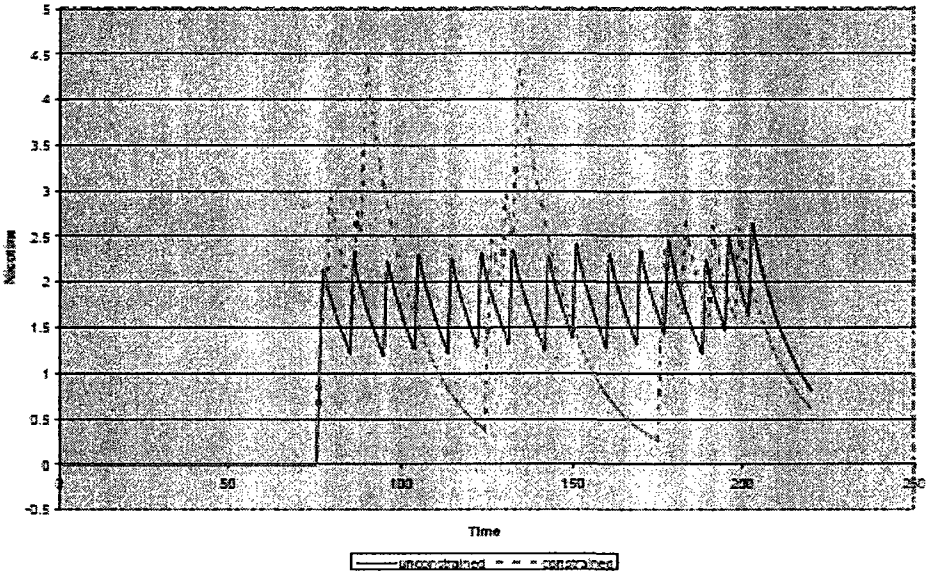
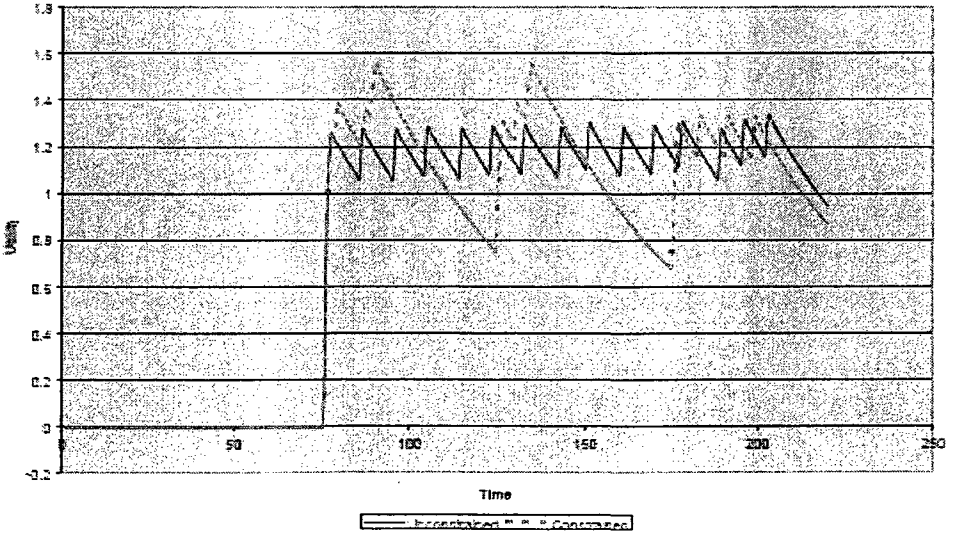


Figure 5.4 Utility path for optimal consumption pattern of 16 cigarettes



5.5 Empirical Evidence

5.5.1 Econometric Framework

In this section, we use micro data to test the predictions of the theoretical model. In particular, we examine (i) the simultaneous impact of workplace and home bans in the same regression, with a view to shedding light on their relative impacts in reducing smoking; (ii) if a workplace ban has stronger impacts on heavy smokers than on light and medium smokers; and (iii) whether high-income individuals respond more to a workplace ban than lower-income individuals on account of their opportunity cost of time.

