Pharmaceutical Drug Regulation and Mortality: Evidence from E-cigarettes *

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Abstract

This study evaluates drug regulation in the United States by examining the effects of the unexpected judicial exemption of e–cigarettes from drug regulation, compared to nicotine replacement therapy, which remained regulated as a drug. We find that this exemption led to significant increases in innovation, as evidenced by a rise in patent applications. Using variation in smoking rates across demographic groups prior to e–cigarette introduction, we estimate that from 2011 to 2019, e–cigarettes saved 677,000 life-years—approximately one-third of the estimated benefit of early HIV/AIDS drugs by year 2000—and increased social surplus by $8 billion annually. We show that reduced smoking is a key mechanism behind this mortality reduction, with statistically significant declines in mortality lagging smoking reductions by approximately four years.

Keywords: drug regulation, mortality, innovation, patents, e–cigarettes, smoking, nicotine replacement therapy

JEL: I12, I18, D69

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1 Introduction

Market forces drive innovation in pharmaceuticals (Acemoglu and Linn, 2004; Dubois et al., 2015) and pharmaceuticals significantly enhance longevity and quality of life (Lichtenberg, 1996, 2022). The Food and Drug Administration (FDA) plays a critical role in regulating pharmaceuticals in the United States, influencing drug diffusion globally.[1] The FDA's primary role is to approve new drugs that demonstrate a positive overall risk–benefit profile in terms of safety and efficacy relative to a placebo (Lakdawalla, 2018), with some ability to consider unmet need (Chandra et al., 2024). The modern–day incarnation of the FDA first appeared in 1938, when the agency was empowered to conduct pre–market review of the safety of all new drugs before these could be brought to market. The FDA's powers were significantly expanded in 1962 by authorizing the FDA to approve efficacy of new drugs in addition to safety (Philipson and Sun, 2008), and by providing the FDA with greater control over the testing process. Peltzman (1973) found that the 1962 FDA expansion led to substantial net costs for consumers through lost innovation.

FDA drug regulation is multi–faceted. It involves a lengthy, costly, and highly–uncertain drug approval process, high regulatory barriers for making even small changes to drugs after initial approval is received, and expensive post–market surveillance. In recent years, the total time it takes to bring a new pharmaceutical compound to market is approximately 15 years, which includes seven years in preparing the original application (e.g., Investigational New Drug [IND] Application) to begin human subjects testing[2] and eight years for marketing approval to be obtained after human subjects testing has started (DiMasi et al., 2016).[3] Only 11.8% of new compounds that are approved for human subjects testing are approved for marketing. Firms are estimated to spend on average $1.4 billion (in 2013 dollars) per approved compound (e.g., drug or vaccine), and $2.9 billion after factoring in post-approval monitoring (DiMasi et al., 2016). These costs have been growing significantly faster than the rate of inflation since the 1970s.

Recent evaluations of FDA drug regulation have primarily focused on the initial drug approval process; in particular, the trade–off between safety/efficacy and speed in the FDA's evaluation of

[1] Fifty–four percent of FDA–approved drugs were launched in the average country within ten years, versus only 19 percent of non–FDA approved drugs (Cockburn et al., 2016).

[2] This stage involves testing the properties of a potential new drug by looking at chemical evidence, animal studies, foreign experience, and use of the drug for treating other conditions (Philipson and Sun, 2008)

[3] This includes the FDA review time, which has been about 1.3 years for drugs approved between 1999 to 2005 (Chorniy et al., 2021).

new drug applications.[4] One welfare evaluation leveraged shortened FDA review times for New Drug Applications mandated by the 1992 Prescription Drug User Fee Act. Authors found that the Act reduced FDA review times and increased social surplus by between $14 to $31 billion per year (or the equivalent of 140,000 to 310,000 life–years saved if the statistical value of life is $100,000 per year), and had an extreme upper bound for adverse events of 56,000 life–years lost, suggesting that gains of faster FDA decisions outweighed costs (Philipson et al., 2008). Several other studies have found that decreasing drug review times has no sizable effect on adverse events (Grabowski and Wang, 2008; Chandra et al., 2024), increases firm R&D expenditures (Vernon et al., 2006), increases drugs in development (Chorniy et al., 2021), and increases market entry of new drug companies (Jia et al., 2023). In addition, Grennan and Town (2020) studies the role of medical device regulation and post-market surveillance between the US and EU, finding that more stringent regulation in the U.S. reduces consumer uncertainty but raises entry costs.

These studies provide important data points for understanding how FDA drug regulation reduces innovation and welfare, but they focus on only one small aspect of the FDA drug regulation process, review times that take on average 1.3 out of 15 years from the time when application preparation starts to when marketing approval is granted (Philipson and Sun, 2008; Chorniy et al., 2021). These findings do not necessarily generalize to the earlier 13.7 years (on average), or to post–market activities. Relatively little causal research exists on FDA drug regulation outside of the drug review process. We have found only one study exploring the effect of post–market FDA drug regulation activities (Garthwaite, 2012), which estimated the effect of sudden withdrawal of a drug approval on labor outcomes. Several studies explore the effect of generic drug entry (Frakes and Wasserman, 2023; Branstetter et al., 2016), but this is generally driven by patent law rather than drug regulation.

In contrast to existing evidence primarily focusing on drug review times, our current study is unique by evaluating the full effect of FDA drug regulation—from initial application preparation to post–market activities—for a single "drug," e–cigarettes, that was unexpectedly exempted from drug regulation at the end of 2010.[5] Drug exemption means that e–cigarette companies were able to bypass the lengthy and costly drug approval process entirely. Additionally, without FDA drug regulation, e–cigarette companies could also freely enter the market, modify products without approval, and

[4]Speed in approving new drugs has the benefits of producer and consumer surplus gains accruing earlier (Philipson and Sun, 2008), but unsafe drugs and/or ineffective drugs represent welfare losses due to premature death and imperfect information.

[5]We use *drug exemption* as a shorthand way to describe the process of e–cigarettes unexpectedly escaping FDA drug regulation by a court ruling that the FDA could not regulate e–cigarettes as drugs, which we describe in subsequent paragraphs.

bypass extensive post–market reporting requirements and quality control standards. By estimating the combined effect of avoiding the drug approval process and post–market drug regulation, our study advances the literature significantly in terms of understanding the full effect of FDA drug regulation. We provide further evidence on the counterfactual path that e–cigarettes would have taken by comparing effects for nicotine replacement therapy that was consistently regulated as a drug by the FDA.

While e–cigarette were no longer constrained by FDA drug regulation, they did face constraints from product liability law. Product liability lawsuits were regularly brought against e–cigarette companies for things like injuries from exploding e–cigarettes and for deceptive marketing practices (Berman, 2021; Willetts, 2023). Our study therefore provides a data point on what could happen if drug regulation disappears but product liability law remains. This concept has been advocated by Philipson and Sun (2008) as sufficient for a safe and well–functioning drug marketplace, but until now has never been empirically tested.

Additionally, our paper builds on existing research examining the uncertainty-innovation trade-off in medical device regulation. For example, Grennan and Town (2020) compare FDA and EU medical device regulation, finding that FDA oversight of coronary stents is near optimal, while EU regulation is too lax. They also highlight the value of post-market surveillance in improving consumer certainty. Similarly, Rogers (2022) show that loosening regulations for devices like spinal implants boosts innovation, market entry, and lowers procedure costs with minimal adverse effects. While our study focuses on FDA drug regulation—specifically of a single drug—our findings contribute to the broader literature on regulatory events and optimal FDA oversight (Carpenter, 2004).

The e–cigarette drug exemption natural experiment also provides the opportunity to quasi–experimentally study the relationship between vaping, smoking, and health.[6] Smoking is estimated to be the largest source of preventable disease and premature death in the United States, resulting in 480,000 deaths per year (US Department of Health and Human Services, 2014),[7] which is similar to the annual death rate during the height of the COVID–19 pandemic of approximately 500,000 per year. Most experts agree that e–cigarettes are safer than cigarettes, but estimates vary.[8] E–cigarettes

[6]Throughout the paper, we refer to the act of using cigarettes as smoking and the act of using e–cigarettes as vaping. This follows colloquial use of these terms, with vaping being thought of as very different from smoking among those that use e–cigarettes.

[7]Darden et al. (2018) and Friedson et al. (2023) also use causal research methods to identify large effects of smoking on mortality.

[8]Two prominent estimates of the relative risks of e–cigarettes to cigarettes are 5% (McNeill et al., 2022) and 37% (Allcott and Rafkin, 2022). A National Academies of Sciences, Engineering, and Medicine (2018) report states, "e–cigarettes contain fewer toxicants and are likely to be far less harmful than combustible tobacco cigarettes."

have become a focal point of controversy, partly because, like many drugs, they can have adverse effects on certain individuals—particularly those who might not have used tobacco if e–cigarettes were unavailable. Not all innovations in e–cigarette design and marketing necessarily lead to health improvements; some may be specifically crafted to attract non-tobacco users into tobacco use. Our natural experiment, combined with data on innovation, smoking behaviors, and mortality rates, aims to remove the emotional bias from this discussion and provide credible population-level estimates that take into account these competing considerations.

To preview our results, using data from the National Vital Statistics System from 1979 to 2019, we find that e–cigarette drug exemption increased life years gained by approximately 74,000 annually from 2011–2019. Using survey data, we find between 7.2 to 8.1 million fewer annual current smoking years, suggesting 1 life year gained for every 100 fewer smoking years. Statistically significant reductions in mortality lagged reductions in smoking by approximately 4 years. We estimate that drug exemption led to approximately 1/3 the benefit in terms of life years saved of early HIV/AIDS drugs through year 2000 (Philipson and Jena, 2006).

2 Background

2.1 E–cigarette History

E–cigarettes were first imported to the United States in August 2006.[9] Starting in September 2008, the FDA began confiscating e–cigarettes at ports of entry, and officially declared them to be an unapproved drug a few months later (Leon, 2010).[10] This declaration of e–cigarettes as a drug is consistent with other historical examples, such as the FDA regulating a nicotine inhaler as a drug, and the FDA having successfully removed other nicotine products from the marketplace for being unapproved drugs, including a non–electronic smokeless cigarette, nicotine hand gel, nicotine lollipops, nicotine lip balm, and nicotine water (Chowdhury and Jockel, 2018). Other countries currently regulate e–cigarettes as drugs; for example, e–cigarettes are currently only available as

[9]The first documented imported e–cigarette to the United States was the Ruyan v8 e–cigarette in Aug. 2006, which was produced in Hong Kong and developed by Hon Lik, a pharmacist. In assigning the product a tariff, U.S. Customs described the purpose of the product as "acting as a nicotine inhaler that has atomized smoke being forced out of the plastic mouthpiece tip" (U.S. Customs and Border Protection, 2006). Notably, these original imports were not flavored. A picture of the original e–cigarette is available in Appendix Figure B1, which can be viewed as baseline for subsequent innovation in e–cigarette space.

[10]The FDA's technical classification of e–cigarettes was they were an unapproved drug-delivery device (like a nicotine patch).

drugs in Australia and South Africa, and can be marketed as drugs in the United Kingdom in addition to as consumer products (Global Tobacco Control, 2024).

Two e–cigarette companies took the FDA to court asking for an injunction against these e–cigarette seizures by claiming the FDA could not regulate them as a drug without authorization from Congress. The FDA had a strong legal argument that they could declare any nicotine product as a drug, unless the federal government had "intended" for the product to be something different than a drug by regulating them differently (for example, by taxing them) (Supreme Court of the United States, 2000).

During deliberations at the district court level, in June 2009, the Family Smoking Prevention and Tobacco Control Act (TCA) became law. This gave the FDA new authority to regulate cigarettes and smokeless tobacco. The legislation also gave the FDA power to regulate other nicotine products, like e–cigarettes and cigars, as tobacco products if the FDA first "deemed" them as tobacco products following the federal rule-making process. While Congress did not require the FDA to regulate e–cigarettes as tobacco products, e–cigarette companies argued that by creating this alternative pathway, Congress *intended* for e–cigarettes to be a tobacco product instead of a drug. In other words, the FDA's previously sturdy, Supreme Court–validated legal strategy became unexpectedly shaky following passage of this new legislation, depending on how the Courts ruled on congressional intent.

Industry lawyers now argued that the FDA could only regulate e–cigarettes as a tobacco product. From industry perspective, being regulated as a tobacco product was far preferable to being regulated as a drug because it was expected to take the FDA years to "deem" e–cigarettes as a tobacco product, during which time e–cigarettes would be unregulated by the FDA. Additionally, even if/when the FDA "deemed" e–cigarettes to be a tobacco product, the regulatory burden of tobacco products was much lower than the regulatory burden of drugs.[11] In contrast, FDA officials preferred to regulate e–cigarettes as drugs so that they had far broader control, including the ability to immediately block importation.

In January 2010, a district court judge sided with industry and ruled that the FDA could not regulate e–cigarettes as a drug and must do so as a tobacco product instead. In a memorandum

[11]New tobacco products can be approved through the Premarket Tobacco Product Application (PMTA) process, which requires that their risks be no greater than those of combustible tobacco—a much lower standard compared to proving drug safety and effectiveness. Initial cost estimates for PMTAs in 2016 were $131,643 for an e–liquid and $466,563 for an e–cigarette delivery system. However, successful applications in 2021-2022 report application costs between $5 to $8 million (Adler and Rich, 2023). While significant, these costs of successful e–cigarette applications are substantially lower than costs of successful drug compounds estimated at $1.4 billion on average (in 2013 dollars) (DiMasi et al., 2016).

opinion, the judge referenced the recent TCA as persuasive (Leon, 2010). The FDA initially won a stay on this ruling, but a three–person panel of the Washington DC Circuit Court unanimously validated the district judge's decision in December 2010, at which point e–cigarettes were drug exempt.

