// NO.24-058 | 09/2024

DISCUSSION PAPER

// CAMILA STEFFENS AND PAULA CARVALHO PEREDA

Dynamic Responses to Smoking Bans: Evidence From Young Adults in a Developing Country

Dynamic Responses to Smoking Bans: Evidence from Young Adults in a Developing Country

Camila Steffens* and Paula Carvalho Pereda§

Abstract

Smoking bans have been widely implemented, despite mixed evidence on their effectiveness in reducing firsthand smoking. This paper provides novel insights into the dynamic impacts of smoking bans in the context of a large developing country, Brazil, that had more than 18.6 million regular smokers in 2013. Our estimation strategy exploits the staggered implementation of comprehensive smoking bans in Brazilian state capitals using an event-study framework. We also leverage the variation in policy enforcement across cities. Our results indicate that bans reduced smoking prevalence by up to 15% among young adults, particularly when rigorously enforced. This effect is primarily driven by smoking cessation, while the impact on initiation is relatively modest. Our analysis suggests that the Brazilian policy prevented roughly USD 53 million in costs in the capitals where it was highly enforced.

Keywords: Smoking bans, Addiction, Policy enforcement, Difference-in-differences, Development.

JEL Classification: D04, D12, I12, I18.

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*ZEW - Mannheim and Universidad Carlos III de Madrid; §University of Sao Paulo. Emails: Steffens (camila.steffens@zew.de) and Pereda (pereda@usp.br). Acknowledgments: We acknowledge the excellent feedback and comments from D. Mark Anderson, Carlos Roberto Azzoni, Stefan Boes, Alan Crawford, Johannes Diederich, Fernanda Estevan, Jesús Fernández-Huertas Moraga, Timo Goeschl, Warn Lekfuangfu, Matilde Machado, Tatiane Menezes, Luigi Minale, Daniel I. Rees, Rudi Rocha, Thomas Siedler, Mateus Souza, Jan Stuhler, and participants at the Heidelberg-Mannheim-ZEW Environmental Economics Brownbag Seminar, the Brazilian Econometric Society (SBE) Meeting, the REAP & SBE International Meeting, PIMES/UFPE academic seminar, the Applied Economics Reading Group at Universidad Carlos III de Madrid, and the Research Seminar in Public Economics at the University of Mannheim. All errors are our own. This project was funded by the Sao Paulo Research Foundation (FAPESP), under grants #2014/50848-9, #2016/18683-5 and #2017/11879-4, and by the CNPq, under grant #304221/2022-8. The opinions, hypotheses, and conclusions or recommendations expressed in this paper are the responsibility of the authors and do not necessarily reflect the views of FAPESP.

1 Introduction

Tobacco use is the leading cause of preventable deaths worldwide, being associated with several afflictions, including cancer, heart disease, and respiratory conditions. Smoking also exerts a significant burden on healthcare systems, and can adversely impact child development and the accumulation of human capital by diverting resources away from essentials, like food and education, an especially critical issue in low- and middle-income countries.[1] Within this context, public health mandates may play an important role in controlling smoking addiction and its associated adverse effects. Specifically, smoke-free environments might reduce exposure to passive smoking in public places, raise the marginal cost of smoking, and shape social norms and attitudes toward smoking by reducing its visibility.

The implementation of smoking bans in indoor public places is widespread, yet their effectiveness in reducing firsthand smoking remains a subject of debate (DeCicca, Kenkel, and Lovenheim, 2022). Studies in the context of Europe often find null or moderate effects on prevalence, but they typically rely on short-term variation in the roll-out of partial bans, which allow for smoke-specific areas/lounges. However, partial bans may not significantly raise the marginal cost of smoking, underscoring the need for evaluation of policies enforcing completely smoke-free places. The United States offers longer-term variation in the implementation of comprehensive bans across states, yet findings are conflicting.[2] One possible explanation is that several anti-smoking policies are determined at the state level, potentially leading to divergent smoking trajectories among states, which might confound the effects of smoking bans (Adda and Cornaglia, 2010).

Our paper contributes to this debate by offering novel evidence on longer-run effects of comprehensive smoke-free places on firsthand smoking in a large country, Brazil, which had almost 19 million regular smokers in 2013. We employ an identification strategy that leverages the staggered introduction of regulations across Brazilian states and their

[1]See, for example, WHO (2008), WHO (2019), and DeCicca, Kenkel, and Lovenheim (2022) for reviews on the health effects of smoking, and Goodchild, Nargis, and d'Espaignet (2018) for the costs imposed to health systems. Block and Webb (2009) provides evidence that expenditure on addictive goods might lead to child malnutrition among low-income families. Heckman (2007) and Hai and Heckman (2022) study the relationship between health/addiction and human capital accumulation.

[2]A recent review by DeCicca, Kenkel, and Lovenheim (2022) highlights the challenge of reconciling mixed findings in the literature and the consensus to evaluate comprehensive bans. By 2020, 70% of countries had introduced smoke-free environments, but only half had implemented comprehensive smoking bans (WHO, 2021).

capitals. Residents of fifteen out of twenty-seven state capitals were exposed to local bans from 2009 to 2013, before a national policy came into force in 2014.[3] These local bans were comprehensive, (i.e., mandated that all closed or partially closed public places were to become entirely smoke-free).[4]

Hence, understanding how these bans operate in Brazil, a country that is often lauded as a success story in tobacco control policies (WHO, 2019), can provide valuable insights into a debated issue. Moreover, we are among the first to delve into the effects of smoking bans in the context of a developing country. Despite bearing a disproportionate burden of the adverse effects of smoking addiction (Goodchild, Nargis, and d'Espaignet, 2018; WHO, 2021), anti-smoking policies of low- and middle-income countries are frequently overlooked in the literature. Brazil's geographical extension and regional diversity also allow us to investigate the role of policy enforcement levels. We compile data from local monitoring agencies regarding the extent to which penalties were applied in case of violation of the bans. Thus, we are able to study the bans' heterogeneous effects according to their level of enforcement, contributing to a broader discussion on policy design, implementation, and evaluation in environments where institutional capabilities are often weaker. Hallward-Driemeier and Pritchett (2015; p.123) summarize the challenges of regulations in such contexts:

> When strict *de jure* regulation and high rates of taxation meet weak governmental capabilities for implementation and enforcement, we argue that researchers and policymakers should stop thinking about regulations as creating "rules" to be followed, but rather as creating a space in which "deals" of various kinds are possible.

We also focus on young adults aged 15 to 29, as defined by the Brazilian Youth Statute, who may be considered the primary target of smoking bans, for two reasons. First, these policies may influence the likelihood of initiation – which typically happens at early ages – by reducing exposure to triggers such as peer influences during social

[3] We build on insights from the recent developments in the difference-in-differences literature (e.g., Sun and Abraham, 2021; Wooldridge, 2021). In our main analysis, we compare twelve capitals treated in 2009 to eleven capitals not treated until 2014. We also show that our results are robust to considering three additional capitals that adopted the policy in 2010 and 2011 in a setting with staggered treatment.

[4] Importantly, other measures such as monitoring, warning labels, advertising bans, cessation programs, and taxation were enacted at the national level, and we provide supporting evidence that they do not confound the identification of the causal effects of smoking bans (Section 2.2).

gatherings. Second, smoking bans can directly raise the marginal cost of smoking by limiting access to smoking areas and reducing social acceptance, thereby encouraging cessation. While studies focusing on adults mainly examined cessation patterns, the addictive nature of smoking suggests that cessation effects may take time to materialize, potentially explaining the mixed findings in the literature. However, during the early stages of addiction, the increased costs associated with smoking bans may accelerate the process of quitting.

We conduct a decomposition analysis which allows us to separately estimate the effects of the bans on smoking cessation and initiation. Our investigation of dynamic effects is possible thanks to the observation of individual-specific smoking trajectories, which we construct using microdata from the 2013 Brazilian Survey of Health (PNS). Our panel contains information on whether each individual was a regular smoker or not at any given point in time between 2005 and 2013, allowing us to estimate the bans' effects depending on years of exposure.[5]

We find that smoking bans led to a 15% reduction in smoking prevalence among young adults after four years of exposure. The impacts were not immediate and became stronger over time, consistent with a scenario in which short-run impacts may not be significant due to delays in enforcement and to the addictive nature of cigarette consumption and its consequent slow adjustment. Importantly, the bans were effective when rigorously enforced, with effects that are similar to (presumably) rigorously enforced bans in the US (Carton et al., 2016) and Europe (Pfeifer, Reutter, and Strohmaier, 2020). In contrast, we found no evidence that weakly enforced bans were effective.

Our findings reveal that the reduction in smoking prevalence is mainly attributed to cessation among young adults that have started smoking shortly before the bans were introduced (i.e., up to three years). Overall, cessation accounts for nearly 70% of the reductions in smoking prevalence. The remaining portion can be attributed to preventing smoking initiation. Although the effects on initiation exhibit a clear decreasing trend, they are less precisely estimated and relatively small in magnitude compared to the effects on cessation. One potential explanation is that individuals in our sample might be introduced to smoking in non-targeted environments, as initiation typically happens

[5]Retrospective data are widely used to study smoking behavior and provide an accurate measure of smoking status, especially among young individuals (Kenkel, Lillard, and Mathios, 2003; Christopoulou and Lillard, 2015). In the Online Appendix A.3, we validate our panel by comparing it to a prior survey conducted in 2008.

before the minimum legal age to purchase cigarettes and alcohol in Brazil (Figure 2). [6]

Related Literature. Our paper makes valuable contributions to the broad literature on the evaluation of tobacco control policies by showing the importance of behavior dynamics, enforcement levels, and adjustment time. Most of the European-based evidence relies on short-term variation. For example, Anger, Kvasnicka, and Siedler (2011), Jones et al. (2015) and Boes, Marti, and Maclean (2015) found no average effect on smoking prevalence within one year of bans implementation in the UK and Germany, and two years in Switzerland, respectively. In line with these findings, we show that the impacts in Brazil were not immediate and became stronger over time, providing novel insights into the medium-run effects of comprehensive smoking bans.

Using long-spanning data from the U.S., Adda and Cornaglia (2010) and Carton et al. (2016) find conflicting results for the effects of smoking bans on firsthand smoking. Adda and Cornaglia (2010) show that the effects on smoking prevalence of workplace and restaurant/bar bans do not persist when accounting for state-specific trends. Instead, their findings suggest that bans might have simply shifted smoking from public to private spaces, potentially causing harmful effects on children exposed to such environments. In contrast, Carton et al. (2016) found that comprehensive smoking bans (i.e., those applied to all restaurants, bars, and workplaces) are related to 3.3% reduction in smoking prevalence in the U.S. In line with our results, they found an 11% reduction in young adults' smoking prevalence due to restaurant and bar bans. To the best of our knowledge, there is only one other paper for a developing country, Catalano and Gilleskie (2021), which suggests that smoking bans might have reduced prevalence in Argentina.

However, the studies for the U.S. and Argentina rely on estimators that assume homogeneous effects over time and across cohorts, not accounting for the recent developments on the difference-in-differences literature (Goodman-Bacon, 2021). Our findings provide supporting evidence that the effects of the bans are not homogeneous in either dimension and highlight the need to properly study trends that might be driving the implementation of the bans. Besides applying a clear and robust estimation approach, our additional extensions are the decomposition of the effects and the heterogeneity by enforcement levels. We show that enforcement is a key factor for the bans' effectiveness in reshaping

[6] This finding aligns with the research by Meier, Odermatt, and Stutzer (2021), which illustrates how teenagers often circumvent sales bans. In contrast, Pfeifer, Reutter, and Strohmaier (2020) found that smoking bans targeting schools are effective in preventing smoking initiation.

smoking behavior in the context of developing countries. Further, their data, consisting of independent cross-sections, did not allow for an examination of how bans relate to different mechanisms of cessation and initiation.

To the best of our knowledge, we are among the first to formally decompose changes in smoking prevalence into initiation and cessation, shedding light on the mechanisms through which the bans influence smoking behavior among young adults.[7] Even though non-negligible, we find that the effects on initiation are modest, possibly because this happens in places not targeted by the bans, given that around 70% of individuals start smoking before the minimum legal age (18 years old). This finding aligns with the research by Meier, Odermatt, and Stutzer (2021), which illustrates how teenagers often circumvent sales bans. In contrast, Pfeifer, Reutter, and Strohmaier (2020) found that smoking bans targeting schools are effective in preventing smoking initiation in Germany, with effects ranging from 14% to 22%.

Motivated by the Theory of Rational Addiction (Becker and Murphy, 1988), we also tested how the effects relate to the level of addiction, measured by the number of years that a given individual has been smoking regularly. Within this framework, whether smoking bans might affect cessation depends on the trade-off between the two channels. On the one hand, current consumption depends on past consumption, which accumulates in the form of an "addictive stock" and *reinforces* addiction. On the other hand, depending on the extent to which individuals discount the future, this stock may also raise the marginal cost of consumption due to expected detrimental health effects. Because young individuals are more present-orientated, they might be more susceptible to addiction through the first channel. This suggests that the bans might be more effective in promoting cessation before addiction is settled (i.e., before the "addictive stock" is too large). Consistently with this prediction, we find that the effects on cessation are driven by individuals with low levels of addiction, i.e., who began smoking no more than three years before the bans were implemented. Conversely, we did not observe any significant effects on cessation among individuals with higher levels of addiction.

Our study also complements recent research by Da Mata and Drugowick (2023), who

[7] This entails running regressions with "asymmetrical outcomes" both before and after treatment. We propose a procedure for estimating the effects of the bans on asymmetrical outcomes, while simultaneously controlling for pre-existing trends that could otherwise introduce bias. Similar procedures may be considered in other applied economics settings with asymmetrical outcomes, such as inflows into employment and outflows to unemployment. See section 5.1.1 and the Online Appendix C for more details.

found that Brazilian smoking bans improved birth outcomes of pregnant women working in occupations strongly affected by the bans. While Da Mata and Drugowick (2023) provide convincing evidence of reduced passive smoking in targeted environments, our findings suggest that the bans were also effective in reducing firsthand smoking, which alleviates concerns about the potential displacement of smoking from public to private places.[8]

In the following section, we first present background information about the smoking bans in Brazil. Subsequently, we discuss additional tobacco control policies and potential threats to identification. We provide supporting evidence that these national-level policies do not confound our estimates of the effects of smoking bans. Our data is comprehensively described in Section 3. Section 4 introduces our empirical strategy and identifying assumptions. The results are presented and discussed in Section 5, and we conclude in Section 6.

2 Smoking Bans in Brazil

Brazil has been implementing policies to reduce cigarette consumption since the 1980s, under the establishment of the National Tobacco Control Program (INCA, 2023a). Smoke-free zones were introduced by a national law in 1996. However, besides allowing smoking areas/lounges, this law and its accompanying regulation did not contain enforcement rules. According to INCA (2010), the 1996 regulation did not follow recommendations from the World Health Organization (WHO) and was not properly adopted by the entertainment and hospitality industries.[9]

As a consequence, in practice, smoke-free areas were not enforced in Brazil until 2009, when some states and municipalities introduced comprehensive local smoking bans. Such policies differed from the national regulation in two dimensions. First, they completely prohibited smoking in every closed or partially open public place, eliminating smoking zones. Second, the local policies established monitoring agencies and penalties for viola-

[8]Smoking bans may also be justified because of the negative externality on passive smokers (DeCicca, Kenkel, and Lovenheim, 2022). However, results from Adda and Cornaglia (2010) suggest that the net effects on secondhand smokers are unclear, depending on the direct effects on cigarette consumption and the degree of substitution between banned and not-banned places.

[9]Smoking areas were supposed to have proper ventilation and to be adequately insulated from non-smoking common areas. Nevertheless, evidence suggests that establishments were not effectively adhering to this legislation (INCA, 2010).

tion of smoking bans, thus institutionalizing its enforcement.[10]

Following the trend of the local policies, a national smoking ban was approved in 2011, replacing the 1996 legislation for the whole country. However, this ban became effective only in December 2014, after its regulation by the federal executive branch. In this paper, we leverage the staggered exposure to comprehensive smoking bans across Brazilian localities between 2009 and 2014 to assess the impacts of this policy on smoking behavior. We focus on bans implemented and their respective effects from 2009 to 2013, as there are no untreated units for comparison from 2014 onward.

State capitals will be the unit of treatment. We consider a state capital to be "treated" if it has adopted a local smoking ban or if it is covered by a state-wide ban. We do not analyze other municipalities because those are not individually identified in our data, which is representative at the capital level.[11] Further, even if a state and its capital did not implement a smoking ban, we cannot exclude the possibility that other cities within the state were treated by local bans.[12] Focusing on state capitals makes the analyses more tractable since their institutional setting is often better understood and observable. Furthermore, most of the urban population and most establishments targeted by the bans are in state capitals, as those concentrate a significant fraction of formal jobs, public services, and cultural and leisure amenities in Brazil.

2.1 Enforcement

Besides sharing a common scope, the comprehensive local smoking bans also have similar enforcement rules. Overall, they are monitored by municipal health agencies subsidiaries of the Brazilian Health Regulatory Agency (ANVISA) and the state Consumer Protection Agency (PROCON). In the case of state-level bans, the state and municipal agencies should act together. Anyone can anonymously report violations to the monitoring agency or the police. After the report, the monitoring agency inspects the establishment and can impose penalties in case of non-compliance. The penalties fall on whoever is responsible

[10] We collected information on restrictions, effective dates, and enforcement rules directly from the laws. A list of local smoking bans was first obtained from the non-governmental organization Tobacco Control Alliance (ACTBr, 2016). We also checked for any changes in these laws and for any unlisted laws through an internet search. Our findings are consistent with those from Furtado and da Silva Filho (2016).

[11] The stratified sample only distinguishes the capital city from other cities within the state. No other cities can be identified.

[12] We cannot observe whether or when local smoking bans were implemented in all 5,570 Brazilian municipalities, as this information is not centralized.

for the establishment where the violation occurred (e.g., the restaurant owner), and can vary from warnings to fines and, in the extreme case of recidivism, closure of the establishment.

However, enforcement depends on the allocation of resources to surveillance, on citizens' awareness and understanding of the law, and on political will to penalize transgressors. Naturally, the existence of a law does not guarantee its effective application, especially in developing countries, which tend to have weaker institutions (Acemoglu, Johnson, and Robinson, 2002). To assess the effective enforcement of the local smoking bans in Brazil, we use data from monitoring agencies across the country, such as the number of violations and the application of penalties. Based on this information, we created a two-level index of enforcement, in which the higher level (level two) refers to smoking bans with high dissemination, surveillance, notifications, and application of fines. The lower level refers to smoking bans with some dissemination and news regarding surveillance but no penalties (level one).[13]

Figure 1 presents the distribution of the local smoking bans across the Brazilian territory, including information about their year of implementation and level of enforcement. The markers show the location of each state capital and represent whether the local ban was introduced by the state, the capital, or both. The assignment of treatment status depends on the dates on which the local smoking ban became effective. For capitals with both state and capital-level bans, treatment assignment was based on the earliest effective date.[14] Once a ban is adopted, it remains effective for the period of study. We note that most of the bans were adopted in 2009 (twelve out of fifteen), and half of them were highly enforced. Ten state capitals and the Federal District were not treated before 2014.[15] Detailed information is provided in the Online Appendix A.

[13] We obtained enforcement data from the agencies in the states of São Paulo (SP) and Rondônia (RO), and in the capitals Curitiba/PR, Campo Grande/MS, Goiânia/GO, Maceió/AL, Salvador/BA, João Pessoa/PB, Aracaju/SE, and Teresina/PI. We complemented with information from newspapers for the remaining localities.

[14] For example, for ten capitals with bans effective at the end of 2009, we assigned 2010 as the first year after treatment, and 2009 as the baseline year. Two additional capitals that implemented smoking bans between February and March of 2010 are also considered in our main analysis with 2009 as the baseline. However, our results are robust to including them in the 2010 cohort in a staggered treatment setting, along with another late-treated capital (Online Appendix Table B.3), and to drop them from the earlier-treated group (Table B.11).

[15] One state, Rondônia (RO), approved a smoking ban in 2008, but never implemented it. The monitoring agency confirmed that the smoking ban was not effective until 2014 when the national ban came into force. For this reason, Rondônia is dropped from our main analysis, but our results are robust to the inclusion of it in the comparison group.