The smoking outcome that we focus on is the log of the number of cigarettes smoked per day per smoker (*CigQ*). Our regressions are of the form:

$$log(CigQ) = \alpha WorkBan + \beta HomeBan + X\Phi + Province\ fixed\ effects + Error \quad (5.15)$$

Workban is a dummy for workplace smoking ban (1 if there is a ban, including complete and partial bans, and 0 if there is none); Homeban is a dummy for restrictions on smoking at home (1 if there is some restriction, 0 otherwise); X is vector of socio-economic variables including gender, age, education level, income, marital status, household size and language of the respondents. We include province fixed effects to capture province-specific differences including cigarette taxes and prices. Therefore, identification of workplace ban and home ban effects is achieved by within-province variation in these two variables. All our regressions use sample weights and adjust

standard errors for clustering at the province level. Equation (5.15) is estimated using three methods. We begin with OLS estimation which provides us with preliminary estimates. Then we apply quantile methods to understand better how different segments of the distribution of smokers respond to bans. Next, given a home ban is likely to be endogenous[36],we instrument it by using dummies indicating whether there are children under 12 years of age in the household, and whether the individual belongs to a voluntary organization.

5.5.2 Data

The data used in our analysis are from the 2003 Canadian Community Health Survey (CCHS)[37]. The cross sectional CCHS surveys are conducted biennially, covering several health aspects of the population. In particular, there is rich coverage of smoker behaviors, including the number of cigarettes smoked per day as well as restrictions on smoking at the workplace and in the home[38]. It also has detailed information on income, education, and other demographic variables.

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[36] Evans et al (1999) propose that a workplace ban may be endogenous due to workers' self selecting into workplaces on the basis of whether or not there may exist a smoking restriction. We think this is possible but is unlikely to be of large magnitude in the modern era given how extensive are such bans. Furthermore, our data do not yield a good instrument for the workplace ban. Most importantly, our focus on the endogeneity of home restrictions is driven by our finding that the effects of the latter are much stronger than those of workplace bans.

[37] CCHS 2003 cycle is chosen for two reasons. First, the question on home smoking restrictions is posed only to non-smokers in previous CCHS cycles. Second, questions on home and workplace ban are asked only in a sub-sample of the 2005 CCHS survey, which therefore suffers from sample size problems.

[38] The question asked on workplace ban is: 'At your place of work, what are the restrictions on smoking?' Possible responses include: (i) Restricted completely, (ii) Allowed in designated areas, (iii) Restricted only in certain places, (iv) Not restricted at all. For the home ban, the question is: 'Are there any restrictions against smoking cigarettes in your home?' and the answers are binary: (i) Yes, (ii) No.

Table 5.2 Summary statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
#Cigs	25109	16.109	8.681646	1	60
ln(#cigs)	25109	2.612448	.6273486	0	4.094345
Work Ban	22567	.5726946	.4946982	0	1
Home Ban	25023	.3707389	.4830123	0	1
Child under 12	25138	.2292545	.4203616	0	1
Member of voluntary Organization	24754	.2256605	.418025	0	1
Male	25109	.4951213	.4999862	0	1
Age	25109	7.233701	3.279057	1	15
Spouse	25031	.4657425	.498835	0	1
Hhsize	25109	2.366602	1.221952	1	5
English	25109	.6576128	.4745178	0	1
Student	25138	.0650808	.2466732	0	1
Income	21716				
< $ 15,000		.3445846	.4752436	0	1
$15,000 - $30,000		.270676	.4443192	0	1
$30,000 - $50,000		.2263769	.4184955	0	1
$50,000 - $80,000		.1227666	.3281767	0	1
> $80,000		.0355959	.1852846	0	1
Education	24592				
Less than secondary		.3305953	.4704371	0	1
Secondary school		.2125895	.4091479	0	1
Some post-secondary		.0797414	.2708979	0	1
Post-secondary		.3770738	.4846635	0	1

Table 5.2 presents summary statistics for the data. Because we study the effects of smoking bans on smoking quantity, our sample consists of daily smokers and thus excludes those categorized as occasional smokers. The average number of cigarettes

smoked per day is 16.1[39]. 57% of workplaces impose smoking bans; the home ban rate is lower, at 37%. Almost half of our sample is male, and 13.7% of respondents' families have one or more children aged five years or younger. The average age of the smokers in our sample is 42[40] and 46% of the sample reports living with a partner. Income is categorized into 5 levels, with 34% of respondents earning less than $15,000 a year and approximately 15% obtaining more than $50,000. Nearly half of the sample has some post-secondary schooling. Lastly, two thirds of the respondents use English as their main language.

5.5.3 Regression Results

5.5.3.1 OLS Estimation

The results from OLS estimation are presented in Table 5.3. Column 1 results contain a workplace ban dummy but not a home ban control. The workplace ban coefficient is negative and statistically significant, indicating that it reduces smoking by about 9% on average - less than two cigarettes per day[41] . In column 2, we keep the socio-economic controls but replace the workplace control by a home ban dummy. The resulting home ban coefficient is also negative and statistically significant. Its effect is almost three

[39] CCHS surveys accept 99 cigarettes per day as maximum. This number is too large to be credible and population representative. We therefore exclude those who report smoking more than 60 cigarettes a day from our sample.