Immediate effects of this ruling were that e–cigarettes were no longer seized at ports and the FDA began the lengthy process outlined by the TCA to deem e–cigarettes as a tobacco product. The FDA finalized their ability to regulate e–cigarettes as tobacco products on August 8, 2016. For almost six years, e–cigarettes were regulated as neither a drug nor a tobacco product by the FDA.[12] Even after the FDA gained tobacco regulatory authority over e–cigarettes, actual regulation was slow in developing. For example, e–cigarette companies were only required to submit PMTAs to the FDA to sell their products four years later, in Sept. 2020, and decisions regarding these applications remain ongoing even today.[13]

2.2 Innovation

We illustrate how innovation has been substantially faster for e–cigarettes that were exempted from FDA drug regulation than for non–e–cigarette smoking cessation drugs that were not. The first FDA–approved smoking cessation product was a 2–mg nicotine gum in 1984 (Shiffman et al., 1997).[14] No new drug innovation occurred through the rest of the 1980s. Perhaps in response to an unmet need for smoking cessation drugs, Favor, a smokeless and vaporless cigarette in which people inhaled liquid nicotine without heating, was sold in select markets between 1985 to 1987.[15] In 1987, the FDA declared Favor an unapproved drug, and removed it from the marketplace. Favor then underwent the FDA drug regulation process and the technology underlying it emerged 5 years later as an FDA–approved prescription nicotine inhaler (Advanced Tobacco Products, 2003).

[12]We are unaware of any other case where the courts prevented the FDA from regulating a new product as a drug. Medical marijuana has some interesting parallels to e–cigarettes, but key differences as well. Primarily, since marijuana is a Schedule 1 controlled substance currently, it is regulated federally by the Drug Enforcement Agency (DEA). Many states have flouted the DEA's authority federally to establish and regulate medical marijuana marketplaces themselves. The DEA has largely kept a hands-off approach to these state marketplaces, but does occasionally intervene. For example, the DEA recently issued letters to Georgia pharmacies warning them that dispensing medical marijuana through the pharmacy violates federal law, even if state law allows it (Manins and Oliviero, 2023).

[13]To reduce confounding from the FDA's escalating efforts to regulate e–cigarettes as tobacco products, we end our analysis in 2019, which is the year immediately prior to PMTAs being due.

[14]Approval came approximately four years after the start of human subjects testing in the United States in 1980 (Reuters, 1980).

[15]Tobacco industry documents reveal significant concern about this new product, with an industry survey in Austin, TX finding that 90% of smokers were aware of the product and 45% of them had tried it within its first 12 weeks (RJ Reynolds Records, 1986).

Seven years after initially authorizing 2–mg nicotine gum, the FDA approved a prescription nicotine patch in 1991, a prescription nicotine inhaler in 1992, and 4–mg nicotine gum in 1993. These products were only used by 11% of smokers during a quit attempt (Babb et al., 2017). In 1996, the FDA approved the patch and gum for over–the–counter (OTC) sale[16] and a prescription nicotine nasal spray. In 1997, OTC nicotine inhalers became available, and a non–nicotine smoking cessation drug, Zyban (bupropion), was approved for smoking cessation. The FDA approved supplemental new drug applications for mint flavored and orange flavored nicotine gum, which were brought to market in 1999 and 2000 respectively.[17] Nicotine lozenges were first approved directly for OTC in 2002 (US Food and Drug Administration, 2013). The second non–nicotine smoking cessation drug, Chantix (varenicline), was approved in 2006.

A key takeaway on the slowness of FDA drug regulation is that it took 9 years before nicotine gum could be sold with a higher nicotine strength, 12 years before it could be sold OTC, and 15 years before it could be sold with a flavor. Further, a recent editorial laments that there has been largely non–existent innovation in FDA–approved smoking cessation drugs since 2006 (Benowitz et al., 2023). In particular, the "world's oldest smoking cessation aid" cyctisine, first brought to market in 1964 in Bulgaria (Prochaska et al., 2013), and with quit success rates exceeding single forms of nicotine replacement therapy (NRT) (Lindson et al., 2023), is not approved as a drug in the United States.[18] This situation raises concern that drugs may be over–regulated in the United States if effective smoking cessation products are not brought to market. Further, the FDA can temporarily or permanently rescind drug approval due to quality control issues or adverse events that are minor compared to the dangers of smoking. For example, large–scale recalls of lozenges occurred in 2014 and Chantix in 2021 over quality control concerns, which led to shortages (Frankel, 2015; Tyko, 2019).

Regulation has been found to impede innovation in many contexts (Aghion et al., 2023). We provide quantitative evidence from Google Patents[19] of rapidly increasing innovation in the e–

[16]While the default is for drugs to be approved for sale over–the–counter (OTC), the FDA can also approve drugs for sale by prescription only. Many drug companies request prescription–only status for their medications during the length of their patent. This approach can increase profits since health insurers are more likely to pay for drugs that require a prescription, rather than those sold over–the–counter.

[17]One study finds a review time for supplemental New Drug Applications of 10 months (Kros and Qian, 2004).

[18]One company did recently announce plans to try to bring cyctisine to market in the United States (Achieve Life Sciences, 2024).

[19]Google Patents is a search engine of filed patents. To find electronic cigarette patents, we used the following search: TAC=(("electronic nicotine delivery system") OR ("electronic cigarette") OR ("e–cigarette")) with "TAC" restricting the search to patent titles, abstracts, or claims. The search for non–e–cigarette smoking cessation drugs was TAC=(("nicotine replacement therapy") OR ("nicotine gum") OR ("nicotine patch") OR ("nicotine lozenge") OR ("nicotine nasal spray") OR ("nicotine inhaler") OR ("cytisine") OR ("Varenicline") OR ("Bupropion" AND "smoking")).

cigarette marketplace following drug exemption compared to the slow innovation that occurred for FDA regulated smoking cessation drugs.[20] In Figure 1, we show patent applications for e–cigarettes and non–e–cigarette smoking cessation drugs. We show these volumes separately for the United States, in which e–cigarettes were exempted from FDA drug regulation, and Australia, in which nicotine–containing e–cigarettes could only be legally accessed through an onerous prescription process requiring multiple stages of approval (Morphett et al., 2023).[21]

There are generally low levels of annual patent applications for non–e–cigarette smoking cessation drugs in the United States (average = 7 per year from 1970–2019; high = 22) and in Australia (average = 3 per year from 1970–2019; high = 13). Similarly, e–cigarette patent applications are low in Australia (average = 9 per year from 2007–2019; high = 22), but are high in the United States where e–cigarettes were exempted from FDA drug regulation (average = 184 per year from 2007–2019; high = 408). In particular, in the United States, e–cigarette patent applications averaged 5 per year during the short–lived phase in which e–cigarettes were imported and the FDA attempted to regulate these as drugs (2007–2009). They increased to 19 in 2010 when the FDA was appealing the district court's decision that they could not regulate e–cigarettes as a drug. They further sharply increased to 226 per year during the phase in which e–cigarettes were unregulated by the FDA. Finally, they remained high (at 306 per year) during the phase in which the FDA could regulate e–cigarettes as a tobacco product. This pattern of results provides evidence of drug regulation sharply restricting innovation, as e–cigarette patents sharply increased in the United States relative to Australia during the years in which e–cigarettes were not regulated as a drug.[22] This also provides evidence that while drug regulation had a large impact on e–cigarette innovation, tobacco regulation between 2016 to 2019 did not.

We distinguished our searches by the "US" and "AU" patent offices, sorted by oldest and "Priority" date, and covered the timeframe of 01/01/1970 to 12/31/2019. The data was downloaded on 09/08/2024. The percentages of applications granted for US e–cigarettes, AU e–cigarettes, US smoking cessation drugs, and AU smoking cessation drugs are as follows: 66.6%, 43.8%, 38.0%, and 41.6%, respectively.

[20] According to Centers for Disease Control and Prevention (2023), innovation has been so rapid that e–cigarettes are commonly described in terms of four "generations" of evolution that affected everything from e–cigarette device types, voltage, heating temperature, battery, flavors, and nicotine formulation. We provide an example of this innovation visually by showing the original Ruyan v8 imported e–cigarette in Appendix Figure B1, and a recent example of hundreds of e–cigarette product offerings in a modern–day e–cigarette vape shop in Appendix Figure B2.

[21] In 2021, e–cigarettes were reclassified such that e–cigarettes could be obtained through a more traditional prescription process, including being sold in pharmacies for the first time (Morphett et al., 2023; Kenkel et al., 2024).

[22] We also estimate this relationship empirically in Table B. Using a difference–in–differences (DD) regression of logged patents for e–cigarettes and non–e–cigarette smoking cessation drugs, we find that e–cigarette applications increased in the United States in the post–drug exemption period by 269% (robust SE=31.9%) more than in Australia. Using a DDD design that includes patents for non–e–cigarette smoking cessation drugs, we find that e–cigarette applications increased in the United States by 234% (robust SE=42.5%).

2.3 Online Search Interest

We also use online search volume data from Google Trends to show that customer interest in e–cigarettes rapidly increased following this large increase in e–cigarette innovation.[23] For context, NRT products are widely available online, and are even sold on Amazon.com. Many states however ban online sales of e–cigarettes or otherwise tightly regulate them such as by requiring ID at purchase (McDonald, 2024). For these reasons, NRT product interest may be over–represented in Google Trends data.

As shown in Figure 2, e–cigarette search volume diverged from search volume for NRT and prescription pills beginning in 2009, reaching maximum search intensity (100) in 2018, relative to maximum search intensities of 2.0 for NRT and 8.0 for prescription pill searches. We attribute this divergence to the ability of e–cigarettes to freely innovate whereas the non–e–cigarette smoking cessation products were largely not able to. We further speculate that the only reason we think of e–cigarettes as "different" from these non–e–cigarette smoking cessation products today is because of the innovation spurred by a vacuum of regulation. The originally imported Ruyan v8 e–cigarette was more similar than different to a little–used, FDA–approved nicotine inhaler. Both were unflavored, and the Ruyan v8 was sold with a nicotine concentration of 16 mg/ml versus 10 mg/ml for nicotine inhalers (versus up to 4 mg/ml for nicotine gum) (Bullen et al., 2010). One early trial found the Ruyan v8 was no more effective than the nicotine inhaler for smoking cessation (Bullen et al., 2010). If the Ruyan v8 had followed the pathway of the nicotine inhaler, and could only be changed slowly and incrementally through supplemental new drug applications, then awareness and use of e–cigarettes today may be just as low as awareness and use of nicotine inhalers today. However, because e–cigarettes were exempted from drug regulation, innovation occurred rapidly,[24] and people generally think of e–cigarettes today as a different product than other NRT products, but this would not have been a–priori predicted when the FDA declared e–cigarettes to be a drug.

[23] Google Trends provides online search volume data for years 2004 to present. Search intensity is indexed from 0 to 100 and equals 100 in the period of greatest search volume. The e–cigarette search category contains the following searches, with search terms in quotations and search topics without quotations: e–cigarette, "electronic cigarette", "vape", Ruyan, Juul, Vuse, MarkTen, NJOY; the NRT search category contains Nicorette, Nicoderm, "nicotine gum", "nicotine patch", "nicotine lozenge"; the Prescription Pill search category contains Chantix, Varenicline, Zyban, and ("Bupropion" AND "smoking").

[24] For example, the typical Juul pod is sold with a nicotine concentration of 59 mg/ml in contrast to the original Ruyan v8 of 16 mg/ml (Prochaska et al., 2022; Bullen et al., 2010), which was made possible by innovation in nicotine salt–based formulations.

3 Data

3.1 Mortality Data

Mortality data come from restricted–use death certificates from the National Vital Statistics System (NVSS).[25] NVSS death certificates provide information on the near–universe of U.S. mortality with high quality ensured by standardized death certificates and coding procedures (Harris et al., 2021). The mortality data are assembled from death certificates submitted each year from states and localities to the National Center for Health Statistics (NCHS), who then prepares the data for publication.

We construct our mortality panel from 1979–2019 for each unique combination of sex, 5 or 3 race/ethnicity groupings, 5–year age bins, census region, and 2013 urban status.[26] Our estimation sample consists of decedents between the ages of 30 and 79.[27] Population counts for the same unique combinations come from the 1969–2020 and 1990–2020 single–year county–level population files from the Surveillance, Epidemiology, and End Results Program (SEER).[28]

We express annual mortality rates for each demographic group as deaths per 1,000 population in that demographic group/year using the underlying cause of death.[29] While we observe information on up to 20 contributing causes of death in the mortality files, for several methodological reasons we rely solely on the underlying cause, as is standard practice (e.g. Ruhm, 2000; Case and Deaton, 2015).[30]

[25]We also use public–use mortality files, which are identical to restricted–use files prior to 1989, from the National Bureau of Economic Research (NBER) public data repository here:

[26]Our mortality panel uses 5–race/ethnicity (White non–Hispanic, Black non–Hispanic, Asian/Pacific Islander non–Hispanic, American Indian non–Hispanic, Hispanic) or 3–race categories (White, Black, other races) depending on survey data source. Both our 3–race and 5–race panels consist of 2 sexes, 4 census regions (Northeast, Midwest, South, West), and 10 5–year age–group bins (30–34, ..., 75–79). We utilize 2013 urban status (RUCC; rural, urban) in both mortality panels.

[27]Smoking–attributable causes of death before the age of 30 are quite rare, consisting of 1.8% of all deaths before the age of 30. Further, age is commonly top–coded in our surveys over the age of 80 or 85, depending on the data source and year, while the mortality data provides single–year age.

[28]We define mortality based on census region and urban status of residence, as opposed to occurence. The 1969–2020 files from SEER use 3–race categories, while the 1990–2020 editions include 5–race/ethnicity groupings. Population counts from SEER are based on county of residence.

[29]Because our unit of observation is 5–year age groupings (by sex, race, urban status, and region), our use of group–specific rates assumes that the average composition within each group is stable over time. We believe this is a reasonable assumption given the granularity that we define our units of observation. We do, however, present adjusted rates using the 2000 standard U.S. population in our descriptive figures, including figures by 15–year age, sex, and our remaining demographic dimensions in Appendix C, and as a robustness check for our national–level specification in Appendix I. We discuss specific and adjusted rates in Appendix D.