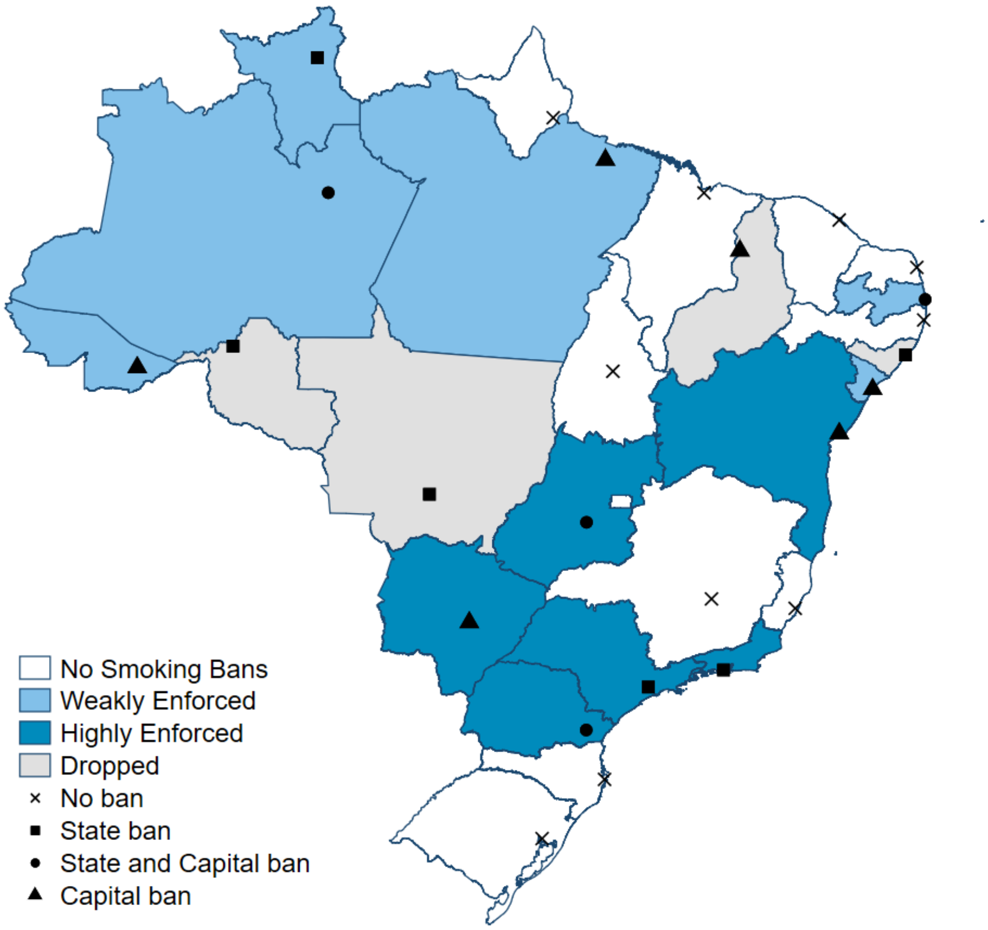


Figure 1: Local Smoking Bans by Year, Unit of Implementation, and Enforcement Level

Notes: The colors represent the effective year and the level of enforcement of local smoking bans. For ease of visualization, the entire state is filled, even if the law was adopted at the capital level. The markers show the geographical location of the state capitals, and whether the smoking ban was adopted by the state, by the capital, or both. "Highly Enforced" bans were strongly disseminated and applied penalties. "Weakly Enforced" bans had some surveillance but no penalties. Rondônia was dropped from our main analysis since the ban approved in 2008 was never implemented ("Not Enforced"). Data on local smoking bans come from ACTBr (2016), municipal and state legislation documents, and internet searches on public records and news. Enforcement levels were obtained from the monitoring agencies. Detailed information is provided in the Online Appendix Table A.2.

2.2 Other Tobacco Control Policies

Brazil is currently one of two countries in the world to have adopted all the tobacco control measures proposed by the World Health Organization at the highest level of achievement (WHO, 2021). Besides smoke-free environments, they include (i) monitoring,[16] (ii) warning (labels and mass media campaigns), (iii) advertising bans, (iv) cessation programs,

[16] Monitoring of smoking behavior was first introduced as a complement of the 2008 Brazilian Household Survey (PNAD), but fully implemented by the 2013 Brazilian Survey of Health (PNS) used in this paper.

and (v) taxation. In what follows, we provide supporting evidence that these policies are not a threat to the identification of the effects of the local smoking bans.

Warning labels and advertising restrictions were also introduced by the 1996's national legislation. Comprehensive policies were adopted in 2011, when cigarette advertisement was totally banned in Brazil and warning labels were set to partially cover the front side of cigarette packages, in addition to the back (INCA, 2023a). Since these are national-level policies and we are not aware of local mass-media campaigns, warnings and advertising should not affect the municipalities differently. Further, in Table 1, we show that individuals residing in capitals treated by a local smoking ban were not more exposed to advertising or warnings in 2013, compared to those in the capitals without smoking bans.[17]

Cessation programs have been available since 2004 under the scope of the Ministry of Health, and the execution is assigned to the municipalities (INCA, 2023b). We do not have access to the number of individuals who received treatment at each locality. However, around 11% of former smokers (4% among young adults) in our data reported to have attended a program to quit smoking (Table 1). Importantly, there is supporting evidence that the participation in cessation programs was not larger in the treated capitals.[18]

Finally, cigarette taxes and prices are also regulated at the national level. A minimum price has been in force in Brazil since 2012, when it was fixed at R$ 3.00, increasing R$ 0.50 each year to its current price of R$ 5.00 (1 USD) (Receita Federal, 2016; INCA, 2023a). Comparing log prices reported by individuals in our survey, we find no evidence that prices were larger in the treated capitals (Table 1).[19] On the contrary, although imprecise, the price difference is negative among young adults. In the Online Appendix Figure B.1, we further show that the distribution of prices is slightly more concentrated in lower prices in treated capitals compared to capitals without smoking bans.

Iglesias et al. (2017) argues that cigarette smuggling has increased in Brazil over the

[17] Based on self-reported exposure in the 2013 PNS.

[18] The coefficient is negative, although only statistically significant at 10% among young adults. Thus, if something, smaller participation in cessation programs would underestimate the effects of the smoking bans. Cessation programs are available in public hospitals and health units in all Brazilian capitals, but less than 8% of young adults who attended a cessation program reported to have done so in the public system.

[19] The 2013 survey asked individuals the price paid for a pack of cigarette in their last purchase. In the Online Appendix Figure B.2, we also show that the evolution of the cigarette price index from 2006 to 2013 was very similar between capitals treated and not treated by a smoking ban as of 2013.

last decades as a response to minimum prices and taxes rise. Measuring smuggling in the same fashion (price paid for a cigarette pack below the 2013 minimum price), we estimate that around 17% of smokers in the Brazilian capitals were likely purchasing illicit cigarettes in 2013.[20] Although imprecise, there is evidence that a larger share of young adults was involved in such behavior in the treated capitals (Table 1).

Since any reported differences in the prevalence of warnings, cessation programs, prices, and smuggling would be in contrast to the adoption of smoking bans, they should not be a concern for the assessment of the effects of local smoking bans in Brazil. If something, these differences would lead to an underestimation of the bans' impacts.[21] We expand on this when discussing our results in Section 5.

Table 1: Prevalence of Other Tobacco Control Policies in 2013

	Adults			Young Adults		
2013 Survey	Untreated	Diff. Treated	N	Untreated	Diff. Treated	N
Advertising	0.308	0.003	23,115	0.315	0.008	8,811
	(0.019)	(0.029)		(0.018)	(0.034)	
Warnings						
News Papers	0.341	-0.058	23,115	0.330	-0.063	8,811
	(0.035)	(0.039)		(0.037)	(0.039)	
TV	0.444	-0.026	23,115	0.429	-0.028	8,811
	(0.017)	(0.026)		(0.018)	(0.023)	
Radio	0.192	-0.014	23,115	0.166	-0.019	8,811
	(0.022)	(0.027)		(0.020)	(0.029)	
Package	0.558	0.003	23,115	0.571	-0.012	8,811
	(0.011)	(0.031)		(0.015)	(0.037)	
Cessation Programs	0.108	-0.025	1,641	0.040	-0.029*	610
	(0.025)	(0.028)		(0.015)	(0.015)	
Log Price	1.554	-0.003	2,765	1.627	-0.046	895
	(0.022)	(0.037)		(0.025)	(0.048)	

Notes: The "*Average*" is the proportion of individuals who reported, at the 2013 PNS, exposure to advertising, warnings, and participation in a cessation program (or log prices) in the capitals that were not treated by a local smoking ban. Prices are for a pack of cigarettes in the last purchase. The columns "*Difference*" compute the differences in the exposure to these other policies among the capitals treated by a local smoking ban. "*N*" is the number of individuals who answered the related question in the 2013 survey. Adults comprehend all individuals from 18 to 64 years old at the moment of the survey. Young adults are individuals from 15 to 29 years old between 2009 to 2013 (those considered in our analysis). Cluster Robust Standard Errors (at capital level) in parentheses.

[20] Our estimate for the Brazilian average (33%) is similar to the one obtained by Iglesias et al. (2017) (31.1%).

[21] These results are robust to comparing only the 12 capitals treated in 2009 to the untreated capitals (Online Appendix Table B.1). In fact, individuals reported to be less exposed to warnings and to attend cessation programs less frequently in the capitals treated with highly enforced bans.

3 Data

One of the challenges of studying smoking policies in Brazil is the absence of individual-level panel data on cigarette consumption. Without tracking individuals over time, for example, it is often not feasible to analyze initiation or cessation decisions. To overcome this limitation, we have created a panel with yearly individual smoking status using retrospective information from the 2013 National Survey of Health (PNS) (IBGE, 2014). This survey contains smoking information from a representative sample of the population of each Brazilian state and their respective capitals.

PNS identifies if each individual was a regular smoker (smoking on a daily basis) at the moment of the survey, or if they had been regular smokers previously in life. In both cases, it contains the age at which the individual started smoking regularly. In case they had quit smoking, the survey asks for how long. In addition, the data identify current and former casual smokers (who never smoked daily), with information on quitting time for the latter. However, the survey does not provide the smoking initiation age for casual smokers. For this reason, we focus on regular smokers in this paper.

Using PNS retrospective information, we created an individual-level panel with smoking status for the period of 2005 up to 2013.[22] In the Online Appendix A.3, we compare the variables obtained for 2008 through our approach (i.e., a "lagged" PNS) to those from the 2008 Tobacco Special Research (IBGE, 2009). Overall, the results indicate that our lagged PNS matches the 2008 real-time survey well, thus suggesting that the panel we create is reliable for the objectives proposed in this paper.

As explained in section 2, our sample is restricted to individuals living in Brazilian state capitals. This sample constitutes around 45% of the individuals in PNS and represents 25% of the Brazilian population (more than 36 million adults in 2013). Given the focus of this paper, we have also restricted our analysis to young adults aged 15 to 29 in each year of the panel. These are the age cutoffs that define "young adults" according

[22]A comprehensive explanation is available in the Online Appendix A. Figure A.1 illustrates the retrospective structure of the survey. A similar approach was also used, for instance, by Boes, Marti, and Maclean (2015) and Christopoulou and Lillard (2015). The main limitation is that we do not observe time-varying individuals' covariates. Nevertheless, we argue that individual-specific effects capture the main sources of heterogeneity in this context.

to the Brazilian Youth Statute.[23] Our final sample constitutes an unbalanced panel data with 11,308 individuals born from 1975 to 1995 and a total of 72,005 observations over the period 2005-2013.[24] Our main findings are robust to considering instead a balanced panel of individuals born from 1975 to 1995 (Online Appendix Table B.12).

3.1 Smoking Behavior in Brazil

Brazil is a continental country, with the seventh largest population in the world and, consequently, a substantial number of smokers: more than 18.6 million regular smokers in 2013, of which almost 3.4 million were young adults. In addition, Brazilian overall smoking prevalence (14.7% in 2013) is similar to that of many developing countries, such as Mexico and other countries in Africa (WHO, 2016; WHO, 2019).

Due to the singular achievement of Brazilian tobacco control policies, the country is considered a success case by the WHO. As of 2020, it was among the only two countries in the world to have fully implemented all the recommended measures (WHO, 2021). In addition, smoking prevalence has been following an expressive downward trend in the last decades in Brazil. For instance, almost 16% of adults in the Brazilian capital cities were regular smokers in 2005. This share decreased by 4.5 percentage points, reaching 11.3% by 2013. The trend is similar among young adults: from 12.5% regularly smoking in 2005 to 8.9% in 2013.

Our sample represents over 1.3 million young adults who were regularly smoking in Brazilian capitals in 2009. While more than 20% have quit smoking by 2013, other 2% of young adults started smoking from 2009 to 2013. Figure 2 highlights an important fact about smoking behavior: initiation happens essentially in the early years. Based on the sample of adults that were current or former regular smokers in 2013 (Panel a), we observe that less than 30% of individuals took up smoking before reaching 15 years old, and 95% started by their 29^{th} year. Hence, approximately 65% of smoking initiation happened within the age range considered in this paper (i.e., 15 to 29 years old). When

[23] National Law n. 1285/2013, which establishes the rights of young people, as well as the principles and guidelines for public policies targeting young adults. Even though the PNS module for smoking behavior was only applied to adults (18 years or older), our retrospective data procedure allows us to assess behavior at earlier ages.

[24] 33.7% of the individuals are observed in all the nine years of the panel; more than 50% are observed for seven periods or more; and around 25% are observed up to 4 periods, with only 5% in one period (those with 29 years old in 2005).

focusing on young adults (Panel b), we further notice that 71% began smoking on a daily basis before the age of 18 (vertical dashed line), which is the minimum age to legally consume cigarettes and alcohol in Brazil.

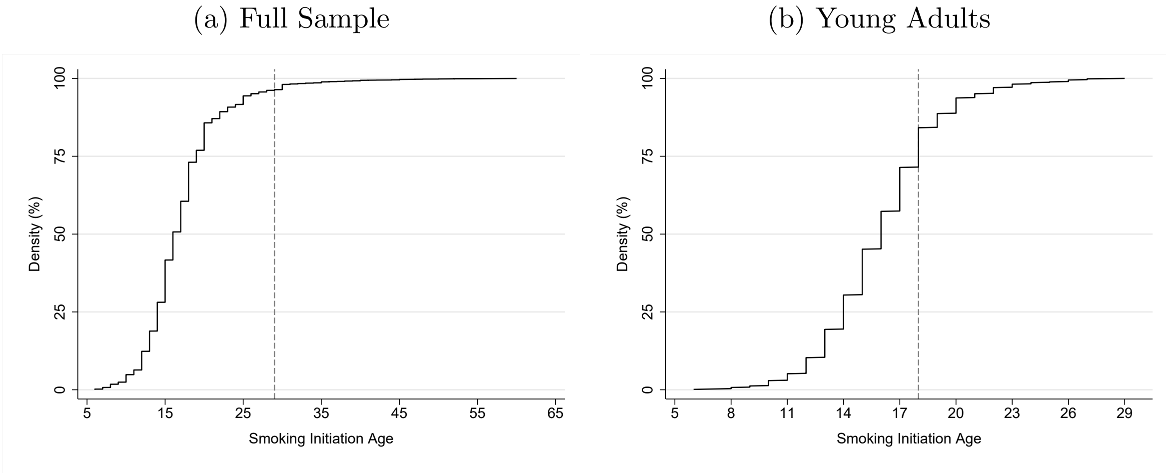


Figure 2: Cumulative Distribution of Smoking Initiation Age

Notes: Smoking initiation age for current and former regular smokers in 2013. Panel (a) is constructed using the sample of adults (18 to 64 years old) residing in Brazilian capitals. Panel (b) is restricted to the sample of young individuals (up to 29 years old) in Brazilian Capitals. The vertical lines highlight that 95% of individuals take up smoking by their 29^{th} year (Panel a), and more than 70% of young adults started smoking daily before being legally allowed (i.e., at the age of 18).

This pattern motivates our definition of the addictive stock as the number of years that an individual has been smoking daily. At early ages, since the addictive stock is relatively small, we should expect cessation to increase as a response to the costs imposed by the smoking bans. In contrast, once addiction is settled, the present orientation of young adults might hinder cessation, highlighting the role of initiation as a mechanism for preventing addiction. In the following sections, we analyze to what extent smoking bans are effective along those dimensions.

4 Empirical Strategy

We estimate the impacts of local smoking bans implemented in Brazil from 2009 to 2013 on young adults residing in the treated capitals. Figure 1 presents the treatment cohorts and the location of state capitals in Brazil. Although our focus on state capitals is due to data availability, the large distance between them attenuates spillover concerns.

Furthermore, since migration is costly in Brazil (Oliveira and Pereda, 2020), it is unlikely that either people or companies would migrate between state capitals as a response to local smoking bans.[25]

In the context of this paper, we allow the impacts of smoking bans to vary across cohorts and over exposure time. This follows from the fact that smoking bans might take some time to be fully assimilated due to adaptation to legislation and the consequent social acceptance and control. Another source of potential heterogeneity is the level of enforcement of the bans, discussed in section 4.2.1.

In our first analysis, we consider all the fifteen treated capitals using a staggered difference-in-differences approach. The comparison group consists of eleven capitals untreated until the national smoking ban came into force in 2014, thus "never-treated" for the purposes of this paper.[26] We provide supporting evidence that the parallel trends assumption holds for the early-treated cohort (i.e., twelve capitals), but smoking followed different trends in the three late-treated capitals. Therefore, in our main analysis, we compare the twelve "early-treated" capitals with the "never-treated" group using a generalized difference-in-differences approach.

4.1 Staggered Difference-in-Differences

Define l as the time relative to the baseline year; e as the cohort related to each baseline year ($e = 2009, 2010, 2011$); and $SB_{i,e}$ as a dummy variable that equals 1 if individual i was residing in the capital in treatment cohort e at the moment of the survey, and 0 otherwise. Following Sun and Abraham (2021), we employ an event-study specification that accounts for heterogeneous effects across cohorts and over time. Specifically, the specification is an extended linear two-way fixed effects interacting period and cohort

[25]Based on a random sample of 3,456,597 individuals (615,544 young adults) from 2010 Census (IBGE, 2010), we observe that migration is more common within rather than between states. While 38% of young adults were living in a different municipality than where they were born, only 15% migrated between states, and even less, 7.8%, between treated and non-treated states.

[26]Recall that smoking bans were approved in Porto Velho/RO in 2008, but were never regulated nor enforced. For this reason, we drop Porto Velho from our main specifications, but our results are robust to its inclusion in the never-treated cohort (Online Appendix Table B.11).

indicators, as follows:[27]

$$y_{i,t} = \mu_i + d_t + \sum_{e \in \{2009,2010,2011\}} \sum_{l \neq 0} \beta_{e,l}(\mathbb{1}\{l = t - e\} \cdot SB_{i,e}) + \epsilon_{i,t} \ , \tag{1}$$

where μ_i are individual fixed effects, d_t are year fixed effects, and $\epsilon_{i,t}$ is an individual random error term. The dependent variable $y_{i,t}$ is a binary variable equal to 1 if individual i is a regular smoker in year t, and equal to zero otherwise.

Within this framework, $\hat{\beta}_{e,l}$ is a consistent estimator for the average treatment effect of smoking bans on the smoking prevalence of the treated cohort e after l years exposed to the bans, under two assumptions: (i) parallel trends, and (ii) no anticipatory effects. The first assumption is that the treatment and the never-treated groups would have followed, in the absence of treatment, the same trend in outcomes. The second assumption is that treatment effects are zero before implementing the bans (i.e., there is no change in behavior in anticipation of treatment).

In the Online Appendix B.1, we provide evidence supporting these assumptions in the early-treated cohort (i.e., twelve capitals treated from 2009 onward). First, using state-level data from a 2008 survey, we show that smoking prevalence was similar between states with treated and "never-treated" capitals before the implementation of local smoking bans. On average, 10.8% of young adults were smoking in 2008, with a (non-statistically significant) smaller prevalence by 1.2 percentage points in the treated group.

As the identifying assumption is based on parallel trends, we also tested the joint significance of pre-treatment coefficients on smoking prevalence from Equation (1) using an F-test (Wooldridge, 2021). We cannot reject the null hypothesis that the pre-treatment parameters are jointly zero for the 2009 cohort (Table B.3). However, the null hypothesis is rejected for the 2010 cohort and the pre-treatment coefficients are large in the 2011 cohort, even after controlling for cohort-specific linear trends. We interpret this as evidence of potential violations of the parallel trends assumption for the late-treated capitals. For this reason, for the remainder of this paper, we drop the 2010 and 2011 cohorts from our main specifications. Our results are robust to considering these late adopters under a staggered treatment design (Online Appendix Table B.4).

[27] In settings without additional covariates, this specification is equivalent to the proposal from Callaway and Sant'Anna (2021).