[40] Age is coded into 15 categories in the dataset.

[41] The dummy variable coefficients are interpretable as percentage differences in the number of cigarettes smoked relative to the 'omitted category' individual in the regression. This individual smokes just very slightly less than the median individual, so we can reasonably interpret the coefficients on the ban variables as percentage impacts relative to a typical median individual.

times larger than that of a workplace ban, suggesting that it might reduce the number of cigarettes smoked on average by four per day.

Because the effect of a workplace ban might be included in the home ban estimated effect, in column 3 we include both home ban and work ban dummies in the following column of results. The effect of the workplace ban decreases slightly but is still statistically significant. The home ban coefficient also drops slightly, but remains three times are large as the workplace ban coefficient. This suggests that home bans play a considerably more important role than workplace bans in reducing smoking. Combined, the overall effect is to reduce daily consumption by 30% - about five cigarettes. This is a large number and we examine the potential endogeneity of the home ban below by instrumenting it.

The remaining variables have the expected effects. Male smokers light up more frequently than their female counterparts. Age and income effects both follow a mildly inverted U pattern. Smokers in middle income groups smoke most heavily. Note that this does not imply that individuals with higher income smoke more, given that the participation rate is much lower among those with higher incomes. Higher education is monotonically correlated with lower number of cigarettes smoked per day. Meanwhile, those who speak English smoke more heavily than those speaking other languages. The dummy Student, included to control for those currently at school, has a large negative coefficient, indicating that students smoke less than those who are not. Its large magnitude compared with the coefficient on college degree group probably indicates a cohort effect. That is, those who already have a college degree used to smoke a lot more as students than those who are currently students.

5.5.3.2 Effects by Income Groups

We now test whether the impact of a workplace ban varies with income. If our behavioral model of smoking is correct, it implies that the real cost of smoking a cigarette is larger for those with higher incomes: the largest part of the total cost of a cigarette in a regime with a workplace ban is the time cost. Hence higher income individuals have a greater incentive to reduce their smoking than those on lower incomes. The results are presented in Table 5.4. While a work ban has no perceptible impact on the lowest income groups, it becomes more effective for higher income groups, and has the largest effect at the top of the income distribution. There thus appears to be a threshold, somewhere below the middle of the income distribution, where a workplace ban becomes more effective on account of time costs. This evidence supports the theoretical model developed in the earlier part of this chapter.[42] The home ban effects are again large, though somewhat more uniform across income groups than the workplace bans.

We also estimate the model for different educational groups. The results are presented in Table 5.5. Given the high positive correlation between income and education, it is not surprising that we find effects similar to those when the sample is disaggregated by income group. Specifically, a workplace ban has no impact on the lowest educational group but becomes more effective for higher educational groups.

[42] Besides the interpretation of higher opportunity costs of time for higher income groups, peer effects may generate this outcome: if higher income smokers hold more important positions in an organization they may be more subject to social pressure to avoid taking smoking breaks at the entrance to their workplace.

Table 5.3 Workplace ban and home ban effect, OLS estimation

Variables	(1)	(2)	(3)
Workban	-0.0946***		-0.0823***
	(0.0163)		(0.0140)
Homeban		-0.242***	-0.231***
		(0.0144)	(0.0146)
Male	0.122***	0.136***	0.127***
	(0.0119)	(0.0104)	(0.0106)
Student	-0.234***	-0.207***	-0.218***
	(0.0424)	(0.0393)	(0.0439)
Age 20-24	0.0884***	0.0855***	0.0821**
	(0.0245)	(0.0250)	(0.0260)
Age 25-44	0.241***	0.224***	0.220***
	(0.0334)	(0.0372)	(0.0371)
Age 45-64	0.326***	0.299***	0.294***
	(0.0483)	(0.0513)	(0.0509)
Age 65+	0.175***	0.0997**	0.145***
	(0.0314)	(0.0344)	(0.0380)
Spouse	-0.00149	0.0254*	0.0181
	(0.0130)	(0.0122)	(0.0126)
Hhsize	-0.0250*	-0.00838	-0.00452
	(0.0120)	(0.0115)	(0.0109)
Income_2	0.0260**	0.00327	0.0224**
	(0.00827)	(0.00856)	(0.00903)
Income_3	0.0641*	0.0339	0.0658**
	(0.0294)	(0.0235)	(0.0286)
Income_4	0.0799***	0.0589**	0.0902***
	(0.0157)	(0.0206)	(0.0176)
Income_5	0.143***	0.111**	0.149***
	(0.0373)	(0.0470)	(0.0443)
Educ_2	-0.0499***	-0.0427***	-0.0357***
	(0.0105)	(0.0120)	(0.0102)
Educ_3	-0.0882**	-0.0892**	-0.0777**
	(0.0358)	(0.0359)	(0.0333)
Educ_4	-0.117***	-0.0960***	-0.0937***
	(0.00768)	(0.00724)	(0.00851)
English	0.0790*	0.0818**	0.0799*
	(0.0407)	(0.0354)	(0.0392)
Constant	2.383***	2.420***	2.432***
	(0.0611)	(0.0585)	(0.0634)
Observations	19824	21295	19816
R-squared	0.082	0.108	0.112