[30]The underlying cause of death is "the disease or injury which initiated the causal chain of morbid events leading directly to death or the circumstances of the accident or violence which produced the fatal injury" (World Health Organization (2010)). Introducing contributing causes–of–death complicates our analysis by requiring a priority–ranking scheme in order to avoid double–counting deaths. Additionally, both the ICD–9 and –10 comparability ratios from Anderson et al. (2001) and smoking–attributable fractions (SAF's) from Lariscy (2019) are intended to be used

Our main outcome of interest is smoking–attributable mortality (SAM). SAM consists of 21 causes of death established as caused by smoking (Lariscy, 2019). While all 21 of these established causes are causally linked to smoking, many of these deaths would still occur even in the absence of smoking at the population–level. To refine our measure of the mortality burden of smoking, we weight each cause by the cause's sex–specific SAF.[31] Our measure of SAM is therefore the weighted sum of all 21 established causes of death, weighted by each cause's SAF. SAM comprises half of all deaths in our sample, and roughly one third of these deaths (32%) are attributable to smoking.[32]

In Appendix D.1, we discuss additional aspects of the NVSS data, including changes to Hispanic ethnicity reporting, availability of county–level FIPS codes, and our use of comparability ratios to address changes in ICD editions. We include a listing of ICD codes in Appendix D, Table D1.

3.2 Survey Data

We construct smoking prevalence, smoking intensity, and smoking frequency outcomes using 3 surveys: the Behavioral Risk Factor Surveillance System (BRFSS), the National Survey on Drug Use and Health (NSDUH), and the National Health Interview Survey (NHIS). We discuss our construction of smoking prevalence outcomes in Appendix Section D.2. We provide an overview of each survey data source in Table D2, and we discuss construction of our daily smoking–share measure (DSSM) in Appendix Section D.3.

3.3 Demographic Variation in Smoking

For each survey data source (BRFSS, NHIS, NSDUH), we construct a panel of current and daily smoking prevalences for each demographic group each year.[33] In addition, we supplement our NHIS and NSDUH panels with per–capita outcomes of average days smoked in the past month and average cigarettes smoked per day. We express our smoking prevalence outcomes as population–level specific rates and express our intensity/frequency outcomes as population–level specific rates per capita.[34]

with underlying causes of death. Using either the underlying–line or any–line on the death certificate are both valid approaches to classifying mortality.

[31] For consistency, we apply SAF's to each cause of death when we examine sub–categories of SAM: Cancers, Cardiovascular, Respiratory, and Metabolic.

[32] Cardiovascular causes of death constitute 53% of unweighted SAM, followed by cancers at 31%, respiratory at 11%, and metabolic at 5%. 49% of cancer deaths are attributable to smoking, 19% of cardiovascular deaths, 47% of respiratory deaths, and 15% of metabolic deaths.

[33] We use daily and heavy smoking prevalence interchangeably.

[34] For example, a value of 0.05 for the current smoking prevalence means that 5% of that group's population is a current smoker. A value of 2 for our per–capita measure of cigarettes smoked per day in the past month reflects an average of 2 cigarettes smoked per day per member of that group in the population. A value of 5 for our per–capita

Our modeling approach hypothesizes that demographics that smoke more prior to drug introduction or exemption will benefit more than demographics that smoke less in terms of their future mortality. In other words, a demographic group's prevalence of daily smokers prior to drug introduction provides a measure of that group's "exposure" to the intervention. We focus on the daily smoking prevalence as our measure of exposure to drug introduction/exemption.[35]

When calculating smoking rates or other statistics from our survey data, we are calculating a sample statistic, specifically, the weighted mean. The degree of uncertainty associated with our sample statistics is the sampling variance, which is directly correlated with the number of survey respondents used to calculate a particular statistic.[36] To account for differences across groups in the statistical uncertainty associated with smoking prevalences and other survey outcomes, we apply Empirical Bayesian Shrinkage (Morris, 1983). We discuss Bayesian Shrinkage further upcoming in Section 4.3.

Our estimation sample for our BRFSS survey panel consists of 719 demographic groups, spans 1997–2019, and is constructed from an average of 270,701 survey respondents in each year. Our BRFSS panel contains current and daily smoking prevalence outcomes, and contains 5–race/ethnicity categories, 5–year age groups, and census region and urban status. The average daily smoking share measure (DSSM) from 2005–2006 is 14.3% (SE = 0.2%).

Our estimation sample for our NHIS survey panel consists of 219 demographic groups, spans 1976–2019 (with gaps), and is constructed from an average of 29,829 survey respondents in each year. Our NHIS survey panel contains current– and heavy smoking prevalence outcomes, and average cigarettes per day and past–month days smoked per capita outcomes. Our NHIS panel contains 3–race categories, 5–year age groups, and census region only. The average daily smoking share measure from 2005–2006 is 16.9% (SE = 0.4%). Since this is the only survey data source with smoking information during the NRT introduction time period (1984), we also use this data source to calculate an average DSSM from 1983 of 27.9% (SE = 0.5%). NHIS did not field smoking questions in 1982, thus forcing us to use a single–year DSSM for this data source.

measure of days smoked in the past month per capita reflects an average of 5 days smoked in the past month per member of that group in the population.

[35]Our use of daily smoking rates over current smoking rates has the advantage of providing greater dispersion across demographic groups and being a stronger predictor of mortality than current smoking rates. People are unlikely to report daily smoking (or any smoking) if they only use e–cigarettes, as users colloquially refer to the act of using e–cigarettes as vaping rather than smoking. Further, since we construct smoking probabilities prior to e–cigarette introduction, e–cigarette use should not impact these smoking measures. We further test the sensitivity of our results to the use of a 2016–2018 daily–e–cigarette use share measure from BRFSS (Allcott and Rafkin, 2022).

[36]For example, demographic groups that make up a larger share of the population will generally contain more survey respondents than groups that make up a smaller share of the population. The mean smoking rate for larger groups will generally be measured with greater precision than groups that consist of fewer observations.

Our estimation sample for our NSDUH survey panel consists of 39 demographic groups, spans 2002–2019, and is constructed from an average of 38,095 survey respondents in each year. Our NSDUH survey panel contains current– and heavy smoking prevalence outcomes, and average cigarettes per day and past–month days smoked per capita outcomes. Our NSDUH panel contains 5–race categories, coarser age–groupings,[37] and no geographic indicators. The average DSSM from 2005–2006 is 16.2% (SE = 1.1%).

Our 3–race mortality panel with urban status spans 1979–2019 and contains 287 unique demographic groups with non–missing smoking–shares. Our 5–race mortality panel with urban status spans 1990–2019 and contains 719 unique groups with non–missing smoking–shares.

3.4 Descriptive Trends

Table 1 presents descriptive means for our smoking–attributable mortality outcomes and for current and daily smoking prevalence in the years before NRT and e-cigarette introduction. SAM and smoking rates have both decreased by roughly 33% in the 22 years between NRT introduction and e-cigarette introduction.

Figure 3 shows trends in logged smoking–attributable mortality since the start of our panel in 1979 for high–smoking (daily smoking rate in 1983: 33.2%; 2006-07: 21.7%) and low–smoking demographic groups (1983: 27.6%; 2006-07: 17.7%). We present analogous figures showing trends in the population–level current and daily smoking prevalence. We pair each figure with deviations in those outcomes from the linearly extrapolated trend (extrapolated using the pre–period years in our event studies).[38]

High–smoking groups experienced slower declines in SAM since 1979 compared to low–smoking groups. In the 9 years following e–cigarette drug exemption (2011–2019), relative declines in SAM increased for high–smoking groups. Current smoking prevalence has likewise declined since 1975, from 43% and 33% for high– and low–smoking groups, to 18% and 12% in 2019, respectively. While current smoking has declined at a similar rate for both groups following NRT drug introduction in 1984, current smoking begins to decline at a greater rate after 2011 for high–smoking groups compared to low–smoking groups, and this reduction is driven almost entirely by reductions in daily smoking.

[37] NSDUH provides ages in the following bins: 30–34, 35–49, 50–64, and 65–99. We include ages 30 and older in our NSDUH panel, unless BRFSS and NHIS, which excludes ages 80 and older.

[38] Our descriptive figures present rates adjusted to the 2000 standard U.S. population.

4 Methods

4.1 Estimation Strategy

To estimate the causal effect of drug introduction/exemption on mortality, we use a dose–response difference–in–differences identification strategy, where the doses are cross-sectional variation in the prevalence of daily cigarette smoking. By using this demographic variation in smoking, our strategy has some features of a "shift–share" identification strategy popularized by Bartik (1991); however, unlike traditional shift–share designs our analysis includes a pre–period that allows testing for unobserved time–varying correlation between the shares and outcome through examination of the parallel trends assumption, thus bolstering our study design (Goldsmith-Pinkham et al., 2020). Our reduced form equation is:

$$\log(y_{it}) = \alpha + \beta \cdot \theta_i \cdot \text{Post}_t + \gamma_i + \phi_{t(a)} + \varepsilon_{it} \tag{1}$$

Above, y_{it} is group i's smoking–attributable mortality rate in year t. SAM is weighted by smoking–attributable fractions and expressed as specific rates per 1,000 population. θ_i is the smoking share variable, measured as the daily smoking prevalence in the years immediately preceding drug introduction: 1983 for NRT drug introduction and 2005–2006 for e–cigarette drug introduction/exemption. Post_t is an indicator equal to unity in years after drug introduction, zero otherwise. β is the before–after change in the association between smoking shares and SAM growth. Positive values of β indicate that drug introduction increases mortality growth, holding smoking shares fixed at their baseline levels; likewise, negative values indicate that drug introduction lowered mortality growth holding smoking shares fixed. We scale β by the mean level of the smoking share so that changes are expressed at the population-average daily smoking share.[39]

γ_i is a unit fixed effect controlling for time–invariant unobserved heterogeneity. γ_i soaks up variation in smoking–attributable mortality induced by time–invariant factors, such as the mortality–age gradient, averages differences in mortality by sex, by geography, etc. Changes in SAM are therefore expressed as within-group changes.[40] Age–group–specific time fixed effects, $\phi_{t(a)}$, where a is one of 10 5–year age bins (30–34, ..., 75–79), control for unobserved shocks to age–specific SAM.

[39] Unless otherwise noted, averages computed are weighted averages, and the weights are population shares. We weight by population shares in the reference period year (year prior to drug introduction) in our regressions.

[40] The age of decedents is measured in the year of death, not the year of drug introduction. Our model therefore estimates changes in period–mortality, not cohort–motrality. Over time, the composition of each age bin changes as individuals grow older.

$\phi_{t(a)}$ serves a few purposes: age explains a disproportionate share of variation in both our smoking share and overall variation in SAM. For example, removing age as a predictor of the smoking share reduces the model's adjusted R–squared by 39pp, roughly half, while removing age as a predictor of logged SAM reduces adjusted R–squared by 88ppt, or a 90% decrease.[41] Age–specific time fixed effects allow us to flexibly control for unobserved shocks to health or SAM that vary with age. Conceptually, $\phi_{t(a)}$ allows the mortality–age gradient to vary freely over time, so that effects of drug introduction are identified off of relative changes with respect to *age–specific* mortality.[42]

Lastly, we examine SAM in logs to better approximate a normal distribution, yielding an interpretation of mortality *growth* (Appendix Figure E1). We weight regressions using population shares in the year prior to drug introduction (reference period), estimate Equation 1 using ordinary least squares, and cluster standard errors at the demographic group level.[43] For our e–cigarette analysis, we estimate both average e–cigarette effects post–introduction (2007–2019) and post–drug exemption (2011–2019).

Equation 1 is identical for our analysis of smoking prevalence outcomes, with two exceptions: first, we examine smoking outcomes in levels, not logs (Appendix Figure E1 for distribution of smoking prevalence). Changes in smoking prevalence are therefore interpreted as level–changes in the population–level proportion of smokers. Second, we control simply for time fixed effects, ϕ_t, both because our regressions using survey data outcomes contain considerably fewer degrees of freedom, and because trends in smoking rates exhibit far less variability across age (Appendix Figure C1).

4.2 Identification

A necessary condition to interpret $\hat{\beta}$ causally is the parallel trend assumption, which states that the association between smoking–shares and SAM growth (or smoking prevalence) remains constant prior to drug introduction. Since our smoking shares are time-invariant, then SAM growth/decay cannot trend differently across groups that experience different levels of exposure to drug introduction/exemption.

To test for violations of the parallel trend assumption and estimate dynamic treatment effects, we modify Equation 1 to estimate the following non–parametric event study:

[41] A regression of 2005–2006 smoking shares on our demographic dimensions (sex, 5–race, region, urban, age) yields an adjusted R–squared of 71.9%. Dropping age–group as a predictor reduces the model's adjusted R–squared by 38.9ppt; dropping race by 16.4ppt; region by 5.9ppt; urban by 4ppt; sex by 2.7ppt. A regression of logged SAM on our demographic dimensions yields an adjusted R–squared of 98.85%. Dropping age–group as a predictor reduces the model's adjusted R–squared by 87.6ppt; dropping sex by 3.3ppt; race by 3ppt; region by 0.2ppt; urban by 0.1ppt.

[42] $\phi_{t(a)}$ is also equivalent to controlling for (median) year–of–birth by age–of–death fixed effects, allowing each cohort to have it's own mortality—age gradient.

[43] Weighting our regressions by population yields an interpretation of a population–weighted average effect, $\hat{\beta}$.

$$\log(y_{it}) = \alpha + \sum_{k=-m}^{n} \beta_k \cdot \theta_i \cdot 1[t - T = k] + \gamma_i + \phi_{t(a)} + \varepsilon_{it} \tag{2}$$

The main difference from Equation 1 is that we replace our reduced–form effect, β, with a series of m event study lags and n event study leads, where T denotes the year of drug introduction (1984 for NRT drug introduction and 2007 for e–cigarette drug introduction). β_k is the effect of drug introduction on SAM growth in the k'th year relative to drug introduction, with period $T-1$ constrained to zero in order to serve as a reference period.[44] Like before, we scale these leads and lags so that changes are estimated at the population–average level of the smoking–share measure.