4.2 Generalized Difference-in-Differences

The main specification in this paper is based on a Generalized Difference-in-Differences design, which compares the 2009 cohort with the never-treated capitals from 2005 to 2013, as follows:

$$y_{i,t} = \mu_i + d_t + \sum_{s \neq 2009} \beta_s(\mathbb{1}\{s = t\} \cdot SB_i) + \alpha(SB_i \cdot Trend) + \epsilon_{i,t} \ , \quad (2)$$

where SB_i is a dummy variable that equals 1 if individual i is exposed to a smoking ban from 2009 onward, and zero if individual i was not exposed to a local smoking ban during the period of study. Even though we are not able to reject the assumption that pre-treatment parameters are jointly or individually null, we are aware that this might be due to the low power associated with pre-trends testing (Roth, 2022). We address potential differences in pre-trends by controlling for treated-cohort specific linear trend, which is captured by the interaction of the treatment dummy (SB_i) with $Trend$ (Wooldridge, 2021). Our results support the linear specification of pre-trends, and we discuss its potential drivers in the next Section. We show that differential trends are driven by increasing initiation trajectories among early adopters, and our results are consistent when removing them from the sample. Our estimates are also consistent to controlling for age square as a time-varying covariate.

Equation (2) assesses the effects of smoking bans on smoking prevalence among young adults residing in the capitals treated by a local smoking. β_s captures post-treatment effects when s = 2010 to 2013, and pre-treatment deviations from linear trends when s = 2005 to 2008. For initiation and cessation, we use the same equation and outcome but apply additional sample restrictions. For example, for initiation, we restrict the sample to non-smokers at baseline (i.e., 2009). Conversely, for cessation, we restrict the sample to regular smokers in 2009.

Our regressions are weighted using PNS sampling weights to obtain an estimation of the average treatment effect on the exposed population of interest.[28] We allow the errors to be correlated among individuals who reside in the same capital by estimating cluster-robust standard errors. Due to concerns about the small number of clusters, we also

[28] Sampling weights are required to obtain aggregated statistics of smoking prevalence that are consistent for the Brazilian sub-population of analysis. Without sampling weights, smoking prevalence among young adults would be underestimated by almost 1 percentage point.

obtain p-values from Wild-Cluster Bootstrap (Cameron and Miller, 2015; MacKinnon and Webb, 2017; Roodman et al., 2019).[29]

4.2.1 Heterogeneity by Enforcement Level

We also study the extent to which the effects of smoking bans depend on their level of enforcement. Among the 2009 cohort, six capitals are treated by strongly enforced bans, while six are treated with low enforcement, as shown in Figure 1. We interact the 2009 treated indicator with indicator variables for each enforcement level (i.e., low and high), as follows:

$$y_{i,t} = \mu_i + d_t + \sum_{s \neq 2009} \sum_{v \in \{low, high\}} \beta_{s,v} \cdot \mathbb{1}\{s = t\} \cdot SB_{i,v} + \sum_{v} \delta_v \cdot SB_{i,v} \cdot Trend + \epsilon_{i,t} \quad (3)$$

where $SB_{i,v}$ is equal 1 if individual i was residing in a capital treated with a local smoking ban enforced with level $v = \{low, high\}$, and zero otherwise. Note that we interact the linear trend variable (*Trend*) with indicators for being weakly or strongly treated by a smoking ban. Therefore, we allow the treatment groups with different enforcement levels to follow specific linear trends.

5 Results

We first estimate the effects of smoking bans on smoking prevalence among young adults in Brazilian capitals from 2005 to 2013. The results shown in Figure 3a indicate that the local smoking bans are associated with a break in their smoking trajectory, manifesting after three years of exposure to the bans. Our lower-bound estimates reveal a 0.6 percentage point reduction in smoking prevalence by 2012 and a 0.8 p.p. reduction by 2013. The latter represents a 6.8% decrease in the average smoking prevalence among young adults in the treated cohort (11.8% in 2009). When accounting for the increasing trend in smoking prevalence, the effects become more pronounced, reaching up to 1.8 percentage points by 2013. This suggests that smoking bans may have reduced smoking prevalence by up to 15% among young adults in the treated cohort after four years of

[29] We implement wild-cluster bootstrap using the `boottest` command built for Stata by Roodman et al. (2019). We thank the authors for this important contribution to the research community.

exposure. The fact that the impacts were not immediate is consistent with a scenario in which short-run impacts may not be significant due to delays in enforcement and due to the addictive nature of cigarette consumption and its consequent slow adjustment. The effects in 2012 and 2013 are also statistically significant according to confidence intervals constructed using wild-cluster bootstrap. These effects are also mostly unchanged after controlling for the square of age (Appendix Table B.5).

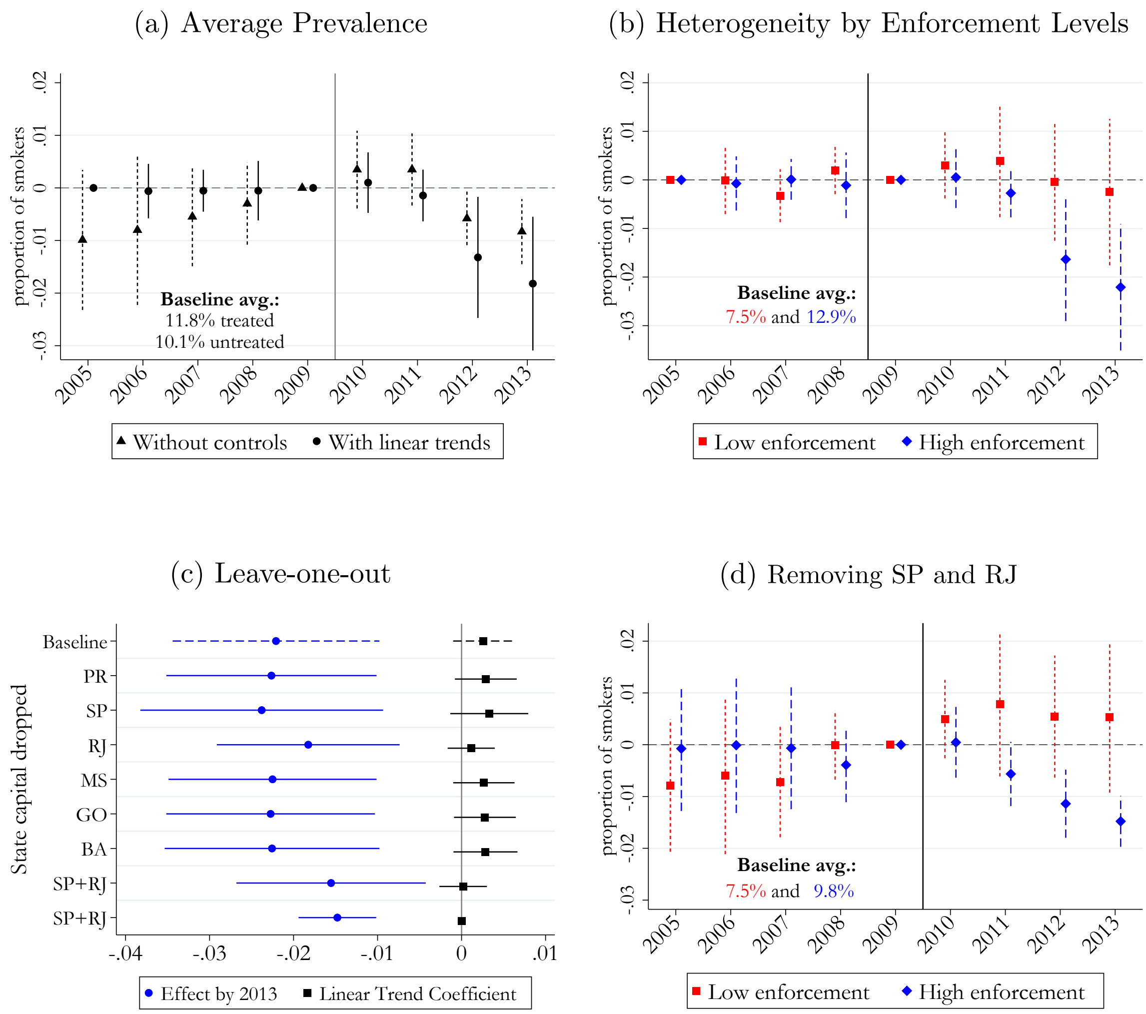


Figure 3: Impacts of Smoking Bans on Smoking Prevalence

Notes: The number of observations is 66,155, with 10,380 individuals. All regressions include year and individual fixed effects and are performed using the PNS sampling weights, and 95% confidence intervals using cluster-robust standard errors at capital. **Panel (a)**: estimates from Equation 2. P-values from wild-cluster bootstrap are available in Online Appendix Table B.5. **Panel (b)**: estimates from Equation 3. Results without linear trends and p-values from wild-cluster bootstrap are available in the Online Appendix Table B.6. In **panel (c)**, only the 2013 coefficients for highly enforced bans are reported. The remaining coefficients, standard errors, and p-values obtained from Wild-Cluster Bootstrap are available in the Online Appendix Table B.7. **Panel (d)** shows estimates without controlling for linear trends, when removing São Paulo (SP) and Rio de Janeiro (RJ).

Importantly, the average effects mask significant heterogeneity regarding the effective implementation of local smoking bans. Figure 3b suggests that the prior results were entirely driven by strongly enforced bans, which lead to a 17% reduction in smoking prevalence after four years of exposure. Remarkably, we find that the effects of weakly enforced smoking bans were virtually zero. These results highlight the importance of considering enforcement levels in policy evaluations in developing countries, where institutional capabilities are often weaker. The experience of Brazilian smoking bans demonstrates that public policies can be effective when properly implemented in such settings. The enforcement mechanisms from the successful capitals may provide valuable insights for other developing countries when implementing similar regulatory policies. Additionally, the success of the Brazilian smoking bans may be informative even for developed countries where such bans are narrower in scope (e.g., some countries in Europe). Our paper helps to disentangle the mechanisms through which comprehensive bans might have effectively transferred property rights over "clean air" from smokers to non-smokers in Brazil, recently even leading to discussions around smoking bans in open common areas (such as parks as public squares).[30]

A possible concern with the prior heterogeneity analysis is that strongly enforced bans are concentrated in capitals with larger smoking prevalence and higher population density. To address this, we implement a leave-one-out analysis, examining whether our results were driven by any particular capital. We re-estimate the heterogeneity specifications on several sub-samples, sequentially removing one capital with a strongly enforced smoking ban. The effects shown in Figure 3c remain consistent with our main findings.[31] We still find that strongly enforced smoking bans reduced smoking prevalence by 15%, even after simultaneously removing both São Paulo (SP) and Rio de Janeiro (RJ), the main cities in Brazil and the leaders in adopting comprehensive smoking bans.

Although the baseline pre-treatment coefficients are not statistically different from zero, the trajectory of smoking prevalence from 2005 to 2011 suggests an increasing trend in the treated cohort. This motivates the inclusion of treated-cohort-specific linear trends

[30] Da Mata and Drugowick (2023) show that the bans reduced passive smoking in targeted environments. Further, the municipality of São Paulo banned smoking from public parks since 2019. Another example is the state of Santa Catarina, which banned smoking from parks and playgrounds in April 2024.

[31] Coefficients for all periods, as well as standard errors and p-values robust to wild-cluster bootstrap, are available in Online Appendix Table B.7.

in our main specifications.[32] In Figure 3c, the coefficient of the linear trend becomes practically null after removing SP and RJ, suggesting that it was driven by these leading regions.

While smoking bans may have been implemented in response to differential smoking trajectories in SP and RJ, Figure 3d offers supporting evidence that adoption in the remaining regions aligns with the parallel trends assumption. After removing SP and RJ, the unconditional pre-treatment coefficients become nearly zero, especially for strongly enforced bans, and there is a decreasing trajectory in smoking prevalence after the treatment, leading to a 12% reduction by 2012 and a 15% reduction by 2013. This finding supports the causal interpretation of our estimates, demonstrating that they are not entirely driven by the main cities, nor dependent on a linear extrapolation of pre-trends.

In what follows, we decompose the effects on smoking prevalence between effects on initiation and cessation and study the response to the bans across these margins of adjustment.

5.1 Decomposition of the Effects on Smoking Prevalence

The change in smoking prevalence from 2009 to year t can be expressed by the difference in the number of smokers normalized by the population (N):

$$\Delta prevalence_t \equiv \frac{\sum_i \mathbb{I}[i \text{ smokes in } t]}{N} - \frac{\sum_i \mathbb{I}[i \text{ smokes in 2009}]}{N} \tag{4}$$

Since each individual that smoked in year t was either smoking or not in 2009, we can rewrite the first numerator as follows:

$$\sum_i \mathbb{I}[i \text{ smokes in } t] = \sum_i \mathbb{I}[i \text{ smokes in } t] \times (\mathbb{I}[i \text{ smokes in 2009}] + \mathbb{I}[i \text{ doesn't smoke in 2009}])$$

Likewise, the number of smokers in 2009 can be expressed in terms of individuals

[32] The linear trend coefficients are estimated as the differences in prevalence growth from 2005 to 2009. There is supporting evidence that the differential pre-trends are fully captured by linear trends. After controlling for it, the remaining pre-treatment coefficients become null, suggesting no deviations from the linear specification (Appendix Tables B.5 and B.6). In addition, when fitting the linear trend using all the pre-treatment periods (i.e., equalizing all coefficients to zero), the estimates for the linear trend and the post-treatment coefficients are unchanged.

continuously smoking in t and those that had quit by t:

$$\sum_i \mathbb{I}[i \text{ smokes in 2009}] = \sum_i \mathbb{I}[i \text{ smokes in 2009}] \times (\mathbb{I}[i \text{ smokes in } t] + \mathbb{I}[i \text{ doesn't smoke in } t])$$

Once taking the difference between these terms, the number of individuals continuously smoking cancels out. Therefore, we can express the numerator in (4) as the difference between the number of individuals smoking in t that were not smoking in 2009, and the number of individuals that were smoking in 2009, but not in t. Normalizing both terms by the number of non-smokers (NS) and smokers (S) in 2009, respectively, we get:

$$\begin{aligned} &\sum_i \mathbb{I}[i \text{ smokes in } t] - \sum_i \mathbb{I}[i \text{ smokes in 2009}] = \qquad (5) \\ &\underbrace{\frac{\sum_i \mathbb{I}[i \text{ smokes in } t,\ \text{doesn't smoke in 2009}]}{NS}}_{\text{Inflow}} \cdot NS - \underbrace{\frac{\sum_i \mathbb{I}[i \text{ smokes in 2009},\ \text{doesn't smoke in } t]}{S}}_{\text{Outflow}} \cdot S \end{aligned}$$

Equation (5) expresses the variation in the number of smokers in terms of inflow and outflow. The inflow is given by the share of individuals in the sample of non-smokers in 2009 who are smokers in t. We denote the inflow as *"Initiation"*. The outflow is the share of non-smokers in t in the sample of individuals who were smoking in 2009. Since our dependent variable assumes one when individual i is smoking, we define *"Cessation"* as smoking prevalence among smokers in 2009, such that *"Cessation"* = - Outflow.

The effect on smoking prevalence is equivalent to the effect on initiation normalized by the share of non-smokers in 2009 (NS/N), plus the effect on cessation normalized by the share of smokers in 2009 (S/N):

$$\Delta prevalence_t = \mathit{Initiation} \cdot \frac{NS}{N} + \mathit{Cessation} \cdot \frac{S}{N} \ , \qquad (6)$$

where $S + NS = N$.

Based on this decomposition, we estimate the effects on *smoking cessation* by restricting the sample to young adults who were smoking in 2009. Therefore, the outcome is equal to one for all selected individuals in 2009. In the subsequent years $t = 2010, ...2013$, the outcome is equal to one if the individual is still smoking, and zero otherwise. An important drawback is that, by restricting the sample to smokers in 2009, the trajectories in the pre-treatment years will not reflect cessation patterns. Likewise, pre-trends in the sub-sample of non-smokers in 2009 will not capture the trajectories in *smoking initiation* before the treatment. The next subsection explains how we study these pre-trends using placebo samples.

5.1.1 Placebo Estimates for Pre-Treatment Trajectories

We compute placebo estimates for the pre-treatment trajectories in cessation and initiation by restricting the sample to smokers and non-smokers in 2005, respectively. We then regress the outcome of these individuals on a treatment indicator interacted with year dummies and with a linear trend using data up to 2009.[33] The results are shown in Table 2.

Based on the sample of non-smokers in 2005, we find supporting evidence that initiation was increasing more in the treated cohort before the introduction of the local smoking bans. Even though imprecisely, this can be observed by the point estimates without controlling for linear trends in column (1), and by the direction and magnitude of the linear trend coefficient in column (2). Remarkably, the null coefficients for the year dummies column (2) indicate that the linear trend is a good approximation to the pre-trend in initiation. In contrast, taking into account that the baseline smoking prevalence is 1.00 in the sample of smokers in 2005, the coefficients on cessation are relatively smaller in magnitude. This supports the validity of the parallel trends assumption on smoking cessation, unconditional on linear trends.

Altogether, these findings point to initiation as the main driver of the slower decrease in smoking prevalence observed in the treated capitals before the bans were introduced. However, when the leading adopters, SP and RJ, are removed from the sample, as shown in column (3), the linear trend slope gets smaller. This indicates that the differential trend is primarily driven by initiation patterns in these two cities, consistent with our findings for prevalence.

A possible explanation for greater initiation rates is that young adults may have been more engaged in risky behaviors in the treated capitals. We provide insight on this by analyzing responses to specific questions of the 2013 PNS which capture these behaviors

[33]For instance, following individuals for who $y_{i,2005} = 1$ from 2005 to 2009, we estimate for cessation: $y_{i,t} = \mu_i + d_t + \sum_{s=2006}^{2008} \beta_s^C \times SB_i \times \mathbb{I}[s = t] + \delta^C Trend \times SB_i + \epsilon_{i,t}$. See Online Appendix C for further discussion and formalization.

Table 2: Placebo Estimates of Smoking Bans on Cessation and Initiation

	Initiation 2005 non-smokers			Cessation 2005 smokers		
	(1)	(2)	(3)	(4)	(5)	(6)
2006	0.004	0.001	0.002	-0.003	-0.007	-0.005
	(0.002)	(0.002)	(0.002)	(0.018)	(0.018)	(0.015)
	[0.187]	[0.675]	[0.398]	[0.861]	[0.705]	[0.779]
2007	0.005	0.0001	0.0002	0.006	-0.001	-0.009
	(0.003)	(0.002)	(0.002)	(0.016)	(0.015)	(0.013)
	[0.151]	[0.940]	[0.942]	[0.781]	[0.923]	[0.587]
2008	0.009	0.001	0.001	0.007	-0.005	-0.019
	(0.005)	(0.002)	(0.002)	(0.021)	(0.020)	(0.019)
	[0.130]	[0.584]	[0.506]	[0.792]	[0.875]	[0.396]
2009	0.010	·	·	0.016	·	·
	(0.007)	·	·	(0.030)	·	·
	[0.208]	·	·	[0.629]	·	·
Trends	·	0.003	0.001	·	0.004	-0.005
	·	(0.002)	(0.001)	·	(0.007)	(0.009)
	·	[0.208]	[0.661]	·	[0.629]	[0.619]
SP + RJ	Yes	Yes	No	Yes	Yes	No
F-stat	3.163	0.525	0.736	0.159	0.110	0.448
P-value	0.165	0.746	0.660	0.984	0.973	0.823
2009 mean	0.040		0.027	0.903		0.851

Notes: The sample for *Initiation* is restricted to non-smokers in 2005 (8,932 individuals, 35,589 observations). The sample for *Cessation* is restricted to smokers in 2005 (1,022 individuals, 4,285 observations). Estimates from Equation (2) using data from 2005 to 2009, where 2005 is the baseline. Columns (2)-(3) and (5)-(6) control for linear trends interacted with the treatment indicator (coefficient *"Trends"*). When controlling for linear trends, coefficients for 2005 and 2009 are normalized to zero. Columns (3) and (6) remove São Paulo (SP) and Rio de Janeiro (RJ) from the sample. All regressions include year and individual fixed effects and are performed using the PNS sampling weights. F-statistics and respective p-values are for tests of the joint significance of the coefficients. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level.

(Table 3). For example, we find that the share of smokers reporting purchasing cigarettes below the minimum legal price, potentially from *smuggling*, was 13 percentage points higher in the group treated by strongly enforced bans, even though prices and taxes are set nationally (subsection 2.2).[34] There is also evidence that a smaller share of smokers tried to quit in the treated capitals.

[34] Access to cheaper cigarettes is consistent with the evidence that young adults are responsive to prices (Carpenter and Cook, 2008; Friedson and Rees, 2020). We do not control for prices because those were only reported in 2013 by smokers in that year. However, evidence from a price index suggests that legal prices did not change differently among the treated and untreated groups over time (Online Appendix Figure B.2).