Notes: Robust standard errors clustered at province level in parentheses; *** p<0.01, ** p<0.05, * p<0.1.

Table 5.4 Workplace ban effect, by income groups

Variables	Lowest income	Low income	Average income	High income	Highest income
Work Ban	-0.005	-0.095***	-0.140***	-0.107	-0.215***
	(0.011)	(0.025)	(0.032)	(0.076)	(0.049)
Home Ban	-0.189***	-0.208***	-0.284***	-0.258***	-0.301***
	(0.012)	(0.034)	(0.023)	(0.038)	(0.039)
Male	0.070**	0.101***	0.152***	0.251***	0.186***
	(0.025)	(0.026)	(0.013)	(0.040)	(0.046)
Student	-0.201***	-0.199***	-0.297**	-0.483***	-0.854**
	(0.049)	(0.041)	(0.127)	(0.066)	(0.347)
Age_2	0.076***	0.050	-0.037	0.045	0.136
	(0.019)	(0.040)	(0.108)	(0.108)	(0.169)
Age_3	0.244***	0.158**	0.130	0.145	-0.062
	(0.033)	(0.063)	(0.107)	(0.106)	(0.114)
Age_4	0.340***	0.259***	0.184	0.200*	0.014
	(0.070)	(0.077)	(0.105)	(0.102)	(0.094)
Age_5	0.234***	0.048	-0.093	0.261*	0.000
	(0.065)	(0.048)	(0.128)	(0.125)	(0.000)
Spouse	0.015	0.019	0.026	-0.041	0.028
	(0.019)	(0.029)	(0.016)	(0.036)	(0.053)
Hhsize	-0.007	-0.014	0.002	0.015	-0.001
	(0.025)	(0.013)	(0.008)	(0.021)	(0.030)
Educ_2	-0.039	-0.078*	-0.016	0.014	-0.090**
	(0.027)	(0.041)	(0.044)	(0.105)	(0.040)
Educ_3	-0.065	-0.129***	-0.079	-0.011	-0.082
	(0.053)	(0.035)	(0.091)	(0.068)	(0.067)
Educ_4	-0.084***	-0.130***	-0.083	-0.028	-0.115
	(0.013)	(0.025)	(0.053)	(0.096)	(0.066)
Langu2	0.071	0.143**	0.025	0.039	0.093*
	(0.055)	(0.064)	(0.044)	(0.039)	(0.048)
Constant	2.375***	2.548***	2.666***	2.594***	2.551***
	(0.088)	(0.081)	(0.130)	(0.087)	(0.220)
Observations	6472	5352	4690	2564	738
R-squared	0.128	0.097	0.105	0.107	0.171

Notes: Robust standard errors clustered at province level in parentheses; *** p<0.01, ** p<0.05, * p<0.1.