Concerning the length of our event study window, we make the following methodological decisions: we estimate Equation 2 using 9 leads and lags wherever possible, 9 being the maximum number of leads we can estimate in our e–cigarette drug exemption analysis (2011–2019) prior to COVID–19. Our e–cigarette drug exemption analysis therefore spans 1997 (9 years before e–cigarette introduction) through 2019 (9 years after e–cigarettte drug exemption). Our NRT drug introduction analysis begins in 1979 (first year of ICD–9) and concludes in 1992 (9 years after 1984). We strictly adhere to this event window unless we are limited by data availability.[45] Lastly, for our regression of smoking prevalence outcomes from BRFSS, we control for group i's share of cell–phone respondents in year t.[46] We further test the sensitivity of our modeling assumptions in Section 5.2.

4.3 Bayesian Shrinkage

As discussed previously, our exposure variable, or DSSM, as well as the other smoking outcomes we examine, contain some degree of measurement error as a consequence of being estimated from a survey. This measurement error is not uniformly distributed across groups because the variance of the smoking–outcome means negatively co–varies with a group's share of the population (and therefore the number of survey respondents within that group).

This type of measurement error has the effect of diluting the true relationship between our smoking–shares and our outcomes. As a consequence, our estimates of the causal effect of drug introduction could be biased and the variance of our estimates would likely be inflated.[47]

[44] For smoking prevalence outcomes, β_k has the same interpretation, but changes are expressed in levels, not growth.

[45] Our NSDUH analysis begins in 2002, the first available year.

[46] BRFSS begins transitioning from land–line to cell–phone survey respondents in 2011, in addition to a change in weighting methodology.

[47] The direction of the bias is ambiguous and depends on whether the mean of our smoking–shares positively or negatively co–vary with their variance. We estimate a weak but significant relationship between our 2005–2006 daily smoking share means and variances: we calculate a pair-wise correlation coefficient of $\rho = 0.22$ ($p < 0.01$), indicating

To ameliorate this concern over measurement error in our survey outcomes, we apply Bayesian shrinkage to our smoking–outcomes and smoking–shares (Morris, 1983).[48] Using the current smoking rate to illustrate, our empirical Bayes procedure works as follows. First, define the current smoking prevalence for group i, estimated from the survey data and weighted using sampling weights, as Y_i, and define the variance of the current smoking prevalence for group i as η_i^2. Define the population–level mean current smoking prevalence (weighted using sample weights) for the adult population as μ and the population variance as σ^2.

We first calculate the group–specific shrinkage factor, λ_i. λ_i is defined as the ratio of the population variance over the sum of the population variance and within–group variance, or $\frac{\sigma^2}{\sigma^2+\eta^2}$. This shrinkage factor is closer to unity for groups featuring current smoking rates estimated with greater precision, and closer to zero for groups with "noisier" estimates of the current smoking rate. Second, we shrink the current smoking prevalence for group i by calculating a weighted average of group i's current smoking rate and the population–level mean current smoking prevalence, weighting by group i's shrinkage factor. The "shrunk" current smoking prevalence, $\hat{Y}_i$, is equal to $\lambda_i \cdot Y_i + (1 - \lambda_i) \cdot \mu$. Bayesian shrinkage therefore adjusts the smoking outcomes of groups that feature greater statistical imprecision (greater within–group variance) closer to the population–mean, while leaving groups with less imprecision relatively unchanged. Unless otherwise noted, we apply Bayesian shrinkage to each outcome we calculate from our survey data sources, including our smoking shares and our per–capita and smoking prevalence outcomes.

4.4 Linear Projection

In estimating Equation 2, we find evidence that the smoking share–to–SAM association increased prior to NRT drug introduction and e–cigarette drug exemption. In other words, the decline in SAM (or decline in smoking prevalence in our survey data analysis) was less steep for groups with higher levels of the smoking share measure, compared to groups with lower levels of the smoking share (Figure 3).

that groups with higher values of the smoking–share are estimated relatively more imprecisely. This evidence suggests that failing to adjust for measurement error in our treatment variable should bias our results towards zero, which we confirm empirically. We show in Figure 5 that our results are robust without applying Bayesian shrinkage.

[48] Empirical Bayes techniques and other regularization methods have frequently been used in the study of crime and education economics (e.g. Weisburst (2024); Aaronson et al. (2007); Chetty et al. (2014)). We further test the sensitivity of our results to bootstrapping and other alternative specifications. We also cluster our standard errors at the group level to account intra–group heteroskedasticity.

We attempt to correct for this by estimating parametric event studies through linear projection (Freyaldenhoven et al., 2019).[49] This and other similar approaches, such as extrapolating group–specific linear trends (Goodman-Bacon, 2021) or the use of control–functions (Wooldridge, 2015) have been proposed and implemented successfully in other contexts.[50] For example, Dobkin et al. (2018) estimates a parametric event study by estimating unit–specific pre–period linear trends in the outcome variable, and then calculating an "adjusted" outcome, which is the deviation in the unadjusted outcome from the extrapolated, pre–period linear trend. A working paper by Finkelstein et al. (2024) likewise uses the control–function approach as part of their event–study analyses.

We implement our linear projection procedure by estimating the following reduced–form model in pre–period years:

$$\log(y_{it}) = \alpha + \Pi \cdot \theta_i \cdot t + \gamma_i + \phi_{t(a)} + \upsilon_{it}. \tag{3}$$

Above, we estimate our reduced–form model from Equation 1 for years prior to treatment ($t - T \leq 0$) and estimate the linear trend in the SAM growth–smoking share association, denoted by $\hat{\Pi}$. $\hat{\Pi}$ characterizes the magnitude and direction of the pre–trend violation, with positive values indicating a positive correlation between SAM growth (or smoking prevalence outcome) and the smoking share measure, and negative values indicating a negative relationship.

The residuals from Equation 3, $\hat{u}_{it}$, are interpreted as deviations in the SAM growth–smoking share association from the linear pre-period trend, extrapolated into the years following drug introduction (conditional on our fixed effects). We estimate our "parametric" reduced-form model and event study by re–estimating Equations 1 and 2 on these residuals:

$$\hat{\upsilon}_{it} = \alpha + \beta \cdot \theta_i + \gamma_i + \phi_{t(a)} + \varepsilon_{it}. \tag{4}$$

In the parametric model above, $\hat{\upsilon}_{it}$ is simply the difference between our observed outcome, $\log(y_{it})$, and our predicted, or *projected*, outcome, $\hat{\log}(y_{it})$. $\hat{\beta}$ is now interpreted as the before–after change in the smoking–share SAM–growth association from the *predicted* trend. Our event study coefficients likewise yield an interpretation of *deviations* in the SAM growth–smoking share association from their (linear) predicted change.

[49] See Freyaldenhoven et al. (2019) for a thorough discussion of pre–trends in event studies and other approaches besides linear projection.

[50] We show in Figure 5.2 that our main results are robust when using group–specific linear trends from (Goodman-Bacon, 2021).

Unless otherwise noted, we provide results from the parametric specification throughout this paper. The new identifying assumption for this model is that deviations from the *predicted* trend in the SAM growth–smoking share association remain constant prior to drug introduction/exemption. Groups with greater smoking–share levels are allowed to exhibit different rates of SAM growth/decay from groups with lower smoking–share levels, as long as those differences–in–growth are roughly *linear*. The effect of drug introduction or exemption, $\hat{\beta}$, is identified from *relative* changes in the smoking–share–SAM growth association (relative to the predicted change).[51]

In implementing this linear projection approach from Freyaldenhoven et al. (2019), we introduce an additional assumption that the predicted change in the SAM growth–smoking share association from the pre–period window is a suitable counterfactual for how SAM growth would have *continued* to trend in the absence of drug introduction/exemption. By relying upon nearly a decade of pre–period years for our e–cigarette drug exemption analysis (5 years for our NRT drug introduction analysis after ICD–9), we attempt to bolster this assumption against short–run or other sporadic changes to mortality that may result from temporary shocks. Nonetheless, this assumption may be more reasonable in years immediately following the treatment event than in years further away; as such, short– and medium–run estimates should be interpreted as more reliable than long–run estimates.[52]

5 Results

In Section 5.1, we present parametric event study results of the effect of NRT drug introduction and e–cigarette drug introduction/exemption on SAM. In Section 5.2, we probe the robustness of results to our modeling assumptions. In Section 5.3, we estimate treatment heterogeneity by sub–groups for e–cigarette drug introduction/exemption and explore heterogeneity by categories of SAM . In Section 5.4, we study how e–cigarette drug introduction/exemption affects smoking behaviors. In Section 5.5, we contrast our approach with that of Allcott and Rafkin (2022). In Section 5.6, we discuss alternative explanations for our results. In Section 5.7, we discuss our e–cigarette introduction and drug exemption results. In Section 5.8, we present the results of our social surplus analysis.

We discuss our calculations of life years and smoking years in Appendix Section 6. We present our consumer surplus calculations in further detail in Appendix Section G and in Tables G1 and G2.

[51]The benefit of this approach versus using group-specific linear time trends across the full time period is that we do not fit the trends using any effect of drug introduction in the post-period, which could otherwise lead to attenuation bias. We also minimize the risk of over–fitting, which may occur through the use of group-specific trends.

[52]We explore an alternative specification using a pre–period unit specific trend (Goodman-Bacon, 2021) in Section I, Figure I3.

5.1 Main Findings

Figure 4 shows the results of our parametric event studies for SAM. E–cigarette drug exemption reduced SAM by 9.91% (SE = 1.15%) on average each year from 2011–2019, or roughly 75,000 life–years (SE = 8,696 LY's) gained per year after 2011. This equates to an average gain of $7 billion in consumer surplus each year after 2011. The introduction of e–cigarettes in 2007 also led to a 7.74% (SE = 1.39%) average reduction in SAM each year after 2007. Further, about 88% of the total life–years gained after e–cigarette introduction in 2007 are gained after e–cigarettes became exempt from drug regulation in 2011.

We find no statistically significant effect on SAM following NRT introduction in 1984, nor do we find any descriptive evidence of a break in SAM trends after 1984.[53] The NRT introduction coefficient is approximately 13% of the e–cigarette coefficient size, and based upon the 95% confidence intervals (CI) we can rule out NRT introduction reducing SAM rates by more than 2.9% per year (6.5k LY's gained per year). Based upon the 95%–CI lower bounds of our NRT and e–cigarette estimates, we can rule out NRT drug introduction having more than 1/3 of the effect of e–cigarette drug exemption on SAM rates, or more than 12% of the effect of e–cigarette drug exemption on life–years gained.[54]

5.2 Robustness & Sensitivity

In Figure 5, we present a summary of percent changes in SAM and the corresponding average number of life–years gained/lost per year in 1,000's for our robustness specifications.[55] Our robustness/sensitivity specifications include an unweighted event study regression, the use of a 2–year daily smoking share measure from earlier in time than the 2 years immediately preceding

[53] In Appendix H1, we also examine the effect of the 1996 introduction of over-the-counter NRT on SAM, which coincided with a 152% increase in NRT sales (Shiffman et al., 1997). We also do not find evidence that NRT becoming OTC reduced SAM.

[54] Our e–cigarette introduction estimate is significantly higher than our NRT drug introduction estimate ($p < 0.01$) as is our e–cigarette drug exemption coefficient ($p < 0.001$).

[55] We show parametric event study results for these robustness specifications separately for NRT drug introduction and e–cigarette introduction/drug exemption in Appendix I. We present results for e–cigarette drug exemption after 2011 in our summary figures.

drug introduction,[56] using a different method to account for pre-trends,[57] nationalizing demographic cohorts by dropping regional categories of Census region and urban/rural status,[58] bootstrapping standard errors, adding additional year–by–demographic dimension (sex, region, urban status, race) fixed effects, a specification using unshrunk daily smoking shares, and a specification including all demographic groups aged 30 and older.

Figure 5 shows a fairly stable pattern of results across our robustness specifications for e–cigarettes. For e–cigarette drug exemption, our estimates of percent reductions in SAM are consistently significant and similar in magnitude, and our estimates of life–years gained are quite comparable. The similar results from the additional fixed–effect specification is reassuring, since this specification effectively controls for unobserved shocks to SAM that vary across sex, race, urban status, and census regions. That the result changes little suggests that these sources of bias are not a major threat to validity. Our national specifications (both specific rates and adjusted rates) feature slightly larger reductions in SAM and life–years gained compared to our main specification. However, to the extent migration across urban status or census region occurs after 2007, this does not appear to result in any meaningful impact.[59]

For NRT drug introduction, percent changes in SAM are clustered around zero, including some positive–signed. None of our robustness results have any serious impact on our interpretation that NRT drug introduction has either no measurable effect on SAM or an effect with a lower bound that is a fraction of our estimated e–cigarette drug exemption effect. Having shown little effect of NRT introduction thus far, in the following sections we draw our attention to e–cigarette introduction/exemption.

[56]The use of smoking share measures from earlier in time is designed to reduce the potential impact from mean–reversion, which does not appear to play any significant role in our e–cigarette analysis. If smoking rates from 2005–2006 are temporarily higher–than typical levels or lower–than typical levels, for example due to some unobserved idiosyncratic shock, then mechanically, SAM rate growth should decrease once smoking rates stabilize over time. This is because the smoking–share SAM–growth association for groups with higher–than typical smoking–shares is biased upward (higher smoking–share than typical) and the association for groups with lower–than typical smoking–shares is biased downward (lower smoking–share than typical). Mean–reversion could therefore explain why the average association between smoking–shares and SAM–rate growth would decline after 2007. By using an alternative share measure estimated earlier in time, from 1997–1998, and finding virtually identical results, this does not suggest mean reversion to be a major source of bias.

[57]In the Goodman-Bacon (2021) time trend specification, we adapt Equation 3 to estimate *unit*–specific trends in our outcome (as opposed to a linear trend in our event study leads) using pre–period observations in our event study window.