Since the above questions about risky behaviors were restricted to smokers in 2013, the observed differences could be partially related to selection. It is likely that young adults who were still smoking in 2013, despite the costs imposed by the bans, were also more prone to risky behaviors. However, results from the bottom panel of Table 3 suggest that selection might not explain all the difference. With a sample that includes both smokers and non-smokers, we find that young adults who faced strongly enforced bans were more likely to consume alcohol and less likely to practice sports regularly. Overall, these results are consistent with young adults in RJ and SP being generally more exposed to situations that can trigger smoking initiation (e.g., social interactions with alcohol consumption, availability of cigarettes at lower prices, etc).

Table 3: Relationship Between Smoking Bans and Other Risky Behaviors in 2013

	Average	Difference for treated		
	Control	High enforced	Low enforced	N
Daily or causal smoker	0.110	0.004	-0.015	5,659
	(0.014)	(0.020)	(0.016)	
		[0.843]	[0.405]	
Smuggling	0.084	0.126**	0.090*	567
	(0.015)	(0.050)	(0.053)	
		[0.043]	[0.066]	
Tried to quit	0.499	-0.082*	-0.113	651
	(0.035)	(0.040)	(0.072)	
		[0.093]	[0.260]	
Starting Age	15.740	0.534	1.794**	505
	(0.247)	(0.266)	(0.617)	
		[0.103]	[0.010]	
Smokers + non-smokers				
Practice sport freq.	0.376	-0.042	-0.045	5,659
	(0.020)	(0.022)	(0.026)	
		[0.136]	[0.128]	
Consumes alcohol freq.	0.129	0.030	-0.024	5,659
	(0.014)	(0.019)	(0.023)	
		[0.158]	[0.414]	

Notes: Based on data from the 2013 PNS. We measure smuggling as a dummy variable that is equal to 1 if the price reported for a pack of cigarettes is below the minimum legal price (i.e., 3.50 Brazilian Reais), and zero otherwise. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level.

5.1.2 Effects on Smoking Initiation and Cessation

By implementing the decomposition described in equation (5), we assess the effects of smoking bans on initiation by restricting the sample to non-smokers in 2009. The results from the previous section suggest that, in the absence of behavioral or environmental changes, a larger share of individuals would have become regular smokers in the capitals that implemented smoking bans. This would lead to the underestimation of the effects of the bans on initiation if not accounted for. We thus estimate the bans' causal effects as deviations from the treated cohort-specific linear trend.

However, because pre-treatment trajectories from the sub-sample of 2009 non-smokers do not capture trends in initiation, our standard event-study approach (Equation 2) will not deliver consistent estimates. For instance, when restricting the sample to smokers in 2009 to study effects on cessation, pre-treatment trajectories would be partially capturing the positive trend in initiation. In our standard estimation, this trend would be abated from the post-treatment outcomes, leading to an overestimation of the effects on cessation. This raises the question of how to control for linear trends when outcomes are asymmetrical before and after the treatment.[35]

Our approach consists of discounting, from the post-treatment outcomes, the linear trend predicted in the placebo samples.[36] Let $\hat{\alpha}^I$ and $\hat{\alpha}^C$ be the linear trend estimates for initiation and cessation from columns (2) and (5) of Table 2 respectively. For each individual i that was not smoking in 2009, we obtain the post-treatment outcome as follows:

$$y^I_{i,t} = y_{i,t} - \hat{\alpha}^I Trend \times SB_i, \text{ for } t \geq 2009 \quad (7)$$

Likewise, the post-treatment outcome for each individual i that was smoking in 2009 is obtained as follows:

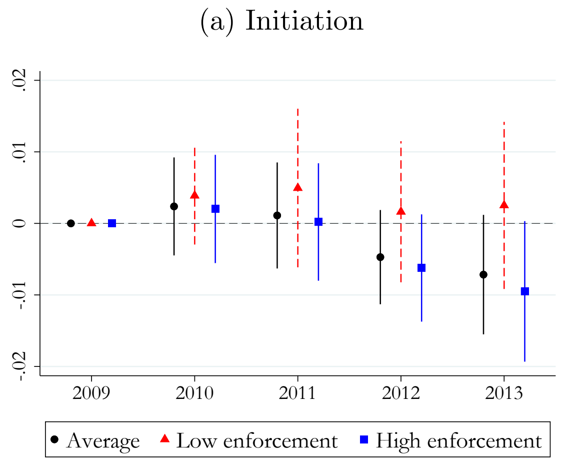
$$y^C_{i,t} = y_{i,t} - \hat{\alpha}^C Trend \times SB_i, \text{ for } t \geq 2009 \quad (8)$$

We then regress $y^I_{i,t}$ and $y^C_{i,t}$ on individual and time fixed effects, and on time dummies

[35] Such outcomes are also common in labor economics when researchers follow workers employed in the baseline of a shock. In this case, post-trends represent outflows from employment to unemployment, while pre-trends represent employment inflows.

[36] The prediction of linear trends in a placebo pre-treatment sample and their imputation in the post-treatment data is also employed by Bhuller et al. (2013) and Goodman-Bacon (2021).

interacted with the treatment indicator using data from 2009 to 2013. For the heterogeneity by enforcement level, the linear trend coefficients are specific for strongly and weakly enforced groups, and are available in the Online Appendix Table B.8.



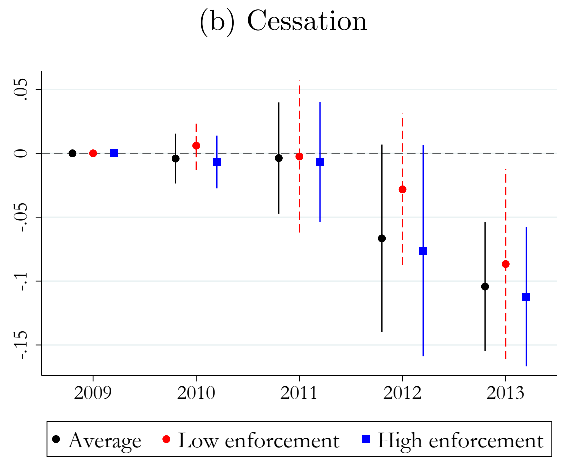


Figure 4: Impact of Smoking Bans on Smoking Initiation and Cessation

Notes: All regressions are weighted by sampling importance in PNS. 95% confidence intervals constructed using cluster-robust standard errors (at capital level), which are robust to inference using wild-cluster bootstrap. The post-treatment coefficients are estimated using the sub-sample of non-smokers and smokers in 2009, and data from 2009 to 2013. Example equations for average estimates: $y_{i,t}^{\{I \text{ or } C\}} = \mu_i + d_t + \sum_{s=2010}^{2013} \beta_s \left(\mathbb{I}(s=t) \cdot SB_i\right) + \epsilon_{i,t}$. The outcomes are $y_{i,t}^{I} = y_{i,t} - \hat{\alpha}^I Trend$ for non-smokers in 2009, and $y_{i,t}^{C} = y_{i,t} - \hat{\alpha}^C Trend$ for smokers in 2009, where $\hat{\alpha}^I$ and $\hat{\alpha}^C$ are the *Trends* coefficients in columns (2) and (5) of Table 2 respectively. Estimates by enforcement level are available in the Online Appendix Table B.8.

The results for post-treatment effects are shown in Figure 4. Although imprecise, the post-treatment estimates shown in Panel (a) suggest a decreasing trend in initiation due to the introduction of smoking bans. The point estimate is -0.007 (p-value = 0.133) by 2013. Importantly, this is driven by strongly enforced bans, which significantly reduced smoking initiation by 1 percentage point by 2013 (almost 25% of a 4% average initiation from 2005 to 2009).[37] Further, following young adults who were regular smokers in 2009, we find that smoking bans were effective in promoting cessation. The negative effect shown in Figure 4b represents an 11% reduction in smoking prevalence among 2009 smokers by 2013. The effects of strongly enforced bans on smoking cessation are similar to the average effects, leading to 11.2% of smokers quitting by 2013.

Overall, our findings suggest that the introduction of smoking bans shifted the trajectory of smoking prevalence mostly through the promotion of smoking cessation. Based

[37] Point estimates and p-values from wild-cluster bootstrap are available in Online Appendix Table B.8.

on the decomposition shown in Table 4, cessation explains almost 70% of the bans' effect on smoking prevalence. The smaller effects on initiation may be partly because, as shown in Figure 2, smoking initiation in Brazil often happens before the minimum legal age for drinking and smoking (i.e., 18 years old). The implication is that initiation might be happening at places that are not targeted by smoking bans. Further, since we do not find immediate effects on smoking prevalence, cessation, and initiation, it could be that bans take time to be assimilated.

Table 4: Decomposition of Smoking Prevalence

			Prevalence	
	Initiation ×0.882	Cessation ×0.118	Decomposition	Estimation
	(1)	(2)	(3)	(4)
2010	0.002	-0.001	0.001	0.001
	(0.003)	(0.001)	(0.003)	(0.001)
	{0.476}	{0.481}	{0.628}	[0.715]
2011	0.001	-0.001	0.0002	-0.001
	(0.002)	(0.003)	(0.003)	(-0.001)
	{0.683}	{0.774}	{0.939}	[0.581]
2012	-0.004	-0.008	-0.013*	-0.013*
	(0.003)	(0.006)	(0.007)	(-0.013)
	{0.219}	{0.132}	{0.091}	[0.075]
2013	-0.006	-0.013**	-0.019**	-0.018**
	(0.004)	(0.006)	(0.008)	(-0.018)
	{0.158}	{0.024}	{0.016}	[0.036]
$N \times T$	30,574	3,375	33,949	66,155
N	7,251	843	8,094	10,380

Notes: The effects on initiation and cessation are estimated using the sub-sample of non-smokers and smokers in 2009 respectively, and data from 2009 to 2013. In columns (1) and (2), we multiply the effects on initiation and cessation by the share of non-smokers ($\frac{NS}{N} = 0.882$) and smokers ($\frac{S}{N} = 0.118$) in the treated group in 2009, respectively. The decomposition of the effect on prevalence in column (3) is equal to (1) + (2). The effects on prevalence - column (4) - are based on Equation 2 using data from 2005 to 2009, where 2005 is the baseline, and controlling for linear trends interacted with the treatment indicator. All regressions include year and individual fixed effects and are performed using the PNS sampling weights. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from a bootstrap of the entire procedure (i.e., accounting for the first state) are shown in curly brackets. Wild-cluster bootstrap p-values in square brackets in column (4). *Statistically significant at 10% level; ** at 5% level; *** at 1% level.

While weakly enforced bans were not effective in preventing initiation (Figure 4a), Panel (b) suggests that the effects on smoking cessation reached almost 9% by 2013. As there is some evidence against the parallel trends assumption for smoking initiation in the weakly enforced group (p-value for joint significance of pre-treatment coefficients = 0.043 in Online Appendix Table B.8), we refrain from interpreting them causally. More individuals were taking up smoking in 2008 and after 2009, but this is unlikely to be related to the bans. This may bias the effects on cessation if those individuals are more likely to quit in the short run. However, as an exercise, assuming that the effects on cessation are causal and the effects on initiation are null, the decomposition would imply a decrease in prevalence by almost 0.7 percentage points [38]. Besides being smaller, this effect is more imprecise compared to the effects of strongly enforced bans. As a conclusion, smoking bans were more effective in shifting smoking prevalence when strongly enforced (if not only effective under this condition). The results for weakly enforced bans are inconclusive, but they might suggest that informal enforcement also has some role in the application of smoking bans.

Finally, the effects on prevalence derived from the decomposition (Equation 6) and displayed in the third column of Table 4 closely mirror our baseline estimates. Additionally, inference remains robust when bootstrapping the entire procedure (p-values shown in curly brackets). This supports the validity of our approach to controlling for linear trends when outcomes are asymmetrical and offers valuable insights for researchers studying similar types of outcomes. We further discuss implications of pre-trends in such settings in the Online Appendix C.

5.1.3 Effects on Cessation by Addiction Level

To better understand the mechanisms behind smoking cessation, we estimate the effects on sub-samples of young adults who were smoking for a different length of time (i.e., with different levels of smoking addiction) when the bans were implemented. The results are shown in Table 5. The average effects discussed before are shown again in column (1) for comparison. In column (2), we look at cessation among young adults who started smoking just before the bans were implemented (i.e., at most three years of smoking by 2009). Columns (3) and (4) present the results for those who had been smoking for at least four years when the bans were implemented.

Our findings imply that the main impact of smoking bans was on promoting smoking cessation among young adults who were smoking for a short period (i.e., at most three years) when the bans were implemented. The effects are statistically significant and increasing over time: cessation of 11% by 2011, 24% by 2012, and 28% by 2013. There

[38] Effect on cessation (-0.087) $\times$ Baseline share of smokers (0.075)

Table 5: Impacts on Smoking Cessation by Addiction Level

	(1)	(2)	(3)	(4)
	Full sample	Addiction level		
	2009 smokers	≤ 3 years	$\{4, 5\}$ years	≥ 6 years
2010	-0.005	-0.017	0.009	-0.004
	(0.010)	(0.032)	(0.016)	(0.016)
	[0.624]	[0.665]	[0.770]	[0.844]
2011	-0.006	-0.108***	0.073	0.029
	(0.022)	(0.030)	(0.090)	(0.025)
	[0.790]	[0.002]	[0.604]	[0.317]
2012	-0.071*	-0.240***	0.050	0.001
	(0.037)	(0.047)	(0.096)	(0.053)
	[0.097]	[0.000]	[0.679]	[0.991]
2013	-0.110***	-0.283***	0.050	-0.044
	(0.026)	(0.043)	(0.095)	(0.041)
	[0.001]	[0.000]	[0.666]	[0.384]
$N \times T$	3,375	854	475	2,046
N	843	178	99	566

Notes: The coefficients are estimated using the sub-sample of smokers in 2009, and data from 2009 to 2013 based on the following equation: $y^C_{i,t} = \mu_i + d_t + \sum_{s=2010}^{2013} \beta_s \left(\mathbb{I}(s = t) \cdot SB_i\right) + \epsilon_{i,t}$. The outcome is $y^C_{i,t} = y_{i,t} - \hat{\alpha}^C Trend$, where $\hat{\alpha}^C$ is the *Trends* coefficients in column (5) of Table 2. All regressions include year and individual fixed effects and are estimated using the PNS sampling weights. The outcomes consist of smoking prevalence among (1) young adults who were smoking in 2009; (2) young adults who had been smoking for up to 3 years in 2009; (3) young adults who had been smoking for 4 or 5 years in 2009; and (4) young adults that had been smoking for 6 or more years in 2009. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. The results are robust to controlling for Age square (Online Appendix Table B.9), and to a single regression interacting the treatment with the addiction level (Online Appendix Table B.10).

are no significant effects of the bans on cessation among individuals who were already smoking for at least four years in 2009.

These findings could be consistent with two interpretations. The first is based on the framework from the theory of rational addiction (Becker and Murphy, 1988). When the level of smoking addiction is still low, the cost imposed by the smoking bans might increase the marginal cost of current consumption above the marginal benefit from addiction reinforcement, thus triggering cessation.

An alternative explanation is that individuals with lower addiction levels are younger than those with larger levels, and thus may go out more frequently and become more exposed to the bans. However, in Online Appendix Table B.10, we present the estimates from a more flexible specification, where we interact the treatment with indicators for each addiction level (1, 2, ..., 5, 6 or more years). When controlling for age square in this specification, we find that the effects of smoking bans are stronger (and statistically different) on individuals with addiction levels up to 3 years compared to those with 4, 5, and 6 or more years. This finding supports our interpretation based on the theory of rational addiction. As a conclusion, once addiction is already settled, the additional marginal cost imposed by the bans might not be large enough to compensate for the addictive effects on individuals who are very present-oriented.

5.2 Robustness

In this paper, we focused on the cohort treated in 2009. However, in Online Appendix Table B.4 we show that our results are very similar when also considering the three capitals treated in 2010 and 2011 under a staggered design, where average effects are obtained according to Sun and Abraham (2021).[39] Our estimates are also based on unbalanced samples of young adults that were between 15 to 29 years old in each year of analysis. In the Online Appendix Table B.12 we show that our results are robust to considering a balanced sample of young adults based on their birth cohort.

In order to verify whether our findings are explained by state- or capital-level bans, we regressed smoking prevalence on the treatment variable interacted with the level of enforcement (low or high) and with the unit of implementation (capital or state). The results can be found in the Online Appendix Table B.13, and are overall robust: in both cases, smoking bans reduced smoking prevalence only when strongly enforced. Since state-level smoking bans are likely exogenous to smoking prevalence among young adults within capitals, this finding supports the causal interpretation of our estimates. Finally, we also estimate the effects of restricting the sample to five treated and five untreated capitals in the Central-Southern region of Brazil, such that they are similar in socio-economic characteristics. The results are presented in Online Appendix Table B.14,

[39] The reason is that since it contains twelve out of fifteen treated capitals, the 2009 cohort has a weight larger than 0.92 in all the years of analysis.

and are consistent with our main findings. Although we lose power due to a smaller sample size, the estimates are consistent with smoking bans reducing prevalence mostly by cessation. When restricting to capitals located in the northern region, we find no effects of weakly enforced smoking bans.

6 Conclusion

This paper evaluated the impacts of smoking bans on the prevalence, initiation, and cessation of cigarette consumption among young adults. We explored the regional and timing variations in the implementation of comprehensive smoking bans across Brazilian capitals from 2009 to 2013. For that, we employed a generalized difference-in-differences design to estimate heterogeneous impacts by the time of exposure to the restrictions and their level of enforcement. Our paper is the first to use nationally representative longitudinal data on cigarette consumption in Brazil. To the best of our knowledge, we are among the first studies to provide a broad assessment of the dynamic effects of smoking bans in the context of a developing country, where policy enforcement is especially relevant.

Our estimates suggest that smoking bans reduced smoking prevalence among young adults by 15% after four years of exposure. These effects are mainly driven by strongly enforced bans, and no significant impacts were found in the cities where the bans were weakly enforced. This effect represents more than 137 thousand young individuals in the cohort treated by local smoking bans in 2009, which can be translated to an avoided cost of USD 81 million in 2015 values.[40] Furthermore, the impacts were driven by smoking cessation, in particular among young adults who had low levels of addiction when the bans were implemented. For this group, cessation increased by more than 28% after four years of exposure to the bans. Therefore, more than 90,000 young individuals have quit cigarette use due to local smoking bans implemented in Brazil, which resulted in USD 53 million of avoided direct and indirect costs in 2015 values.

We have also shown the robustness of our results through various sensitivity tests and have ruled out the influence of other tobacco control policies. Therefore, we have provided compelling evidence that public policies like smoking bans can effectively reduce smoking

[40] Pinto et al. (2019) estimate direct and indirect costs of USD 588 per smoker in Brazil in 2015.

prevalence among young adults, particularly those who have low levels of addiction. However, initiation patterns suggest that their capacity to circumvent legal restrictions may pose a challenge to preventing smoking addiction. Overall, this paper stresses the importance of considering enforcement in policy design, implementation, and empirical evaluation.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used ChatGPT in order to improve language in specific sentences. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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Appendix – For Online Publication

A Data Appendix

In order to overcome the lack of longitudinal data available on consumption of addictive goods, many studies on smoking behavior rely on retrospective information available in national surveys to observe individual consumption over time.[41] In this paper, we created a panel with yearly individual smoking status for the period of 2005 to 2013 using retrospective information from the 2013 National Survey of Health (PNS) (IBGE, 2014), which is representative of Brazilian states and capitals.

We focus on regular smokers (or former regular smokers). A person is considered a regular smoker at year t if the difference between their current age and their smoking initiation age is greater or equal to the difference between 2013 and year t. If this person was not a smoker in 2013, we also consider their smoking quitting time and compare it with the difference between 2013 and year t. We do not observe quitting time for those who reported being casual smokers in 2013, but who are also former regular smokers. For this reason, we omit casual smokers from our main definition of regular smokers.

Regarding smoking quitting time, individuals were asked to provide the number of years and/or the number of months since they quit. Due to the timing of the survey (mid of 2013), individuals who had quit smoking for less than six months were considered smokers in 2013. In contrast, if they had quit smoking for more than six months, they were considered as former smokers in 2013.[42] In the Online Appendix Table B.15, we show that our results are robust to the change of this cutoff to nine and three months respectively.[43]

To illustrate how our data was constructed, consider three individuals: A (never

[41] See, for instance, Boes, Marti, and Maclean (2015); Palali and Ours (2019); Palali and Van Ours (2017); De Walque (2010); Kenkel, Lillard, and Liu (2009); Guindon (2014); Kostova, Husain, and Chaloupka (2017); Kidd and Hopkins (2004); Nicolás (2002); Boes, Marti, and Maclean (2015); Jones (1994); Bünnings (2017); Liu (2010); Forster and Jones (2001).

[42] As an illustration, consider four individuals and their respective quitting time: A (5 months), B (9 months), C (1 year and 3 months) and D (1 year and 8 months). A received a null quitting time and, for this reason, was not considered a former smoker in 2013. B was considered to have quit for 1 year. Consequently, B was a former smoker in 2013 but was a smoker in 2012. The same for C. Finally, D was considered to have quit for 2 years, thus was considered a smoker up to 2011, but a non-smoker after that.

