The effects of territorial smoking restrictions on individual smoking behaviour in Australia

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Declaration

I hereby declare that this submission is my own work and any contributions or materials by other authors used in this thesis have been appropriately acknowledged. This thesis has not been previously submitted to any other university or institution as part of the requirements for another degree or award.

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Abstract

While smoking is the leading preventable cause of death in Australia, tobacco consumption is so heavily regulated that existing policy options, except for bans on smoking at public places, seem to have little room for expansion. Several US-based studies conclude that legal or voluntary restrictions on smoking at designated places (territorial smoking restrictions) provide a disincentive to smoke. It remains to be explained, however, whether the disincentive operates by limiting opportunities to smoke or changing social norms regarding smoking.

Eight new smoking bans introduced in six different Australian jurisdictions over 2003 and 2005 provide an interesting topic for evaluation. First, the determinants of smoking behaviour have been rarely studied in Australia and second, all but two of the regulatory changes involved public place bans only. The effectiveness of the bans will provide justifications for expanding smoking bans at public venues and lend support to the view that territorial bans operate by changing social norms regarding smoking since most people spend only a small fraction of time at the affected sites.

My two-part model estimates using 4 waves of the HILDA survey, however, indicate that the smoking bans affected neither the decision to smoke nor the decision on how much to smoke. The results are robust to alternative specifications and estimation methods. It seems appropriate to reconsider a third view that the disincentive comes from publicity on the smoke-related health risks which accompanies the introduction of smoking bans. This mechanism was absent during the period under my study.

1. Introduction

There is no lack of epidemiological evidence and statistics which establish strong links between smoking and a variety of adverse health consequences to smokers. In addition, following the US Surgeon General's Report in 1986, an increasing number of findings on the impact of non-smokers' exposure to passive smoking has come to public attention. Nor is it still the case that, as policy makers often seem to believe, smoking results from an ill-informed choice. Smokers are well aware of the health risked posed by tobacco consumption – perhaps too well. A casual search through scholarly databases returns a large number of recent risk perception studies concluding that smokers tend to overestimate the health risks posed by smoking; it is symbolic that in one study, the risk of lung cancer was overestimated by four-fold (Goldfarb et al., 2001).

While estimates differ across surveys a recent ABS statistic (2006) indicates that in 2004-2005, 26% of men and 23% of women among the Australian adult population were reported as current smokers. Other statistics show that in 1998 smoking was responsible for 15% of all deaths in Australia (ABS, 2006) including 82% of all drug related ones (QuitSa, 2005), making it the leading preventable cause of death. During the same period, the social cost of smoking was estimated at $21.6 billion after taking into account tangible costs such as output loss due to sickness and deaths and intangible costs including consumption opportunities lost by premature death (QuitSA, 2005). In Queensland alone, 21 children aged 0 to 4 died annually between 1999 and 2001 due to exposure to passive smoking (Queensland Government, 2006).

Even though the current prevalence of smoking places Australia among the least smoking countries in the world, there is a little reason for optimism. While a combination of successful anti-smoking regulations and increasing public awareness of the health risks had reduced the prevalence among males to roughly ¼ of the post-WWII level by the mid-1990s, the downward trend faded out once men's smoking rate caught up with women's, which has remained fairly stable for more than half a century. Furthermore, tobacco consumption is so heavily regulated in Australia -with the second most expensive cigarette price in the world, indoor smoking bans at private worksites and almost a complete ban on advertising- that traditional policy options seem virtually exhausted.

A recent trend analysis commissioned by the Department of Health and Aging (Social Research Centre, 2006) concludes using a series of comparable surveys that after remaining static around 20.40% from 1999 to 2002, the smoking prevalence among Australians aged above 17 resumed its downward trend and declined to 18.4% in 2005. The analysis also notes that the decline between 2004 and 2005 was statistically significant at the 5% level. To place the figure in perspective, it is useful to follow the approach taken by Evans et al. (1999). Based on the price elasticity of smoking prevalence commonly estimated by US researchers, -0.20, it requires a 50% increase in prices to induce a 10% drop in the prevalence; or since taxation account for 70c per dollar of Australian cigarette prices, taxes need to be raised by 70% to produce the same change. Moreover, given that cold-turkey quitting is rare, such a drop implies a considerable decline in the number of cigarettes consumed during the same period.

Available regulatory information reveals an interesting fact. Six different Australian states/territories introduced new bans on smoking at selected places between 2003 and 2005, with a majority of the changes taking effect in 2005. Real cigarette prices remained fairly stable over the same period and there was no other noticeable government intervention. The existing evaluation studies, mostly based on the USA, suggest that smoking bans are significantly associated with a decline in tobacco consumption.

My primary objective is to evaluate the effects of the new smoking bans on individual smoking behaviour in Australia, or more specifically an individual's decision to smoke and a smoker's decision on the amount of tobacco to consume. My major contribution to the relevant economic literature comes in two aspects.

First, the determinants of individual cigarette demand have been traditionally an under-investigated topic in Australia and there has been only one previous effort to evaluate the impact of smoke-free laws on smoking behaviour. My results will have important implications for policy making, for under the current Australian regulatory regime conventional anti-smoking policies have little room for expansion. While it is still technically possible to raise excise taxes substantially, taxation is always unpopular and complicated by the fact that more economically disadvantaged groups are more likely to smoke. To the contrary, smoking bans are approved not only by non-smokers, but also by a substantial fraction of smokers too (VicHealth Centre for Tobacco Control,

2002). Moreover, smoking bans at public places have much room for expansion, as evidenced by Queensland's new regulation of smoking at selected outdoor areas in 2005.

Second, the mechanisms through which smoking bans, originally designed to curb passive smoking, may affect smoking behaviour are still poorly understood. While some researchers believe that the effects come from limiting opportunities to smoke others argue that bans operate by changing social norms regarding smoking (Levy and Friend, 2003). US-based studies in general have not used an evaluation method which could disentangle the impact of workplace smoking bans from that of public place bans. Since the latter type of ban provides a much shorter duration of exposure to regulation and all but two changes under my study involved legislations on smoking at public places only, my results will be able to offer useful insight into the underlying policy mechanism.

The remainder of this thesis is structured as follows. Section 2 presents an overview of the developments of tobacco regulations in Australia and outlines the regulatory changes under my study; given that the regulatory information is hard to obtain, the outline in itself may be viewed as a minor contribution to the literature. Section 3 reviews alternative theoretical perspectives on smoking, empirical modelling approaches and the empirical literature on the effects of tobacco regulations with a particular emphasis on smoking bans. Section 4 develops a two-part model of smoking behaviour and discusses a difference-in-differences approach to be used for my evaluation. Section 5 provides a description of a 4-year panel of Australian individuals from the HILDA Survey which provides primary data for my empirical analysis.

Section 6 presents my empirical findings which stand in stark contrast to US-based studies; none of the regulatory changes had a statistically detectable impact on the probability and intensity of smoking, and even economically significant policy coefficients typically had associated p-values above 0.500 which did not warrant much confidence in the point estimates. Section 7 discusses why such a big difference may have arisen and concludes with some suggestions for future research.

2. Tobacco regulation in Australia

2.1 Tobacco regulation in Australia prior to 2002

Figure 1 : Australian smoking prevalence trends 1945-2004

Source: Reproduced directly from QuitSA (2005)

Early research into casual links between smoking and its adverse health effects culminated in recognition by the US Surgeon General's Report in 1964, stimulating anti-smoking sentiment internationally. The Australian government was initially slow to respond, while the public continued to be informed of the potential health risks by the media (QuitTAS, undated). In the following decades, however, the Australian government has crafted one of the most successful anti-smoking regulatory regimes in the world. As Figure 1 shows, the men's smoking prevalence declined impressively between 1945 and the mid-1990s until it caught up with the women's rate which had

remained surprisingly stable over the same period. The chart needs to be interpreted with caution, however, since it plots data from a number of non-comparable surveys.

The first notable intervention was the introduction of mandatory warning labels on cigarette packs in 1972; these labels were replaced twice subsequently in 1987 and 1995, increasing in the size and clarity of health-related messages (White et al., 2003). Efforts to inform the public were stepped up in the 1980s and 1990s, leading to the establishment of a cessation advisory service in each state, and a series of mass media public education campaigns were carried out, following New South Wales's lead in 1982/1983.

The assaults on the informational front extended to regulations on tobacco advertising. Starting from the removal of cigarette advertising from the broadcast media between 1973 and 1976 (White et al., 2003), a number of Commonwealth and State legislations came into effect during late 1980s through 1990s, heavily restricting published advertisements, promotion through sponsorship, point of sale advertising and value-added promotions of all tobacco products. Most notably, The Commonwealth Tobacco Advertising Prohibition Act 1992, which was intended to provide benchmark national regulation standards, phased out virtually all forms of tobacco advertising in Australia by 1995. White et al. (2003) state that by 1991, 80% of Australians were covered by advertising bans, and Bardsley and Olekalns (1999) found that the effect of advertising on aggregate tobacco consumption came to nil after 1993.

Taxation has also been employed to discourage tobacco consumption. Bardsley and Olekalans (1999) estimated that the real cigarette price rose by more than 175% over 1962/63 to 1995/96, with various taxes accounting for 65% of cigarette prices at the time when their study was published. By simulating the effects of several tobacco control programs based on rational addiction model estimates, the authors conclude that a substantial decrease in aggregate consumption during the relevant period was mainly driven by the increase in the real prices. In a recent 30-country comparison, Australia is found to have the second highest real cigarette prices (Lal and Scolo, 2002).

In the mid-1980s amid growing concerns over environmental tobacco smoke (ETS), more colloquially known as passive smoking, the federal government legislated on smoking bans at its workplaces and Australian airlines (White et al., 2003). Private businesses also responded to the increasing public demand for smoke-free environment and tightening occupational health regulations by adopting workplace smoking restrictions voluntarily or in compliance with law. While no nationwide tally is available, data from Victoria shows that the percentage of indoor workers covered by total smoking bans increased from 17 to 70% over 1988-1999, and a substantial level of protection is reported in Western Australia and New South Wales too (VicHealthCentre for Tobacco Control, 2001). The trend toward smoke-free environments continued into the late 1990s, as a series of State and Territory legislations came into effect, extending smoking bans to enclosed public venues where more general population was exposed to the risk of ETS. By 2002, a majority of State and Territories placed formal restrictions on smoking in indoor public venues including restaurants and shops, although the details and relative strictness of regulations varied from jurisdiction to jurisdiction.

Restrictions on smoking in Australia had much room for improvement, however. Most notably, existing smoking bans in general did not cover bars and gaming venues in which the concentration of ETS tended to be the highest and anti-smoking organisations called for closing loopholes arising from a variety of regulatory exemptions. Moreover, Northern Territory, the jurisdiction with the highest smoking prevalence in Australia failed to legislate on indoor smoking bans. South Australia had a relatively limited smoking ban without formalising workplace smoking prohibition. On the other hand, Victoria and Tasmania could be considered as national leaders in tobacco control.

2.2 Smoke-free legislations and regulations after 2002

Table 1 outlines the new wave of smoke-free laws/regulations which came into effect after 2002 and prior to September 2005. US researchers often refer to an official source of regulatory information which summarises different types of smoking bans in each American state. Such information is not readily available in Australia. In the absence of government-provided data outlining which legislation came into effect in which state at which date, the construction of the table greatly benefited from information compiled by Buddelmyer and Wilkins (2005), Lewis (2007), ASH Australia (2005;2007) and Drabsch (2005). All information contained in the table was subsequently confirmed as being true and comprehensive by PhD Frontdesk at the Deparment of Health and Ageing.

It seems reasonable to view the regulatory changes jointly as a natural experiment. Available regulatory data shows that there was no other major intervention by the governments during the relevant period. Insofar as media campaigns are concerned QuitVictoria's new TV commercials in 2003 and the Cancer Institute NSW's campaigns to inform certain sub-groups in 2004 and 2005 are the more significant ones; both by common sense and on the basis of econometric studies to be discussed in the next section, these items are likely to have had only a negligible impact on smoking behaviour, if any. Since the policy changes were state governments' responses to the Health Department's National Tobacco Strategy that called for the need to reduce exposure to passive smoking, a correlation between regulatory intervention and some smoke-related shocks to individuals in a particular state is highly unlikely. It is also difficult to imagine that a non-chimerical individual living outside the elegant world of rational addiction in Becker and Murphy (1988) would have invested time in going through Hansard to check on an upcoming smoke-free legislation for the purpose of making an optimal prior adjustment to his/her smoking behaviour; an illustrative anecdote comes from a prominent public health expert contacted during my information collection stage who admitted of not being well informed on recent smoking bans in Australia.

In light of the pre-existing regulations as outlined in 2.1, the effects of Northern Territory's intervention in 2003 and South Australia's in 2004 will be difficult to interpret. They may capture the impact of formalising workplace bans to the extent that occupational health laws alone had provided little incentive to impose smoking bans at worksites. Thus, their proximity to pure public ban effects will depend on the

unobserved prior diffusion of voluntary workplace smoking bans in the two jurisdictions.

Table 1: Timeline of newly imposed smoking bans, 2003-2005

2003
January & May *Northern Territory* introduces smoking bans in enclosed public venues including restaurants, shops, dining areas in hotels and bars, and indoor worksites except licensed premises. **July** *New South Wales* implements Phase One of the voluntary 'Share the Air' agreement with the industry; smoking is prohibited at bar or service counters and a non-smoking area should be designated within one bar area.
2004
July *New South Wales* implements Phase Two of the Share the Air agreement; one full smoke-free bar is to be designated in multiple-bar venues and there is a similar provision for recreational and gaming areas. **December** *South Australia* tightens its existing ban, prohibiting smoking in all enclosed public places, workplaces and shared areas; partial smoking bans apply to bars and gaming venues.
2005
January *Queensland* prohibits smoking in several outdoor areas, including sport stadiums, patrolled beaches, and areas in proximity to residential buildings and playgrounds; smoking bans in enclosed venues were tightened, requiring two thirds of licensed premises to be smoke-free before October. *Tasmania* extends smoke-free areas to a nightclub or cabaret, a gaming area and 50% of outdoor dining areas. *Western Australia* bans smoking within a metre of a bar in clubs and hotels, and within five metres of the entrance of government buildings including courts and hospitals. **January & July** *New South Wales* implements a new legislation in January, mainly formalising the Share the Air agreement. From July onwards, smoking is to be permitted in only one bar or gaming area in each premise.