Table 5.5 Workplace ban effect, by education groups

Variables	< Secondary	Secondary	Some post secondary	College and university
Workban	-0.0485	-0.0828***	-0.0780	-0.104***
	(0.0347)	(0.0131)	(0.0519)	(0.00935)
Homeban	-0.242***	-0.197***	-0.238***	-0.246***
	(0.0258)	(0.0298)	(0.0555)	(0.0274)
Male	0.103***	0.119***	0.120**	0.142***
	(0.0159)	(0.0298)	(0.0477)	(0.00770)
Student	-0.194**	-0.135*	-0.241***	-0.248***
	(0.0782)	(0.0612)	(0.0619)	(0.0550)
Age 20-24	0.0916**	0.131***	0.0341	0.121
	(0.0311)	(0.0309)	(0.0635)	(0.0954)
Age 25-44	0.216**	0.292***	0.201***	0.232*
	(0.0814)	(0.0578)	(0.0512)	(0.127)
Age 45-64	0.194*	0.358***	0.299***	0.344**
	(0.0899)	(0.0634)	(0.0493)	(0.153)
Age 65+	0.0193	0.299***	0.195	0.216
	(0.0643)	(0.0602)	(0.136)	(0.143)
Spouse	0.0584*	0.0170	0.00434	-0.00962
	(0.0277)	(0.0175)	(0.0452)	(0.0193)
Hhsize	-0.0360**	-0.0116	-0.00865	0.0176
	(0.0158)	(0.0129)	(0.0206)	(0.0127)
Income_2	0.0574***	0.0121	0.00228	0.0122
	(0.0171)	(0.0283)	(0.0298)	(0.0224)
Income_3	0.0670	0.0899**	0.0550	0.0531**
	(0.0788)	(0.0302)	(0.0568)	(0.0169)
Income_4	0.0637	0.0959**	0.0992**	0.0843***
	(0.0911)	(0.0332)	(0.0445)	(0.0207)
Income_5	0.244***	0.155**	0.216***	0.112*
	(0.0646)	(0.0609)	(0.0660)	(0.0595)
English	0.143**	0.0972**	0.0393	0.0379
	(0.0514)	(0.0423)	(0.0851)	(0.0504)
Constant	2.438***	2.342***	2.569***	2.335***
	(0.0570)	(0.0740)	(0.116)	(0.153)
Observations	5843	4351	1673	7949
R-squared	0.126	0.098	0.190	0.098

Notes: Robust standard errors clustered at province level in parentheses; *** p<0.01, ** p<0.05, * p<0.1.

5.5.3.3 Heavy Smokers and Lighter Smokers: Quantile Regressions

We now test the second prediction of our theoretical model - that workplace bans have larger impacts on heavy smokers, by estimating a quantile regression which includes both workplace and home ban controls. The results for selected quantiles are presented in Table 5.6. The effects of a workplace ban are quite small throughout, though broadly increasing in going from the low to the high quantiles. The 2.8% reduction at the twentieth quantile amounts to essentially no real impact, despite a significant coefficient. Given that the number of cigarettes smoked per day in this range is in the region of six to seven, the coefficient amounts to stating that the average impact is to take a couple of puffs less per day. At the mid and upper mid ranges the impact becomes more meaningful and averages about 6% - implying a numerical reduction of a little more than one cigarette. In contrast, at the ninety fifth percentile a 9% impact implies a reduction in excess of three cigarettes per day. In sum, the overall effects are again surprisingly small, with meaningful reductions achieved only at the very upper end of the distribution. Furthermore, the results are remarkably consistent with the output of the theoretical model in the preceding section. A smoker smoking 18 cigarettes per day - the value used in our illustrative simulation - lies between the sixtieth and seventieth percentiles, and the simulation indicated that such a smoker would reduce intake by two per day. We were initially surprised that the reduction was so modest, yet there appears strong support for it in the data.

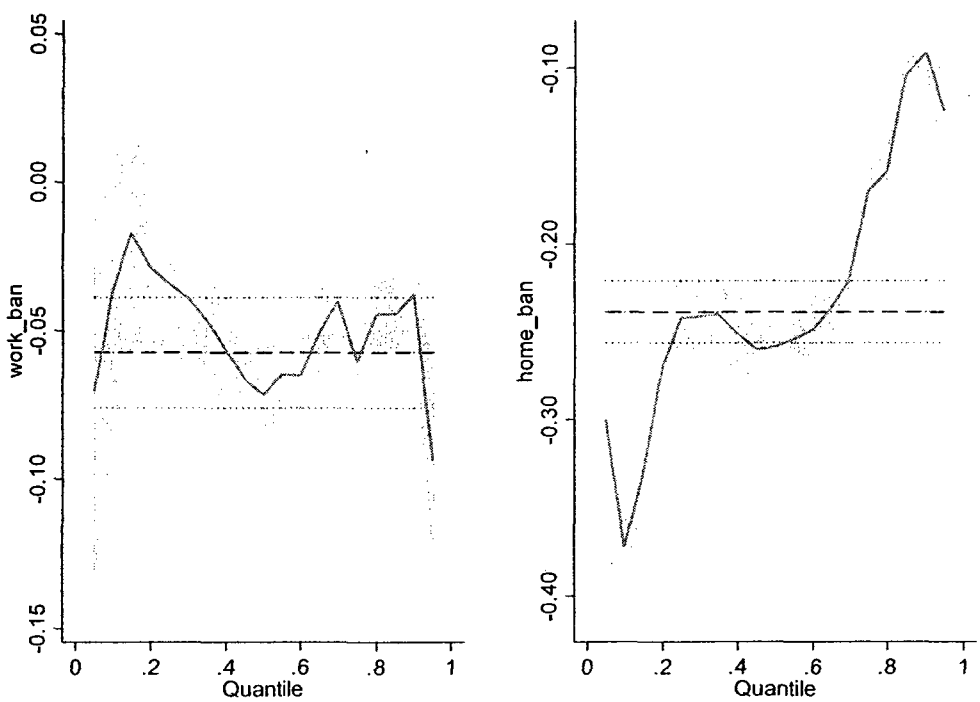
Table 5.6. Smoking ban effects at different quantiles