[58]For adjusted rates, we weight each national group by the 2006 population share of census region and urban status.

[59]Our specific rate results allow population by region and urbanicity to vary freely over time when calculating national–level SAM rates, while our adjusted rate results hold the share of population by region and urbanicity fixed at 2007 levels.

5.3 Heterogeneity

5.3.1 Heterogeneity by Demographics

In Figure 6, we examine heterogeneity by demographic characteristics and 15–year age groups.[60] We find that e–cigarette drug exemption had a fairly widespread effect on SAM across sub–groups in the population, with each sub–group's share of total life–years gained strongly correlated with that group's share of SAM prior to 2007, indicating that drug exemption generated proportional public health gains. In interpreting Figure 6, we consider both the percent reductions in SAM, as well as the share of the total life year gains in relation to each sub–group's baseline share of total SAM.

By sex, we estimate a 9.7% reduction in male SAM and an 11.8% reduction in female SAM, which are not statistically different from each other ($p = 0.35$). Men had a higher mean DSSM from 2005–2006 (15.4% for men) compared to women (13.3% for women), while men constituted roughly 60% of all SAM deaths in 2006 and roughly half of life–years gained after 2011.

By race, we observe significant reductions across all sub–groups except for Asian/Pacific Islanders and Hispanics.[61] White non–Hispanics constitute nearly 90% of life–years gained after 2011, followed by Black non–Hispanics, with these two groups collectively making up nearly 93% of all SAM in 2006. American Indians had the highest mean DSSM from 2005–2006 at 19.6%, and the highest rate of ever–vape use from 2016–2018 at 22.8%.

By urban status, we find that e–cigarette drug exemption primarily benefited urban residents, who constituted 80% of the life–year gains and likewise nearly 80% of all SAM deaths in 2006. Our rural and urban percent SAM reductions are also not statistically different.

By age, we find similar percent reductions in SAM mortality across 15–year age groups. Age groups 30–44, 45–64, and 65–79 respectively made up 4%, 36%, and 60% of total life–years gained after e–cigarette drug exemption. These life year gains by age group are roughly proportional to the SAM mortality burden prior to e–cigarette introduction: age groups 30–44, 45–64, and 65–79 respectively made up 3%, 34%, and 63% of total SAM in 2006.

In Appendix Figure J4, we document a significant reduction in SAM deaths among ages 18-29, but the average annual life–years gained of approximately 400 life–years among youth through 2019 is less than 0.5% of our estimate from our main sample of ages 30–79 (75,000 average annual life–years gained).

[60] We present accompanying parametric event–studies in Appendix J.

[61] Asian/Pacific Islanders and Hispanics contain the lowest mean DSSM at 6.5% and 9.8%, respectively. These two groups also have the lowest rates of ever–vape use from 2016–2018 at 15.4% and 15.6%, respectively.

5.3.2 Heterogeneity by Cause of Death

In Figure 7, we present estimates of the impact of e–cigarette drug exemption by categories of SAM: cancers, cardiovascular diseases, respiratory conditions, and metabolic disorders.[62] All 4 sub–categories are mutually exclusive, and like before, we weight each cause of death by sex–specific SAF's.

In Figure 7, we find that cancer SAM makes up the majority of SAM reductions after 2011, followed by respiratory, cardiovascular, and metabolic SAM. We also find that respiratory causes constitute a greater–than–predicted share of life–years gained (compared to their share of SAM deaths in 2006), while cardiovascular causes make up a smaller–than–predicted share of life–years gained. Cancers and metabolic–cause shares are roughly proportional to their baseline mortality burden of SAM deaths.

We estimate that respiratory–SAM was reduced by 21.5% (31,000 annual life–years gained) on average each year from 2011–2019; for cancer–SAM, we estimate a 12.5% reduction on average (53,000 annual life–years gained); for cardiovascular–SAM, we estimate a 4.6% reduction (8,200 annual life–years gained); for metabolic–SAM, we estimate a 4.3% reduction (1,400 annual life–years gained), but this last estimate is not statistically significant.

We offer several possibilities for our pattern of results. First, we observe that both respiratory and cancer SAM, which comprise a disproportionate share of life–years gained, have a stronger association with smoking.[63] Second, there is abundant evidence of the unique harms of carcinogens from combustible tobacco on respiratory conditions and cancer formation (e.g. Hecht, 1999).[64] In contrast, the association between smoking and cardiovascular SAM is driven primarily by the pharmacologic effects of nicotine (Benowitz and Burbank, 2016): substitution of combustible tobacco for e–cigarettes (containing nicotine) may therefore offer more modest risk reductions in contrast to other causes of death.[65]

[62] Refer to Table D1 for category definitions. We present accompanying parametric event studies in Appendix Section K.

[63] In 2006, 49.8% of cancer deaths were attributable to smoking, while 48.4% of respiratory deaths were attributable to smoking. In contrast, only 18.5% of cardiovascular deaths, and 15% of metabolic deaths, were attributable to smoking.

[64] Peto et al. (2000) finds that quitting smoking before middle-age reduces tobacco–related cancer risk by over 90%. Additionally, Hurt et al. (2000) finds that one in three carcinogenic biomarkers decreased significantly within 24 weeks of reduced smoking.

[65] Reductions in overall mortality could consist of reductions in SAM, plus increases in SAM from causes of death that are also affected by e–cigarette use (e.g., cardiovascular causes of death). Our estimates should therefore be interpreted as *net* reductions.

5.3.3 Heterogeneity by Acute– and Chronic– Onset of Risk Reduction

In classifying smoking–attributable causes by onset of risk reduction, we look primarily to Godtfredsen et al. (2002), who estimate cause–specific hazard ratios among former smokers between 1967 and 1988 for nearly 20,000 persons in three prospective cohort studies.[66] Cause–specific survival increases more rapidly after quitting smoking among certain "acute–onset" causes: these include most cardiovascular causes, certain types of cancers such as oral and lung cancers, respiratory causes such as chronic obstructive pulmonary disease, and diabetes. In contrast, other causes take longer before cause–specific survival improves: these "chronic–onset" causes include most cancers and certain types of heart and arterial diseases.

In Figure K2, we estimate the impact of e–cigarette drug exemption on acute–onset and chronic–onset of risk reduction causes.[67] We estimate a reduction in acute–onset cause SAM of 12.2% on average each year from 2011–2019, or 76,100 annual life–years on average. For chronic–onset causes, we estimate a 4.7% reduction on average from 2011–2019, or 5,500 annual life–years gained on average.

Acute–onset of risk reduction causes comprise the majority of life–year gains, roughly 93% from 2011–2019, while chronic–onset causes comprise only 7% of life–year gains. Acute–onset causes account for only 85% of SAM deaths in 2006, while chronic–onset causes account for the remaining 15%. Our pattern of results suggest that e–cigarette drug exemption had the greatest impact on causes of death with stronger associations to smoking, and causes of death in which smoking–cessation generates relatively quicker risk–reductions.

5.4 Mechanisms

We now apply our empirical strategy to estimate whether e–cigarette introduction/drug exemption reduces cigarette smoking, which we hypothesize is the primary channel through which e–cigarette introduction/exemption reduces SAM. To do this, we use the same modelling approach, and replace our SAM outcome with current and daily smoking prevalence and per–capita measures of past–month

[66] Godtfredsen et al. (2002) conduct 5–10 year follow-ups of individuals who lower their heavy smoking intensity by at least 50% and those who quit smoking, compared to those who remain heavy smokers. The authors examine nearly 20,000 subjects from three prospective cohort studies: the Copenhagen City Heart Study, the Glostrup Population Studies, and the Copenhagen Male Study. After conducting a record linkage using death certificates, the authors estimate adjusted hazard ratios (HR's) for former smokers by sub–category of SAM. For cardiovascular causes, they estimate a HR of 0.88 (95% CI $[0.68, 1.15]$); for tobacco–related cancers, an HR of 0.36 [0.22, 0.59]; for respiratory causes, an HR of 0.77 [0.44, 1.35].

[67] Refer to Table D1 for category definitions. We present accompanying parametric event studies in Appendix Section K. Causes of death are weighted by smoking–attributable fractions.

average days smoked and average cigarettes smoked per day (NHIS and NSDUH only).For consistency, we construct our daily smoking measure from the data source whose outcome we examine. We control for the share of cell–phone respondents in our BRFSS regressions.

Similar to our calculation of average annual life–years gained/lost (Equation 7), we calculate average annual smoking years gained/reduced from Equation 9.[68]

5.4.1 Smoking Prevalence

In Figure 8, we present our event study results for current smoking and daily smoking prevalence in each of our 3 data sources. For the effects of e–cigarette drug exemption on current smoking prevalence, we estimate a 3.9ppt reduction from BRFSS (SE = 0.19ppt), a 4.3ppt reduction from NHIS (SE = 0.26ppt), and a 4.9ppt reduction from NSDUH (SE = 0.38ppt). For daily smoking prevalence, we estimate a 3.1ppt reduction from BRFSS (SE = 0.15ppt), a 4.2ppt reduction from NHIS (SE = 0.25ppt), and a 6.6ppt reduction from NSDUH (SE = 0.30ppt). Our estimate of average smoking–years reduced per year varies across data sources: for reductions in current smoking, our estimates range from 6.4 million annual smoking–years reduced from 2011–2019 in BRFSS to 8.5 million in NSDUH, and a similar pattern for smoking–years reduced from reductions in daily smoking.

The only survey data source in which the reduction in current smoking and daily smoking are statistically different from each other is NSDUH. For NSDUH, we estimate a significantly larger reduction in daily smoking rates (6.6ppt reduction) than the reduction in current smoking rates (4.9ppt reduction), indicating an average *increase* in the light smoking rate of 1.7ppt. NSDUH reports the lowest share of daily smoking prevalence among current smokers of all 3 data sources, which may explain this difference.[69]

Our BRFSS event studies are slightly attenuated from the 2011 sample design change, which results in an upward level–shift in our event study lags in year 2011. Our results are attenuated somewhat as a result so we interpret BRFSS results cautiously. We are reassured, however, that our BRFSS event studies otherwise show similar level–shifts and changes in trends to the other 2 surveys.

[68]We discuss details of our smoking–year formula in Appendix Section F. We provide additional information on the surveys and outcomes in Appendix Section D.2 and Appendix Table D2.

[69]In both NHIS and BRFSS, the share of current smokers that smoke less than daily is consistently below 4% of current smokers. For NSDUH, this share is closer to 15% on average.

5.4.2 Smoking Intensity & Frequency

In Figure 9, we document a consistently significant pattern of reduced smoking intensity and cigarettes smoked. For NHIS, we document a reduction of 0.4 cigarettes per day per capita (SE = 0.06) after 2011, or 12 cigarettes per month per capita (SE = 1.8). For NSDUH, we document a larger reduction of 0.9 cigarettes per day per capita (SE = 0.09) after 2011, or 27 cigarettes per month per capita (SE = 2.7). We estimate an average reduction of 2 days smoked in the past month per capita (SE = 0.12) in NHIS and 1.6 fewer days smoked per capita (SE = 0.11) in NSDUH, after e–cigarette drug exemption.

Our estimates are equivalent to a population–level reduction of 65 million cigarettes smoked per day in NHIS (158 million fewer cigarettes in NSDUH) and a population–level reduction of 194 million days smoked in the past month in NHIS (270 million fewer days smoked in the past month in NSDUH).[70]

In summary, we document consistent evidence of e–cigarette drug introduction/exemption reducing smoking. Our preferred estimates indicate drug exemption reduced current smoking by 4.3ppt and daily smoking by 4.2ppt.[71]

5.5 Comparison to Allcott and Rafkin (2022)

Our main finding is that e–cigarette introduction and drug exemption reduces smoking among adult demographic groups with higher baseline smoking rates in 2005–2006. Allcott and Rafkin (2022), in contrast, use vaping rates in 2016–2018 for their share measure.[72] This decision has important implications. In Appendix L, we re-estimate our specification using the same 2016–2018 e–cigarette use share measure, and find null results for smoking and SAM, which is consistent with Allcott and Rafkin (2022). This differs from our previously-shown results finding reductions in smoking and SAM using 2005–2006 smoking rates for the share measure.

Allcott and Rafkin (2022)'s e–cigarette use share measure directly measures e–cigarette use using data from 2016–2018, but heterogeneity could exist in terms of why different groups of people use e–cigarettes. In particular, some groups, such as the elderly, may be more likely to use e–cigarettes

[70] We estimate larger magnitude reductions in NSDUH likely due to larger population counts (we use ages 30 and older in NSDUH due to coarser age-bins).

[71] Reductions in smoking–prevalence from NHIS generally lie between those from BRFSS and those from NSDUH. We use our estimates from NHIS as our preferred set of results, as current and daily smoking rates are more comparable between NHIS and BRFSS (the latter contains larger sample sizes). In addition, we prefer NHIS over BRFSS due to the 2011 sampling change in BRFSS, while NHIS also contains a greater pre-period compared to NSDUH.

[72] E–cigarette questions were not asked regularly in surveys until 2016.

for health-improving reasons than the non-elderly that use e–cigarette for mostly consumption. This heterogeneity in reasons for e–cigarette use could lead to e–cigarettes being particularly important for reducing smoking and SAM among some groups even if e–cigarette use rates are low. Our approach of using a smoking share measure addresses this issue by modeling the potential for e–cigarettes to disproportionately benefit some groups over others based on smoking rates from before the e–cigarette marketplace emerged.[73] Our smoking share measure is moderately positively correlated with 2016–2018 current e–cigarette use rates ($\rho = 0.54$, $p < 0.001$).