3. Literature Review

3.1 Theoretical perspectives on smoking

Until relatively recently, the dominant view held that smoking was irrational behaviour. Addicts were regarded as completely myopic in the sense that they would inertly consume today what they had consumed in the past, without considering the adverse future consequences. Myopic behaviour may arise from 'incompetence', the lack of the ability to make optimal choices, and/or incapacitation, the loss of such competence due to addiction; the distinction between the two concepts can be summarised by a primrose path model from psychology, where minors pick up smoking due to the lack of discretion and as they reach adulthood as addicts, they are deprived of the ability to reverse earlier decisions (Goldfarb et al., 2001). While there has been no formal economic model of myopic addiction, Goel and Nelson (2006) classify cigarette demand applications of the partial adjustment model, which define addiction as a depreciated stock of past consumption, as the closest things in economics.

The depiction of smokers as simply irrational individuals is not immune from criticism, especially because some smokers are known to quit voluntarily and a considerable fraction of people smokes in any country. Becker and Murphy (1988) pioneered the efforts to explain smoking within a rational decision-making framework. According to their model, an individual rationally takes into account all inter-temporal dependence of cigarette consumption and choose the lifetime consumption path today to maximise lifetime utility, by balancing at each time period expected marginal benefits

against full marginal costs of addiction, including monetary price, decline in earnings and utility losses due to the deterioration of health. As in the partial adjustment applications, addiction is modelled as a stock of past consumption. The key feature of this model is adjacent complementarity, the assumption that cigarettes consumed at different periods are complements. For example when a price fall is anticipated, a smoker will adjust up his consumption today, so as to obtain a greater enjoyment in the future.

The rational addiction model of Becker and Murphy (1988) has its own critics since it implies that smokers can quit at will if it is optimal to do so. Such implication is inconsistent with the demand for cessation aids and the existence of several smokers who repeatedly fail to quit (Goldfarb et al., 2001). In addition, Suranovic et al. (1999) find it implausible to assume that individuals have the 'superhuman' ability to optimise the lifetime smoking path by rationally forecasting all future cigarette prices and earnings. Instead, they look at current utility-maximising smokers who are fully aware of the adverse health consequences of smoking and as a result may derive a negative current utility from cigarettes. The smokers are held back from quitting, however, because withdrawal effects will cause their utility to fall even further when cigarette consumption is cut below the habitual level. A substantial increase in excise taxes is required to induce a cutback in consumption so that smokers find it more worthwhile to suffer adjustment costs than pay monetary costs.

The adjustment costs approach, however, leaves unexplained why the eventual onset of withdrawal effects could not be anticipated before smokers became fully

addicted. Goldfarb et al. (2001) provide a review of competing theories of smoking and related empirical evidence, concluding that the 'under-determination of theory by data' problem is present as no single perspective fully describes the observed smoking behaviour. Jones's (1999) more technical discussion also agrees that there is a theoretical impasse.

One surprising commonality is that no theory provides positive insight into how smoking bans may affect individual smoking behaviour, notwithstanding a growing body of empirical findings on their effectiveness as anti-smoking regulations. Given the detachment of empirical modelling from theory to be discussed in next subsection, applied papers rarely dwell on filling the theoretical gap and usually present findings without considering the mechanisms through which smoking bans may operate. Among the rare explanations is the argument by Bardsley and Olekalns (1999) that smoking bans increase the real cost of smoking by imposing inconvenience costs or decrease the stock of addiction by forcing current consumption down. Chaolupka (1992), on the other hand, speculates that the passage of smoke-free legislations raises the public awareness of smoke-related problems, thereby reducing the level of smoking.

The main theoretical implications on smoking bans are therefore of a normative kind. According to the rational addiction theory, each individual is making the optimal smoking decision for himself/herself and therefore government intervention should solely aim at correcting for negative externalities (Goldfarb et al., 2001). A controversy over whether the level of tobacco taxation is too high relative to negative externalities (Chaloupka and Warner, 1999) implies that smoking bans may be criticised as a form of

majority-imposed discrimination against the minority. On the other hand, non-rational perspectives may provide justifications for smoking bans on paternalistic grounds; purely myopic smokers do not know what is best for them and current utility-maximisers are unwilling to trade off short-term utility losses for long-term gains.

3.2 Empirical methods

In aggregate-level tobacco demand studies, myopic addiction or partial adjustment models have been applied since as early as the mid-1970s, and following Becker and Murphy's (1988) influential publication a reduced form rational addiction model has been estimated by several researchers. As both approaches agree that one period lag of consumption is a significant predictor of current consumption, the point at issue has been whether to include one period lead of consumption too as implied by rational addiction theory. To be consistent with the theoretical model, the lead's coefficient needs to be interpreted as the product of the lag's coefficient and a discount factor; Baltagi and Griffin (2001) are concerned that the implied discount rate may range from a negative number to 223% depending on which instruments are used to account for the endogeneity of the lead and the lag terms.

Since the early 1990s, a growing body of the empirical literature has taken interest in modelling individual smoking behaviour, particularly in anti-smoking program evaluation context. Analytical approaches used in aggregate-level studies could not be applied directly because individual-level cigarette consumption data is characterised by a mixed distribution, with a large number of zeros and a small number

of positive values, whereas aggregate consumption is always positive. It is very difficult to take into account the mixed distribution in a dynamic context even with a rich dataset and the problem becomes intractable if the equation includes multiple endogenous regressors as in the rational addiction model (Chaloupka, 1992). A pragmatic microeconometric solution has been to use a static two-part model. In the first part, the probability of participating in smoking is modelled by a binary response model and in the second part the consumption of cigarettes conditional on having participated is linearly estimated. While limited in the ability to reflect state-dependence in smoking behaviour, this method enjoys flexibility in allowing individual characteristics to exert different influences on the participation and conditional consumption decisions.

Less common microeconometric methods include hazard regression (Kidd and Hopkins, 2004) and a dynamic probit model (Dorsett, 1999) which offers no insight into the conditional consumption decision, an individual rational addiction model which intentionally ignores the mixed distribution of micro-level data (Chaloupka, 1992) and a Heckman selectivity model and a Cragg model (Blaylock and Blissard, 1992) which share much in common with the static two-part approach.

Overall, theory has offered little guidance for empirical modelling except in a few empirical studies mainly aimed at testing the rational addiction theory. It is symbolic that most of the existing papers do not even make a casual reference to different theoretical perspectives on smoking.

3.3 International evidence on smoking regulations

Chaloupka and Warner (1999) provide a detailed review of the US-based studies of smoking. The estimated price elasticity of cigarette demand generally fell within the range of -0.3 to -0.5, regardless of the type of data or econometric method used, implying that consumers responded to price disincentives to smoke. On the other hand, empirical evidence on the effectiveness of advertising bans and government's anti-smoking advertising is summarised as being inconclusive. A newer elasticity estimate of -0.2 by Taurus (2006) is below the range because, according to the author, state fixed effects which may affect both individuals' awareness of health issues and cigarette prices have been controlled for and the retransformation problem, to be discussed in details in 4.3, has been taken into account. In other recent studies, aggregate expenditure on tobacco control programs (Marlow, 2006) and on anti-smoking advertising (Bardsley and Olekalns, 1999) are found to have no discernible effects on tobacco sales. Khwaja et al. (2006) conclude that smokers do not even respond to spouses' smoke-related health shocks and thus are not likely to be affected by general anti-smoking information.

The earliest form of smoking ban introduced in the latter half of the 1980s, either voluntarily or in compliance with legislation, targeted private worksites. Several monitoring surveys which looked at changes in smoking behaviour following the introduction of smoking bans at selected worksites are available. Comprehensive reviews of these studies (Brownson et al.,1997; Hopkins et al., 2001; Brownson et al., 2002; Levy and Friend, 2003) generally agree that worksite smoking regulation is associated with a decrease in the quantity of cigarettes consumed by workers, the level of non-smokers' exposure to passive smoking, and to a lesser extent in the smoking

prevalence. As Evans et al. (1999) state, however, the findings from these studies are not free from selection bias and thus cannot be generalised to the entire working population. For example, a majority of surveys were conducted at hospitals in the United States, 96% of which had adopted smoking bans by 1993 (Brownson et al., 2002), but hospital employees, especially doctors, are likely to have a different tendency to smoke from other workers.

Evans et al. (1999) provide arguably the most convincing evaluation of smoking bans to date. Using nationally representative individual-level data from the USA, they initially found that workplace smoking bans were associated with 5.7 percentage point decline in smoking participation and, for smokers, 2.5 less cigarettes smoked per day. These findings were successfully replicated using an alternative dataset. Even after taking into account potential endogeneity of smoking bans via the 2SLS model or controlling for health/lifestyle variables, the initial results remained mostly unchanged. Furthermore, the effects were found to increase in work hours, confirming that causality ran from the intervention. Another econometric study by Morozumi and Ii (2006) based on a cross-section of Japanese workers from Kanto and Kansai found that total smoking prohibition was associated with 10 percentage point decrease in the propensity to smoke and 4.11 less cigarettes consumed per day, while simple separation of smoking areas affected neither. In Greece, Raptou et al. (2005) report a much weaker result that total prohibition only affected the conditional demand.

In response to findings on the negative health consequences of passive smoking, indoor smoke-free laws in the United States have grown at dramatic rates; since mid-

80s, the cumulative number of such regulations increased by three- and eight-fold at state and local levels, respectively (Brownson et al., 1997). Since the effects of smoke-free laws extend to wider segments of the population than current workers, a growing body of the econometric literature has analysed their impact on smoking behaviour. In individual-level studies, reverse causality is likely to be trivial since any given individual's smoking behaviour is too trivial to affect government policy (Wasserman et al., 1991; Chaloupka, 1992). A few authors expressed concern that some unobserved state-specific sentiments might affect both individual smoking behaviour and state smoke-free legislation. Controlling for such possibility using instrumental variables for policy (Ohsfeldt et al., 1998) or adding state dummies (Tauras, 2006) did not produce qualitatively different outcomes.

One important gap in the literature is that the policy impact has been mostly discussed in terms of statistical significance only. This is not surprising, given the nature of the policy variable which has been almost invariably used by US researchers. Most of the studies initially intend to include dummy indicators for smoking bans at different venues and use cross sectional variations to estimate the impact of each. As a given state is likely to have different types of bans simultaneously, however, all policy effects tend to be imprecisely estimated. A common practice is to replace the policy dummies by a regulation index similar in spirit to one used in Wasserman et al. (1991), which takes one of five possible values. The highest score, 1, is assigned to states with workplace smoking restrictions and the second highest, 0.75, is given to those with the restriction at restaurants. The remaining positive scores, 0.5 and 0.25, are distributed according to the number of other types of restrictions in force and 0 is reserved for

regulation-free states. Other recent variants include a simple (Czart et al., 2001) or weighted tally (Tauras, 2006) of the number of smoke-free laws in a state. The use of such an 'index' is highly problematic, for it does not permit any meaningful and natural interpretation of the policy impact and drawing distinction between workplace bans and public place bans.

In two-part model applications, an increase in the 'index' has been found to be significantly associated with a decline in the adults' probability of smoking and conditional demand (Wasserman et. al., 1991) or only in the conditional demand (Tauras, 2006). Chaloupka and Wechsler (1997) and Czart et al. (2001) find that the index significantly affects the conditional demand by university students, but not the decision to smoke. However, Tauras and Chaloupka (1999), whose work stands out for being a fixed-effects analysis using a panel of young adults, has estimated the impact of the index with statistical precision in both parts. Ohsfeldt et al. (1998) is noteworthy for having directly addressed the concern that the effects of smoking bans on the decision to smoke may simply reflect the substitution of smokeless tobacco for cigarettes; while their analysis does not include the second part, they find that an increase in the index leads to a significant decline in both the probability of smoking and of 'snuff' use.

In a micro-level rational addiction model application, Chaloupka (1992) created a separate dummy for each possible positive index value. He reports that while each dummy is significant, coefficient sizes are not proportional to regulatory stringency; 0.5 and 0.75 groups had the same and biggest impact on the demand while 0.25 and 1.00 groups had smaller effects.

3.4 Australian evidence

The demand for cigarettes has been an under-investigated topic in Australia. Economics databases return only a handful of articles on Australian tobacco consumption. Bardsley and Olekalns (1999) used national-level consumption data to estimate a rational addiction model by restricting the discount factor at 0.98. Their simulation shows that the aggregate consumption became price-elastic in the early 1990s, a result which the authors attribute to advertising bans. On the other hand, direct effects of advertising bans, product advertising, anti-smoking advertising and health warning labels are found to be trivial. In individual-level studies, two-part model applications have been extremely rare, if not non-existent. Using a probit regression, Cameron and Williams (2001) estimated the own price elasticity of the probability of smoking at -0.436 between 1988 and 1995. Kidd and Hopkins (2004) failed to detect any robust price effect on the decision to smoke and the decision to quit using a sample of individuals aged 27-37 from the 1990 National Health Survey.

Apart from a couple of monitoring surveys of federal government employees from the early 1990s which reported 3.5 to 7.9 cigarettes less smoked per day and a 1 to 3% decline in smoking prevalence (Brownson et al., 1997) there seems to be no other individual-level study looking at the impact of workplace smoking bans. Bardsley and Olekalns (1999) estimate that workplace bans produced a 5% decline in aggregate tobacco consumption between the late 1980s and 1995.

Buddelmeyer and Wilkins (2005) estimated the impact of smoke-free laws on the decision to smoke and the decision to quit, using a 3-year panel from the HILDA survey. In their trivariate probit model of smoking status in the first period, sample retention between two successive periods and smoking status in the second period, a dichotomous intervention variable was found to exert no significant effect on the initiation probability, and marginally increase the quitting rate for only those aged 14-17 and above 60. This study is limited by the fact that the same binary variable was used to encompass all changes even though each state's intervention differed with respect to scope and restrictiveness and also that the impact on the conditional demand was left unexamined. In the next section, I will discuss an alternative modelling approach which can overcome these two limitations.

4. Modelling framework

4.1 Latent smoking behaviour

The use of a two-part model (2PM) to describe individual smoking behaviour has been a practical choice to take into account the mixed distribution of cigarette consumption, rather than an application of a particular theory of smoking. The statistical appeal of a 2PM relative to the Tobit model lies in the former's flexibility in allowing variables to have different effects on the decision to participate in smoking and the participant's level of consumption, in terms of both signs and magnitudes.

A discussion by Blaylock and Blisard (1992) on the latent process behind observed smoking behaviour offers intuitive justification for the separation of participation and consumption decisions. Treating zero consumption as a standard corner solution or result of unfavourable price and income is inappropriate for goods with adverse consequences like tobacco, as a big majority of individuals never consider consuming them regardless of price and income.