Variables	q20	q40	q60	q75	q85	q95
Workban	-0.028***	-0.057***	-0.065***	-0.061***	-0.044***	-0.094***
	(0.001)	(0.009)	(0.022)	(0.019)	(0.013)	(0.010)
Homeban	-0.269***	-0.252***	-0.249***	-0.170***	-0.104***	-0.124***
	(0.004)	(0.004)	(0.001)	(0.011)	(0.001)	(0.013)
Male	0.191***	0.223***	0.184***	0.121***	0.087***	0.160***
	(0.003)	(0.012)	(0.006)	(0.001)	(0.007)	(0.004)
Student	-0.240***	-0.185***	-0.159***	-0.186***	-0.175***	-0.105***
	(0.052)	(0.049)	(0.013)	(0.011)	(0.011)	(0.032)
Age 20-24	0.130***	0.117***	0.125	0.114	0.083	0.051
	(0.004)	(0.035)	(0.080)	(0.073)	(0.052)	(0.042)
Age 25-44	0.335***	0.305***	0.328***	0.341***	0.198***	0.128***
	(0.056)	(0.003)	(0.030)	(0.031)	(0.008)	(0.034)
Age 45-64	0.394***	0.427***	0.458***	0.401***	0.254***	0.231***
	(0.038)	(0.000)	(0.038)	(0.032)	(0.002)	(0.006)
Age 65+	0.192***	0.202***	0.279***	0.280***	0.179***	0.119***
	(0.031)	(0.058)	(0.060)	(0.046)	(0.007)	(0.023)
Spouse	0.026***	0.011	-0.013***	-0.008**	-0.015**	-0.026
	(0.001)	(0.012)	(0.004)	(0.004)	(0.007)	(0.019)
Hhsize	0.002	0.006***	0.011***	0.000	0.007**	-0.005
	(0.004)	(0.000)	(0.002)	(0.000)	(0.003)	(0.006)
Income_2	0.045***	0.034**	0.015	0.000	0.002	-0.019***
	(0.006)	(0.013)	(0.020)	(0.009)	(0.005)	(0.004)
Income_3	0.076***	0.058***	0.037	0.008	0.004	-0.010
	(0.018)	(0.013)	(0.025)	(0.018)	(0.012)	(0.009)
Income_4	0.109***	0.095***	0.073***	0.030*	0.015***	-0.002
	(0.009)	(0.007)	(0.024)	(0.016)	(0.003)	(0.019)
Income_5	0.132***	0.130***	0.099***	0.045**	0.037***	0.085***
	(0.033)	(0.033)	(0.035)	(0.021)	(0.005)	(0.017)
Educ_2	-0.033***	-0.041**	-0.030***	-0.030**	-0.030***	-0.041***
	(0.009)	(0.019)	(0.008)	(0.012)	(0.002)	(0.009)
Educ_3	-0.042**	-0.073***	-0.070**	-0.038***	-0.039***	-0.044
	(0.020)	(0.011)	(0.027)	(0.011)	(0.001)	(0.057)
Educ_4	-0.096***	-0.109***	-0.085***	-0.068***	-0.051***	-0.062
	(0.010)	(0.006)	(0.006)	(0.001)	(0.007)	(0.041)
English	0.039***	0.038***	0.033***	0.030***	0.024***	0.011***
	(0.007)	(0.008)	(0.007)	(0.002)	(0.005)	(0.000)
Constant	1.867***	2.246***	2.509***	2.742***	2.996***	3.355***
	(0.035)	(0.002)	(0.027)	(0.021)	(0.016)	(0.012)
Obs	19816	19816	19816	19816	19816	19816

Notes: Robust standard errors clustered at province level in parentheses; *** p<0.01, ** p<0.05, * p<0.1.

In contrast to a workplace ban, a home ban is considerably more important throughout the whole range of the distribution. The effects in the bottom 60% of the distribution are such as to reduce smoking by one quarter. The percentage reductions decline as we move to the higher percentiles, but the absolute impact increases: a 25% reduction at the lower level may result in a reduction of just two cigarettes, whereas a 12% reduction at the top end reduces the number by as much as five cigarettes per day. Figure 5.5 describes the impact of each ban at every percentile in the distribution.