[43] With three months, both A, B and C would be considered to have quit for 1 year, while D would be considered to have quit for 2 years (former smoker in 2013 and 2012). With the 9-month cutoff, both A and B would be considered smokers in 2013, while C and D would be considered as smokers up to 2012 (thus having quit for only 1 year).

smoker), B (current smoker) and C (former smoker). A will be considered as a non-smoker in all the years of the panel (dummy variable of smoking status assuming zero). If B is forty years old and answered that he started smoking at the age of 35, then B will be considered a smoker in 2013 and in the prior five years (2012-2008). So he will not be considered a smoker in 2007 and before. Finally, consider that C is also 40 years old and started smoking by the age of 35, but quit in 2011, thus 2 years before the survey. Then C also started smoking in 2008, but will not be considered a smoker in 2013 and 2012. A simplified schematic for these definitions is presented in Figure A.1. We provide an assessment of the reliability of our retrospective data in Online Appendix A.3.

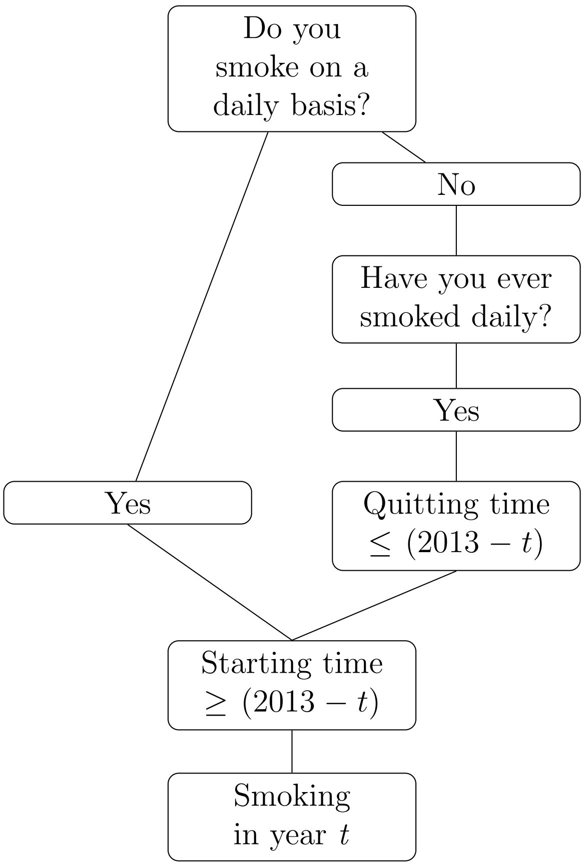

Figure A.1: Database Scheme to Generate the "Smoking" Variable

Notes: We use retrospective information of PNS (2013) to create an individual panel with smoking status for the period of 2005 to 2013. A person is considered a regular smoker in a specific year t if the difference between their current age and the smoking initiation age is greater or equal to the difference between 2013 and the year t. If this person was not a regular smoker in 2013, we also consider their smoking quitting time and compare it with the difference between 2013 and the year of analysis (t).

A.1 Treatment Cohorts

Table A.1 shows the effective date of the implementation of the smoking bans and their assigned baseline year. We assigned the baseline such that the ban would be in action in the following year. When the effective date was by the end of 2009, we assigned 2009 as the baseline. When the effective date was at the beginning of a given year, we considered this year as one year of exposure and the previous year as the baseline. That is the case of Rio Branco/AC and Campo Grande/MS, which introduced the laws on February 2010 and March 2010 respectively, thus having 2009 as the baseline; and Maceió/AL, with the effective date of the smoking ban in February 2012, thus baseline in 2011.

For the capitals with local smoking bans that belong to states that also adopted it, we consider the first one implemented to define the effective date. This only affects the baseline year for Goiânia/GO, which adopted municipal bans in 2009, and state bans only later in 2012. Rio de Janeiro/RJ was assigned a baseline of 2009 (state ban) because its municipal ban of 2008 was suspended based on a claim of unconstitutionality.

Table A.1: Baseline year for each capital based on the effective implementation of the smoking ban

Treated Unities	Effective date	Baseline year
São Paulo/SP	August, 2009	2009
Boa Vista/RR	November, 2009	2009
Curitiba/PR	November, 2009	2009
Belém/PA	November, 2009	2009
João Pessoa/PB	November, 2009	2009
Rio de Janeiro/RJ	November, 2009	2009
Aracaju/SE	December, 2009	2009
Salvador/BA	December, 2009	2009
Goiânia/GO	December, 2009	2009
Manaus/AM	December, 2009	2009
Rio Branco/AC	February, 2010	2009
Campo Grande/MS	March, 2010	2009
Teresina/PI	November, 2010	2010
Cuiabá/MT	June, 2011	2011
Maceió/AL	February, 2012	2011

Notes: Treated capitals are denoted as capital name/acronym of the state. “Effective date” is the date that the ban became effective. “Baseline year” refers to the relative time to the treatment equal to zero.

A.2 Enforcement

Regarding the bans considered in the low enforcement level, we received information from Maceió/AL, Aracaju/SE, and João Pessoa/PB. Aracaju and Maceió's monitoring agencies informed they carried out awareness-raising activities when the law were adopted and never received any reports. João Pessoa's consumer protection agency informed they are responsible for supervising smoking bans, but did not provide further information. Based on news in the media, we found that smoke-free areas were disclosed to the population, and some surveillance was put into practice in Maceió, Aracaju, and João Pessoa, as well as Manaus/AM, Rio Branco/AC, Boa Vista/RR, and Belém/PA. However, no penalties were applied. Porto Velho/RO received a zero-level indicator, due to the absence of enforcement confirmed by the monitoring agency and by the news. Rondônia's monitoring agency informed that the law was still not enforced and that they were preparing to enforce the federal law introduced in 2014. Therefore, this municipality was dropped from our main specifications.[44]

With respect to the smoking bans considered as high enforcement, we received data on fines applied from the monitoring agencies in São Paulo/SP, Curitiba/PR and Campo Grande/MS. For instance, in São Paulo, the agency inspected almost 14 thousand establishments in 2010, and more than 20 thousand in 2012. Of those, 49 received a fine in 2010, and more than 200 in 2012. More than 100 establishments were fined yearly in 2013 and 2014. The law states that establishments should be banned in case of recidivism. One establishment was closed for 48 hours in 2011, and for 30 days in 2012. In Curitiba, 99, 108, and 48 fines were applied in 2010, 2011, and 2012 respectively. After that, only 8 fines were reported in each year for 2013 and 2014. Finally, the monitoring agency of Campo Grande applied two penalties in 2011 (warnings), and 5 warnings plus one fine in 2012.

The municipal health agency of Goiânia/GO provided a list of all the notifications related to smoking bans at the workplace each year. In 2010, for example, there were 454 notifications. However, the agency did not provide information on whether there were application of fines. For Teresina/PI and Salvador/BA, we received the information that the agencies warned some establishments. In Teresina, only one establishment received a

[44] Our results are robust for the inclusion of Porto Velho/RO in the control group, as we show in the Online Appendix Table B.11.

warning from 2012 to 2017. Salvador's monitoring agency informed us that they applied fines, but the system could not provide that data in detail. We confirmed the information on surveillance, notifications, and fines with news on media. Even though we did not receive a response from the monitoring agencies in Cuiabá/MT and Rio de Janeiro/RJ, the news are clear in reporting that notifications and fines were applied in those regions.

Table A.2: Smoking bans description and enforcement levels

State [a]	**Municipality** [b]	**State Law** [c]	**Monitoring Agency** [d]	**Agency answer** [e]	**Law enforcement** [f]	**Level** [g]
SP	São Paulo	Yes	State health agency and PROCON	Complete	News and agency data: surveillance, notifications and fines.	2
PR	Curitiba	Yes	State and municipality health agency	Complete	News and agency data: surveillance and fines.	2
RJ	Rio de Janeiro	Yes	State and municipality health agency	No	News: surveillance and penalties, but no data. **Municipal law: suspended.**	2
MS	Campo Grande	No	Municipality agency	Complete	News and agency data: surveillance and few penalties.	2
MT	Cuiabá	Yes	State PROCON	No	News: surveillance and penalties, but no data.	2
GO	Goiânia	Yes	State and municipality health agency and PROCON	Yes	Agency: data on fines and notifications for work bans. News: no surveillance for smoking areas.	2
PI	Teresina	No	Municipality health agency and PROCON	Yes	News and agency data: surveillance and notifications.	2
BA	Salvador	No	Municipality PROCON	Yes. No data.	News: surveillance, notifications and fines.	2
AM	Manaus	Yes	State and municipality health agency and PROCON	No	News: surveillance, but no penalties applied.	1
AC	Rio Branco	No	Municipality health agency	No	News: surveillance, but no penalties applied.	1
SE	Aracaju	No	Municipality health agency and PROCON	Partial	News and agency: few surveillance, but no penalties applied.	1
PB	João Pessoa	Yes	State and municipality health agency and PROCON	No	News: surveillance, but no penalties applied.	1
RR	Boa Vista	Yes	State health agency and PROCON	No	News: surveillance, but no penalties applied.	1
PA	Belém	No	Municipality health agency	No	News: surveillance, but no penalties applied.	1
AL	Maceió	Yes	State and municipality health agency	Yes. No penalties	No news. Agency: surveillance, but no penalties applied.	1
RO	Porto Velho	Yes	No regulation of the law	Yes. Not enforced	News and agency: no enforcement.	0

Notes: information from ACTBr (2016), cited legislation and internet search on public sources and news. For enforcement: data from the monitoring agencies and news on media.
[a] Acronym of the State [b] Name of the state capital [c] Whether the unity is treated or not by a state level law [d] Agency responsible by the enforcement of the law [e] Whether the monitoring agency answered to our requirement [f] Description of the enforcement of the local smoking ban [g] Level of enforcement, where (0) represents not enforced smoking bans; (1) low enforcement; and (2) high enforcement.

A.3 Panel Data Validation: PNAD 2008 vs. lagged PNS

In this Appendix, we assess the reliability of our panel by comparing retrospective information from the 2013 PNS to contemporaneous data from 2008 National Household Survey (PNAD) (IBGE, 2009). We define the *2008 PNAD* as the baseline to which the *lagged PNS* (2008 retrospective data obtained from 2013) is compared. Following Kenkel, Lillard, and Mathios (2003), first, we compare average smoking prevalence among two cohorts: young adults from 15 to 29 years old; and adults from 15 to 65 years old. Then, we extend the analysis by applying goodness-of-fit tests to compare the distribution of variables related to smoking behavior in both samples. We use the two sample Pearson and Kolmogorov-Sminorv tests to the following variables: the age of current smokers; the initiation age of current and past smokers; and the time of cessation for those that quit smoking up to 2008.[45] The age of current smokers from the *lagged PNS* relies on the correct classification of smokers in 2008 and the correct calculation of their age in 2008, based on the information reported in 2013. The quitting time is a measure of the length of time, in years, since the individual quit smoking, as reported in 2013 and discounted by five years to obtain the respective time in 2008.

In addition to the goodness-of-fit tests, we implement a two-sample multiple testing procedure proposed by Goldman and Kaplan (2018) to contrast the equality of the distributions at different values. The advantage of this test is that it provides the ranges of the distribution at which the differences are significant. However, the main limitation related to this paper is that this procedure is not expected to perform well with discrete

[45] A goodness-of-fit test is performed to estimate the agreement between a distribution of the two samples of interest and the theoretical distribution. The main tests in the statistical literature are the Pearson Chi-Square and the Kolmogorov-Smirnov tests (Slakter, 1965). The Pearson $\hat{\chi}^2$'s statistic is used in this paper to test that two random samples (PNAD and PNS) come from the same population. The $\hat{\chi}^2$ "evaluates whether the difference between the observed and expected frequencies in each cell arose by chance" (Delgado and Vainora, 2021: p.2). Kolmogorov (1933) suggested a test to compare whether a sample is drawn from a population with continuous distribution function F. The test measures the maximum distance between the empirical distribution from a given sample to the populational distribution(Darling, 1957; Massey Jr, 1951). Massey Jr (1951) argues that the advantage of the Kolmogorov-Smirnov test compare to Pearson-χ^2 is that the former can detect smaller differences. However, the KS test is known to be conservative when applied to discrete data (Massey Jr, 1951; Slakter, 1965; Darling, 1957).

data.[46]

Overall, the results from this Appendix show that the retrospective data is reliable to measure smoking prevalence, supporting the absence of misclassification error. We also argue that the prevalence of recall errors is minimized in this paper, given the focus on young adults.[47] Moreover, according to Kenkel, Lillard, and Mathios (2003), while smokers use to under-report the number of cigarettes they smoke, smoking status information is known to be more accurate.[48] In this paper, we do not explore the number of cigarettes consumed, since this variable is not available for the period. Moreover, Kenkel, Lillard, and Mathios (2003) highlights that divergences in reported information were mostly from casual smokers, which are not considered in our main definition of regular smokers. Our results show evidence of heaping in the quitting time, mostly for 5 years quitting in PNS 2013. Since the Smoking Bans were introduced in 2009, heaping in 5 years should not bias the estimates. Moreover, we show evidence that the distribution of smoking quitting time is not statistically different between the states with treated and never-treated capitals in the *lagged PNS*. Our results suggest that heaping is not correlated with the treatment status.

A.3.1 Results

First, we point out that the measure of smoking prevalence from our data is consistent with official information from IBGE. Based on *2008 PNAD*, the proportion of regular

[46] Goldman and Kaplan (2018) propose a two-sample multiple testing procedure across different values of the distribution function. The motivation of the test is related to the fact that we might be interested not only in whether two samples have different distributions but also in where they differ. The paper derives the Kolmogorov-Smirnov-based multiple testing procedure and proposes a new procedure, the two-sample Dirichlet approach, in order to deal with the low sensitivity (low power) of the Kolmogorov-Smirnov procedure in the tails of the distribution. We apply the Goldman and Kaplan (2018)'s two-side multiple testing procedure using the *Stata* command `distcomp`. The command provides a graph that compares the two-sample cumulative distributions at each data point, indicating the ranges where the null hypotheses are rejected. The command also provides the p-values for the global goodness-of-fit test (Kaplan, 2019). The results in this Appendix show that the Pearson χ^2 and the Kolmogorov-Smirnov tests give very similar conclusions, regardless of the fact that the data is discrete. However, the results from Goldman and Kaplan (2018)'s multiple test procedure is not consistent with the former ones, which might be a consequence of the high prevalence of ties, given the discrete nature of the data.

[47] In a study to Vietnam, Guindon (2014) argues that exploring cross-sectional retrospective data for young age individuals reduce the possibility of recall errors.

[48] When investigating the reliability and validity of retrospective reported data to study smoking behavior using different data sets from the United States from 1970–1998, Kenkel, Lillard, and Mathios (2003) found an agreement rate in smoking status above 80%. They also found that around 80% of the respondents reported initiation ages consistently.

smokers in Brazil was 15.6% in 2008 among individuals from 15 years old on.[49] According to IBGE, in 2008, the proportion of smokers was 17.2% among individuals from 15 years old on, where only 12.2% were causal smokers.

In order to proceed with the comparisons, we restrict our retrospective panel based on 2013 PNS to the cross-section in 2008 (*lagged PNS*) and compare it to the contemporaneous data from *2008 PNAD*. We use the baseline definition of regular smokers of the paper to perform the comparisons on smoking prevalence in this Appendix. Our tests are performed on a sample of young adults (15 to 29 years old) and adults (15 to 65 years old). The sample sizes for each age cohort in each sample are shown in Table A.3. We can observe that the samples are large, even when restricted to young individuals. Therefore, the sample size is large enough to justify the asymptotic properties of the Pearson-χ^2 test statistics.

Table A.3: Sample size, PNAD 2008 vs Lagged PNS 2008

	(1) All sample	(2) Age 15 to 65	(3) Age 15 to 29
2008 PNAD	39,180	35,055	12,223
Lagged PNS	57,833	53,357	19,472
Observations	97,013	88,412	31,695

Notes: The *2008 PNAD* is the survey from 2008, which is used here to assess the reliability of the retrospective data from PNS. The sample from *Lagged PNS* is the one backtracked to 2008.

The results for the average differences' tests are shown in Tables A.4 and A.5. We test whether the differences are significant using a t-test from the regression of the variable of interest on an indicator variable that assumes one if the sample is from the *lagged PNS* and zero in the case of PNAD. The regression procedure allows using standard errors robust to heteroskedasticity and clustered at the state level. The regressions are performed using the survey data structure (weight for each individual according to their frequency in the population). The estimated coefficients for the indicator function in the tables give the results for the average difference for the *lagged PNS*, where the baseline is the average in the *2008 PNAD* sample.

[49] We measured the proportions of smokers using sample weights of individuals selected for tobacco surveys in 2008 and 2013.

[49] Available in `https://censo2010.ibge.gov.br/noticias-censo.html?view=noticia&id=1&idnoticia=1505&busca=1&t=17-2-brasileiros-fumam-52-1-deles-pensam-parar`.

In Table A.4 we observe that the PNS sample has a lower proportion of males, capital residents and rural area households among adults compared to PNAD. The average age among adults is 36 years old, and it is not statistically different across both samples. Most importantly to this project, we can observe that the proportion of smokers observed in the retrospective data of PNS is not statistically different from the true proportion in 2008 (16% in PNAD). Furthermore, the average age of smokers (40.45) is not statistically different. However, the proportion of former smokers and the average age of smoking initiation are significantly smaller in the *lagged PNS*, while the average quitting time is larger compared to the *2008 PNAD* sample (13.38 years versus 12.21 years).

Table A.4: Average differences for the sub population up to 65 years old

	Age	Male	Capital	Rural	Smokers
1(PNS=1)	0.124	-0.011	-0.272	-0.017	0.007
	(0.207)	(0.002)	(0.039)	(0.007)	(0.005)
Average PNAD	36.31	0.483	0.519	0.154	0.160
Observations	88,414	88,414	88,414	88,414	88,414
	Former smokers	Initiation age	Smokers age	Quitting time	
1(PNS=1)	-0.025	-0.663	-0.562	1.172	
	(0.005)	(0.069)	(0.340)	(0.318)	
Average PNAD	0.114	17.35	40.45	12.21	
Observations	88,414	22,758	14,145	8,613	

Notes: The indicator variable is equal one if the sample is from the *lagged PNS* and zero in case of PNAD. The baseline is the average for the *2008 PNAD* sample. The regressions are performed using the survey data structure (weight for each individual according to their frequency in the population). Cluster Robust standard errors in parentheses (state cluster).

The results for the cohort of young individuals are similar, as we can observe in Table A.5. Even though the proportion of smokers is not statistically different (10.1%), the lagged data from PNS has a smaller proportion of former smokers (1.7% versus 2.8% in PNAD) and a smaller smoking initiation age on average (15.8 versus 16.4). The average age of smokers is 23 years old, and it is not statistically different across the samples. The time of cessation for former smokers is almost one year larger in the *lagged PNS* (4.75 versus 3.78 years in PNAD).

In order to improve the comparison of the *lagged PNS* to the PNAD data beyond simply testing average differences, we compare the distributions of three variables: age of smokers, smoking initiation age, and time since quitting smoking. The two first variables are measured in age (years), and smoking quitting time is measured in the number of years since the individual quit smoking up to 2008. We present the histogram for each variable and discuss the results of the Pearson and the Kolmogorov-Smirnov tests, which

Table A.5: Average differences for the sub population up to 29 years old

	Age	Young adults	Male	Capital	Rural
1(PNS=1)	0.254	-0.006	-0.015	-0.269	-0.023
	(0.062)	(0.006)	(0.007)	(0.040)	(0.008)
Average PNAD	21.82	0.377	0.501	0.520	0.157
Observations	31,695	88,414	31,695	31,695	31,695
	Smokers	Former smokers	Initiation age	Smokers age	Quitting time
1(PNS=1)	0.012	-0.011	-0.611	0.004	0.971
	(0.008)	(0.002)	(0.114)	(0.211)	(0.258)
Average PNAD	0.101	0.028	16.41	23.24	3.78
Observations	31,695	31,695	4,181	3,411	770

Notes: The indicator variable is equal one if the sample is from the *lagged PNS* and zero in case of PNAD. The baseline is the average for the *2008 PNAD* sample. The regressions are performed using the survey data structure (weight for each individual according to their frequency in the population). Cluster Robust standard errors in parentheses (state cluster).

are shown in Table A.6.