The authors consider a utility function of the form:

$$\mathrm{U} = \mathrm{U}(D \cdot c, q; W) \qquad [1]$$

where D=1 for a current smoker and 0 otherwise, c and q refer to consumption levels of tobacco and a composite good with unit price of one respectively, and W is a vector of

non-price and non-income factors affecting smoking. Time and individual subscripts have been suppressed for simplicity.

The current consumption decision depends on the indirect utility function:

$$\mathrm{v}(p_c, m) = \max_{D,c,q}[\mathrm{U}(D \cdot c, q; W)] \text{ s.t. } p_c \cdot c + q = m \qquad [2]$$

where p_c denotes the tobacco price and m refers to the current income. In words, v(.) results from an individual's optimal choice over feasible bundles of tobacco and the composite good. Given maintained separability assumption on U(.), D is governed by the following process:

$$\begin{aligned} &D = 1 \text{ if } y^* > 0, \text{ and } = 0 \text{ otherwise} \\ &y^* = [\mathrm{v}_1(.) - \mathrm{v}_0(.)] + [W_1 - W_0] = \Delta \mathrm{v} + \Delta W \end{aligned} \qquad [3]$$

where v_k refers to the maximised utility when D = k, and W_1 and W_0 are factors contributing to smoking and non-smoking, respectively. [3] implies that non-participation or zero consumption is not necessarily a consequence of unfavourable price and income summed up by $\Delta v < 0$. It can also arise due to an individual's aversion to smoking as captured by $\Delta W < 0$.

One important limitation of this latent variable approach is that with many common preference structures, [2] will imply positive income effects on current smokers' tobacco consumption. To the contrary, income is found to have negative

effects in many empirical studies. Formal microeconomic theories of smoking provide explanations for negative income effects. In the rational addiction model (Becker and Murphy, 1988), an increase in smoking intensity leads to a decline in the smoker's future labour productivities, while the adjustment cost theory (Suranovic et al., 1999) models future consequences as a loss of life years and hence lifetime consumption opportunities. Thus, if current income is positively related to future earnings, the real cost of smoking increases in current income.

4.2 Basic empirical approach

My empirical analysis takes the dominant approach in the anti-smoking policy evaluation literature, the two-part model (2PM), as given. This approach assumes the conditional independence of the decision of how much to consume once the participation decision has been made. This approach assumes independence of participation and consumption decisions. In the context of medical care expenditure, there has been a debate over the relative merits of the 2PM and a sample selection model such as the Heckit, which assumes bivariate normality of disturbances in the two decisions. Jones (2000, pp.285-289) summarises that while each side on the debate regard the other's distributionary assumption untenable, empirical evidence does not clearly favour a particular side. This unsettled debate and other considerations provide a little reason to deviate from the existing paradigm; conceptually tobacco consumption is observed if the person smokes and non-smoking implies no consumption, and practically valid exclusion restrictions to avoid the collinearity problem in a Mills-ratio-corrected conditional demand equation are unknown. Treating the two-part decisions as

a joint, rather than sequential, process opens room for estimating hurdle models but a comparative analysis of the various possible approaches is beyond the scope of my study. As Mullahy (1998) states a more relevant question for policy inferences may be, given that the 2PM has been demonstrated to provide useful estimates in several contexts, how its coefficients should be interpreted in the presence of the retransformation problem to be discussed below.

In part one, the participation decision in [3] is modelled by a probit model of the form:

$$\begin{aligned} y_{it}^{*} &= X_{it}^{'}\beta + u_{it} \\ \mathrm{E}(D_{it} = 1 \mid X_{it}) &= \Pr(c_{it} > 0 \mid X_{it}) = \Pr(y_{it}^{*} > 0 \mid X_{it}) = \Phi(X_{it}^{'}\beta) \end{aligned} \qquad [4]$$

where i and t are individual and time subscripts respectively, X represents a vector of characteristics and includes 1, u is a standard-normally distributed error term and Φ(.) is the standard-normal cumulative density. [4] is estimated by maximum likelihood estimation.

In part two, the optimal level of c for a smoker resulting from [2], is linearly approximated. In words, the second tier models the demand for cigarettes conditional on being a current smoker. The log transformation of the heavily skewed and strictly positive dependent variable provides a convenient way to mitigate heteroskedasticity, moderate the impact of potential outliers, and impose non-negativity and non-zero constraints on the predicted conditional demand in arithmetic units:

$$\log(c_{it} \mid c_{it} > 0) = X_{it}^{'}\gamma + e_{it}$$
$$\mathrm{E}[\log(c_{it}) \mid X_{it}, c_{it} > 0] = X_{it}^{'}\gamma \qquad [5]$$

where log(c) is the natural log of c and e is a random error term differing from u. [5] is estimated separately from [4], using an OLS regression of log(c) on X.

For policy analysis, the parameter of primary interest is the impact of policy on the arithmetic mean, $E(c_{it} \mid X_{it}, c_{it}>0)$, not on the logarithmic mean. The often-neglected retransformation problem (Manning, 1998; Manning and Mullahy, 2001) arises because e has zero mean conditional on X in logarithmic, not arithmetic units. The property of a log-normal variable, x, is illustrative; if $\log(x) \sim N(0, \sigma^2)$, then $E(\log(x)) = 0$ but $E(x) = 0.5 \cdot \sigma^2$. Hence in general,

$$\mathrm{E}(c \mid X, c > 0) = \exp(X'\gamma) \cdot \mathrm{E}(\exp(e) \mid X, c > 0) = \exp(X'\gamma) \cdot \mathrm{r}(X) \neq \exp(X'\gamma) \qquad [6]$$

for representative values of X, even if $E(e \mid X, c>0) = 0$. One approach is to assume $e \sim N(0, \sigma^2)$, in which case $r(X) = r = 0.5 \cdot \sigma^2$

A non-parametric and less restrictive approach that relaxes the distributional assumptions on e is to employ a smearing estimator of r(X) (Manning, 1998; Tauras, 2005). This approach will be used in my analysis. It can be expressed as:

$$\hat{r}(X) = \hat{r} = \frac{1}{P}\sum_{p=1}^{P}\exp(\hat{e}_p) \qquad [7]$$

where p is the subscript for observations with c > 0, P is the number of such observations, and $\hat{e}$ is the OLS residual from estimating [5]. Thus, the expected tobacco consumption estimated from my two-part model can be written as:

$$\hat{E}(c \mid X) = \hat{\Pr}(c > 0 \mid X) \cdot \hat{E}(c \mid X, c > 0) = \Phi(X'\hat{\beta}) \cdot \exp(X'\hat{\gamma}) \cdot \frac{1}{P}\sum_{p=1}^{P}\exp(\hat{e}_p) \qquad [8]$$

for representative X.

4.3 Extensions

If e_{it} is heteroskedastic its geometric function including the exponential is no longer constant across X_{it}. [6] implies that when a log prediction is retransformed by using a constant retransformation factor as in [7], heteroskedasticity not only invalidates OLS standard errors of $\hat{\gamma}$ but also biases the retransformed prediction, $\hat{E}(c \mid X, c > 0)$. The size of bias may be substantial if the error variance, and thus the smearing estimate, is large.

An alternative approach models and estimates the form of heteroskedasticity. Specifically, Manning and Mullahy (2001) propose a simpler alternative for part two which bypasses the need for retransformation:

$$\begin{aligned} &\mathrm{E}(c_{it} \mid X_{it}, c_{it} > 0) = \exp(X_{it}^{'}\mu) \\ &\log(\mathrm{E}(c_{it} \mid X_{it}, c_{it} > 0)) = X_{it}^{'}\mu \end{aligned} \qquad [9]$$

which can be consistently estimated as a GLM with log-link using the quasi-likelihood approach. The GLM requires choosing a heteroskedastic variance function of the form:

$$\mathrm{var}(c_{it} \mid X_{it}, c_{it} > 0) = \kappa \cdot [\exp(X_{it}^{'}\gamma)]^{\lambda}$$

where $\kappa > 0$ and $\lambda \geq 0$. An incorrectly specified variance leads to inefficiency, but not inconsistency. The authors use a modified Park test as a way to form the basis for choosing a suitable variance function:

$$\log((c_{it} - \hat{c}_{it})^2 \mid c_{it} > 0) = \lambda_0 + \lambda_1 \log(\hat{c}_{it}) + v_{it} \qquad [10]$$

where $\hat{c}_{it}$ is an initial GLM prediction of c_{it} with any variance function and v_{it} is a random error in the auxiliary linear regression. If $\hat{\lambda}_1 \approx 1$, [9] is re-estimated with the Poisson distribution. $\hat{\lambda}_1 \approx 2$ and $\hat{\lambda}_1 \approx 3$ suggest re-estimation with gamma and inverse Gaussian distributions, respectively. Tauras (2005) recommends a Gaussian GLM if $\hat{\lambda}_1 \approx 0$.

Simulations by Manning and Mullahy (2001) show that GLMs are consistently estimated in the presence of heteroskedasticty, whereas the OLS-based retransformation

with a smearing estimator is subject to appreciable bias; yet GLM estimates can be considerably imprecise under certain data generating mechanisms. Accordingly I will estimate a GLM as a complement, rather than substitute, to a log-linear model.

Another source of bias lies in the presence of unobserved individual heterogeneity, reflecting past experiences or personality traits which may affect an individual's perception of smoking and costs of addiction. Examples include past exposure to certain cultural values, the circle of friends and the self-assessed risk of smoking among others. These factors may be correlated with observed characteristics but can be reasonably assumed to be constant over a short span of time under my study.

The use of panel data allows me to address this issue. Specifically, assume that the error terms in [4] and [5] can be decomposed as:

$$u_{it} = a_{i1} + \upsilon_{it}$$
$$e_{it} = a_{i2} + \varepsilon_{it} \qquad [11]$$

where υ_{it} and ε_{it} are random disturbances orthogonal to X_{it} and a_{i1} and a_{i2} are time-invariant individual-specific effects possibly correlated with X_{it}. Ignorance of these latter effects will bias parameter estimators.

I will assume that individual heterogeneity is linearly related to observed characteristics as follows:

$$a_{i1} = \psi_1 + \overline{X}_i'\xi_1 + w_{i1},\ w_{i1} \mid \overline{X}_i \sim N(0, \sigma_a^2)$$
$$a_{i2} = \psi_2 + \overline{X}_i'\xi_2 + w_{i2},\ \mathrm{E}(w_{i1} \mid \overline{X}_i) = 0 \qquad [12]$$

where $\overline{\mathrm{X}}_{\mathrm{i}}$ is a vector of averages of any time-varying regressors except time dummies for each individual. Under [12], both parts can be consistently estimated as correlated random-effects models as follows:

$$\Pr(c_{it} > 0 \mid X_{it}, w_{i1}) = \Phi(X_{it}'\beta + \psi_1 + \overline{X}_i'\xi_1 + w_{i1}) \qquad [13a]$$

$$\mathrm{E}(\log(c_{it} \mid X_{it}, w_{i2}, c_{it} > 0)) = X_{it}'\gamma + \psi_2 + \overline{X}_i'\xi_2 + w_{i2} \qquad [13b]$$

where X no longer includes 1. In other words, the source of bias and inconsistency in [12] is directly controlled for, leaving well-behaving disturbances as unobservables. Once again, both parts will be estimated separately. Unlike traditional random effects models which assume away potential inconsistency, [13a] and [13b] use information from panel data to model and estimate correlations between unobserved heterogeneity and observed characteristics. While fixed-effects models are more flexible in allowing any arbitrary correlation, not only a linear form as assumed here, they are not used in this study due to the non-linear nature of my model.

4.4 Evaluation method

As discussed in 2.2, public place smoke-free laws in Australia fall under the jurisdiction of state and territory governments and a number of new smoking

restrictions had become effective between 2002 and 2005. Exogenous variation in the treatment status across states and over time, coupled with the availability of an individual-level panel spanning the relevant period, provides situations favourable to the use of a natural experiment approach as follows:

$$\begin{aligned} y_{it} &= \mathrm{f}(R_{it}) + X'_{it}\varsigma + \eta_{it} \\ \eta_{it} &= \tau_t + \iota_i + \mu_{it} \end{aligned} \qquad [14]$$

where y is a measure of smoking linear in parameters, R is the unobservable stringency index of anti-smoking policy in i's state of residence, f'(R)<0, τ is the common temporal effect, ι is the time-invariant individual heterogeneity, and μ is the temporary idiosyncratic zero-mean error.

Abstracting from X and assuming that there are only two periods and two states, the expected changes in outcomes over time can be written as:

$$\mathrm{E}(y_{it+1} - y_{it} \mid S = 1) = [\mathrm{f}(R_{it+1}) - \mathrm{f}(R_{it})] + [\tau_{t+1} - \tau_t] = \delta + \theta \qquad [15a]$$

$$\mathrm{E}(y_{it+1} - y_{it} \mid S = 0) = [\tau_{t+1} - \tau_t] = \theta \qquad [15b]$$

where S=1 for the state with a new smoking ban (ie an increase in R) and =0 otherwise.

A difference-in-differences (DID) estimator can be obtained by subtracting [15b] from [15a], thereby isolating $\delta = [\mathrm{f}(R_{it+1}) - \mathrm{f}(R_{it})] < 0$. This identification strategy depends critically on the assumption that in the absence of the new intervention,

smoking behaviour in both states will change by θ. For the strategy to be valid, it must be the case that $E(\mu_{it} \mid S = 1) = E(\mu_{it} \mid S = 0)$; this assumption is reasonable since smoking bans were part of national efforts to curb passive smoking, not each state's response to its own contemporaneous or looming smoking problem. It also has to be assumed that R will remain constant over time if not for the smoking ban; available regulatory data outlined in 2.2 shows no other policy change which might have affected R non-trivially, both for control and treatment groups. In light of the stability in the Australian smoking prevalence during years preceding 2002 and the absence of concrete counterexamples, it is not unreasonable to assume that the secular trend, $[\tau_{t+1} - \tau_t]$, will be constant across states.

The undesirability of comparing single-period outcomes can be seen from:

$$\begin{aligned} E(y_{it} \mid S = 1) - E(y_{it} \mid S = 0) = [f(R_{it} \mid S = 1) - f(R_{it} \mid S = 0)] + \\ [E(\iota_i \mid S = 1) - E(\iota_i \mid S = 0)] \end{aligned} \quad [16]$$

Thus, a single-period comparison cannot distinguish the impact of new smoking bans from that of previous anti-smoking interventions, and is subject to selection bias from pre-existing state-specific differences in tastes for smoking.