Figure 5.5 Workplace ban and home ban effects, by quantiles



5.5.3.4 Home Ban Effects on Smoking: An IV Analysis

To this point it appears that if a smoker is subject to both a work place and a home ban, he will reduce his intake substantially. However, workplace bans, despite the commonly held view, are of less value, and have very little impact outside the top of the distribution. To see if this finding is robust to endogeneity concerns we now present the results for an IV estimation.

The presence or absence of home restrictions could arise from several unobservable sources: first, it may result from negotiations between family members (where the smoker is not classified as an 'individual'), including the smoker. Unfortunately our data base has no information on the smoking behaviour of a partner or spouse. A second channel may arise through home restrictions being a type of commitment device used by an individual as a result of poor health or advice from a physician.

Our main instrument for dealing with the endogeneity of the home ban is a dummy indicating whether households have one or more children less than twelve years old. We believe this is a strong instrument: worrying about the effect of exposure to smoke by offspring, parents are more likely to put in place restrictions against smoking at home. This instrument is also likely to be valid, because we expect the only way young children affect their parents' smoking is through pressuring them not to smoke at home, which is captured by the home ban. Another instrument we use is whether a respondent is a member in a voluntary organization. Being a member of voluntary organization, one would be more likely to adopt a home smoking ban if there are smoking restrictions in

the voluntary organizations themselves, and if other members already have smoking restrictions at their homes. Additionally, such membership may denote that an individual is more concerned about the externalities that attend his (smoking) behavior.

The results of the IV regression are shown in Table 5.7. The first two columns of the table use one of the two instruments, the third column results are based on both instruments being included. The coefficients on home ban from these two just-identified 2SLS regressions are negative, statistically significant and a bit larger than the OLS estimates. This is not at all surprising, because in the context of heterogenous treatment effects, the IV estimate here is LATE (local average treatment effect), and estimates the impact of a home ban on the complier group (i.e. those who impose home ban if having children under 12 years old and those who do not if having no children under 12). This complier group is most likely to respond to the home ban. In contrast, OLS estimates the mean effect on the whole population. The F statistics for excluded instrument from first-stage regressions are 227 and 29 which exceed the conventional critical value of 10 used to assess weakness of instruments. Thus, they are not weak instruments.

We next include both instruments in our regressions. The home ban coefficients do not deviate much from the just-identified cases. More importantly, there is little difference between the results estimated by 2SLS and LIML. This is reassuring because it is well known that 2SLS is likely to be biased, especially in the presence of weak instruments, and that LIML provides better estimates than 2SLS in finite samples. Also, the tests indicate that the nulls of weak instruments are easily rejected and the nulls of valid instruments cannot be rejected.

Table 5.7. IV Estimation of Home Ban Effects

Variables	IV=Children	IV= Member	IV= Children & Member; 2SLS	IV= Children & Member; GMM	IV= Children& Member; LIML
Homeban	-0.391***	-0.292*	-0.366***	-0.356***	-0.366***
	(0.082)	(0.172)	(0.077)	(0.084)	(0.077)
Workban	-0.074***	-0.079***	-0.076***	-0.076***	-0.076***
	(0.014)	(0.022)	(0.016)	(0.018)	(0.016)
Male	0.130***	0.128***	0.130***	0.130***	0.130***
	(0.009)	(0.011)	(0.009)	(0.009)	(0.009)
Student	-0.207***	-0.221***	-0.216***	-0.216***	-0.216***
	(0.044)	(0.056)	(0.050)	(0.050)	(0.050)
Age 20-24	0.078***	0.076***	0.075**	0.075**	0.075**
	(0.027)	(0.028)	(0.030)	(0.030)	(0.030)
Age 25-44	0.207***	0.208***	0.201***	0.202***	0.201***
	(0.039)	(0.028)	(0.038)	(0.036)	(0.038)
Age 45-64	0.270***	0.279***	0.269***	0.271***	0.269***
	(0.051)	(0.034)	(0.046)	(0.043)	(0.046)
Age 65+	0.124***	0.128***	0.119***	0.120***	0.119***
	(0.043)	(0.021)	(0.035)	(0.033)	(0.035)
Income_2	0.020**	0.024***	0.023***	0.023***	0.023***
	(0.010)	(0.008)	(0.009)	(0.009)	(0.009)
Income_3	0.066**	0.066**	0.067**	0.066***	0.067**
	(0.027)	(0.026)	(0.026)	(0.026)	(0.026)
Income_4	0.098***	0.096***	0.100***	0.099***	0.100***
	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)
Income_5	0.155***	0.155***	0.158***	0.156***	0.158***
	(0.048)	(0.038)	(0.043)	(0.041)	(0.043)
Educ_2	-0.026**	-0.031***	-0.026***	-0.027***	-0.026***
	(0.012)	(0.010)	(0.010)	(0.009)	(0.010)
Educ_3	-0.070**	-0.075**	-0.072**	-0.072**	-0.072**
	(0.031)	(0.036)	(0.031)	(0.031)	(0.031)
Educ_4	-0.078***	-0.091***	-0.084***	-0.084***	-0.084***
	(0.013)	(0.011)	(0.008)	(0.008)	(0.008)
Constant	2.468***	2.458***	2.474***	2.470***	2.474***
	(0.063)	(0.030)	(0.052)	(0.044)	(0.052)
First stage F	F(1,10) =227.66	F(1,10) =29.33	F(2,10) =185.58	F(2,10) =185.58	F(2,10) =185.58
Test of Overidentify restriction			Score chi2(1) = 0.49 (p = 0.48)	Hansen's J = .18 (p = 0.67)	A-R chi2(1) = .47 (p = 0.48)
Observations	19816	19594	19594	19594	19594
R-squared	0.098	0.110	0.101	0.103	0.101