Another significant difference between our studies is the use of 2007 or 2013 as the introduction date. Allcott and Rafkin (2022) use 2013 since this is the year e–cigarettes began to be sold in sizable numbers in Nielsen–tracked stores.[74] However, a sizable number of people had purchased e–cigarettes from non-traditional retail stores and used these prior to 2013.[75] In contrast, we proxy e–cigarette access based on natural experiments of e–cigarette introduction in 2007 and drug exemption in 2011, with the latter being a largely unexpected event (based on legislation and subsequent court decisions). This difference has little impact on the conclusion, though, because we show in Appendix M that using a 2013 introduction date and our standard daily smoking share results in statistically significant reductions in smoking and SAM, though attenuated compared to using earlier years.[76]

5.6 Competing Explanations

Many features of our findings support a causal link between e–cigarette drug exemption and SAM reductions. For example, we show large reductions in smoking among the same people experiencing reductions in SAM. Additionally, we find large effects of e–cigarette drug exemption on respiratory and cancer SAM, with causes of death that generate the most rapid onset of risk-reduction from smoking-cessation, so called "acute–onset" causes, consisting of 93% of the life–year gains.

[73] Our approach also allows e–cigarettes to benefit (or harm) demographic groups through spillover effects separate from direct use of e–cigarettes. For example, if a husband and wife both smoke, and if a wife is more likely to try e–cigarettes and quit, then the husband has increased odds of quitting as well even if unaided by e–cigarettes. Our estimates incorporate such spillover effects. Our use of past values of our outcome as a share measure follows other studies, such as Alpert et al. (2018) and Callison and Pesko (2022)'s Table 6.

[74] The authors also show their results are insensitive to using 2012 as the introduction date.

[75] Tracked-stores do not include online stores, vape shops, or smaller retailers like bodegas and mall kiosks. Nationally representative surveys found that 4.7% of high school students had tried e–cigarettes by spring 2011 (Corey et al., 2013) and that 12% of U.S. adult smokers had tried e–cigarettes by 2012 (Shi et al., 2016). This suggests a sizable number of people obtained e–cigarettes from non–traditional retailers (e.g., online, vape shops) prior to 2013.

[76] Two other differences between their analysis and ours that do not appear consequential: 1) There are some differences in our demographic groups. We use 10 age categories instead of 3. We also do not use education and income, since this information is not available on death certificates. 2) We use a pre–trend adjustment procedure from Freyaldenhoven et al. (2019) instead of using a trend fit across both the pre–period and post–periods. Goodman-Bacon (2021) warns that the latter can cause the model to throw out useful variation from the post-period.

As is the case with any DD model, the main threat to our identification is the potential for other concurrent changes correlated with treatment status (e.g., smoking rates at baseline) and outcomes (e.g., SAM). For several reasons, we believe that e–cigarettes are the primary explanation for the effects we observe, though we can never fully rule out the influence of competing effects. In this section we describe potential competing explanations and rule these out as major influencers one–by–one.

Could concurrent new availability of non–e–cigarette smoking cessation products explain our findings? E–cigarette introduction in 2007 was near in timing to Chantix (varenicline) introduction in 2006 and mini–lozenge NRT introduction in 2009. This does not seem likely to drive our findings for several reasons. First, Tobacco Use Supplement of the Current Population Survey (TUS–CPS) data finds only a small increase in prescription pill use and lozenge use between 2003 and 2010-2011 (5.7pp increase and 1.1pp increase respectively)[77] for current and recent former smokers, which is small compared to 30.2% of current/former smokers that used e–cigarettes in their most recent quit attempt in 2014-2015.[78] Second, Google Trends search volume shown in Figure 2 indicates some modest increase in prescription pill search volume in 2006 and 2008, before falling to baseline low levels comparable to NRT, which exhibited no movement in search volume during the time period. E–cigarette search volume meanwhile clearly overtakes NRT and prescription pill search volume in 2010, which is also the year that SAM began its steady decline. Third, in early 2014, GlaxoSmithKline paused production and issued a recall of Nicorette lozenges for quality reasons (Frankel, 2015), leading to a significant shortage of these drugs, and since we do not observe evidence of SAM reductions stagnating on or shortly after 2014 this suggests lozenges are incapable of explaining the reductions in SAM we document. Finally, our effects cannot be accounted for by these competing drugs (Chantix/varenicline and mini–lozenges) being more effective. A recent Cochrane review finds that e–cigarettes are at least as effective as varenicline and more effective than NRT for planned quit attempts (Lindson et al., 2024), and e–cigarettes meanwhile may also reduce smoking through unplanned quit attempts as well (Kasza et al., 2021).

Could concurrent improvements in medical technology affecting SAM explain our findings? We find dispersion in the types of deaths averted that is consistent with smoking cessation story affecting many types of SAM rather than a story of a single medical intervention that reduces a specific type of SAM.

[77] Based on author calculations of TUS–CPS data.

[78] The TUS–CPS is generally collected every three to four years. Smoking cessation pill and lozenge questions were not collected in 2006. E–cigarette use questions for current/former smokers was first collected in 2014-2015.

Could state or national policies or economic events drive our findings? Our identification strategy reduces the potential for state-specific policies or economic factors to influence our findings because our analysis is performed at the demographic group level. Further, our findings are unchanged whether we use incorporate Census region or not in creating the demographic cohorts. Further, our results are similar when we use population weights or not, thus reducing concern over a correlation between demographic group presence in a large state and an omitted policy from that large state. California, importantly, did not increase their cigarette taxes between 2000 to 2016.

2009 was an active year federally with passage of the TCA and a cigarette tax increase from $0.33 to $1.01 a pack. We do not believe this to be a major source of bias, however. Our event studies for smoking show evidence of smoking–prevalence and intensity reductions immediately after e–cigarette drug introduction in 2007 (Figures 8 and 9), indicating both a level–shift and downward shift in trends. The 2009 tax increase did not appear to accelerate what was already set in motion by drug introduction. A doubling of cigarette taxes in 1983, close in timing to the 1984 NRT drug introduction, did not lead to reductions in SAM (Figure 4).

5.7 Unique Effects of E–cigarette Introduction and Exemption

We find evidence that patents began increasing in the period of time after e–cigarette introduction, before drug exemption, in the United States relative to Australia (Figure 1). However, patents increased approximately 10-fold in the drug exemption period. In terms of smoking, we find evidence that approximately half of the reduction in smoking occurred in the transition period, and the other half after e–cigarette exemption (Figures 8 and 9). This suggests that early e–cigarettes were effective and subsequent innovation made them even more effective. Had the FDA won authority to regulate e–cigarettes as drugs, cleared the marketplace of e–cigarettes, and forced e–cigarette companies to start the drug approval process, we speculate that these early gains would have disappeared. As such, we believe our choice of using a pre-period before e–cigarette introduction to evaluate the effect of drug exemption is the best choice, as few if any of the e–cigarettes on the market in the early years would have likely survived the drug approval process, and certainly not without significant delay.

It is difficult to disentangle the effect of e–cigarette introduction and drug exemption on SAM given the lagged nature through which smoking affects SAM (Figure 4). We find the first statistically significant reductions in smoking in 2007, and the first statistically significant reductions in SAM in 2011. This suggests approximately a four-year lag.

5.8 Social Surplus

In our consumer surplus calculations, we utilize our estimates of cumulative life–years gained/lost from Equation 6. We discuss our procedure for social surplus in Appendix Section G and present tables of our consumer surplus calculations in Appendix Tables G1 and G2.

For e–cigarette drug introduction (2007–2019), we calculate the net cumulative change in life–years as 760,621 life–years *gained*, with a 95% confidence interval ranging from 434,968 life–years to 1.09 million life–years gained. We estimate an annual average change of 58,509 life–years gained, with a 95% confidence interval of 33,459 life–years to 83,559 life–years gained.

From e–cigarette drug introduction, we calculate a total consumer surplus of $73.55 billion [$39.2b, $107.9b] from 2007–2019, for an average consumer surplus of $5.7 billion per year [$3.0b, $8.3b]. We calculate a total producer surplus of $14.29 billion, for an average of $1.10b per year. We calculate a total social surplus of $87.84 billion from 2007–2019, or $6.75 billion annually, of which consumer surplus constitutes 84% of social surplus.

For e–cigarette drug exemption (2011–2019), we calculate the net cumulative change in life–years as 669,828 life–years *gained*, with a 95% confidence interval ranging from 499,172 life–years gained to 840,483 life–years gained. We estimate an annual average change of 74,425 life–years gained, with a 95% confidence interval ranging from 55,463 life–years to 93,387 life–years gained.

For e–cigarette drug exemption, we calculate a total consumer surplus of $63.66 billion [$47.0b, $80.3b] from 2011–2019, for an average consumer surplus of $7.1 billion per year [$5.2b, $8.9b]. We estimate a total producer surplus of $14.29 billion, for an average of $1.59 in producer surplus per year. We calculate a total social surplus of $77.94 billion from 2011–2019, or $8.66 billion annually, of which consumer surplus constitutes 82% of social surplus. We calculate that the social surplus generated after 2011 constitutes 89% of the total social surplus generated after e–cigarette introduction in 2007.[79]

Comparing our estimates to the Philipson and Jena (2006), who find that HIV/AIDS therapies introduced in the 1980's generated roughly $1.39 trillion in social surplus ($1.33 trillion in consumer surplus and $63 billion in producer surplus), we find that consumers captured a slightly smaller share of social surplus following e–cigarette drug exemption, roughly 82% compared to a share of 96% from Philipson and Jena (2006). The authors also estimate 200,000 annual life–years gained of HIV/AIDS

[79]For NRT drug introduction (1984–1992), we calculate the net cumulative change in life–years as 92,746 life–years *gained*, with a 95% confidence interval from 148,049 life–years lost to 333,542 life–years gained, or 10,305 life–years gained per year. We calculate a total consumer surplus of $19.74 billion [-$33.1b, $72.6b] from 1984–1992, for an average consumer surplus generated each year of $2.19 billion [-$3.7b, $8.1b].

drugs for all HIV–infected individuals through 2000, whereas we estimate 74,426 annual life–years gained from 2011–2019, suggesting that e–cigarettes have approximately 1/3 the population health benefit of early HIV/AIDS drugs.

6 Conclusion

Our study provides evidence of the effect of FDA drug regulation by taking advantage of e–cigarettes' unprecedented and unexpected exemption from FDA drug regulation. Our results join other literature in showing how government regulation can increase mortality overall (Broughel and Chambers, 2022; Blalock et al., 2007; Neidell et al., 2021; Jarvis et al., 2022; Alpert et al., 2018). Our study is perhaps most similar to Alpert et al. (2018) finding that the FDA's ban on crushable Oxycotin increased mortality; however, in our case we estimate effects of FDA drug regulation writ large instead of effects of a change in a specific FDA guideline.

In particular, our paper makes several contributions. First, we show that e–cigarette innovation sharply increased when e–cigarettes were exempted from drug regulation. In particular, e–cigarette patent applications increased sharply in the United States between 2011–2019, compared to e–cigarette applications in Australia where e–cigarettes have been regulated as drugs, and compared to applications for non–e–cigarette smoking cessation drugs in both countries. These non–e–cigarette smoking cessation products may have experienced greater innovation, like e–cigarettes, if not regulated as drugs by the FDA. E–cigarettes, meanwhile, experienced so much innovation that many people do not associate them as drugs today. It is easy to forget that at their introduction point, e–cigarettes were similar in key ways to little-used FDA–approved nicotine inhalers (Bullen et al., 2010).[80]

Our second major contribution is to identify effects of e–cigarette introduction and drug exemption on SAM. We find that e–cigarette drug exemption reduced SAM by approximately 10%. As a point of comparison Friedson et al. (2023) previously found that a $1 increase in cigarette taxes experienced between the ages of 14–17 years of age reduces later–life all–cause mortality by 4%.[81] Further, we show evidence that the link between post–drug exemption e–cigarettes and SAM is largest for those causes of death with the most immediate risk–reduction upon quitting smoking, and for cancers and respiratory causes that are closely linked to smoking (US Department of Health

[80] Both were unflavored; 16 mg/ml for the Ruyan v8 versus 10 mg/ml for nicotine inhalers.

[81] On average, roughly 1 in 5 all–cause deaths are caused by smoking in our sample. Scaling our results produces an average reduction in all–cause mortality of around 2% per year.

and Human Services, 2020). We also show that the first FDA drug–approved NRT product, 2–mg nicotine gum, did not reduce SAM in the same way upon its introduction in 1984.

Our third major contribution is to provide evidence from three separate surveys that e–cigarettes reduced smoking. Our median estimates document a reduction of current smoking by 4.3ppt and daily smoking by 4.2ppt.

Our findings present a data point in support of FDA drug deregulation and support the notion, advocated by Philipson and Sun (2008), that product liability law is sufficient for a well-functioning drug marketplace. It would be useful to have further evaluation opportunities to observe if the socially beneficial effects of drug deregulation we estimate within the smoking cessation therapeutic drug class are generalizable to effects for other therapeutic drug classes, perhaps through pilot initiatives that release select poorly–performing therapeutic drug classes from drug regulation. Expanded access and right–to–try laws already allow patients with serious conditions access to unapproved experimental drugs under certain conditions (U.S. Food and Drug Administration, 2022). Our results support increasing the use of these and similar initiatives, and implementing new initiatives that allow the release of under–performing therapeutic drug classes.[82] Such a law change could also have a secondary benefit of reducing the extent to which demand exists without legal supply, which incentivizes the creation of illicit markets.[83]

Alternative market–based approaches to drug regulation could maintain many of the benefits of the current FDA drug approval process in terms of drug safety/effectiveness, while allowing for faster action and more innovation. For example, firms could be allowed to operate outside of the drug approval process provided they carry sufficiently large product liability insurance. Such an approach would effectively make the drug firm's insurer the new regulator, though likely a faster and more flexible one.

Our study also makes an important contribution to tobacco product regulation by showing that e–cigarette introduction sizably improved public health. We estimate that e–cigarette introduction and subsequent drug exemption saved 760,000 life years through 2019, which translates to roughly 1

[82]The FDA does have four accelerated drug approval processes: Fast Track, Breakthrough Therapy, Accelerated Approval, and Priority Review. All tracks are aimed at expediting the approval of drugs for serious conditions. However, these programs all retain some version of the drug approval process, which may reduce benefits compared to opportunities to access drugs outside of this process. Similar to our findings from e–cigarette drug exemption, the Breakthrough Therapy program has been linked to a surge in innovation (Hermosilla, 2024).