In a linear context, δ can be estimated in a regression model:

$$E(y_{it}) = X_{it}'\varsigma + \alpha S_i + \theta T + \delta(S_i \cdot T) \quad [17]$$

where T=1 for period t+1, X includes 1 and other covariates to reduce the error variance and to control for changing individual characteristics over time. It is straightforward to extend [17] to a multi-period, multi-treatment case as follows:

$$\mathrm{E}(y_{it}) = X_{it}^{'}\varsigma + [\sum_{s=1}^{6}\alpha_s S_{si} + \sum_{h=1}^{3}\theta_h T_h + \sum_{k=1}^{8}\delta_k ST_k\ ] \qquad [18]$$

where T_h=1 at and after period h and $ST_k = S_{si} \cdot T_h$ if state s introduced new smoking bans at h. As outlined in Table 1, six jurisdictions excluding Victoria and Australian Capital Territory introduced new smoking bans over 2003 - 2005 and New South Wales' intervention had been phased in through three different years. Adding terms in [.] to [4] or [5] shows the DID approach assumes that the policy impact takes the form of a permanent shift in the intercept on the latent variable or log of consumption. For simplicity, assuming a two-period two-state case, the policy impact on the participation and consumption decisions can be respectively written as:

$$\begin{aligned}&[\Pr(c>0 \mid T=1, S=1, X) - \Pr(c>0 \mid T=0, S=1, X)] - \\ &[\Pr(c>0 \mid T=1, S=0, X) - \Pr(c>0 \mid T=0, S=0, X)]\end{aligned} \qquad [19a]$$

$$\begin{aligned}&[\mathrm{E}(c \mid T=1, S=1, X, c>0) - \mathrm{E}(c \mid T=0, S=1, X, c>0)] - \\ &[\mathrm{E}(c \mid T=1, S=0, X, c>0) - \mathrm{E}(c \mid T=0, S=0, X, c>0)]\end{aligned} \qquad [19b]$$

where both [19a] and [19b] are conditioned on a representative vector X.

One limitation of this evaluation method is that it cannot isolate the impact of restriction at a particular venue because the treatment ST is invariably a package of different types of smoking restrictions. As discussed in 2.2 and 3.3, available regulatory data is not detailed enough to tabulate what type of ban is in force in which jurisdiction and even if such information is available high multicollinearity among restrictions will make it difficult to identify venue-specific effects. Subject to these limitations, the DID approach is preferable to arbitrary regulation indices used by US-based studies in terms of the ability to clarify what is being measured.

5. Data description

5.1 Data sources

The primary data for my analysis comes from the Household, Income, Labour Dynamics Australia Survey General Release 5.1. The HILDA Survey is a large indefinite panel of households across Australia starting from 2001. If any member of a household provided a wave 1 interview, then all members of his/her family are pursued over time subject to changes to the household composition (eg. marriage, death and emigration) and refusal to provide subsequent interviews.

The Survey is conducted during September of each year. On top of the household questionnaire for collecting household-level information, the Survey administers the person questionnaire (PQ) to household members aged 15 or above, asking for their personal information. The PQ is supplemented by the self-completion questionnaire (SCQ) which covers more sensitive personal topics, including smoking behaviour. The SCQ is filled out in absence of the survey administrator, and collected at a later date or mailed back. Information collected from the three questionnaires is stored in *Combined Files*, available to users in Stata format.

My analysis is based on waves 2 through 5, spanning 2002 through 2005, which provide information required for estimating the 2PM. Of 70,664 observations in total, 19,728 had not responded to the PQ and SCQ, mostly for being below 15; 4,689 had not returned the SCQ; and further 511 observations had not identified their current smoking

status. After deleting these, 45,736 observations were retained. My baseline analysis, which treats the data as pooled cross sections, uses 44,654 observations excluding those with partial non-responses. For a sensitivity check and random effect anaylsis, 28,634 observations which remained in the sample without inter-state migration during the relevant period are used. More information is provided below.

State-specific price data, including tobacco price indices and deflators for nominal variables, come from the Consumer Price Index Standard Data Report: Capital City Index Numbers by Expenditure Class September Quarter (ABS, 2004; ABS, 2005), released by the Australian Bureau of Statistics in late October each year.

5.2 Dependent variables

From wave 2 onwards, the SCQ asks whether respondents smoke cigarettes or other tobacco products. Possible responses include ‘never smoked’, ‘no longer smoke’, ‘smoke daily’, ‘smoke at least weekly’ and ‘smoke less often than weekly’. Those who belong to the latter three categories are asked to state their usual weekly consumption of tobacco products, in terms of the number of cigarettes. In wave 1, respondents were asked to state their current smoking status and weekly expenditure on tobacco products. Given the limited price information, it is not possible to convert expenditure into the number of cigarettes or vice versa; consequently, wave 1 is not used in this study.

For part one, a dichotomous indicator of current smoking status is required. SMOKE, which equals 1 for those who smoke daily, weekly or less than weekly and 0

otherwise, was created accordingly. As Table 2 shows, a big majority of self-identified smokers smoked daily. Using this variable would overstate the policy impact on tobacco use, to the extent that smokers switch to smoke-less tobacco products, such as 'snuff' in Oshfeldt et al. (1998).

Table 2: Smoking status of observations in the HILDA waves 2 - 5

	Estimation sample 1		Estimation sample 2	
Smoking status	No. of obs.	Percent	No. of obs.	Percent
Never smoked	22,131	49.56%	14,173	49.50%
No longer smoke	12,109	27.12%	8,426	29.43%
Smoke daily	8,561	19.17%	4,970	17.36%
Smoke at least weekly (but not daily)	1,045	2.34%	588	2.05%
Smoke less often than weekly	808	1.81%	477	1.67%
Total	44,654	100.00%	28,634	100.00%

In part two, the dependent variable is the logged quantity smoked. The two-tiered smoking-related questions in the SCQ introduce a slight complication. As Table 3 shows, 227 out of 10,414 smokers failed or refused to provide appropriate information. Ideally, the 2PM requires information on consumption decisions of all participants. The observations are retained in the sample, as they still provide valuable information for part one. Another minor complication arises from 55 smokers who reported zero weekly consumption. While 49 of them smoked less than weekly, it seems more natural to view zeros as refusal to respond, rather than indicators of 'social' smokers who do not smoke regularly, given the noise in the data to be discussed below. For estimation, LN(QTTY) has been created which is the natural log of the number of cigarettes smoked per week by current smokers who reported positive numbers.

Table 3: The number of cigarettes smoked per week in the HILDA waves 2 – 5; self-reported response types for smokers

	Estimation Sample 1		Estimation Sample 2	
Responses	No. of obs.	Percent[(a)]	No. of obs.	Percent[(a)]
Implausible value	1	0.01%	1	0.02%
Refused/not stated	212	2.09%	102	1.73%
Don't know	14	0.14%	5	0.08%
None	54	0.53%	29	0.49%
Mean positive weekly consumption	10133	82.25	5898	84.06
Total	10414	-	6035	-

(a) A percentage of self-reported smokers who made each inconsistent response or the mean number of cigarettes smoked by smokers who reported positive consumption

Overall, the data on the quantity smoked is less than ideal. As noted by Wasserman et al. (1991), self-reported consumption tend to be less than what is implied by actual sales data and the incentive for heavy smokers to underreport by greater amounts may have implications for the consistency of estimators. The SCQ does not seem to be immune from underreporting; there are several daily smokers who reported less consumption than less frequent smokers, or alternatively the frequency itself could have been understated. Log-transformation may mitigate the problem only to the extent that all quantities are underreported by a constant proportion. Another limitation of the SCQ data is that it fails to adjust for the potential substitution of cigarettes with heavier tobacco contents for lighter ones in response to policy intervention, the impact of which will be overestimated as a consequence. These problems are common to most of surveys and the extent to which microeconometric findings are invalidated is unknown.

5.3 Policy variables

The HILDA dataset provides state of residence (*hhstate*) and wave (*wave*) identifiers for each observation. State dummies were created using the former. Since wave n was collected in year 200n, the time dummy for 200n was set to unity if *wave* ≥ n, so that policy variables would capture a permanent shift in the intercept. On the basis of regulatory information in Section 2, policy variables were created by multiplying state dummies and relevant year dummies. South Australia's intervention in 2004 took effect in December, after the HILDA wave 4 interviews, and hence the South Australia dummy was interacted with the 2005 dummy instead.

The state dummy in equation [18] was indexed by i but not t, because in principle the DID approach requires the same individuals to be present in each group before and after an intervention, so that individual fixed effects can be cancelled out. Interstate migration is analogous to undesirable changes in treatment status. Since the current study covers 4 years and the HILDA panel is unbalanced, placing such requirements on my sample will lead to the loss of many observations.

To maximise the sample size and check for selection bias, my baseline analysis treats the data as pooled cross sections of 44,654 observations. In the evaluation literature, a DID estimator is often applied to *independently* pooled cross sections by making an extra assumption that the *expected* individual effect within each group remains constant over time even though different individuals are sampled at each period. This assumption is far less restrictive for my study since the dataset still includes a large number of individuals who provided interviews through relevant waves and did not move to other states.

For a sensitivity check and random effects analysis, the estimation sample was restricted to 28,634 observations or 7,169 individuals who 1) remained in the sample from wave 2 until relevant regulations took effect and 2) did not move across states. As listed in Table 1, most changes occurred in 2005. In effect, the new estimation sample is a balanced panel of individuals from all jurisdictions but Northern Territory; those from Northern Territory were retained so long as they were present in both waves 2 and 3.

5.4 Tobacco price index

The ABS's tobacco price index per state capital was matched with each observation, based on state and wave identifiers. The index has been used by Cameron and Williams (2001) and Kidd and Hopkins (2004) previously. It is based on the quality-adjusted retail price of one cigarette, where quality refers to the amount of tobacco content (Kidd and Hopkins, 2004). The nominal index was converted into real index and log-transformed for my analysis as follows:

$$LN(PRICE) = \log\left(\frac{tobacco_{s,t}}{cpi_{s,t}} \cdot cpi_{s,2002} \right) \qquad [20]$$

where tobacco and cpi are tobacco price index and consumer price index per state capital, s and t are state and year subscripts, $cpi_{s,2002}$ is the relevant state capital's cpi in 2002. The price data is far less detailed than those used in the US studies which contain both interstate and within-state variations.

US studies pay attention to 'informal bootlegging of cigarettes' (Wasserman et al., 1991) across states with vastly different tobacco taxes. A common solution is to exclude people living in vicinity to state borders. While the HILDA General Release does not provide detailed enough location information to make such adjustment, the border problem is likely to be trivial in Australia. As reported in Appendix 1, tobacco prices are very similar across Australian states.

Given the features of the price data, two a priori expectations can be formed. First, minimal interstate variations, coupled with trivial real changes over time, will make it hard to estimate the price impact with precision; the tobacco index grew by 13% between 2002 and 2005, in comparison to the CPI growth of 11.6%. Second, since the price data is state-specific while the DID estimator assumes a constant policy impact, there is some possibility that the price variable may pick up dynamic components of regulatory effects. Earlier US studies which ignored other tobacco regulations tended to find higher price elasticity estimates (Chaloupka and Warner, 1999).

5.5 Control variables

The HILDA survey provides a rich set of individual characteristics which can be used as control variables. Both nominal family disposable income (*hifip*) and loss (*hifin*) in dollars were available. A new family income variable, in '0,000s of 2002 dollars, was created as follows:

$$FAMINC = \left(\frac{hifip_{i,t} - hifin_{i,t}}{10000 \cdot cpi_{s,t}} \right) \cdot cpi_{s,2002} \qquad [21]$$

where i and t are individual and time subscripts, and s refer to the state of i's residence at t. The data on self-reported satisfaction with life (*losat*) was available on a 0-to-10 scale, where 0 indicates total dissatisfaction, 5 indifference, and 10 complete satisfaction. Two dummy variables were created from this, indicating dissatisfaction with life or LFDIS=1 if *losat*<4, and satisfaction with life or LFSAT=1 if *losat*>6.

Other personal characteristics to be used include family size, gender, being an Indigenous Australian, employment status, working in the hospitality sector, marital status, education, physical exercise, social club membership, socialising frequency and current drinking status. Variable definitions and summary statistics are reported in Table 4 and Table 5 respectively.

Apart from income and age which are postulated to affect the size of the eventual adverse consequences of smoking and the extent to which such consequences are discounted, no other characteristic features prominently in the theoretical models of smoking. Accordingly the existing empirical studies provided the primary guidance for choosing the control variables which can be related to risk preferences and exogenous shocks to smoking abstractly discussed in the theoretical literature. While some less commonly used variables were also used in my initial regression, they were dropped from the final analysis. More information will be provided in the next section.

As a final note, there were a relatively small number of observations with non-responses on one or more of the reported control variables. When my baseline 2PM was estimated over 45,736 observations with a missing value dummy for each of such variables, the results were not different from estimation over 44,654 observations with complete information. For reporting simplicity and to reduce the amount of time required to estimate the correlated random effects model, all observations with partial non-responses were dropped.

Table 4: Variable definitions

Variable	Source[(a)]	Description
SMOKE	lssmkf	=1 if smoke; =0 otherwise
QTTY	lstbcn	(positive) number of cigarettes smoked per week by a smoker
LN(QTTY)	lstbcn	natural log of QTTY
FMSIZ	hhpers	number of persons in the household
MJURBAN	hhsos	=1 if resides in a major urban area
RURAL	hhsos	=1 if resides in a rural area
MALE	hgsex	=1 if male
INDIG	anatsi	=1 if of Aboriginal or Torres Strait Islander origin
EMP	esbrd	=1 if currently employed
UNEMP	esbrd	=1 if cureently unemployed
HOSP	jbmind2	=1 if works in the hospitality industry
MARRIED	mrcurr	=1 if currently married legally or de facto
DIVORCED	mrcurr	=1 if currently divorced or separated
WIDOWED	mrcurr	=1 if currently widowed
PSTUGRD	edhigh	=1 if holds higher qualifications than an undergraduate degree
UGRD	edhigh	=1 if holds an undergraduate degree
YR11	edhigh	=1 if the highest qualification is year 11 or below
LFDIS	losat	=1 if dissatisfied with life
LFSAT	losat	=1 if satisfied with life
PHYACT	lspact	=1 if does physical exercise at least once a week
CLUB	lsclub	=1 if an active member of sporting/hobby/community-based associations or clubs
SOCWK	lssocal	=1 if socialises with friends or non-resident family members at least once a week
DRINK	lsdrkf	=1 if drinks
LN(PRICE)	-	logged real tobacco price index for the person's state
AGE	hgage	age as at 30 June each year
AGESQ	-	square of AGE
FAMINC	hifdip,hifdin	real family income in '0,000s of constant 2002 dollars
FAMINCSQ	-	square of FAMINC
Y*N*	wave	=1 if year $\geq 200N$; eg Y4=1 if surveyed in 2004 or 2005
Abbreviated state names	hhstate	=1 if resides in that state or territory; for example, WA=1 if the person lives in Western Australia
NT*Y3	-	NT·Y3 or Nothern Territory's regulatory intervention in 2003; other policy variables are defined similarly

(a) Variables in the HILDA dataset on which the defined variables are based.