Notes: Robust standard errors clustered at province level in parentheses; *** p<0.01, ** p<0.05, * p<0.1. Regression also controls for spouse, household size and English language.

5.6 Conclusions

It is important to recognize that this chapter is about behavior and incentives. It is not about social well being, nor is it about the appropriate role for governments in controlling tobacco use. This given, the results are remarkably clearcut. If we take seriously the idea that smokers should substitute from periods when smoking is prohibited to periods when it is not, then the imposition of bans on smoking in the workplace should be small for most smokers. Our theoretical model has additional predictions: (i) heavy smokers should be the ones most heavily impacted by a workplace ban, (iii) higher income smokers experience a higher time cost when a workplace ban is imposed and therefore should exhibit greater reductions, and (iii) smokers have an incentive to smoke their reduced number of cigarettes more intensively. Our empirical work indicates that the groups most affected by bans (in an absolute sense) are those at the top of the smoker distribution and at the top of the income distribution, the former because substitution becomes more challenging, and the latter on account of their elevated time costs.

A new finding in this research is that the impact of restrictions on smoking in the home is an order of magnitude larger than the impact of workplace bans. The growing spread of restrictions on smoking in the home means that workplace bans are more effective now than in an era when such home restrictions were rare: ultimately the effectiveness of government-imposed work bans depend upon the inability of smokers to switch their smoking to the home or extra-workplace environment. Consequently, the direct impact of government decrees on workplace bans as stand-alone policies would

appear to be modest. These results are consistent with, yet distinct from, those of Evans, Farrelly and Montgomery (1998). They found that the impact of a workplace ban was to reduce smoking by 10% among smokers, whereas we find a reduction in the neighbourhood of 6% for a median smoker. Our data are for a much more recent period (2003) than the data used by Evans et al (1992 and 1993). The number of cigarettes smoked per day has declined dramatically among continuing smokers in that time interval, on account of higher real prices in both jurisdictions (the US and Canada) and evolving social norms. The larger declines they obtain may be a function of the greater difficulty in avoiding bans, given the greater number of cigarettes smoked per day in 1992 and 1993 by a typical smoker.

Finally, how can the health consequences of all of this be assessed? The answer hinges critically upon whether health costs are convex or concave in toxin intake. The severity of the health impact of smoking increases with the amount of smoking: smoking for a greater number of years or smoking more cigarettes per day increases the lifetime probability of tobacco-related morbidity. For example, Godfredsen et al (2005) find that quitters reduce their probability of disease relative to continuing smokers, and also that moderate smokers have lower risks than heavy smokers. Specifically, they find a near exact proportionate relationship in the relative disease probability between smokers who smoke fewer cigarettes and smokers who smoke more. However, if low quantity smokers smoke more intensively than higher-quantity smokers, their finding implies that health consequences are convex in the amount of nicotine-correlated toxins in the body. Our quantile regression results indicate that the biggest impact of workplace bans is at the upper tail of the distribution of smokers. As a consequence, a reduction in toxin intake of

a given amount in this range of the distribution may lead to a greater improvement in health than an equal reduction at reduced smoking rates. Consequently, even if workplace bans do not reduce toxin intake substantially when smokers consume a relatively small number of cigarettes per day, health improvements may still materialize as a result of heavy smokers smoking less, given the observed convexities.

As a last word of caution, it must be recognized that more work needs to done in assessing econometrically the intensity response of smokers to these two types of bans. It is critically important to understand if the impact of home and workplace restrictions may be moderated by such responses.

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