[83]As an illustrative example, clustered regularly interspaced short palindromic repeats (CRISPR) gene–editing technology was awarded the 2020 Noble Prize in chemistry and has been heralded as technology that "may make the dream of curing inherited diseases come true" (The Nobel Prize Organization, 2020). Despite the foundation for modern–day CRISPR research being established in 2012 (Jinek et al., 2012), the FDA did not approve a CRISPR drug for human subjects trials until 2019 (Saey, 2019). During this period of time a community of biohackers formed and began to sell do–it–yourself CRISPR kits (Schultz, 2018).

life–year gained for every 100 fewer smoking–years, or a smoking–year to life–year pass–through of 1%. Our results complement microsimulation modeling predicting that a hypothetical vape ban in 2019 would lead to 11 million fewer life–years over the life course for individuals age 12 and up in 2019 (Hui, 2024), and a second predicting that e–cigarettes will save nearly 40 million life–years between 2013 and 2060 (Levy et al., 2021). While the prediction that e–cigarettes will save lives is not new, we are the first to show ex–post that e–cigarettes did in fact do so.

The FDA formally asserted regulatory authority over e–cigarettes in August 2016 by "deeming" them to be tobacco products under the TCA, and e–cigarette companies were required to submit PMTAs in Sept. 2020. For this, the companies need to show the FDA that their products are "appropriate for the protection of public health," which means that these companies need to demonstrate that the population–level health impact of these products is on net positive. Our study finds that e–cigarettes are providing important public health benefits by reducing SAM; as such, they meet the public health standard of the TCA. Many other quasi–experimental studies have similarly found that e–cigarettes have public health benefit by reducing smoking.[84]

Out of the 6.7 million e–cigarette applications that the FDA received by Sept. 2020, they have authorized only 34 e–cigarette products, few of which are flavored or have high nicotine concentrations. This authorization rate of 0.000005% is small compared to the success rate for drug compounds approved for human subjects testing of 11.8% (Chorniy et al., 2021). At the moment many unauthorized e–cigarette products remain on the marketplace, but should the FDA be successful in clearing the marketplace of unauthorized e–cigarettes, the observed reductions in SAM could considerably lessen. The FDA may wish to authorize e–cigarettes at a far higher rate than currently to protect these public health benefits. The FDA may also wish to reduce regulatory barriers to allow e–cigarettes to be approved as a drug, essentially creating a parallel e–cigarette prescription marketplace to the consumer marketplace, which one study finds could lead to social surplus gains (Kenkel et al., 2024).

One previously unmentioned limitation of our study is that we observe SAM effects over thirteen years after e–cigarette introduction, instead of over the full life–course. Theoretically, e–cigarettes could reduce SAM initially, but increase it at some later point in time. Several factors lead us to believe that our findings are likely to persist into the future rather than be reversed at any point

[84]For example, Pesko (2023) found substitution between e–cigarettes and cigarettes in 14 of 16 reviewed studies of e–cigarette taxes, minimum legal sales age laws, and advertising restrictions. More recent studies regulating e–cigarette flavors also supports e–cigarettes and cigarettes being substitutes (Chen et al., 2024; Friedman et al., 2024, 2025; Saffer et al., 2024; Cotti et al., 2024).

in time. First, we document life years saved only through 2019. Deaths averted through 2019 will likely lead to additional life years saved beyond 2019; further, e–cigarettes will likely continue to avert new deaths far into the future, thus providing additional sources of life years saved. Second, we do not see any evidence in looking at changes in SAM for people 18–to–29 years of age to suggest the pattern of results may be different than the pattern of results for older people (Appendix J4). Third, most available evidence suggests that e–cigarettes are displacing cigarette use among youth. Descriptive data from the National Youth Tobacco Survey shows youth cigarette use near zero in 2023, and overall tobacco product use rates half of what it was in 2011 (Appendix N). These rates of decline in cigarette use have occurred during the e–cigarette era far faster than optimistic government projections (Pesko and Warman, 2022). Quasi–experimental work also generally supports e–cigaretes displacing cigarettes among youth (Pesko and Currie, 2019; Pesko, 2023; Friedman, 2015; Dave et al., 2019; Abouk et al., 2023; Pesko and Warman, 2022). All of these factors lead us to believe that e–cigarettes are likely to continue saving life years, compared to a counterfactual world where e–cigarettes are not available or are strictly regulated as drugs, into the future. However, our inability to precisely evaluate these future implications is a limitation of our work.

Our results do not suggest that drug deregulation or e-cigarettes are without harm—some individuals are undoubtedly harmed by both. However, evidence from this natural experiment indicates that the benefits outweigh the costs by a sizable margin. Drug regulation may be a promising area for reform, offering opportunities to reduce regulatory costs while improving health outcomes.

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7 Tables & Figures

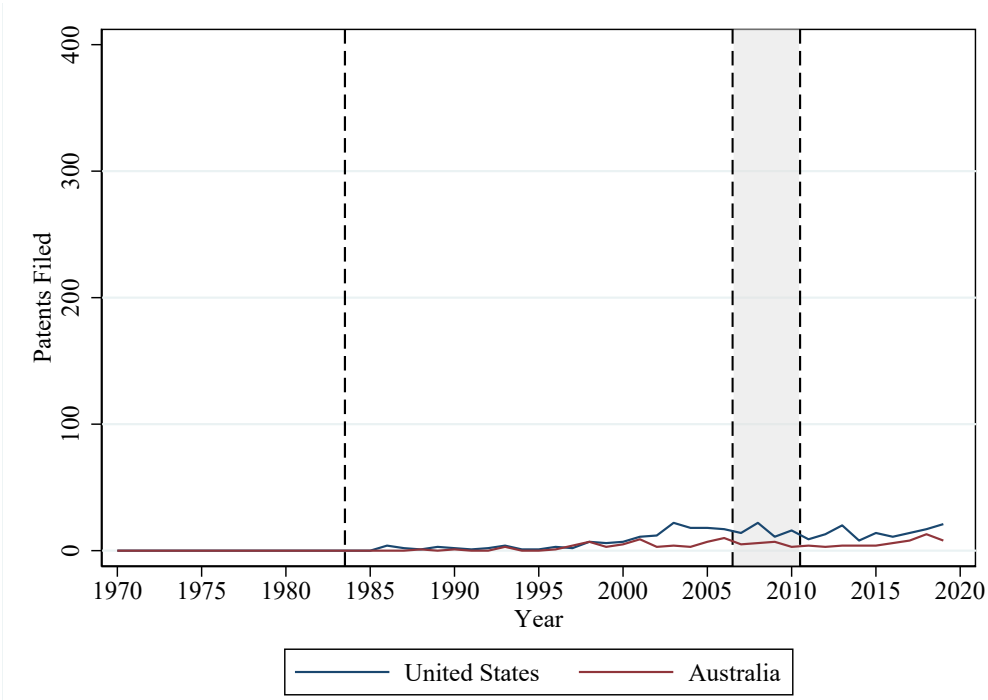
Table 1: Descriptive Means

	NRT Drug Introduction (1983)	E–cigarette Drug Introduction (2005–2006)
Smoking–Attributable Mortality:	1.97	1.33
	(0.17)	(0.08)
Cancers:	0.91	0.71
	(0.07)	(0.04)
Cardiovascular:	0.74	0.33
	(0.06)	(0.02)
Respiratory:	0.22	0.20
	(0.02)	(0.01)
Metabolic:	0.03	0.05
	(0.003)	(0.004)
Current Smoking (NHIS):	34.4%	24.2%
	(0.40%)	(0.23%)
Daily Smoking (NHIS):	30.6%	19.8%
	(0.38%)	(0.20%)

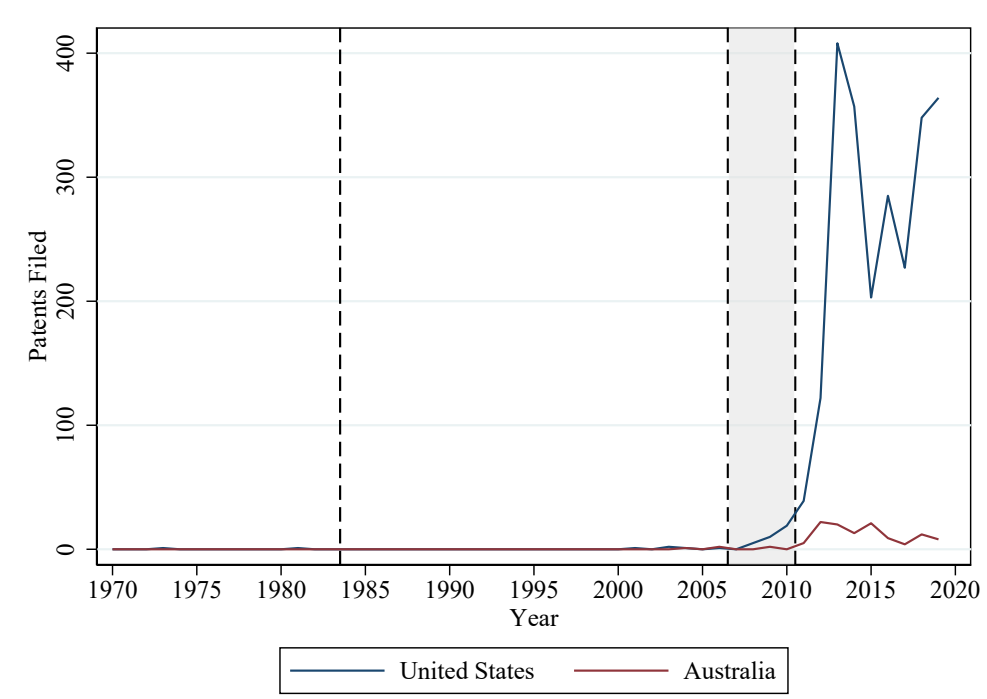
Notes: Data is from NVSS and NHIS for our estimation sample of ages 30-79. Means and standard errors are shown for mortality and smoking outcomes. Mortality rates are expressed as deaths per 1,000 population. Rates are adjusted to 2000 Standard U.S. population. Smoking outcomes are weighted using NHIS weights and Bayesian shrinkage.

Figure 1: Historical Patent Applications

(a) Non–E–cigarette Smoking Cessation Drug Patents

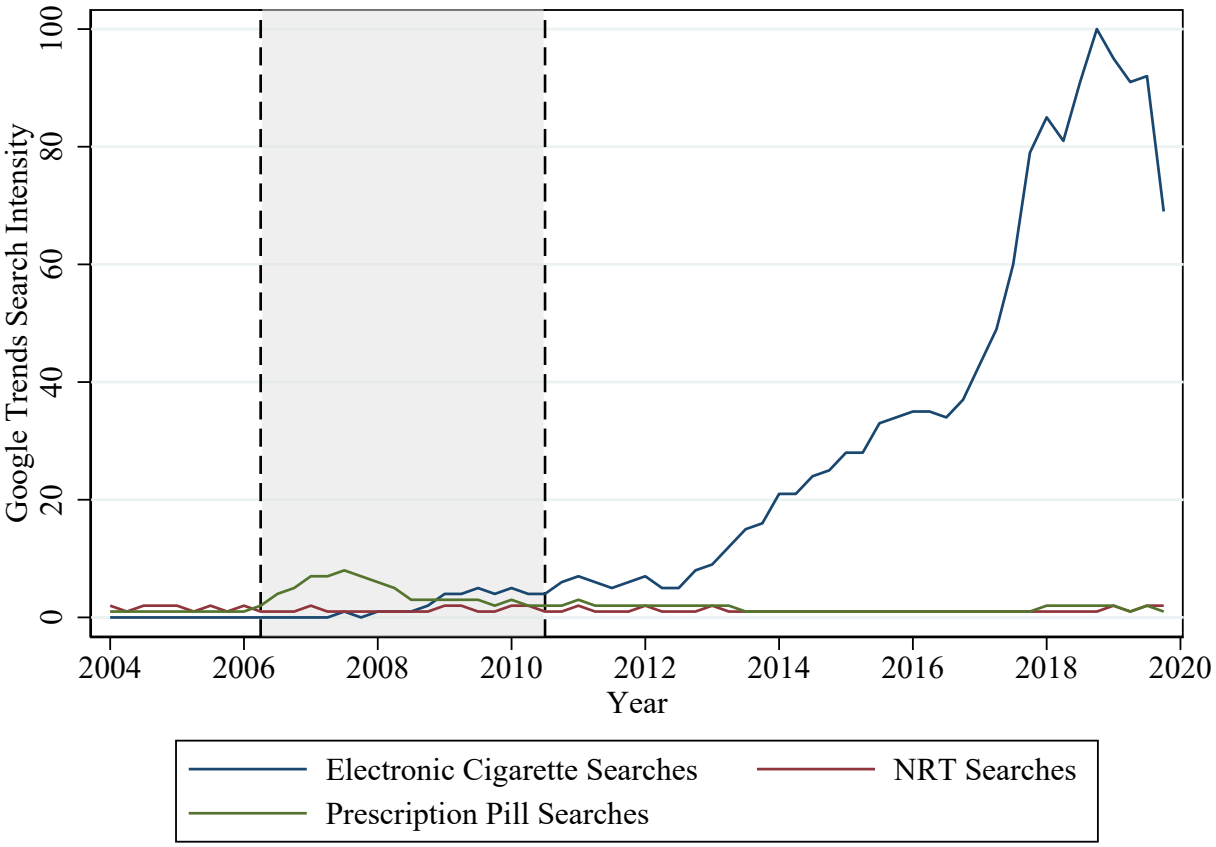


(b) E–cigarette Patents



Notes: Google Patent application intensity is shown for e–cigarettes and non–e–cigarette smoking cessation drugs from 1970 to 2019, for both the United States and Australia.