Table 5: Summary statistics

	Estimation Sample 1[a]				Estimation Sample 2[b]			
Name	Mean	Std. Dev.	Min	Max	Mean	Std. Dev.	Min	Max
SMOKE	0.233	0.423	0.000	1.000	0.211	0.408	0.000	1.000
QTTY[c]	82.248	71.580	1.000	1200.000	84.058	70.637	1.000	600.000
LN(QTTY)[c]	3.863	1.286	0.000	7.090	3.887	1.299	0.000	6.397
FMSIZ	2.892	1.452	1.000	13.000	2.828	1.421	1.000	13.000
LN(PRICE)	5.892	0.041	5.791	5.952	5.892	0.042	5.791	5.952
AGE	43.744	17.717	15.000	93.000	45.899	16.605	15.000	93.000
FAMINC	5.596	4.229	-65.650	46.815	5.557	4.121	-65.650	46.815

Estimation Sample 1[a][d]				Estimation Sample 2[b][d]			
Name	Mean	Name	Mean	Name	Mean	Name	Mean
MJURBAN	0.604	PHYACT	0.730	MJURBAN	0.602	PHYACT	0.735
RURAL	0.125	CLUB	0.399	RURAL	0.127	CLUB	0.412
MALE	0.470	SOCWK	0.305	MALE	0.460	SOCWK	0.283
INDIG	0.017	DRINK	0.834	INDIG	0.014	DRINK	0.847
EMP	0.639	Y3	0.749	EMP	0.646	Y3	0.750
UNEMP	0.034	Y4	0.494	UNEMP	0.025	Y4	0.500
HOSP	0.041	Y5	0.246	HOSP	0.037	Y5	0.250
MARRIED	0.633	NSW	0.297	MARRIED	0.681	NSW	0.290
DIVORCED	0.091	QLD	0.203	DIVORCED	0.096	QLD	0.207
WIDOWED	0.048	SA	0.095	WIDOWED	0.046	SA	0.094
PSTUGRD	0.077	WA	0.099	PSTUGRD	0.089	WA	0.099
UGRD	0.122	TAS	0.033	UGRD	0.131	TAS	0.036
YR11	0.372	NT	0.006	YR11	0.346	NT	0.006
LFDIS	0.014	ACT	0.020	LFDIS	0.012	ACT	0.021
LFSAT	0.872			LFSAT	0.882		

(a) No. of observations = 44,654 ; No. of individuals = 15,007
(b) No. of observations = 28,634 ; No. of individuals = 7,169
(c) Summary statistics are calculated over observations with positive consumption in each estimation sample.
(d) All listed variables are binary.

6. Empirical findings

6.1 Specification search strategy

A priori similar factors are likely to affect both the decision to smoke and the intensity of smoking. Following the common practice, the same set of explanatory variables is included in both parts of my model. All estimates presented in this section have been produced by Stata 10 SE. Because the primary objective of my study is the evaluation of smoking bans, the coefficients on demographic characteristics are of secondary interest. Accordingly my specification search process involved the two steps outlined below. Only cross sectional probit and log-linear models were employed in this process, to avoid the computational burden of estimating GLM and random-effects extensions.

First to ensure the consistency of policy coefficient estimates, a cross sectional two-part model was initially specified with a richer set of control variables than the final model. The set contained characteristics which are reasonably likely to affect smoking behaviour but rarely used in the empirical literature: the continent of birth, the extent to which physical disability limits social life, experience of own child's death, and experience of parents' divorce or separation. In addition variables reported in Table 4 were specified in greater detail; for example two separate dummies were used to distinguish those who drink fewer than 5 times a week and 5 times or more.

Second for reporting simplicity and statistical precision, the initial model was simplified to the extent that doing so did not alter key inferences. For illustration, the less commonly used variables had practically negligible effects on smoking behaviour and excluding them did not affect other estimates. Merging the two drinking dummies into a single indicator did not compromise the statistical and relative economic significance of drinking as a demand determinant. The related empirical studies were consulted as a benchmark for how parsimonious the final model should be.

My final specification differs slightly from equation [18] in that the dummy for Australian Capital Territory (ACT), a control group, is included. Although the control group consists of Victoria and ACT, the two jurisdictions without new regulatory intervention during the relevant period, 874 observations from ACT in my sample has the smoking prevalence of 14.42%, far below the full sample average. Treating both regions as a homogeneous group resulted in misleading price and state fixed effects in the participation equation. The intercept without ACT dummy was -30.23, three times as large as the one reported, inducing offsetting increases in the magnitudes of the coefficients on LN(PRICE) and other state dummies to keep the rest of coefficients qualitatively intact.

6.2 Diagnostics

After simplification, my baseline two-part model in columns (1) and (2) of Table 6 was subjected to a specification link test of the form:

$$\Pr(SMOKE_n = 1) = \Phi(\beta_0 + \beta_1 \hat{I}_n + \beta_2 \hat{I}_n^2)$$
$$\log(QTTY_n \mid SMOKE_n = 1) = \gamma_0 + \gamma_1 \hat{Y}_n + \gamma_2 \hat{Y}_n^2 + v_n \quad [22]$$

using the *linktest* command from Stata, where $\hat{I}_n$ and $\hat{Y}_n$ are predicted probit index and OLS fitted values for nth observation, respectively. $\hat{\beta}_2$ and $\hat{\gamma}_2$ were statistically significant at the 5% level, rejecting the null hypothesis of no functional form misspecification. The test results were the same for the more general initial model and for alternative specifications which incorporated various interactions of control variables and those of control and policy variables.

It seems that the source of the failure is related to age. When the final model was re-estimated after dropping AGE and AGESQ, the null could not be rejected at any conventional level. On the other hand, adding higher powers of AGE to the final model or repacling AGE and AGESQ by 5-year interval age dummies led to the rejection of the null. The test results were robust to excluding observations with outlying predicted values and estimating the model separately over gender- and various age-based subgroups. Testing at the x% significant level reflects the willingness to reject the null wrongly x% of times. It seems that the rejection of the null was not a consequence of misspecification per se. A logit version of part one passes the specification test at any conventional level. Since both logit and probit are based on arbitrary distributionary assumptions, it is hard to postulate a priori why such difference should occur.

Table 7 presents a prediction success table for my baseline probit model using the 23-77 criterion, where the classification threshold has been chosen to reflect the low

sample mean of SMOKE. The model correctly classifies 72.44% of smokers and 61.41% of non-smokers. The related literature tends not to report the predictive performance of participation models. Blaylock and Blissard (1992) allude to that using the 50-50 criterion 65% of observations, roughly their sample frequency of non-smokers, were correctly classified. On the basis of the same criterion, my model correctly classifies 77.76% of all outcomes and only 12.70% of smokers.

The Breusch-Pagan test rejects the null of homosekdasticity in the baseline conditional demand model at the 1% level. The constant smearing estimate has a size of 1.672. As discussed in 4.3, a GLM model with log-link was estimated for extension. Stata command for this procedure is provided in Manning and Mullahy (2001). Since the consistency of the GLM does not depend on the variance function, $\hat{\lambda}_1$ from equation [10] was 1.3 regardless of initial variance functions, suggesting that the Poisson distribution would be a good fit. Accordingly, the GLM has been re-estimated with the Poisson variance function.

6.3 Baseline cross sectional results

Table 6 presents my baseline model and its GLM extension. Column (1) lists probit estimates for the participation equation. (2) reports OLS estimates for the conditional log demand equation along with heteroskedasticity-robust standard errors. (3) reports the log of the expected conditional demand, as estimated by the GLM, along with sandwich standard errors which are robust to variance function misspecification. Suppressed coefficient estimates can be found in Appendix 2.

Table 6: Selected cross sectional analysis results[a]

	Participation	Conditional demand	
	Probit (1)[b]	OLS (2)[c]	GLM (3)[d]
MALE	0.226***(0.014)	0.234***(0.026)	0.208***(0.0173)
HOSP	0.101***(0.033)	-0.129**(0.054)	-0.105***(0.0385)
AGE	0.047***(0.003)	0.065***(0.005)	0.052***(0.003)
AGESQ[e]	-0.001***(0.000)	-0.001***(0.000)	-0.001***(0.000)
FAMINC	-0.020***(0.003)	-0.013***(0.005)	-0.005*(0.003)
FAMINCSQ[f]	0.000***(0.000)	-0.000(0.000)	-0.000(0.000)
NSW	0.031(0.100)	0.170(0.176)	0.069(0.116)
QLD	-0.001(0.043)	0.251***(0.078)	0.203***(0.051)
SA	0.043(0.030)	0.184***(0.051)	0.083**(0.035)
WA	0.047(0.208)	0.235(0.378)	0.056(0.247)
TAS	0.293(0.223)	0.319(0.402)	0.093(0.264)
NT	0.288(0.214)	0.539*(0.302)	0.136(0.216)
ACT	-0.207*(0.115)	0.201(0.210)	0.136(0.141)
Y3	-0.040(0.034)	-0.071(0.058)	-0.060(0.041)
Y4	-0.023(0.043)	-0.016(0.077)	0.021(0.049)
Y5	-0.010(0.048)	-0.021(0.090)	-0.037(0.059)
NT*Y3	-0.164(0.204)	-0.165(0.284)	0.192(0.191)
NSW*Y3	0.020(0.052)	0.021(0.095)	0.044(0.062)
NSW*Y4	-0.006(0.047)	-0.057(0.083)	-0.083(0.056)
SA*Y5	-0.043(0.063)	-0.046(0.112)	0.018(0.071)
QLD*Y5	0.002(0.047)	-0.006(0.081)	-0.008(0.055)
TAS*Y5	0.091(0.104)	0.043(0.166)	0.022(0.110)
WA*Y5	-0.017(0.096)	-0.063(0.179)	-0.063(0.117)
NSW*Y5	-0.009(0.051)	-0.058(0.094)	0.016(0.064)
Constant	-10.53(12.71)	-4.156(23.06)	4.803(15.00)
Observations	44654	10133	10133

(a) Other coefficients can be found in Appendix 2. ***, **, * indicates statistical significance at 1, 5, 10% levels.

(b) Standard errors in parentheses. Coefficients on probit index. Pseudo-R^2 = 0.111, Log-likelihood = -21571.

(c) Heteroskedasticity-robust standard errors in parentheses. Coefficients on E(ln(QTTY)). R^2 = 0.103.

(d) Sandiwich standard errors in parentheses. Coefficients on ln(E(QTTY)). Log pseudo-likelihood = -297722.

(e) Actual entries are -0.00072(0.00003), -0.00062(0.00005) and -0.00051(0.00004) in (1), (2) and (3), respectively.

(f) Actual entries are 0.00030(0.00008), -0.00018(0.00016), and -0.00015(0.00009) in (1), (2) and (3), respectively.

Table 7: Prediction successes from Table 6 (1) using 23:77 criterion

	Actual		
Predicted	1	0	Total
1	7,544	13,212	20,756
0	2,870	21,028	23,898
Total	10,414	34,240	44,654

% of 1s correctly predicted	72.44%
% of 0s correctly predicted	61.41%
% of SMOKE correctly predicted	63.99%

Three key findings from my analysis are as follows. First, the probit estimates confirm the previous finding by Buddelmeyer and Wilkins (2005) that the Australian smoking bans in 2003 had a statistically insignificant impact on the decision to smoke. The same is the case with additional smoking bans unique to my study, regardless of the relative levels of stringency. Second, microeconomic theories predict that over time smokers with a lower level of consumption are more likely to quit eventually (Becker and Murphy, 1988; Suranovic et al., 1999). As discussed in 3.3, several US-based studies conclude that smoking bans affect the intensity of smoking even when they have no impact on the decision to smoke. My OLS and GLM estimates, however, indicate that the results cannot be generalised to Australia. Finally, my policy coefficient estimates tend to have practically negligible magnitudes and inconsistent signs across regressions. Contextual considerations do not lend support to the effectiveness of smoking bans either. New South Wales' intervention in 2004 (NSW*Y4) was the second phase of an industrial agreement in 2003 (NSW*Y3) and the coefficients on the two variables need to be added up within each column to obtain the overall impact of this policy change. As a result, Western Australia's legislation in 2005 (WA*Y5) is the only regulation which is found to have negative signs across all regressions. It was, however, the least extensive intervention under consideration, prohibiting smoking within a metre of bars and five metres of government buildings.

Overall my analysis indicates that public place smoking bans are not likely to be an effective tool to discourage tobacco consumption. As a normative corollary, any further expansion of smoking bans should be justified solely on grounds of protecting non-smokers, not by the paternalistic purpose of compelling smokers to quit and the

government needs to take an entirely new perspective on developing new anti-smoking policy which can complement the existing regulatory regime. In the remainder of this subsection, I will elaborate on the results reported in Table 6.

The eight policy variables are insignificant at any conventional level, both individually and jointly; the Wald test statistics for joint significance from columns (1) and (3) are 2.97 and 4.53 respectively, while the F-statistic from (2) is 0.37. On the other hand, the seven state dummies are jointly significant at the 1% level in all cases. Statistical insignificance is not likely to be a consequence of insufficient variations in the policy variables. Working in the hospitality sector (HOSP) has a statistically significant impact at the 1% level across columns, despite its low sample mean of 0.0408, and suggests that the hospitality workers are more likely to participate in smoking as public health experts have often worried about.

Relative to the coefficient on MALE in each equation, roughly the median of coefficients on binary indicators, all policy variables are practically negligible except for Northern Territory's intervention in 2003 (NT*Y3) which has negative and non-trivial coefficients in (1) and (2) but positive and non-trivial one in (3).