Figure A.2 shows that the distribution of the smokers' age is very similar between the *2008 PNAD* and the *lagged PNS*, especially in the cohort of young adults (panel (b)). The age of smokers in the *2008 PNAD* is the observed age of those that were actually smoking in 2008, while the age of smokers in the *lagged PNS* is based on two retrospective information: (i) age of smokers in 2008 given as the observed age in 2013 discounted of 5 years; (ii) the sub-sample of individuals that were smoking in 2008 was obtained from the retrospective information provided in 2013. Thus, the fact that the distribution of the age of smokers looks similar for young adults, in addition to the finding that the proportion of smokers is not statistically different, gives evidence that the retrospective data is a good approximation of the prevalence of current smokers in each year, at least for the short run.

(a) Adults

(b) Young adults (15-29 years old)

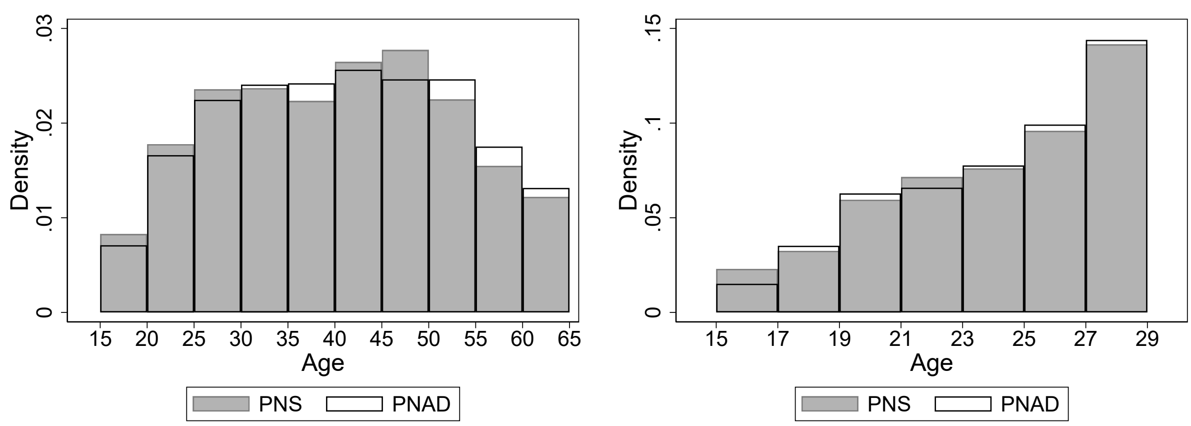

Figure A.2: Histogram Comparing the Age of Smokers in Both Samples

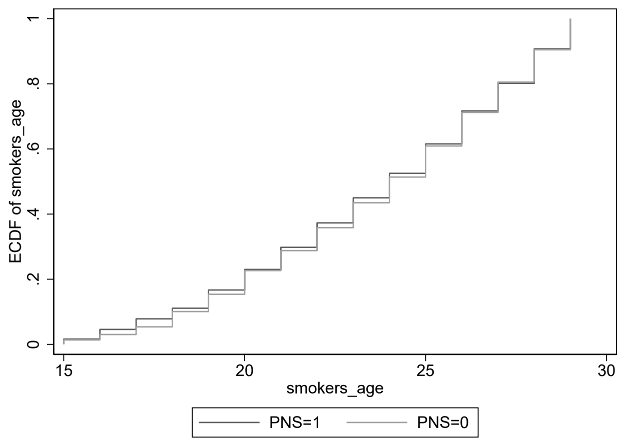

Figure A.3: Comparing Distribution of Smokers' Age Using Goldman and Kaplan (2018)

The results observed in the histograms for smokers' age are corroborated by the goodness-of-fit test statistics in the second column of Table A.6. In Panel A, we can see that the adjusted p-value of the Pearson test comparing both distributions is larger than 0.05 for all the cohorts, achieving 0.32 for the cohort of young adults. The p-value for smokers' age from the Kolmogorov-Smirnov test in Panel B shows the same results. In conclusion, for the cohort of young adults, which is the focus of the paper, we cannot reject the null hypothesis of identical distributions for the smokers' age in PNAD and PNS based on the Pearson-χ^2 and Kolmogorov-Smirnov tests. The results of Goldman and Kaplan (2018)'s procedure also give a similar conclusion: according to Figure A.3, we cannot reject that the cumulative distribution is equal at all the ranges in the samples.

Regarding smoking initiation age, the histograms plotted in Figure A.4 show that the PNS data is shifted to the left, in the direction of smaller ages, compared to the PNAD data. This is consistent with the previous finding that the initiation age is on average more than 0.5 years smaller in PNS compared to PNAD. The Pearson and the Kolmogorov-Smirnov test statistics are higher than the critical values at the 1% significance level (p-values are very smaller than 0.01 for both cohorts, as shown in the third columns of Table A.6). The largest difference in the cumulative distribution observed for the sub-sample of young adults is in the order of 0.092. On contrary, the Goldman and Kaplan (2018)'s test does not provide evidence to reject the null hypothesis that the distributions of initiation age for young adults are equal at every point (Figure A.5).

We highlight that the differences found in smoking initiation age can be partially explained by the different measures used in both surveys. In PNS, the smoking initiation

(a) Adults (b) Young adults (15-29 years old)

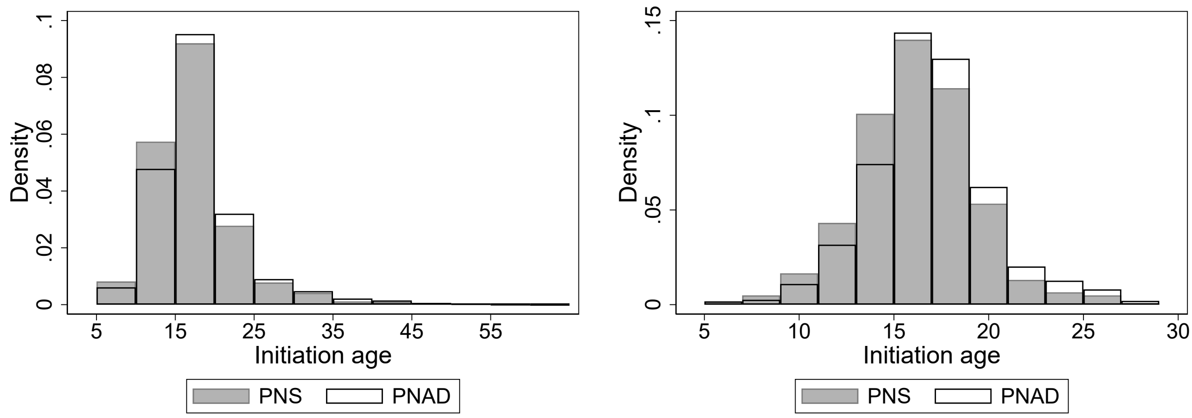

Figure A.4: Histogram Comparing Smoking Initiation Age in Both Samples

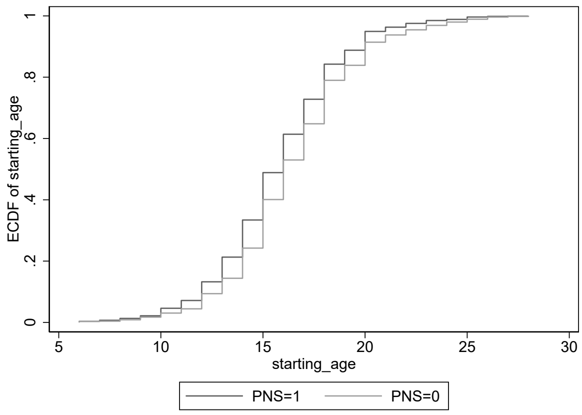

Figure A.5: Comparing Distribution of Initiation Age Using Goldman and Kaplan (2018)

age was reported as the age that the individual started smoking on a daily basis. This information should be accurate for young individuals, who are likely to have a smaller recall bias. In PNAD, the initiation age was obtained as the difference between the reported age and the reported smoking time. Therefore, we can not conclude that the findings in this Appendix indicate that the retrospective data is not reliable, since the differences could be given by measurement error in the baseline data, which would not bias our results. In addition, when we restrict the sample to the young adults in the lagged PNS, we find that the distribution of smoking initiation age is not statistically different between states with and without local Smoking Bans (Table A.7).

Finally, with regards to smoking quitting time, from the histograms in Figure A.6 we can observe that individuals round up the reported time in multiples of 5. Since PNS was 5 years lagged to be compared with PNAD, many individuals that had quit smoking for one year in 2008 (the first bar in the histogram) and should have reported 6 years of quitting time in 2013, actually reported 5 years and thus were considered as smokers in 2008. We also observe a higher concentration of individuals who reported 10 years of smoking quitting time in 2013 (thus a bunching in 5 years in 2008). Again, even though the cumulative density is higher for the PNAD sample compared to the PNS sample (Figure A.7), the Goldman and Kaplan (2018)'s test does not reject the null hypotheses of equality of the distributions at all values. However, both the Pearson and the Kolmogorov-Smirnov tests give p-values that reject the null hypothesis at 5% of significance (fourth column of Table A.6). The maximum difference between the cumulative distributions is found in the sub-sample of young individuals in capitals, in the order of 0.13.

(a) Adults (b) Young adults (15-29 years old)

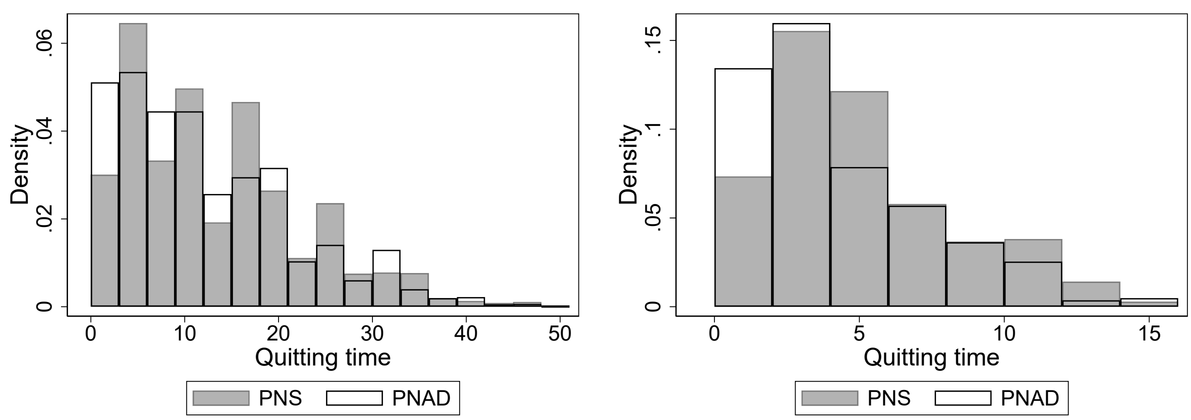

Figure A.6: Histogram Comparing Smoking Quitting Time in Both Samples

We argue that the main difference in quitting time is related to the fact that individuals who had quit for one year in 2008 were reported to have quit for five years in 2013,

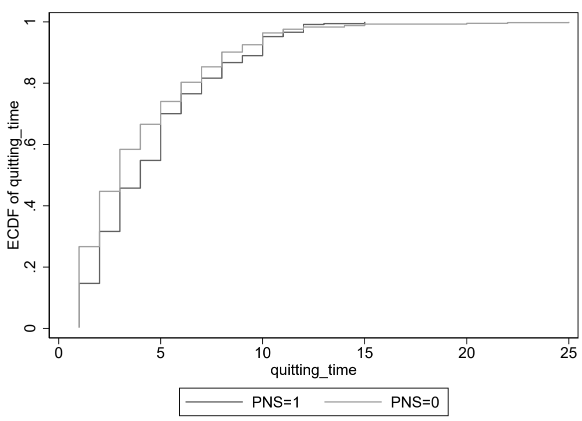

Figure A.7: Comparing Distribution of Quitting Time Using Goldman and Kaplan (2018) due to rounding. Given that the main interest of our paper is to track individuals who quit smoking up to four years in 2013 (i.e, after the introduction of smoking bans from 2009 on), the different findings related to 2008 should not bias our findings. Moreover, when we compare the distribution between the states with and without Smoking Bans in the *lagged PNS*, we can not reject the null hypothesis that the distribution of smoking quitting time is equal in both sub-samples, as shown in Table A.7.

Overall, the results from this Appendix show that the retrospective data is reliable to measure smoking prevalence, supporting the absence of misclassification error, at least for a small range of backtracking years (four years in our case). Moreover, we show that the distribution of smoking initiation age and quitting time do not differ between the sub-sample of individuals in states with treated and never-treated capitals in the *lagged PNS*. Our results suggest that 5 and 10 years of heaping in quitting time is not correlated with the treatment status (p-value = 0.39). Therefore, the comparison performed in this Appendix provides supporting evidence that the prevalence of smokers is well estimated in the period of interest, especially for the cohort of young adults.

Table A.6: Goodness-of-fit test for the distributions

Panel A: Pearson test			
	Smokers' age	Initiation age	Quitting time
Adults	1.35 (0.055)	2.45 (0.0)	5.5 (0.0)
Young Adults	1.13 (0.32)	1.84 (0.015)	1.35 (0.003)

Notes: Adjusted test statistics with p-values in parentheses.

Panel B: Kolmogorov-Sminorv test			
	Smokers' age	Initiation age	Quitting time
Adults	0.026 (0.011)	0.058 (0.0)	0.078 (0.0)
Young Adults	0.025 (0.374)	0.092 (0.0)	0.131 (0.001)

Notes: Maximum absolute difference with respective p-value in parentheses.

Table A.7: Goodness-of-fit test for the distribution of young adults in treated and not treated states in the *lagged PNS*

Panel A: Pearson test	Smokers' age	Initiation age	Quitting time
All treated	1.19 (0.28)	0.96 (0.51)	1.37 (0.19)
2009 treated	1.27 (0.23)	0.97 (0.49)	1.47 (0.15)

Adjusted test statistics with p-values in parentheses.

Panel B: Kolmogorov-Sminorv test	Smokers' age	Initiation age	Quitting time
All treated	0.04 (0.19)	0.02 (0.52)	0.05 (0.70)
2009 treated	0.05 (0.11)	0.03 (0.50)	0.07 (0.50)

Maximum absolute difference with respective p-value in parentheses.

Notes: This table provides the results of the comparison of the distribution between the sub-sample of young adults in states with treated and never-treated capitals (with respect to the implementation of local smoking bans from 2009 on) in the *lagged PNS* only. All treated groups compare the cohorts of 2009, 2010, and 2011 smoking bans with the never-treated group. 2009 treated excludes the 2010 and 2011 cohorts, in order to compare the treated in 2009 with the never-treated group.

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B Additional Results

Table B.1: Prevalence of Other Tobacco Control Policies in 2013 Across Treated and Untreated Capitals

	Adults				Young Adults			
		Enforcement				Enforcement		
	Average	High	Low	N	Average	High	Low	N
Advertising	0.308	0.014	-0.033	21,054	0.315	0.020	-0.033	8,109
	(0.019)	(0.031)	(0.031)		(0.018)	(0.039)	(0.033)	
Warnings								
News	0.341	-0.065	-0.005	21,054	0.330	-0.071*	-0.021	8,109
	(0.035)	(0.039)	(0.041)		(0.037)	(0.040)	(0.038)	
TV	0.444	-0.049**	0.034	21,054	0.429	-0.045*	0.004	8,109
	(0.018)	(0.022)	(0.043)		(0.018)	(0.022)	(0.023)	
Radio	0.192	-0.011	-0.004	21,054	0.166	-0.016	-0.012	8,109
	(0.022)	(0.030)	(0.031)		(0.020)	(0.033)	(0.036)	
Package	0.558	0.010	-0.048	21,054	0.571	-0.007	-0.064	8,109
	(0.011)	(0.036)	(0.041)		(0.015)	(0.044)	(0.039)	
Cessation Programs	0.108	-0.022	-0.027	1,501	0.040	-0.027*	-0.032*	566
	(0.025)	(0.029)	(0.036)		(0.015)	(0.015)	(0.016)	
Log Price	1.554	-0.016	0.063	2,547	1.627	-0.079	0.076	834
	(0.022)	(0.043)	(0.087)		(0.025)	(0.056)	(0.086)	

Notes: Cluster Robust Standard Errors (at capital level) in parentheses.

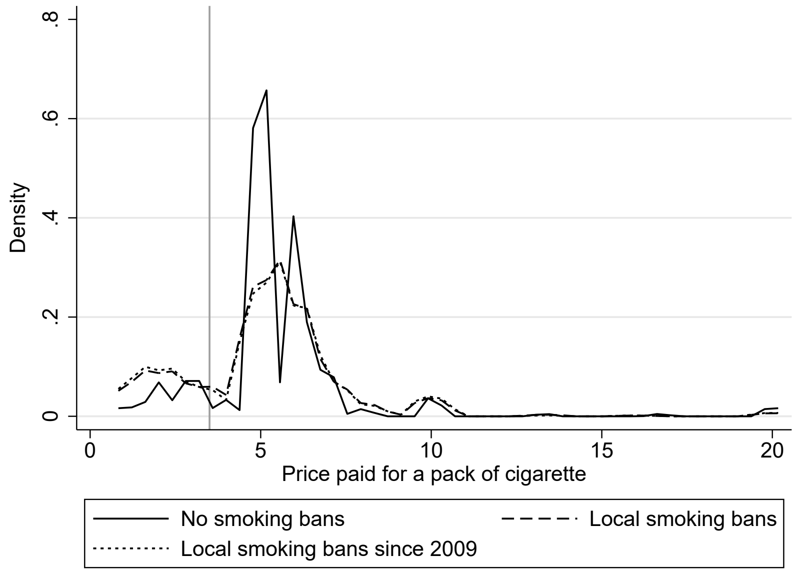

Figure B.1: Distribution of Prices Paid for a Pack of Cigarette in 2013 Among Young Adults

Notes: Density of prices reported by young adults in the 2013 PNS in their last purchase (price for a package of cigarette in Brazilian Reais).

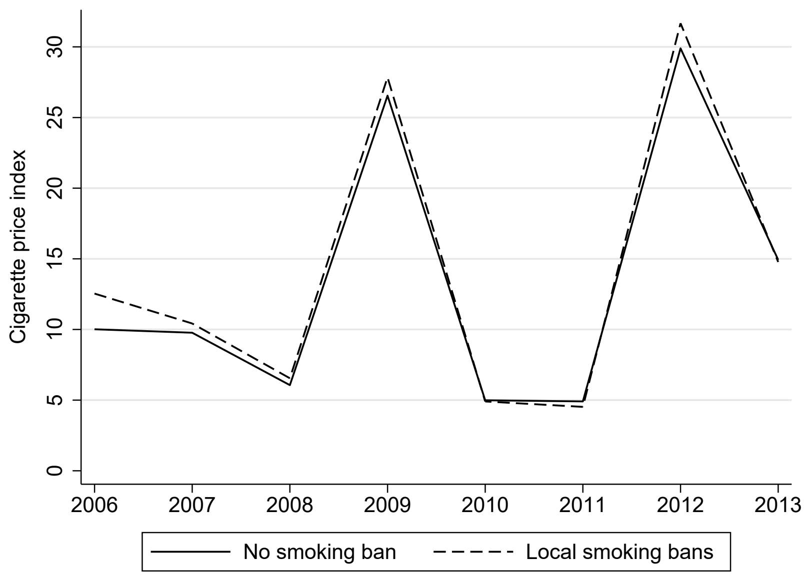

Figure B.2: Yearly Variation of Cigarette Price Index in Main Brazilian Metropolitan Areas and Capitals

Notes: The yearly accumulated variation in the cigarette price from 2006 to 2013 is from (IBGE, 2017). The index is available for nine metropolitan areas and two capitals, from which four metropolitan areas and one capital were not treated by a local smoking ban as of 2013.

B.1 Staggered Design and Identifying Assumptions

Table B.2: Smoking Prevalence in 2008 in States with Treated and Untreated Capitals

	(1) Adults	(2) Youths
All treated municipalities	-0.009	-0.012
	(0.010)	(0.011)
	[0.489]	[0.310]
2009 cohort of muicipalities	-0.009	-0.012
	(0.011)	(0.011)
	[0.492]	[0.331]
Average	0.165	0.108
Observations	34,489	11,996

Notes: The coefficients are obtained from a simple regression on an indicator of the treatment group using sampling weight. The sample is from 2008 PNAD (IBGE, 2009). Since PNAD is not representative at the capital level, we consider entire states. Cluster robust standard errors in parenthesis, and p-value from wild-cluster bootstrap in brackets. Column (1) shows the results for all the samples of adults (15 to 65 years old), while the results for young adults are shown in Column (2).