For more concrete discussion, marginal effects of covariates on expected outcome variables have been calculated. A change in the expected probability of smoking or conditional demand for cigarettes in response to a 0-to-1 increase in a dummy variable, B, can be written as:

$$\Delta \hat{\Pr}(SMOKE = 1 \mid X, \Delta B) = \Phi(X'\hat{\beta} + \hat{\beta}_B) - \Phi(X'\hat{\beta}) \quad [23a]$$

$$\Delta \hat{\mathrm{E}}(QTTY \mid X, \Delta B, QTTY > 0) = \hat{r}[\exp(X'\hat{\gamma} + \hat{\gamma}_B) - \exp(X'\hat{\gamma})] \text{ or } = \exp(X'\hat{\mu} + \hat{\mu}_B) - \exp(X'\hat{\mu}) \quad [23b]$$

where X is a vector of covariates other than B fixed at reference values, $\hat{\beta}$, $\hat{\gamma}$ and $\hat{\mu}$ are coefficient estimates from (1), (2) and (3) respectively, $\hat{r}$=1.672 and subscript B marks the coefficient on B. It can be shown using the chain rule of differentiation that the marginal effect of a continuous covariate, K, with quadratic specification is:

$$\frac{\partial \hat{\Pr}(SMOKE = 1 \mid X, K)}{\partial K} = \phi(X'\hat{\beta} + \hat{\beta}_K K + \hat{\beta}_{K^2} K^2) \cdot (\hat{\beta}_K + 2 \cdot \hat{\beta}_{K^2} K) \quad [24a]$$

$$\frac{\partial \hat{\mathrm{E}}(QTTY \mid X, K, QTTY > 0)}{\partial K} = \exp(X'\hat{\gamma} + \hat{\gamma}_K K + \hat{\gamma}_{K^2} K^2) \cdot \hat{r} \cdot (\hat{\gamma}_K + 2 \cdot \hat{\gamma}_{K^2} K) \text{ or } = \exp(X'\hat{\mu} + \hat{\mu}_K K + \hat{\mu}_{K^2} K^2) \cdot (\hat{\mu}_K + 2 \cdot \hat{\mu}_{K^2} K) \quad [24b]$$

where X is a vector of covariates other than K and K^2 held at reference values, subscript K and K^2 mark the coefficient on each variable, ϕ is the standard normal pdf, and others are defined as previously. The average treatment effect of NT*Y3 at the time of treatment have been calculated as follows:

$$[\Pr(SMOKE = 1 \mid TA) - \Pr(SMOKE = 1 \mid TB)] - [\Pr(SMOKE = 1 \mid CA) - \Pr(SMOKE = 1 \mid CB)] \quad [25a]$$

$$[\mathrm{E}(QTTY \mid QTTY > 0, TA) - \mathrm{E}(QTTY \mid QTTY > 0, TB)] [\mathrm{E}(QTTY \mid QTTY > 0, CA) - \mathrm{E}(QTTY \mid QTTY > 0, CB)] \quad [25b]$$

where the details of the expected outcomes are similar as before, the conditioning sets TA=(NT=1, NT*Y3=Y3=1, X), TB=(NT=1, NT*Y3=Y3=0, X), CA=(NT=NT*Y3=0, Y3=1, X), CB=(NT=NT*Y3=Y3=0, X), and X is a vector of covariates other than time, state, policy dummies held at reference values. In a similar fashion, the effects of WA*Y5 have also been computed. Further details of calculating the policy effects are provided in Appendix 3.

Given that each policy effect has been so imprecisely estimated, the cumbersome procedure of calculating the average of treatment effects across observations in the sample has been avoided. Instead, the policy effects and other selected marginal effects have been evaluated at the typical values of explanatory variables in the sample. A reference individual for my analysis is described as: a 44-year old married woman living in a major urban area in Victoria at 2002, in a family of three earning $56,000 per year, employed, satisfied with life, doing physical exercise, drinking, and facing the log real cigarette price of 5.892. For this reference individual, the marginal effects of the reported personal characteristics are reported in Table 8. The policy impact is reported in Table 9. NT*Y3 and WA*Y5 have been chosen because they can be viewed as the more practically relevant cases, the former in terms of economic significance and the latter in terms of consistency of signs.

Table 8 indicates that despite earlier initial theoretical concerns, inferences from log-linear and GLM estimates are similar. Indeed the conditional demand of the reference individual predicted by OLS and GLM estimates are 65.79 and 70.11

cigarettes per week, respectively. This contrasts with Tauras (2005) whose comparative analysis shows a substantial retransformation bias in the US tobacco consumption data, and suggests that the extent of the retransformation problem may be case-specific.

Table 8: Effects of covariates on expected outcomes

	Participation[(a)]	**Conditional Demand[(b)]**	
	Probit E(SMOKE=1)	OLS E(QTTY|QTTY>0)	GLM E(QTTY|QTTY>0)
MALE	0.073	17.346	16.209
HOSP	0.031	-7.963	-6.988
FMINC	-0.005	-0.998	-0.467
AGE	-0.005	0.675	0.497

(a) Changes in the predicted probability of smoking. The base probability = 0.226.
(b) Changes in the predicted number of cigarettes smoked per week by a smoker.
The base conditional demand is 65.793 for OLS and 70.106 for GLM.

Table 9: Effects of smoking bans on expected outcomes

	Participation[(a)]	**Conditional Demand[(b)]**	
	Probit E(SMOKE=1)	OLS E(QTTY|QTTY>0)	GLM E(QTTY|QTTY>0)
NT*Y3	-0.057	-19.206	15.427
WA*Y5	-0.006	-6.337	-4.459

(a) Changes in the predicted probability of smoking
(b) Changes in the predicted number of cigarettes smoked per week by a smoker

As in other studies a substantial gender effect is present possibly because males are more risk-tolerant or due to more negative social attitudes to female smokers; a man otherwise identical to the reference individual is 7 percentage points more likely to smoke and predicted to consume 17 more cigarettes than she conditional on participation. Had she worked in the hospitality sector, she would be more likely to smoke by non-trivial 3 percentage points, although her conditional demand would have

been somewhat lower by 7 to 8 cigarettes. The economic significance of age and family income falls short of their statistical significance. Appendix 4 illustrates that age effects tend to be more cumulative than marginally dramatic while income effects are trivial both cumulatively and marginally. Yet the results should not be interpreted as evidence against the popular notion that economically disadvantaged groups are substantially more likely to smoke; the estimated income effects have been conditioned on other characteristics correlated with income and hence equity-based arguments against tobacco taxation may be still relevant. In both participation and consumption decisions the penchant for smoking initially increases in age and declines later, consistent with theoretical predictions by Suranovic et al. (1999); the peak comes around 32 in the participation equation and 50 in the conditional demand models. The penchant declines in current family income, as in other cross sectional studies; the rate of decline in the participation equation approaches nil as income rises to $320,000 but that in the conditional demand equations are predicted to accelerate over the entire range.

The economic significance of WA*Y5 is rather trivial. The impact of NT*Y3 on the participation and the conditional demand is considerable, even though its sign in the conditional demand equation is not robust to methods used. Even after abstracting from statistical imprecision which does not warrant much confidence in the point estimates, the results from the first two columns must be interpreted with caution; Northern Territory's intervention included the formalisation of workplace smoking bans, not only public place bans. Coincidently, the point estimates are similar to what Evans et al. (1999) found in a study of US workplace smoking bans, -5.7 percentage point reduction in participation and 17.5 per week cigarettes less smoked.

Since the application of a two-part model to the Australian data has been non-existent, other estimates reported in Appendix 2 deserve some discussion and may warrant further research in a non-evaluation context. One interesting pattern is that stress factors, including unemployment, divorce, spouse's death and overall dissatisfaction with life, are found to increase the participation probability but exert no further impact on the conditional demand for cigarettes. Similarly, while drinkers are more likely to smoke than otherwise identical non-drinkers, drinking smokers do not consume more cigarettes than non-drinking smokers. A more formal form of socialising through social clubs is associated with a lower participation rate and a less conditional demand, whereas frequent socialising in an informal setting has the opposite effects. The more educated are both less likely to smoke and more likely to be a light smoker if the person smokes; overall satisfaction with life and participation in physical exercise have similar qualitative effects as being more educated. As expected, the price effects have been too imprecisely estimated to draw a definite conclusion.

6.4 Correlated random-effects results

Table 10 reports estimates from correlated random-effects probit and GLS, where time-invariant individual heterogeneity is assumed to be linearly related to the within-individual averages of time-varying regressors, including policy variables but not time dummies. Suppressed coefficients are reported in Appendix 5. In brief, the use of alternative estimators does not alter my general conclusion from 6.3 that there is not enough evidence to endorse the effectiveness of smoking bans.

Table 10: Selected Correlated Random-Effects Analysis Results[a]

	Participation	Conditional demand
	RE Probit (1)[b]	RE GLS (2)[c]
MALE	0.873***(0.110)	0.265***(0.0637)
HOSP	-0.162(0.167)	-0.040(0.082)
AGE	0.133***(0.024)	0.066***(0.011)
AGESQ[e]	-0.002***(0.000)	-0.001***(0.000)
FAMINC	0.031**(0.015)	0.0013(0.004)
FAMINCSQ[f]	-0.000(0.000)	-0.000***(0.000)
Y3	-0.236**(0.096)	-0.072*(0.043)
Y4	-0.089(0.121)	-0.015(0.060)
Y5	-0.177(0.135)	0.011(0.067)
NSW	-0.370(0.481)	0.203(0.261)
QLD	-0.694*(0.388)	0.028(0.234)
SA	0.384(0.541)	-0.043(0.271)
WA	0.821(0.790)	0.086(0.483)
TAS	-0.572(0.885)	0.033(0.448)
NT	-0.268(0.900)	0.080(0.445)
ACT	-0.349(0.532)	0.519(0.320)
NT*Y3	0.315(0.585)	-0.197(0.190)
NSW*Y3	0.085(0.149)	0.058(0.073)
NSW*Y4	0.094(0.134)	-0.084(0.062)
SA*Y5	0.146(0.169)	-0.095(0.084)
QLD*Y5	-0.069(0.132)	-0.156**(0.064)
TAS*Y5	0.283(0.296)	0.008(0.117)
WA*Y5	0.170(0.270)	-0.118(0.137)
NSW*Y5	-0.048(0.146)	-0.050(0.070)
Constant	-66.81(56.08)	-3.341(43.76)
Observations	28634	5898
Individuals	7169	1865
σ_c[d]	4.812	1.142
σ_e[d]	1	0.689

(a) Other coefficients can be found in Appendix 5. ***, **, * indicates statistical significance at 1, 5, 10% levels.
(b) Standard errors in parentheses. Coefficients on probit index. Log-likelihood =-6809.
(c) Standard errors robust to individual clustering in parentheses. Coefficients on E(ln(QTTY)). Overall $R^2 = 0.121$.
(d) σ_c denotes model standard deviation due to individual heterogeneity, σ_e due to random error.
(e) Actual entries are -0.00215(0.00029) in (1) and -0.00063(0.00012) in (2).
(f) Actual entries are -0.00010(0.00042) in (1) and -0.00028(0.00010) in (2).

Effects of smoking bans on conditional demand[g]

	NT*Y3	QLD*Y5	WA*Y5
E(QTTY | QTTY>0)	-17.015	-10.053	-6.826

(g) Changes in the number of cigarettes smoked per week by a smoker

In both regressions the time-demeaned averages are jointly significant at the 1% level, rejecting the null of strict exogeneity. The correlated random-effects 2PM has been estimated over the restricted sample of individuals as described in 5.3. When the baseline 2PM was estimated over the same restricted sample, no qualitative change occurred and any difference to be discussed below is not a consequence of using a different sample.

As previously, the eight policy variables are jointly insignificant at any level in both models. All individual policy effects have been imprecisely estimated in the participation equation. Furthermore, no policy intervention has the expected sign, except for Queensland's regulation in 2005 (QLD*Y5) which is practically negligible (Table 1 indicates that NSW*Y5 cannot be viewed independently from the previous two changes in the same state). The negative and economically significant coefficient on NT*Y3 from the pooled probit turns out to be sensitive to estimation method.

In the conditional demand equation, there is one policy variable, QLD*Y5, which is statistically significant at the 5% level and considerable in magnitude. It also has a negative sign as it does in columns (2) and (3) of Table 6. While this policy intervention had a greater coverage than most others by imposing smoking bans at several selected outdoor areas overemphasis on its statistical significance needs to be avoided, given that the regression includes a larger number of variables than previously and no other policy coefficient is close to being even marginally significant. Other practically non-trivial interventions with expected signs include NT*Y3 and WA*Y5, same as in the pooled log-linear conditional demand model.

For the same reference individual as in 6.3, changes in the expected conditional demand in response to each of the three interventions have been calculated as in Appendix 6**.** The calculations are slightly more complicated because of the need to incorporate the deterministic components of unobserved heterogeneity. The smearing estimate for the random-effects regression is 1.933. The estimated effects of NT*Y3 and WA*Y5 are very similar to the pooled results, 17.0148 and 6.8264 less cigarettes smoked per week, respectively. The impact of QLD*Y5 is estimated to be 10.0534 less cigarettes smoked.

Age effects on both parts are qualitatively the same as in the pooled model with roughly the same turning points. Correlated random effects models are similar to fixed effects models in that the statistical significance of time-variant regressors depends on within-individual deviations from their time-demeaned averages. Insignificant coefficients on HOSP may be understood in this context. The conditional demand is estimated to decrease in family income at increasing rates after the peak at a relatively low income, $23,750.

On the other hand, the participation probability's response to income seems to contradict the notion that those from lower income families are more likely to smoke; the probability increases in income at decreasing rates over any reasonable range of income. A possible explanation may be that the negative estimates of income effects on the participation from other cross sectional studies and my own have been confounded by unobserved and stable factors which are negatively correlated with the decision to

smoke and positively correlated with income or vice versa. The extent to which smoking is perceived negatively by peer groups is one example. Interestingly in a fixed-effects linear probability model of smoking estimated over young American adults, Tauras and Chaloupka (1999) also find significant and positive income effects on the participation decision.

6.5 Robustness checks

The previous two subsections provide very weak empirical evidence for endorsing the effectiveness of the smoking bans. In most cases, policy coefficients were indistinguishable from zero both statistically and practically. Moreover, a few practically significant coefficients were too imprecisely estimated in general to allow any definite conclusion. To ensure that the results are unrelated to subjective choices made in empirical modelling, several alternative specifications, policy variable definitions and estimation samples have been explored using pooled cross sectional analysis. In brief, none of the resulting changes affect my conclusion or warrant further investigations based on time-demanding correlated random-effects analysis. It is illustrative to note that a drastic alternative excluding all policy variables produced qualitatively the same set of coefficients as in Table 6. All regression results discussed below are available on request.

6.5.1 Interactions

As discussed in 6.2 during the specification search process several alternative models with various interaction terms of policy and control variables had been estimated. Following the line of reasoning by Buddelmeyer and Wilkins (2005), of particular interest among these would be ones incorporating interactions between each policy variable and 1) drinking, 2) hospitality employment, and 3) weekly socialising (SOCWK) indicators as individuals with these characteristics are more frequently or persistently exposed to smoking bans. The three sets of interaction terms were jointly insignificant in both parts at any conventional level and took on ad hoc signs. By choosing a certain subset of interaction terms it was possible to find some individually significant variables with expected signs at the 5% level but such efforts fall nothing short of being outright data-mining; NT*Y3*SOCWK in a probit model with interaction types 2) and 3) is one such example.

6.5.2 Alternative policy variables

A traditional DID approach assumes that the policy impact can be captured by a permanent shift in the intercept in each part. But any regulation triggers efforts to avoid it. To allow for such possibility, two alternative approaches had been taken: 1) redefining year dummies so that the policy impact disappears after one year ie setting YN to 1 in 200N only, and 2) re-estimating the baseline model using each pair of two successive waves. In the second case, treatment groups consisted of states which implemented smoking bans a year after the base year specific to each regression. Either approach produced more insignificant coefficients, both statistically and economically.

Adjusting smoking behaviour is known to be difficult and arguably there is some possibility that smoking bans might have acted with a lag. To consider such scenario, state and time dummies were interacted as if interventions had taken effect a year later than actually. When the two-part model was re-estimated, all retained policy variables, NT*Y3, NSW*Y3 and NSW*Y4 were highly insignificant in both parts, both practically and statistically.

Multicollinearity is often unduly blamed for statistical imprecision. To keep the large number of 2005-related policy variables above suspicion a single treatment status dummy, BAN = 1 for an observation residing in any of treatment groups, was created as in Buddelmeyer and Wilkins (2005). The two-part model was re-estimated after allowing BAN and its interactions with year dummies to replace all state and policy variables. The three new policy variables were jointly and individually insignificant at any conventional level in both parts, and had practically negligible coefficients.

6.5.3 Estimation over subsamples

Given that the smoking prevalence among Australian women has remained relatively stable for a few decades, a reasonable hypothesis is that women are less likely to respond to policy changes than men. To verify this, the model was estimated over males and females separately. In the participation equation, NT*Y3 was economically significant and negative only in the probit over men; in the conditional demand equation, NT*Y3 was practically important and negative only in the log-linear OLS over women. Yet, the policy variables including NT*Y3 were jointly and individually insignificant at

any conventional level in all four estimated equations and as before, it was impossible to draw any definite conclusion.

If the level of addiction to cigarettes is related to the number of years since the age of initiation, then young people are, on average, less likely to be strong addicts and therefore more likely to respond to policy changes. Readers are reminded that my model failed the specification test whenever age-related variables were added and as a by-product an exhaustive set of age-based alternatives had been explored. No matter how one defines 'young people' there is no age-based subsample over which significant policy effects can be estimated. Moreover, in most of the cases, interacting a policy variable with age, its square or arbitrarily defined age group dummies is not likely to lead to a sensible and precisely estimated coefficient on the resulting interaction term.

7. Conclusion

Empirical evidence strongly refutes the hypothesis that smokers are irrational individuals whose present consumption is completely governed by inertia due to addiction. Smokers voluntarily quit, seek cessation aids and, just as those who are not fully initiated into smoking, do respond to price disincentives to smoke. As tobacco taxation has been traditionally the most popular form of government intervention, economic theories have tended to focus on explaining mechanisms behind the small but negative and statistically significant price elasticity as estimated by a big majority of econometric studies.

At a theoretical level, little is understood about how territorial restrictions on smoking, originally introduced to curb non-smokers' exposure to passive smoking in the late 1980s, may affect the demand for cigarettes. Nonetheless, a growing body of the empirical literature, both economic and non-economic, has taken interest in the effectiveness of smoking bans as anti-smoking policy. A wide range of monitoring surveys and available econometric studies generally agree that the earliest type of smoking ban, the regulation of smoking at private worksites, is significantly associated with a decline in both the smoking prevalence and the number of cigarettes smoked by a smoker.

Some applied studies have extended the scope of evaluation to encompass both workplace and public place smoking bans. In US-based studies, it has been extremely difficult to disentangle the impact of each type of ban from another because of

multicollinearty among policy variables. A common and problematic solution has been to construct a 'regulation index' which numerically summarises the stringency of each state's smoke-free legislations. Virtually all US-based studies conclude that the 'regulation index' has a significant and negative effect on individual smoking behaviour. Two-part model applications report that an increase in the 'regulation index' is associated with a lower probability of smoking and/or a lower level of cigarette consumption conditional on smoking. Such increase, however, has no natural interpretation and not surprisingly the estimated policy impact has been rarely discussed in terms of economic significance.

Eight smoking bans introduced in six different Australian states/territories over 2003 and 2005 provided an interesting topic for evaluation in two regards. First, there has been only one known econometric analysis of the effects of smoking bans on Australian smoking behaviour and second, all but two of the regulatory changes involved public place smoking bans only. Given that under Australia's tight regulatory regime traditional anti-smoking policy options seem exhausted, the effectiveness of the smoking bans would have offered justifications for expanding smoking bans at public places. Furthermore since most people are exposed to public place bans for a relatively short duration of time, significant public ban effects would have lent support to the view that territorial bans affect smoking behaviour by affecting social norms regarding smoking.

My empirical analysis, however, finds no overall statistical evidence that the smoking bans affected either the decision to smoke or the demand for cigarettes by

continuing smokers. While Queensland's smoke-free law in 2005 has a significant effect on the conditional demand in the random-effects regression, the result is not robust to alternative estimation methods. In terms of practical significance, a big majority of the smoking bans had practically negligible effects on participation and consumption decisions. In both cross sectional and panel regressions, Western Australia's regulation in 2005 and Northern Territory's intervention in 2003 are found to have induced continuing smokers to smoke 4~7 and 16~19 fewer cigarettes per week; yet the level of statistical imprecision is too high to warrant any confidence in the point estimates because p-values associated with the two policy variables are well above 0.500. The results from alternative model specifications and sub-sample analysis indicate that no particular population sub-group responded to the new smoking bans. In addition, readers are reminded that if anything using the HILDA or any standard survey data on smoking risks overestimating policy effects due to the failure to account for substitution between 'smoky' and 'smokeless' tobacco and between heavier and lighter cigarettes.

Such results may leave one to puzzle over the large number of US publications which found the significant impact of smoke-free laws. Apart from reporting bias, the following three possibilities seem to offer reasonable explanations on the basis of potential mechanisms through which smoking bans may operate.

Smoking bans may affect individual smoking behaviour directly by limiting opportunities to smoke. Under this view public place bans are not likely to be effective since most people spend only so much time at affected areas, and conversely workplace

smoking bans are highly likely to be effective. Given the definition of the 'regulation index', it may not be surprising if the variable was significant entirely because of workplace ban effects. Evans et al. (1999) found that the effects of US smoking bans tended to increase in the number of working hours. It may not be pure coincidence that Northern Territory's legislation in 2003 (NT*Y3) was practically significant whenever it took on a negative sign; since South Australia's legislation in December 2004 (SA*Y5), which also formalised workplace bans, always had practically negligible coefficients, interpreting NT*Y3 this way requires the assumption that voluntary worksite bans were very common in SA and rare in NT due to differences in occupational health laws. On the other hand, I found no significant interaction between policy variables and personal characteristics related to the duration of exposure to public place bans.

Another related possibility is that workplace smoking bans may work because they signal to employees what employers expect. It is useful to note that in the USA health insurance is employment-related, and that the number of worksites is too large to be monitored effectively by regulators and consequently enforcement efforts may differ across employers.

Finally it is worth reconsidering Chaloupka's (1992) speculation that 'one unmeasured factor which may be being captured by the law indicators is publicity on the negative health consequences of cigarette smoking which accompanies the passage of a clean indoor air law'. In other words, the introduction of smoking bans affects people's risk perception, rather than social norms. As a sensible alternative to the

'regulation index', he defined a group dummy for each possible index point. His finding was that while all group dummies were significant, coefficient sizes were not proportional to regulatory stringency. Coincidently most of existing US-based studies covered years from the late 1980s to the mid-1990s, when information on the risk of passive smoking and smoke-related lawsuits were coming to public awareness for the first time. To the contrary during the period under my study, there was no major update on smoke-related health risks which might have caught people's attention.

Before closing it needs to be emphasised that smoking has always been one of the greatest hazards to public health and a substantial proportion of the population in any country does still smoke despite well publicised findings on smoke-related adverse health consequences. In Australia and some other developed economies, traditional anti-smoking policies targeting the general population seem to have reached natural limits. It seems fair to state that relative to the magnitude of the looming problem the economic literature has provided too few basic inputs into understanding individual smoking behaviour. Some suggestions for future research are provided below.

At a theoretical level, a theoretical framework which may gain wide acceptance and provide useful guidelines for empirical modelling is yet to come. In the empirical literature, while the effects of existing regulations have been examined by a large number of studies including my own, little attention has been paid to a more fundamental question over why people decide to smoke or quit which may have broad implications on designing new anti-smoking policies. Given that governments tend to spend considerable resources on scare tactics against smoking, one useful direction for

future research may come from examining what type of risk information smokers respond to; a recent study by Khwaja et al. (2006) concludes that smoking behaviour is affected by own health shocks but not by spouses'. In addition, if smoking is a way to satisfy a subtler desire than the demand for nicotine per se (Jones, 1999), then a direct regulation of smoking may not be the only policy lever to pull; a multivariate probit analysis of the decision to smoke and other lifestyle choices, such as drinking or physical exercise, may provide useful insight in this regard. Finally survey designers can aid empirical analysis by developing alternative measures of tobacco consumption, including a more general 'tobacco use' instead of smoking frequency and 'standard tobacco products' comparable to standard drinks.

8. Appendices

Appendix 1: ABS price indices per state capital 2002-2005

Tobacco Price Index per State Capital as at September of each year									
Year	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	Weighted Average of 8 Capital Cities
2002	347.50	362.00	370.20	362.00	327.40	328.10	341.90	346.70	354.00
2003	363.60	376.10	382.20	382.50	343.80	339.20	357.60	362.40	369.50
2004	376.40	391.70	399.40	394.70	352.60	355.40	376.20	375.60	383.10
2005	395.60	408.70	417.00	411.10	362.10	364.10	390.20	388.00	399.60

Consumer Price Index per State Capital as at September of each year									
Year	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	Weighted Average of 8 Capital Cities
2002	139.60	137.80	139.20	140.30	135.80	137.50	135.40	138.10	138.50
2003	142.40	141.80	143.30	145.40	138.60	141.10	137.80	141.90	142.10
2004	146.20	144.20	146.80	149.00	142.00	145.00	140.80	145.50	145.40
2005	150.50	148.60	150.90	153.40	147.80	150.10	144.70	149.70	149.80

Sources: ABS (2004) and ABS (2005)

Appendix 2: Additional cross sectional analysis results

	Participation	Conditional demand	
	Probit	OLS	GLM
	(1)[a]	(2)[b]	(3)[c]
FMSIZ	-0.066***(0.006)	-0.010(0.010)	-0.008(0.007)
MJURBAN	-0.098***(0.016)	-0.197***(0.028)	-0.110***(0.019)
RURAL	-0.124***(0.024)	-0.136***(0.041)	-0.076***(0.028)
INDG	0.471***(0.048)	0.063(0.062)	0.058(0.060)
EMP	-0.064***(0.019)	-0.025(0.032)	-0.029(0.022)
UNEMP	0.280***(0.038)	0.082(0.053)	0.039(0.040)
MARRIED	-0.020(0.022)	0.049(0.035)	0.024(0.025)
DIVORCED	0.330***(0.030)	0.030(0.045)	0.048(0.031)
WIDOWED	0.215***(0.047)	0.135(0.088)	0.096*(0.056)
PSTUGRD	-0.544*(0.032)	-0.612***(0.082)	-0.410***(0.053)
UGRD	-0.376***(0.024)	-0.438***(0.056)	-0.243***(0.036)
YR11	0.155***(0.016)	0.183***(0.026)	0.097***(0.018)
LFDIS	0.158***(0.057)	0.054(0.080)	0.092*(0.051)
LFSAT	-0.272***(0.021)	-0.166***(0.032)	-0.108***(0.022)
PHYACT	-0.158***(0.016)	-0.180***(0.027)	-0.137***(0.018)
CLUB	-0.204***(0.015)	-0.133***(0.028)	-0.054***(0.018)
SOCWK	0.118***(0.016)	0.113***(0.028)	0.064***(0.019)
DRINK	0.422***(0.021)	-0.056(0.036)	-0.002(0.027)
LN(PRICE)	1.632(2.157)	1.164(3.914)	-0.242(2.546)
Observations	44654	10133	10133

(a) Continued from column (1) of Table 6 : Selected cross sectional analysis results
(b) Continued from column (2) of Table 6 : Selected cross sectional analysis results
(c) Continued from column (3) of Table 6 : Selected cross sectional analysis results

Appendix 3: Computing policy effects in Table 9

To facilitate discussion, assume that NT and WA are the only two treated states. The probit, OLS, and GLM predictions can be written as in [A3.1a], [A3.2a] and [A3.3a], respectively:

$$\hat{\Pr}(SMOKE=1) = \Phi(X'\hat{\beta} + \hat{\alpha}_1 NT + \hat{\alpha}_2 WA + \hat{\theta}_1 Y3 + \hat{\theta}_2 Y4 + \hat{\theta}_3 Y5 + \hat{\delta}_1 NT*Y3 + \hat{\delta}_2 WA*Y5) \quad [A3.1a]$$

$$\hat{E}(QTTY \mid SMOKE=1) = \exp(X'\hat{\gamma} + \hat{\alpha}_1 NT + \hat{\alpha}_2 WA + \hat{\theta}_1 Y3 + \hat{\theta}_2 Y4 + \hat{\theta}_3 Y5 + \hat{\delta}_1 NT*Y3 + \hat{\delta}_2 WA*Y5)\cdot \hat{r} \quad [A3.2a]$$

$$\hat{E}(QTTY \mid SMOKE=1) = \exp(X'\hat{\mu} + \hat{\alpha}_1 NT + \hat{\alpha}_2 WA + \hat{\theta}_1 Y3 + \hat{\theta}_2 Y4 + \hat{\theta}_3 Y5 + \hat{\delta}_1 NT*Y3 + \hat{\delta}_2 WA*Y5) \quad [A3.3a]$$

where X is a vector of the reference individual's characteristics including 1, $\hat{r}$ is the smearing estimator, and notations for year, state and policy coefficients have been abused for simplicity. The impact of WA*Y5 on each prediction has been calculated as in [A3.1b], [A3.2b] and [A3.3b]:

$$\Delta\hat{\Pr} = [\Phi(X'\hat{\beta} + \hat{\alpha}_2 + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3 + \hat{\delta}_2) - \Phi(X'\hat{\beta} + \hat{\alpha}_2)] - [\Phi(X'\hat{\beta} + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3) - \Phi(X'\hat{\beta})] \quad [A3.1b]$$

$$\Delta\hat{E}_{OLS} = \{[\exp(X'\hat{\gamma} + \hat{\alpha}_2 + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3 + \hat{\delta}_2) - \exp(X'\hat{\gamma} + \hat{\alpha}_2)] - [\exp(X'\hat{\gamma} + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3) - \exp(X'\hat{\gamma})]\}\cdot \hat{r} \quad [A3.2b]$$

$$\Delta\hat{E}_{GLM} = [\exp(X'\hat{\mu} + \hat{\alpha}_2 + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3 + \hat{\delta}_2) - \exp(X'\hat{\mu} + \hat{\alpha}_2)] - [\exp(X'\hat{\mu} + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3) - \exp(X'\hat{\mu})] \quad [A3.3b]$$

The impact of NT*Y3 on the probit prediction at the time of treatment is:

$$\Delta\hat{\Pr} = [\Phi(X'\hat{\beta} + \hat{\alpha}_1 + \hat{\theta}_1 + \hat{\delta}_1) - \Phi(X'\hat{\beta} + \hat{\alpha}_1)] - [\Phi(X'\hat{\beta} + \hat{\theta}_1) - \Phi(X'\hat{\beta})] \quad [A3.1c]$$

An extension to the OLS or GLM prediction is straightforward.