Table B.3: Impact of Smoking Bans on Smoking Prevalence by Cohort

	(1)			(2)			(3)		
Cohorts:	2009	2010	2011	2009	2010	2011	2009	2010	2011
$\hat{\beta}_{-4}$	-0.010	-0.005	-0.017	.	.	.	.	.	.
	(0.007)	(0.008)	(0.007)						
	[0.191]	[0.633]	[0.204]						
$\hat{\beta}_{-3}$	-0.008	-0.001	-0.024	-0.001	0.002	-0.012	-0.001	0.004	-0.012
	(0.007)	(0.006)	(0.012)	(0.003)	(0.001)	(0.008)	(0.003)	(0.002)	(0.008)
	[0.309]	[0.918]	[0.319]	[0.816]	[0.461]	[0.644]	[0.768]	[0.438]	[0.644]
$\hat{\beta}_{-2}$	-0.005	-0.005	-0.021	-0.001	-0.003	-0.012	-0.000	-0.003	-0.012
	(0.005)	(0.005)	(0.010)	(0.002)	(0.003)	(0.008)	(0.002)	(0.003)	(0.008)
	[0.319]	[0.508]	[0.199]	[0.798]	[0.515]	[0.611]	[0.815]	[0.517]	[0.612]
$\hat{\beta}_{-1}$	-0.003	-0.004	0.002	-0.001	-0.002	0.006	-0.000	-0.004	0.006
	(0.004)	(0.003)	(0.002)	(0.003)	(0.002)	(0.003)	(0.003)	(0.002)	(0.003)
	[0.495]	[0.475]	[0.505]	[0.866]	[0.519]	[0.298]	[0.901]	[0.446]	[0.276]
$\hat{\beta}_{1}$	0.003	-0.000	-0.009	0.001	-0.001	-0.013	0.001	-0.005	-0.013
	(0.004)	(0.002)	(0.004)	(0.003)	(0.004)	(0.006)	(0.003)	(0.003)	(0.006)
	[0.397]	[0.969]	[0.255]	[0.750]	[0.767]	[0.201]	[0.643]	[0.342]	[0.200]
$\hat{\beta}_{2}$	0.004	-0.002	-0.017	-0.001	-0.004	-0.026	-0.001	-0.009	-0.026
	(0.003)	(0.003)	(0.009)	(0.003)	(0.007)	(0.012)	(0.003)	(0.005)	(0.012)
	[0.325]	[0.671]	[0.357]	[0.557]	[0.615]	[0.204]	[0.642]	[0.347]	[0.204]
$\hat{\beta}_{3}$	-0.006**	-0.001	.	-0.013*	-0.005	.	-0.013*	-0.010	.
	(0.003)	(0.003)		(0.006)	(0.008)		(0.006)	(0.006)	
	[0.043]	[0.806]		[0.065]	[0.682]		[0.077]	[0.345]	
$\hat{\beta}_{4}$	-0.008**	.	.	-0.018**	.	.	-0.018**	.	.
	(0.003)			(0.006)			(0.007)		
	[0.040]			[0.038]			[0.042]		
Trends	.	.	.	0.002	0.001	0.004	0.002	0.002	0.004
				(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
				[0.191]	[0.641]	[0.226]	[0.219]	[0.193]	[0.225]
F-stat	0.999	4.285	1.696	0.027	5.666	1.566	0.029	6.653	1.712
P-value	0.427	0.009	0.182	0.994	0.004	0.222	0.993	0.002	0.190

Notes: Coefficients from the three cohorts are estimated from a single regression, based on Equation 1. Models (2) and (3) include cohort-specific linear trends (coefficient *"Trends"*). Two capitals treated in the beginning of 2010 are considered in the 2009 cohort in models (1) and (2), and in the 2010 cohort in model (3). Cluster robust standard errors in parenthesis, and p-value from wild-cluster bootstrap in brackets. All regressions include year and individual fixed effects and are estimated using the PNS sampling weights. F-statistics and p-values are for tests of the joint significance of pre-treatment coefficients. The average smoking prevalence in 2009 for each cohort is 0.117 (2009 cohort); 0.113 (2010 cohort); and 0.080 (2011 cohort).

Table B.4: Average Impact of Smoking Bans on Smoking Prevalence of Treated Cohorts with Staggered Treatment

	(1)	(2)
$\hat{\beta}_{-4}$	-0.010	.
	(0.007)	
$\hat{\beta}_{-3}$	-0.009	-0.001
	(0.007)	(0.002)
$\hat{\beta}_{-2}$	-0.006	-0.001
	(0.005)	(0.002)
$\hat{\beta}_{-1}$	-0.003	-0.000
	(0.004)	(0.003)
$\hat{\beta}_{1}$	0.003	0.000
	(0.003)	(0.003)
$\hat{\beta}_{2}$	0.003	-0.003
	(0.003)	(0.003)
$\hat{\beta}_{3}$	-0.006**	-0.013**
	(0.003)	(0.006)
$\hat{\beta}_{4}$	-0.008**	-0.018***
	(0.003)	(0.006)
Trends	No	Yes
Average	0.115	
$N \times T$	70,257	

Notes: The estimates for all the cohorts, based on Equation 1, are aggregated using the weights from Sun and Abraham (2021). The number of untreated clusters is 11, and the number of treated clusters is 15. Cluster-robust standard errors (at capital level) are in parentheses. All regressions include year and individual fixed effects and are estimated using the PNS sampling weights. "Trends" indicates whether linear trends specific for each treated cohort were included in the regression. "Average" denotes the prevalence of smokers in 2009 among the treated cohorts. *Statistically significant at 10% level; ** at 5% level; *** at 1% level.

B.2 Main Results

Table B.5: Impacts of Smoking Bans on Smoking Prevalence

	(1)	(2)	(3)	(4)	(5)	(6)
2005	-0.010	-0.010	.	.	.	.
	(0.007)	(0.007)				
	[0.229]	[0.217]				
2006	-0.008	-0.008	-0.001	-0.001	.	.
	(0.007)	(0.007)	(0.003)	(0.003)		
	[0.336]	[0.318]	[0.813]	[0.812]		
2007	-0.005	-0.006	-0.001	-0.001	.	.
	(0.005)	(0.005)	(0.002)	(0.002)		
	[0.323]	[0.312]	[0.802]	[0.770]		
2008	-0.003	-0.003	-0.001	-0.001	.	.
	(0.004)	(0.004)	(0.003)	(0.003)		
	[0.527]	[0.511]	[0.882]	[0.882]		
2010	0.003	0.003	0.001	0.001	0.001	0.001
	(0.004)	(0.004)	(0.003)	(0.003)	(0.004)	(0.004)
	[0.427]	[0.431]	[0.768]	[0.820]	[0.760]	[0.791]
2011	0.004	0.004	-0.001	-0.001	-0.001	-0.001
	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
	[0.356]	[0.329]	[0.564]	[0.616]	[0.718]	[0.787]
2012	-0.006*	-0.006*	-0.013*	-0.013*	-0.013*	-0.013*
	(0.003)	(0.003)	(0.006)	(0.006)	(0.005)	(0.006)
	[0.054]	[0.056]	[0.061]	[0.061]	[0.071]	[0.080]
2013	-0.008**	-0.008**	-0.018**	-0.019**	-0.018**	-0.018**
	(0.003)	(0.003)	(0.006)	(0.007)	(0.006)	(0.007)
	[0.039]	[0.035]	[0.045]	[0.041]	[0.045]	[0.048]
Trends	.	.	0.002	0.003	0.002	0.003
			(0.002)	(0.002)	(0.002)	(0.002)
			[0.197]	[0.185]	[0.211]	[0.209]
Age^2	No	Yes	No	Yes	No	Yes
F-stat	0.994	1.090	0.027	0.033	.	.
P-value	0.632	0.588	0.992	0.993	.	.

Notes: The number of observations is 66,155, with 10,380 individuals. Estimates from Equation 2, including linear trend interacted with the treatment indicator in columns (3) to (6) (coefficient *"Trends"*). Columns (2), (4), and (6) control for the age square on the right-hand side of the equation. The pre-treatment coefficients are assumed to be zero in columns (5) and (6). All regressions include year and individual fixed effects and are performed using the PNS sampling weights. There are 12 treated and 11 untreated clusters. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. F-statistics and respective p-values are for tests of the joint significance of pre-treatment coefficients. The baseline smoking prevalence in the treated group is 0.118. Cohorts 2010 and 2011 are dropped from the sample.

Table B.6: Impacts of Smoking Bans on Smoking Prevalence by Enforcement Level

	(1)		(2)		(3)
Enforcement:	High	Low	High	Low	High
2005	-0.010	-0.008	.	.	.
	(0.007)	(0.006)			
	[0.219]	[0.278]			
2006	-0.009	-0.006	-0.001	-0.001	-0.001
	(0.008)	(0.007)	(0.003)	(0.003)	(0.003)
	[0.335]	[0.497]	[0.818]	[0.988]	[0.791]
2007	-0.005	-0.007	0.0001	-0.003	0.0001
	(0.005)	(0.005)	(0.002)	(0.003)	(0.002)
	[0.374]	[0.209]	[0.969]	[0.256]	[0.963]
2008	-0.004	-0.001	-0.001	0.002	-0.001
	(0.004)	(0.003)	(0.003)	(0.002)	(0.003)
	[0.473]	[0.986]	[0.792]	[0.477]	[0.778]
2010	0.003	0.005	0.001	0.003	0.001
	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)
	[0.490]	[0.227]	[0.881]	[0.418]	[0.881]
2011	0.002	0.008	-0.003	0.004	-0.003
	(0.004)	(0.007)	(0.002)	(0.006)	(0.002)
	[0.590]	[0.419]	[0.284]	[0.569]	[0.298]
2012	-0.009***	0.005	-0.016**	-0.001	-0.016**
	(0.002)	(0.006)	(0.006)	(0.006)	(0.006)
	[0.007]	[0.521]	[0.020]	[0.941]	[0.028]
2013	-0.012***	0.005	-0.022**	-0.003	-0.022***
	(0.002)	(0.007)	(0.006)	(0.007)	(0.006)
	[0.006]	[0.510]	[0.014]	[0.775]	[0.006]
Trends	.	.	0.003	0.002	0.003
			(0.002)	(0.002)	(0.002)
			[0.225]	[0.269]	[0.187]
F-stat	0.927	2.141	0.080	2.175	0.078
P-value	0.677	0.330	0.966	0.293	0.971

In models (1) and (2), the coefficients for low and high enforcement were estimated in a single regression, based on Equation 3. Individuals in a capital with low enforced bans were dropped from model (3). Models (2) and (3) controls for linear trend interacted with the indicators for each enforcement level (coefficient *"Trends"*). All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. The number of observations is 66,155, with 10,380 individuals in models (1) and (2), and 49,269 observation with 7,763 individuals in model (3). Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. The baseline smoking prevalence is 0.129 in the treated group with high enforcement, and 0.075 in the treated group with low enforcement.

Table B.7: Impact of Smoking Bans on Prevalence on the Treated Cohort with High Enforcement Level: Leave-one-out

	(1) BA	(2) RJ	(3) SP	(4) PR	(5) MS	(6) GO	(7) RJ/SP
2010	0.001	-0.001	0.003	0.001	0.001	0.000	0.000
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
	[0.860]	[0.700]	[0.447]	[0.821]	[0.884]	[0.943]	[0.941]
2011	-0.002	-0.004	-0.003	-0.002	-0.003	-0.003	-0.006
	(0.002)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.003)
	[0.458]	[0.176]	[0.272]	[0.359]	[0.261]	[0.266]	[0.211]
2012	-0.017**	-0.012*	-0.020**	-0.017**	-0.017**	-0.016**	-0.012*
	(0.007)	(0.005)	(0.007)	(0.006)	(0.006)	(0.006)	(0.005)
	[0.027]	[0.058]	[0.025]	[0.023]	[0.024]	[0.033]	[0.076]
2013	-0.023**	-0.018**	-0.024**	-0.023***	-0.023***	-0.023***	-0.016*
	(0.007)	(0.006)	(0.007)	(0.006)	(0.006)	(0.006)	(0.006)
	[0.021]	[0.033]	[0.023]	[0.008]	[0.008]	[0.008]	[0.059]
Trends	0.003	0.001	0.003	0.003	0.003	0.003	0.000
	(0.002)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)
	[0.207]	[0.496]	[0.348]	[0.177]	[0.209]	[0.195]	[0.903]
F-stat	0.274	0.013	1.459	0.029	0.067	0.125	0.800
P-value	0.891	0.999	0.435	0.996	0.987	0.953	0.726
Average	0.139	0.127	0.115	0.129	0.128	0.131	0.098
$N \times T$	62,873	62,571	59,967	63,173	63,701	64,023	56,383
N	9,858	9,826	9,391	9,907	9,985	10,049	8,837

Notes: The table presents the estimates from Equation 3 interacting the treatment variable with the level of enforcement (low and high), leave one capital with high enforcement smoking bans out at a time. Only coefficients for high enforcement are shown, but the coefficients for low enforcement are also estimated in the same regression, controlling for linear trend interacted with the treatment indicator by enforcement level (coefficient *"Trends"*). All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. "Average" denotes the baseline smoking prevalence in each treated group, after leaving one capital out.

Table B.8: Impact of Smoking Bans on Cessation and Initiation by Enforcement Level

Panel A - Placebo: 2005 non-smokers and smokers				
	Initiation		Cessation	
Enforcement:	High	Low	High	Low
2006	0.001	0.001	-0.010	0.012
	(0.002)	(0.003)	(0.020)	(0.014)
	[0.727]	[0.792]	[0.686]	[0.426]
2007	0.001	-0.002	-0.000	-0.009
	(0.002)	(0.002)	(0.015)	(0.014)
	[0.696]	[0.407]	[0.984]	[0.545]
2008	0.001	0.004*	-0.004	-0.008
	(0.002)	(0.002)	(0.020)	(0.019)
	[0.830]	[0.081]	[0.888]	[0.784]
Trends	0.003	0.0003	0.003	0.011
	(0.002)	(0.001)	(0.008)	(0.009)
	[0.164]	[0.874]	[0.744]	[0.325]
F-stat	0.173	6.886	0.210	1.290
P-value	0.937	0.043	0.940	0.479

Panel B - 2009 non-smokers and smokers				
	Initiation		Cessation	
Enforcement:	High	Low	High	Low
2010	0.002	0.004	-0.007	0.006
	(0.004)	(0.003)	(0.011)	(0.010)
	[0.612]	[0.306]	[0.511]	[0.572]
2011	0.000	0.005	-0.007	-0.003
	(0.004)	(0.006)	(0.024)	(0.030)
	[0.978]	[0.496]	[0.800]	[0.937]
2012	-0.006	0.002	-0.076	-0.028
	(0.004)	(0.005)	(0.042)	(0.030)
	[0.125]	[0.763]	[0.109]	[0.386]
2013	-0.010*	0.003	-0.112***	-0.087*
	(0.005)	(0.006)	(0.028)	(0.038)
	[0.096]	[0.645]	[0.003]	[0.095]

Notes: The coefficients for low and high enforcement were estimated from a single regression, based on Equation (3). Panel A restricts the sample to non-smokers ($N = 8,932$) and smokers ($N = 1,022$) in 2005 for Initiation and Cessation respectively, following them from 2005 to 2009. Panel B restricts the sample to non-smokers ($N = 7,251$) and smokers ($N = 843$) in 2009 for Initiation and Cessation respectively, following them from 2009 to 2013. The outcome in Panel B discounts the linear trends specific to the enforcement cohort estimated in the placebo sample (Panel A- coefficient *"Trends"*). All regressions include year and individual fixed effects, and are estimated using the PNS sampling weights. Cluster-Robust Standard Errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients.

Table B.9: Impact of Smoking Bans on Smoking Cessation by Addiction Level: Controlling for Age Square

	(1)	(2)	(3)	(4)
	Full sample	Addiction level		
	2009 smokers	≤ 3 years	$\{4, 5\}$ years	≥ 6 years
2010	-0.005	-0.017	0.011	-0.002
	(0.010)	(0.032)	(0.016)	(0.015)
	[0.621]	[0.660]	[0.655]	[0.926]
2011	-0.006	-0.109***	0.077	0.033
	(0.022)	(0.030)	(0.090)	(0.025)
	[0.791]	[0.002]	[0.583]	[0.270]
2012	-0.071	-0.241***	0.056	0.003
	(0.039)	(0.049)	(0.096)	(0.054)
	[0.102]	[0.000]	[0.660]	[0.927]
2013	-0.110***	-0.285***	0.057	-0.046
	(0.029)	(0.045)	(0.095)	(0.046)
	[0.005]	[0.000]	[0.632]	[0.411]
$N \times T$	3,375	854	475	2,046
N	843	178	99	566

Notes: The coefficients are estimated using the sub-sample of smokers in 2009, and data from 2009 to 2013 based on the following equation: $y^C_{i,t} = \mu_i + d_t + \sum_{s=2010}^{2013} \beta_s \left(\mathbb{I}(s = t) \cdot SB_i\right) + \gamma Age_i^2 + \epsilon_{i,t}$. The outcome is $y^C_{i,t} = y_{i,t} - \hat{\alpha}^C Trend$, where $\hat{\alpha}^C$ is the *Trends* coefficients in column (5) of Table 2. All regressions include year and individual fixed effects and are estimated using the PNS sampling weights. The outcomes consist of smoking prevalence among: (1) young adults that were smoking in 2009; (2) young adults that had been smoking for up to 3 years in 2009; (3) young adults that had been smoking for 4 or 5 years in 2009; and (4) young adults that had been smoking for 6 or more years in 2009. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. The results are robust to a single regression interacting the treatment with the addiction level (Online Appendix Table B.10).

Table B.10: Impact of Smoking Bans on Smoking Cessation: Interaction with Addiction Level

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
			P-values from test of equality of coefficients					
Addiction	Estimated effects		=1	=2	=3	=4	=5	=6
1	-0.149***	-0.243***		0.232	0.846	0.0018	0.019	0.024
	(0.051)	(0.085)						
	[0.005]	[0.002]						
2	-0.246***	-0.313**			0.800	0.0057	0.015	0.017
	(0.068)	(0.110)						
	[0.009]	[0.011]						
3	-0.218	-0.272**				0.0187	0.006	0.018
	(0.103)	(0.079)						
	[0.301]	[0.017]						
4	-0.002	-0.010					0.671	0.408
	(0.044)	(0.041)						
	[0.965]	[0.820]						
5	-0.016	-0.033						0.832
	(0.041)	(0.041)						
	[0.722]	[0.432]						
6	-0.081*	-0.043						
	(0.041)	(0.029)						
	[0.069]	[0.160]						
Age^2	No	Yes						
F-stat	12.46	17.11						
P-value	8.20e-06	6.21e-07						
F-stat joint	14.66	17.80						
P-value joint	1.07e-06	2.00e-07						

Notes: The coefficients from column (1) and (2) are estimated using the sub-sample of smokers in 2009, comparing a pre-(2009) and a post-treatment period (2013), based on the following equation: $y^C_{i,t} = \alpha_i + d_{2013} + \sum_{a=1}^{6} \beta_a \left(\mathbb{I}[t = 2013] \cdot SB_i \cdot \mathbb{I}[addiction_i = a]\right) + \epsilon_i$. The outcome is $y^C_{i,t} = y_{i,t} - \hat{\alpha}^C Trend$, where $\hat{\alpha}^C$ is the *Trends* coefficients in column (5) of Table 2.The regression includes year and individual fixed effects and is estimated using the PNS sampling weights. Column (2) also controls for age square in the right-hand side of the equation. Cluster-Robust Standard Errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. Columns (3) to (8) present the p-value related to the test where the null hypotheses are that both coefficients are equal in Column (2). *F-stat* and *P-Value* are for the test of equality of all coefficients. *F-stat joint* and *P-value joint* are for the test of joint significance of all coefficients. Since our panel is unbalanced, when restricting the analysis to 2009 and 2013, the number of observations ($N = 832$) is smaller than when considering all the post-treatment period ($N = 843$). However, the average estimated effect for 2013 using this approach is almost identical to the main results discussed in the paper: -0.114 (p-value = 0.005).