Appendix 4: Age and income effects profiles*

Figure 2: Probability of smoking profile by age for the reference individual

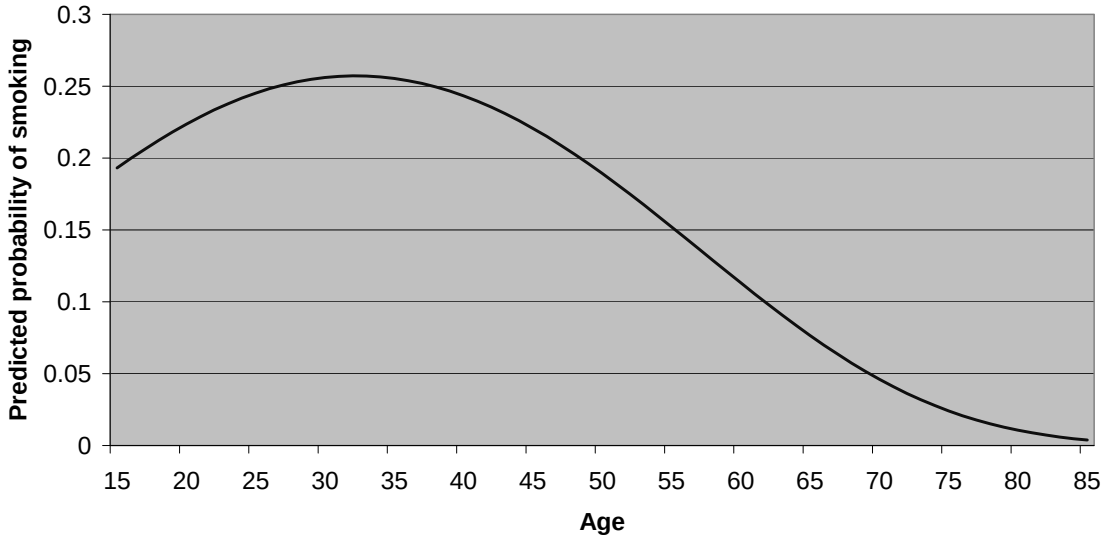
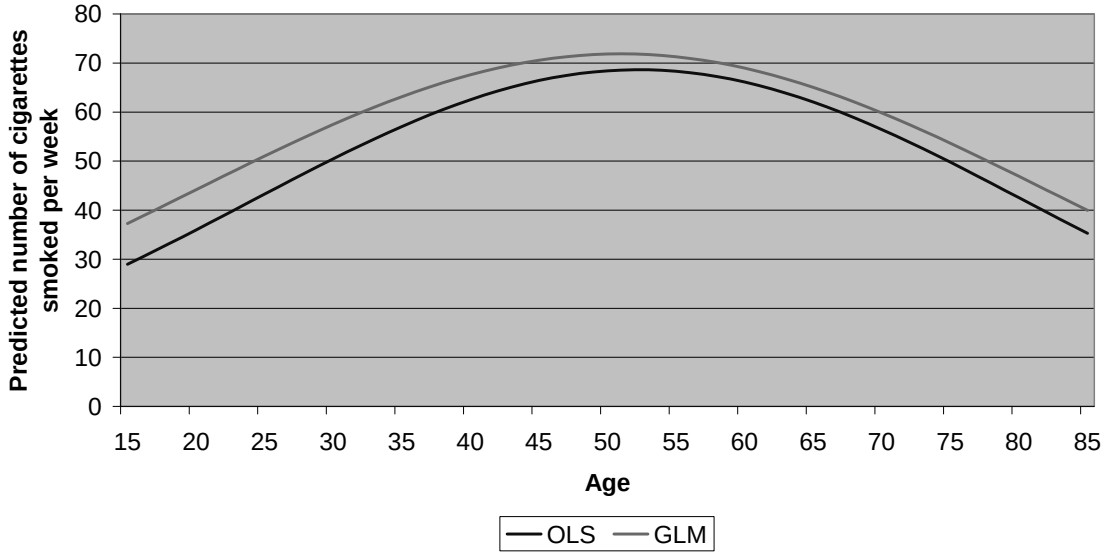


Figure 3: Cigarette consumption profile by age for the reference individual



* The following figures are based on the results discussed in 6.3.

Appendix 4: Age and income effects profiles (ctd.)

Figure 4: Probability of smoking profile by income for the reference individual

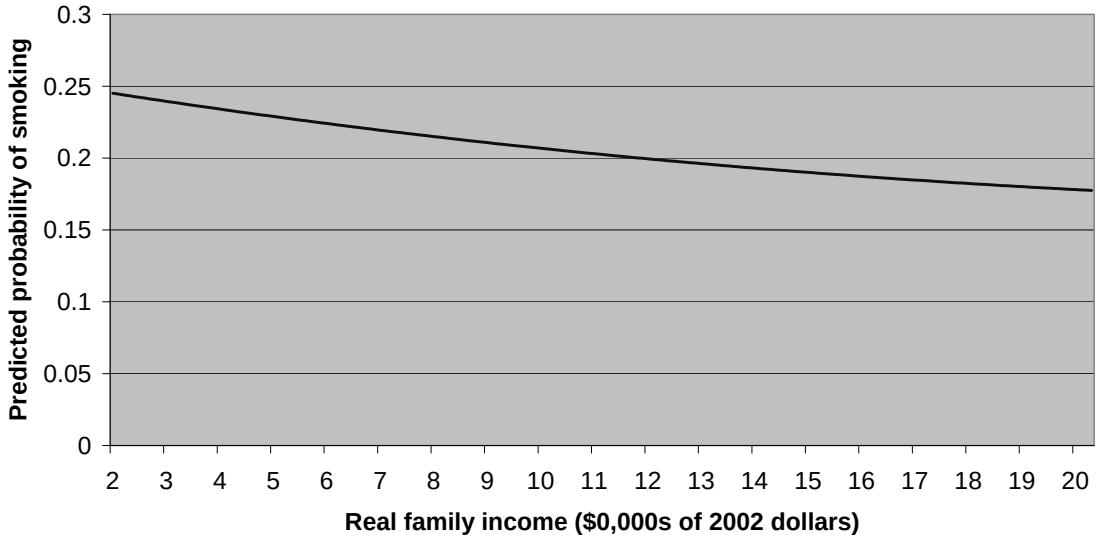
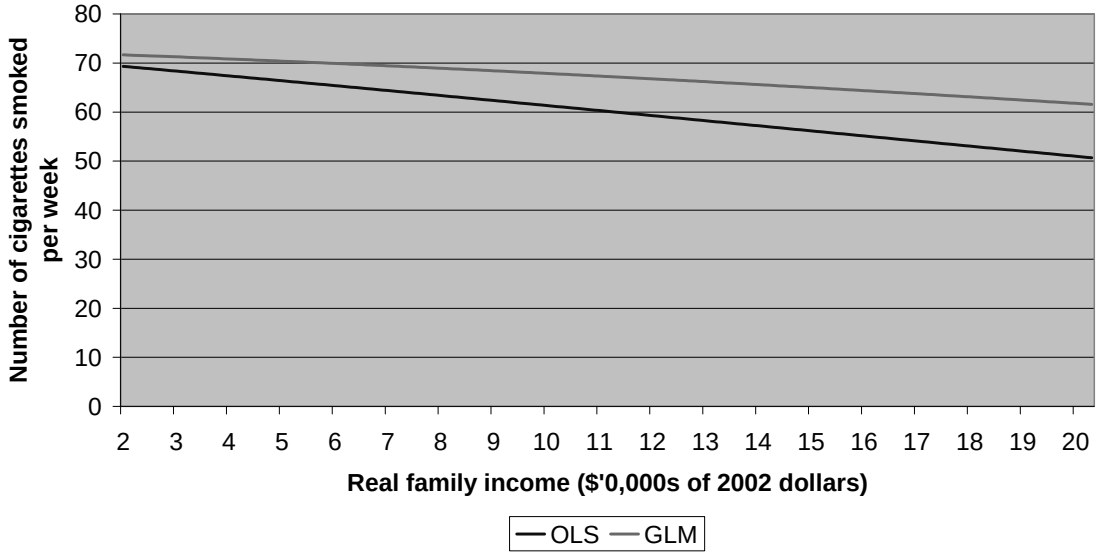


Figure 5: Cigarette consumption profile by income for the reference individual



Appendix 5: Additional Correlated Random-Effects Analysis Results[a]

	Participation	Conditional demand
	RE Probit (1)[b]	RE GLS (2)[c]
FMSIZ	-0.158***(0.045)	0.043**(0.021)
MJURBAN	-0.168(0.159)	0.062(0.072)
RURAL	-0.428**(0.175)	-0.025(0.079)
INDG	1.658***(0.480)	0.439***(0.156)
EMP	0.310***(0.100)	0.028(0.048)
UNEMP	0.224(0.148)	-0.034(0.061)
MARRIED	0.094(0.148)	0.047(0.072)
DIVORCED	0.586**(0.231)	0.124(0.107)
WIDOWED	1.714***(0.478)	-0.112(0.210)
PSTUGRD	-0.989**(0.476)	-0.172(0.235)
UGRD	-0.430(0.289)	-0.426**(0.211)
YR11	-0.468**(0.193)	-0.463***(0.106)
LFDIS	0.191(0.201)	-0.051(0.073)
LFSAT	0.013(0.082)	-0.037(0.034)
PHYACT	-0.126**(0.063)	-0.022(0.031)
CLUB	0.015(0.068)	-0.042(0.033)
SOCWK	0.060(0.066)	0.088***(0.030)
DRINK	0.439***(0.116)	0.085(0.059)
LN(PRICE)	5.386(6.074)	0.983(3.009)
Observations	28634	5898
Individuals	7169	1865

(a) Coefficients on time-demeaned averages have been suppressed and are available on request.
(b) Continued from column (1) of Table 10: Selected Correlated Random-Effects Analysis Results
(c) Continued from column (2) of Table 10: Selected Correlated Random-Effects Analysis Results

Appendix 6: Computing policy effects in Table 10

To facilitate discussion, assume that NT and WA are the only two treated states. The GLS conditional demand prediction can be written as:

$$\begin{aligned}\hat{\mathrm{E}}(QTTY \mid SMOKE = 1) = \exp(&X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\alpha}_1 NT + \hat{\alpha}_2 WA + \hat{\theta}_1 Y3 + \hat{\theta}_2 Y4 \\ &+ \hat{\theta}_3 Y5 + \hat{\delta}_1 NT*Y3 + \hat{\xi}_{NT*Y3}\overline{NT*Y3} + \hat{\delta}_2 WA*Y5 \\ &+ \hat{\xi}_{WA*Y5}\overline{WA*Y5}) \cdot \hat{r}\end{aligned} \qquad \text{[A6.1a]}$$

where X is a vector of the reference individual's characteristics excluding 1, $\overline{\mathrm{X}}$ is a vector of the reference individual's average time-variant characteristics which are held at the same values as her time-variant characteristics in X for the current purpose, $\hat{\xi}_{\mathrm{NT*Y3}}$ and $\hat{\xi}_{\mathrm{WA*Y5}}$ are the coefficients on the time-demeaned averages of respective policy variables. The effects of NT*Y3 and WA*Y5 have been calculated as in [A6.1b] and [A6.1c], respectively:

$$\begin{aligned}\Delta\hat{\mathrm{E}}_{GLS} = \{&[\exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\alpha}_1 + \hat{\theta}_1 + \hat{\delta}_1 + \hat{\xi}_{NT*Y3} \cdot 0.75) \\ &- \exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\alpha}_1 + \hat{\xi}_{NT*Y3} \cdot 0.75)] \\ &- [\exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\theta}_1) - \exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2)]\} \cdot \hat{r}\end{aligned} \qquad \text{[A6.1b]}$$

$$\begin{aligned}\Delta\hat{\mathrm{E}}_{GLS} = \{&[\exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\alpha}_2 + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3 + \hat{\delta}_2 + \hat{\xi}_{WA*Y5} \cdot 0.25) \\ &- \exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\alpha}_2 + \hat{\xi}_{WA*Y5} \cdot 0.25)] \\ &- [\exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2 + \hat{\theta}_1 + \hat{\theta}_2 + \hat{\theta}_3) - \exp(X'\hat{\gamma} + \overline{X}'\hat{\xi}_2 + \hat{\psi}_2)]\} \cdot \hat{r}\end{aligned} \qquad \text{[A6.1c]}$$

The impact of QLD*Y5 has been computed in a similar fashion as [A6.1c]. The related spreadsheet output is available on request.

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