B.3 Robustness

Table B.11: Impact of Smoking Bans: Alternative Treatment and Control Groups

	Excluding 2 units treated in 2010			Porto Velho in control group		
	Prevalence	Cessation	Initiation	Prevalence	Cessation	Initiation
	(1)	(2)	(3)	(4)	(5)	(6)
2010	0.001	-0.007	0.003	0.001	-0.004	0.002
	(0.003)	(0.010)	(0.004)	(0.003)	(0.010)	(0.003)
	[0.765]	[0.472]	[0.530]	[0.786]	[0.665]	[0.575]
2011	-0.002	-0.009	0.001	-0.001	-0.006	0.001
	(0.003)	(0.023)	(0.004)	(0.002)	(0.022)	(0.004)
	[0.532]	[0.705]	[0.740]	[0.523]	[0.801]	[0.816]
2012	-0.014*	-0.079*	-0.005	-0.013*	-0.069*	-0.005
	(0.006)	(0.039)	(0.003)	(0.006)	(0.037)	(0.003)
	[0.060]	[0.092]	[0.220]	[0.075]	[0.093]	[0.178]
2013	-0.019**	-0.116***	-0.007	-0.018**	-0.108***	-0.007
	(0.006)	(0.026)	(0.004)	(0.006)	(0.026)	(0.004)
	[0.031]	[0.005]	[0.151]	[0.039]	[0.003]	[0.117]
Trends	0.003	0.004	0.003	0.002	0.004	0.003
	(0.002)	(0.008)	(0.002)	(0.002)	(0.007)	(0.002)
	[0.204]	[0.630]	[0.211]	[0.210]	[0.660]	[0.162]
F-stat	0.035	0.142	0.606	0.024	0.129	0.462
P-value	0.994	0.963	0.700	1.000	0.964	0.793
$N \times T$	60,784	3,020	28,120	69,087	3,439	32,019
N	9,530	756	6,682	10,826	858	7,593

Notes: Estimates from Equation 2, including linear trend interacted with the treatment indicator (coefficient *Trends*). Estimates for *Cessation* are obtained from a sample of smokers in 2009 for post-treatment, and a sample of smokers in 2005 for pre-treatment and linear trend. Estimates for *Initiation* are obtained from a sample of non-smokers in 2009 for post-treatment, and a sample of non-smokers in 2005 for pre-treatment and linear trend. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. Number of observations from the post-treatment sample in initiation and cessation, and full sample for prevalence. All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. In columns (1), (2), and (3) there are 10 clusters treated in 2009, and 11 untreated clusters. In columns (4), (5), and (6), there are 12 treated and 12 untreated clusters. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. Average baseline smoking prevalence in the treated group is 0.117 in column (1) and 0.118 in column (4).

Table B.12: Impact of Smoking Bans: Balanced Sample of Individuals

	(1)	(2)	(3)	(4)	(5)	(6)
	Prevalence		Cessation		Initiation	
2010	-0.003	-0.001	-0.005	-0.001	0.002	0.003
	(0.002)	(0.003)	(0.009)	(0.019)	(0.002)	(0.003)
	[0.203]	[0.826]	[0.577]	[0.969]	[0.443]	[0.377]
2011	-0.007**	-0.005	-0.013	-0.040	0.001	0.001
	(0.003)	(0.004)	(0.026)	(0.032)	(0.003)	(0.005)
	[0.043]	[0.286]	[0.658]	[0.247]	[0.771]	[0.823]
2012	-0.013	-0.015	-0.050	-0.092**	-0.004	-0.005
	(0.007)	(0.008)	(0.039)	(0.033)	(0.003)	(0.005)
	[0.142]	[0.118]	[0.365]	[0.011]	[0.215]	[0.346]
2013	-0.017*	-0.021*	-0.066**	-0.137***	-0.006*	-0.008
	(0.008)	(0.009)	(0.025)	(0.027)	(0.003)	(0.005)
	[0.086]	[0.073]	[0.015]	[0.001]	[0.087]	[0.157]
Trends	0.003*	0.004	0.006	0.005	0.002	0.003
	(0.001)	(0.002)	(0.008)	(0.008)	(0.001)	(0.002)
	[0.077]	[0.144]	[0.284]	[0.578]	[0.164]	[0.181]
F-stat	0.647	0.485	0.815	0.092	2.205	2.540
P-value	0.772	0.842	0.623	0.976	0.269	0.200
Pre	1975/95	1980/95	1975/90	1980/90	1975/90	1980/90
Post	1975/95	1980/95	1980/95	1985/95	1980/95	1985/95

Notes: The table presents the estimates from Equation 2, including linear trend interacted with the treatment indicator (coefficient *Trends*). The post and pre-treatment coefficients for prevalence are estimated in a single regression, where the sample is restricted to individuals that were born from 1975 to 1995 ($N = 10,687$) in column (1) and from 1980 to 1995 ($N = 7,874$) in column (2). The pre-treatment estimates for *Cessation* are obtained from a sub-sample of smokers in 2005 and born between 1975 to 1990 ($N = 1,058$) in column (3) and between 1980 and 1990 ($N = 645$) in column (4). Post-treatment estimates are obtained from a sample of smokers in 2009 and born from 1980 to 1995 ($N = 803$) in column (3) and from 1985 to 1995 ($N = 430$) in column (4). The pre-treatment estimates for *Initiation* are obtained from a sub-sample of non-smokers in 2005 and born between 1975 to 1990 ($N = 7,649$) in column (5) and between 1980 and 1990 ($N = 5,249$) in column (6). Post-treatment estimates are obtained from a sample of non-smokers in 2009 and born from 1980 to 1995 ($N = 7,071$) in column (5) and from 1985 to 1995 ($N = 4,463$) in column (6). F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. *"Pre"* and *"Post"* represent the cohorts considered in the sample. N_{Pre} is the number of observations for the pre-treatment period in initiation and cessation, and the full sample in prevalence. N_{Post} is the number of observations for the post-treatment period in initiation and cessation, and the full sample in prevalence. All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. Average baseline smoking prevalence in the treated group is 0.122 for the cohort born between 1975 and 1995, and 0.111 for the cohort born between 1980 and 1995.

Table B.13: Impact of Smoking Bans by Enforcement Level and Unit of Implementation

	High enforcement		Low enforcement	
	Capital	State	Capital	State
2010	-0.004	0.002	0.004	0.006
	(0.004)	(0.004)	(0.004)	(0.006)
	[0.446]	[0.624]	[0.336]	[0.357]
2011	-0.012	0.0002	0.012**	0.002
	(0.006)	(0.002)	(0.004)	(0.005)
	[0.269]	[0.941]	[0.033]	[0.749]
2012	-0.017	-0.015**	0.006	0.001
	(0.007)	(0.005)	(0.004)	(0.004)
	[0.169]	[0.013]	[0.309]	[0.870]
2013	-0.025**	-0.020**	0.006	-0.002
	(0.008)	(0.005)	(0.005)	(0.006)
	[0.041]	[0.010]	[0.288]	[0.795]
Trends	0.002	0.002	0.003	0.003
	(0.002)	(0.002)	(0.001)	(0.001)
	[0.212]	[0.212]	[0.295]	[0.295]
F-stat	0.998	0.306	0.393	7.130
P-value	0.804	0.868	0.879	0.160
Average	0.081	0.138	0.079	0.071

Notes: The table presents the estimates from Equation 3 interacting the treatment variable with the level of enforcement (low and high) and the unit of implementation (capital or state). All the coefficients are estimated in a single regression, controlling for linear trend interacted with the treatment indicator by enforcement level (coefficient *"Trends"*). All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. There are 66,155 observations across 10,380 individuals. There are 11 untreated clusters. The 12 treated clusters are as follows: 2 capitals treated with high enforcement bans; 4 capitals treated with high enforcement bans from the state; 3 capitals treated with low enforcement bans; and 3 capitals treated with low enforcement bans from the state. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. "Average" denotes the baseline smoking prevalence in each treated group.

Table B.14: Impact of Smoking Bans: Central-Southern and Northern Regions

	Central-Southern			Northern		
	Prevalence	Cessation	Initiation	Prevalence	Cessation	Initiation
2010	0.003	0.002	0.003	-0.001	0.031	-0.003
	(0.003)	(0.006)	(0.004)	(0.002)	(0.021)	(0.003)
	[0.379]	[0.808]	[0.455]	[0.574]	[0.287]	[0.368]
2011	0.001	-0.020	0.005	-0.008	-0.017	-0.006
	(0.003)	(0.017)	(0.004)	(0.011)	(0.041)	(0.011)
	[0.792]	[0.358]	[0.355]	[0.523]	[0.701]	[0.636]
2012	-0.012	-0.078	-0.001	-0.004	-0.010	-0.001
	(0.009)	(0.045)	(0.003)	(0.008)	(0.049)	(0.010)
	[0.379]	[0.163]	[0.802]	[0.741]	[0.826]	[0.946]
2013	-0.017	-0.120**	-0.003	-0.002	-0.054	0.001
	(0.008)	(0.027)	(0.004)	(0.009)	(0.040)	(0.011)
	[0.227]	[0.014]	[0.643]	[0.853]	[0.271]	[0.930]
Trends	0.002	0.001	0.002	0.001	-0.002	0.0003
	(0.002)	(0.005)	(0.003)	(0.001)	(0.008)	(0.002)
	[0.567]	[0.832]	[0.481]	[0.568]	[0.854]	[0.858]
F-stat	0.917	0.944	0.588	12.940	0.132	7.947
P-value	0.634	0.728	0.759	0.068	0.968	0.204
$N \times T$	31,901	2,069	14,066	20,467	849	9,927
N	5,037	510	3,365	3,172	215	2,322

Notes: Estimates from Equation 2, including linear trend interacted with the treatment indicator (coefficient *"Trends"*). Estimates for *Cessation* are obtained from a sample of smokers in 2009 for post-treatment, and a sample of smokers in 2005 for pre-treatment and linear trend. Estimates for *Initiation* are obtained from a sample of non-smokers in 2009 for post-treatment, and a sample of non-smokers in 2005 for pre-treatment and linear trend. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. Number of observations from the post-treatment sample in initiation and cessation, and full sample for prevalence. All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. There are 5 treated and 5 untreated clusters in Central-Southern region, and 4 treated and 3 untreated clusters in the Northern region. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. The baseline smoking prevalence is 0.139 and 0.123 in the treated and untreated groups in the Central-Southern region respectively; and it is 0.08 and 0.07 in the treated and untreated groups in the Northern region respectively.

Table B.15: Impact of Smoking Bans: Alternative Thresholds for Smoking Cessation

	9 months threshold			3 months threshold		
	Prevalence	Cessation	Initiation	Prevalence	Cessation	Initiation
	(1)	(2)	(3)	(4)	(5)	(6)
2010	0.001	-0.010	0.002	-0.002	-0.027*	0.002
	(0.003)	(0.011)	(0.003)	(0.003)	(0.014)	(0.003)
	[0.836]	[0.435]	[0.545]	[0.623]	[0.079]	[0.525]
2011	-0.002	-0.006	0.001	-0.004	-0.031	0.001
	(0.003)	(0.022)	(0.004)	(0.003)	(0.020)	(0.004)
	[0.558]	[0.809]	[0.817]	[0.275]	[0.132]	[0.780]
2012	-0.014*	-0.071*	-0.005	-0.012*	-0.060	-0.005
	(0.006)	(0.038)	(0.003)	(0.006)	(0.036)	(0.003)
	[0.066]	[0.093]	[0.175]	[0.092]	[0.130]	[0.191]
2013	-0.017*	-0.105***	-0.006	-0.016*	-0.078*	-0.007
	(0.007)	(0.029)	(0.004)	(0.007)	(0.034)	(0.004)
	[0.052]	[0.008]	[0.155]	[0.084]	[0.065]	[0.152]
Trends	0.002	0.004	0.003	0.002	0.003	0.003
	(0.002)	(0.007)	(0.002)	(0.002)	(0.007)	(0.002)
	[0.208]	[0.665]	[0.210]	[0.214]	[0.713]	[0.225]
F-stat	0.014	0.137	0.525	0.041	0.109	0.588
P-value	0.999	0.963	0.746	0.991	0.979	0.699
$N \times T$	66,160	3,375	30,579	66,141	3,365	30,574
N	10,381	843	7,252	10,377	841	7,251

Notes: The table presents the estimates from Equation 2, including linear trend interacted with the treatment indicator (coefficient *Trends*). Estimates for *Cessation* are obtained from a sample of smokers in 2009 for post-treatment, and a sample of smokers in 2005 for pre-treatment and linear trend. Estimates for *Initiation* are obtained from a sample of non-smokers in 2009 for post-treatment, and a sample of non-smokers in 2005 for pre-treatment and linear trend. F-statistics and respective p-values are for tests of joint significance of pre-treatment coefficients. Number of observations from the post-treatment sample in initiation and cessation, and full sample for prevalence. All regressions include year and individual fixed effects, and are performed using the PNS sampling weights. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level. Average baseline smoking prevalence in the treated group is 0.118.

Appendix B References

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C Controlling for Linear Trends when Outcomes are Asymmetrical

We want to study the effects of a policy (SB) on individuals who were smoking in $t = 0$, such that $y_{i,0} = 1$ for all i. In this case, post-treatment trajectories capture smoking cessation ($1 \rightarrow 0$) among smokers in $t = 0$. One could consider estimating equation (2), following baseline smokers for the periods before and after the treatment. However, in this case, pre-treatment trajectories in smoking prevalence will not identify pre-trends in smoking cessation. This asymmetry in the outcome before and after the treatment requires caution when controlling for linear trends.

As an alternative, we estimate the effects on smoking cessation using data from $t = 0, ..., 4$ for the sub-sample of smokers in $t = 0$. The equation controlling for linear trends interacting with the treatment indicator is:

$$y_{i,t} = \alpha_i + \gamma_t + \sum_{s=1}^{4} \beta_s^C \times SB_i \times \mathbb{I}[s = t] + \delta^C Trend \times SB_i + \epsilon_{i,t}, \text{ for } i : y_{i,0} = 1, t \geq 0 \quad \text{(C.1)}$$

Where δ^C is the coefficient for linear trends in smoking cessation. Note that we cannot estimate δ^C from equation C.1, as it would also capture treatment effects. Instead, our approach is based on estimating $\hat{\delta}^C$ using the sub-sample of smokers in $t = -4$ and data from $t = -4$ to $t = 0$:[50]

$$y_{i,t} = \alpha_i + \gamma_t + \sum_{s=-3}^{-1} \beta_s^C \times SB_i \times \mathbb{I}[s = t] + \delta^C Trend \times SB_i + \epsilon_{i,t}, \text{ for } i : y_{i,-4} = 1, t \leq 0 \quad \text{(C.2)}$$

By discounting the linear trend obtained from C.2, we compute a residual outcome for individuals smoking in $t = 0$:

$$\tilde{y}_{i,t} = y_{i,t} - \hat{\delta}^C Trend \times SB_i, \text{ for } i : y_{i,0} = 1, t \geq 0 \quad \text{(C.3)}$$

We thus estimate the treatment effect of smoking bans on cessation following smokers in $t = 0$ by regressing $\tilde{y}_{i,t}$ as the outcome, in a modified version of equation (C.1):

$$\tilde{y}_{i,t} = \alpha_i + \gamma_t + \sum_{s=1}^{4} \beta_s^C \times SB_i \times \mathbb{I}[s = t] + \epsilon_{i,t}, \text{ for } i : y_{i,0} = 1, t \geq 0 \quad \text{(C.1')}$$

The underlying assumption is that linear trends in smoking prevalence of individuals

[50] A similar approach is also employed by Bhuller et al. (2013) and Goodman-Bacon (2021).

who were smoking in $t = -4$ are a good counterfactual for what would have been the linear pre-trends in the sample of interest (i.e., smokers in $t = 0$).

C.1 Application

In Figure C.1, we present estimates of the effects of smoking bans on initiation in Panel (a), and on cessation in Panel (b). The biased estimates are obtained from an event-study regression (Equation 2), where we estimate the linear trend using pre-treatment data of the same sample. The adjusted coefficients are estimated from the approach described above. The results highlight the need to take into account asymmetries in the outcomes when controlling for linear trends. Not properly accounting for linear trends would lead to an overestimation of the impacts on smoking cessation (reduction of prevalence among smokers by 15%), while point estimates on initiation would be positive (around 0.4 percentage points, although not statistically significant). In contrast, using the approach discussed above, we find that the policy reduced smoking prevalence among individuals that were smoking in the baseline by 11%, and, despite imprecise, might also have affected smoking initiation (-0.7 percentage points). When not properly accounted for, linear trends in smoking initiation would lead to negative effects on initiation being wrongly estimated as effects on cessation.

The point estimates and p-values using wild-cluster bootstrap for Panel (a) and (b) are shown in Table C.1, columns (1)-(2) and (4)-(5) respectively. The linear trends coefficients are also provided. Although imprecise, the linear trend coefficient in column (1) suggest that smoking initiation was following an increasing trend in the treated group. By not controlling for this differential trend, we therefore obtain positive coefficients for the effects on initiation in column (3). In addition, the fact that the coefficients in column (3) are very similar to those in column (2) reinforces linear trends in initiation are not properly estimated using the pre-treatment data of non-smokers in $t = 0$. Further, the linear trends in smoking cessation (column 4) are smaller in magnitude, which implies that the effects when not controlling for linear trends (column 6) are not as biased as in initiation (11% vs. 9.4% reduction in smoking prevalence in columns 4 and 6 respectively).

(a) Initiation (b) Cessation

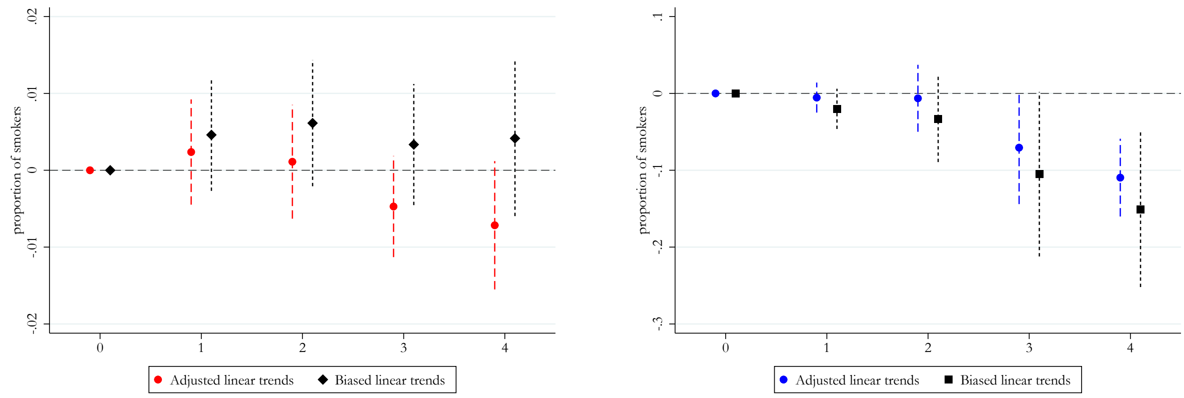

Figure C.1: Impact of Smoking Bans on Initiation and Cessation when Controlling for Linear Trends

Notes: The biased estimates are obtained from an event-study regression (Equation 2) using the sub-sample of non-smokers in $t = 0$ in Panel (a) and the sub-sample of smokers in $t = 0$ in Panel (b). The adjusted coefficients are estimated using our approach described in Appendix C. All regressions are weighted by sampling importance in PNS. 95% confidence intervals constructed using cluster-robust standard errors (at capital level), which are robust to inference using wild-cluster bootstrap. Point estimates and p-values are available in Table C.1.

Table C.1: Results for Linear Trends with Asymmetrical Outcomes

	Initiation			Cessation		
	Adjusted (1)	Biased (2)	No LT (3)	Adjusted (4)	Biased (5)	No LT (6)
$t = 1$	0.002	0.005	0.005	-0.005	-0.020	-0.002
	(0.003)	(0.004)	(0.003)	(0.010)	(0.013)	(0.010)
	[0.535]	[0.261]	[0.219]	[0.624]	[0.147]	[0.881]
$t = 2$	0.001	0.006	0.006	-0.006	-0.033	0.001
	(0.004)	(0.004)	(0.004)	(0.022)	(0.029)	(0.022)
	[0.803]	[0.176]	[0.145]	[0.790]	[0.313]	[0.946]
$t = 3$	-0.005	0.003	0.003	-0.071*	-0.105*	-0.059
	(0.003)	(0.004)	(0.003)	(0.037)	(0.055)	(0.037)
	[0.195]	[0.421]	[0.396]	[0.097]	[0.085]	[0.169]
$t = 4$	-0.007	0.004	0.003	-0.110***	-0.151**	-0.094**
	(0.004)	(0.005)	(0.004)	(0.026)	(0.052)	(0.026)
	[0.133]	[0.455]	[0.526]	[0.001]	[0.018]	[0.010]
Trends	0.003	0.0001	.	0.004	0.013	.
	(0.002)	(0.001)		(0.007)	(0.010)	
	[0.210]	[0.837]		[0.650]	[0.277]	

Notes: The biased estimates in columns (2) and (5) are obtained from an event-study regression (Equation 2) using the sub-sample of non-smokers in $t = 0$ and the sub-sample of smokers in $t = 0$ respectively. The adjusted coefficients in columns (1) and (4) are estimated using the approach described above. The coefficients in columns (3) and (6) are not controlling for differential pre-trends. All regressions include year and individual fixed effects and are estimated using the PNS sampling weights. Cluster-robust standard errors (at capital level) are in parentheses, and p-values from wild-cluster bootstrap are in square brackets. *Statistically significant at 10% level; ** at 5% level; *** at 1% level.

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ZEW – Leibniz-Zentrum für Europäische Wirtschaftsforschung GmbH Mannheim

ZEW – Leibniz Centre for European Economic Research

L 7,1 · 68161 Mannheim · Germany
Phone +49 621 1235-01
info@zew.de · zew.de