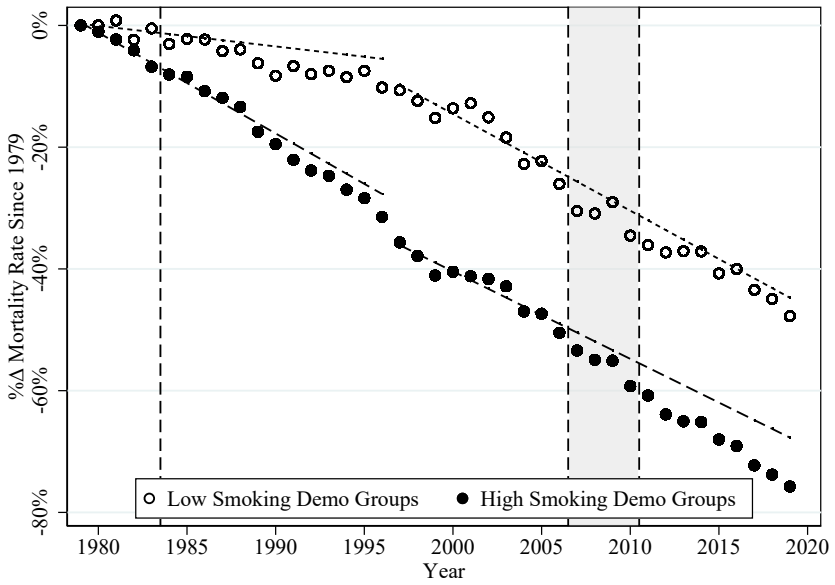
Figure 2: Smoking Cessation Drug Google Searches



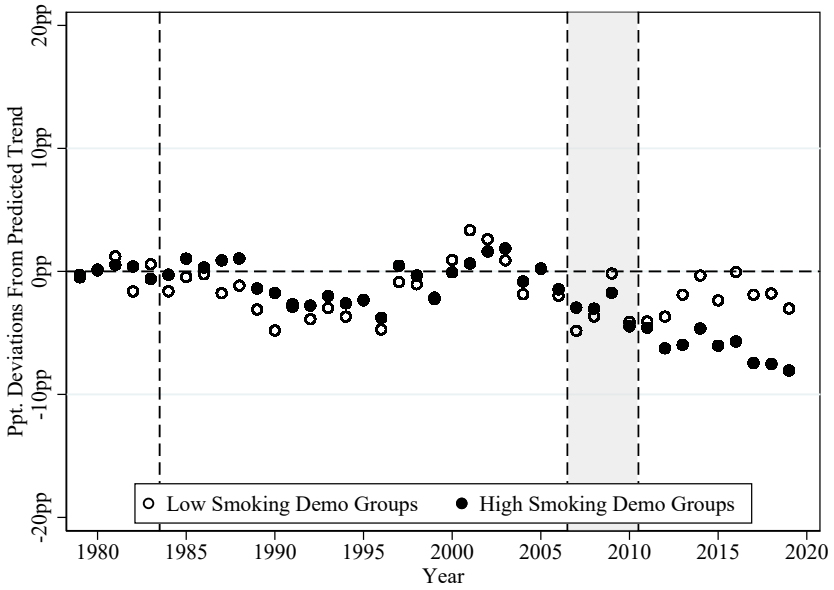
Notes: Google Trends search intensity is shown for electronic cigarette searches, NRT searches, and prescription pill searches from the earliest date available, 2004, through 2019, for the United States. Data is collapsed from monthly to quarterly using the maximum monthly search interest in each quarter. Dashed lines denote dates of e–cigarette introduction (August 2006) and drug exemption (December 2010).

Figure 3: Trends in Smoking–Attributable Mortality and Smoking

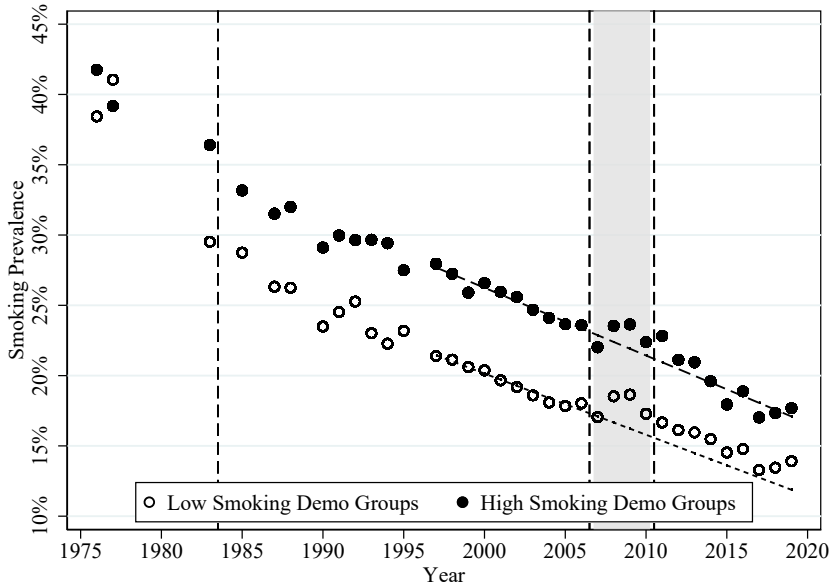
(a) Trends in Smoking–Attributable Mortality



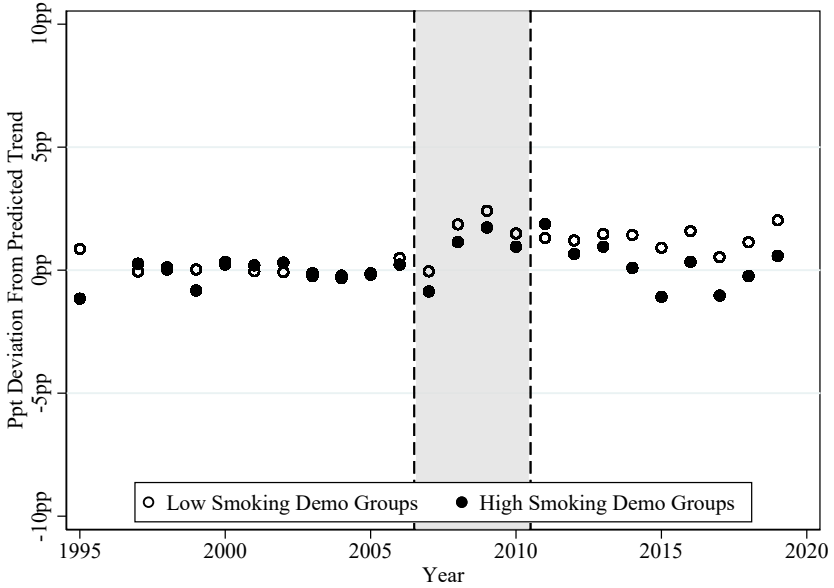
(b) Deviations From Predicted SAM Trends



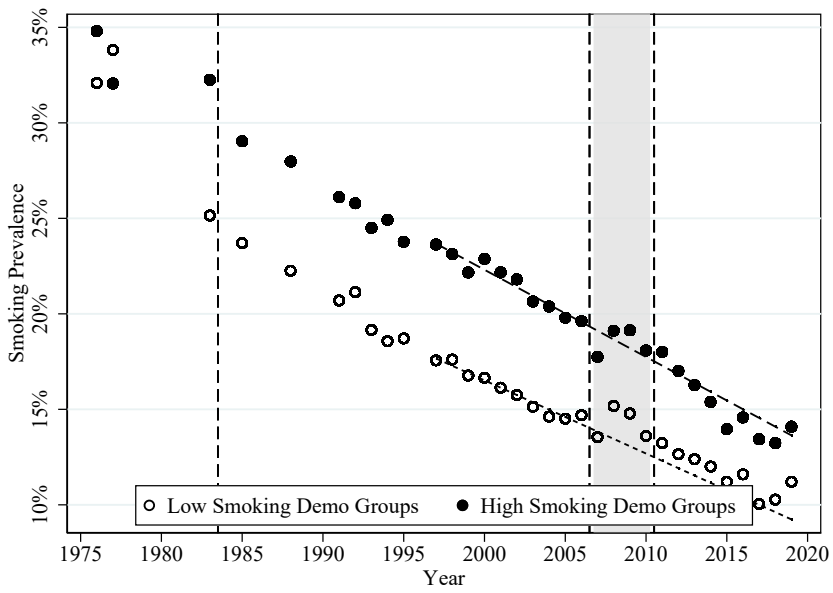
(c) Current Smoking Prevalence



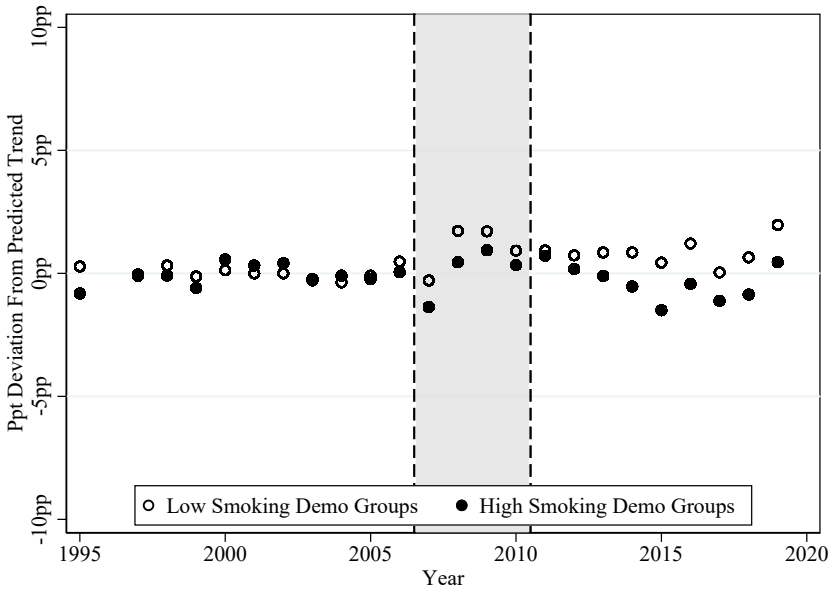
(d) Deviations From Predicted Current Smoking Trends



(e) Daily Smoking Prevalence



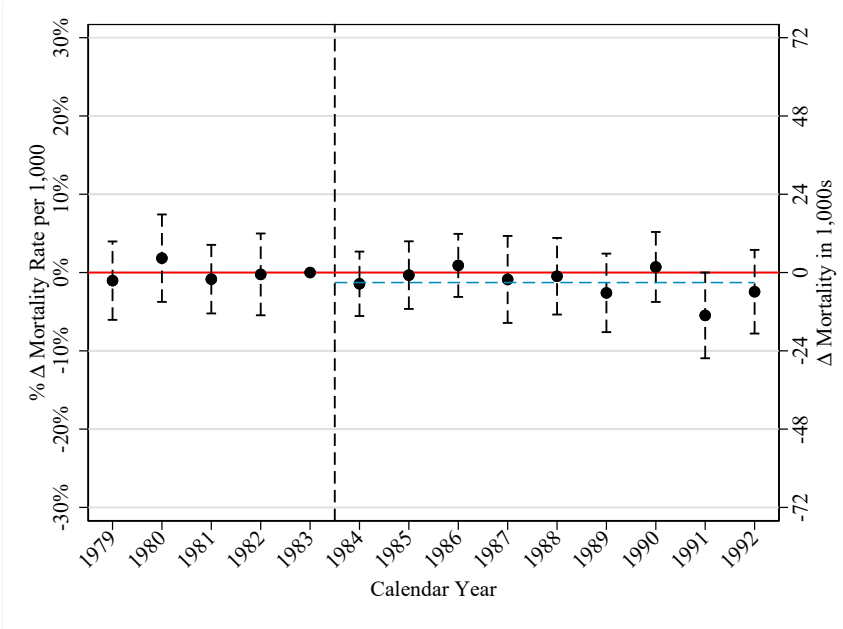
(f) Deviations From Predicted Daily Smoking Trends



Mortality data is from NVSS. Smoking data is from NHIS. Population data is from SEER. Mean SAM rates per 1,000 population are shown for adults ages 30–79. Mean adjusted smoking prevalences are shown for adults ages 30–79. Rates are age-adjusted using U.S. year 2000 standard population. Smoking outcomes are weighted using NHIS weights and Bayesian shrinkage.

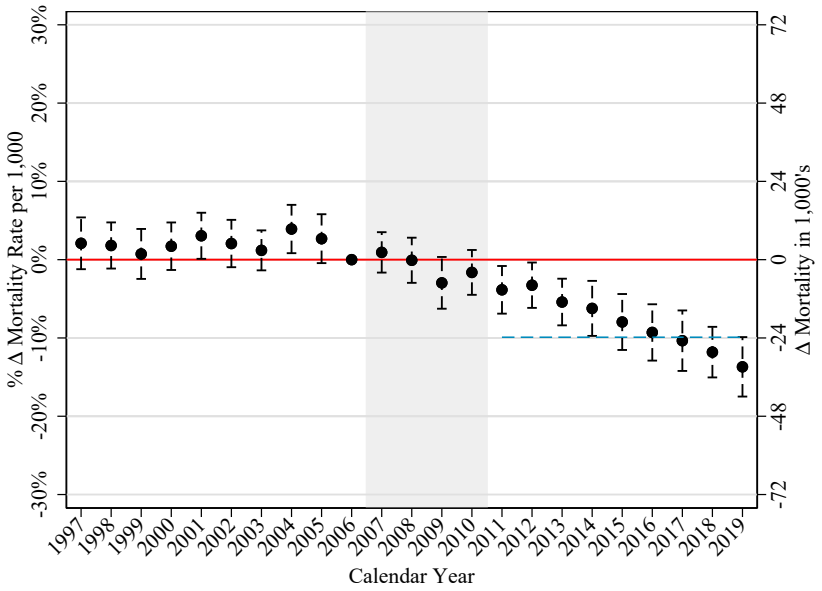
Figure 4: Event Studies for Smoking–Attributable Mortality

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = -1.28\%\,(1.75\%)$; Δ LY $= 21,852$

(b) E–cigarette Drug Exemption

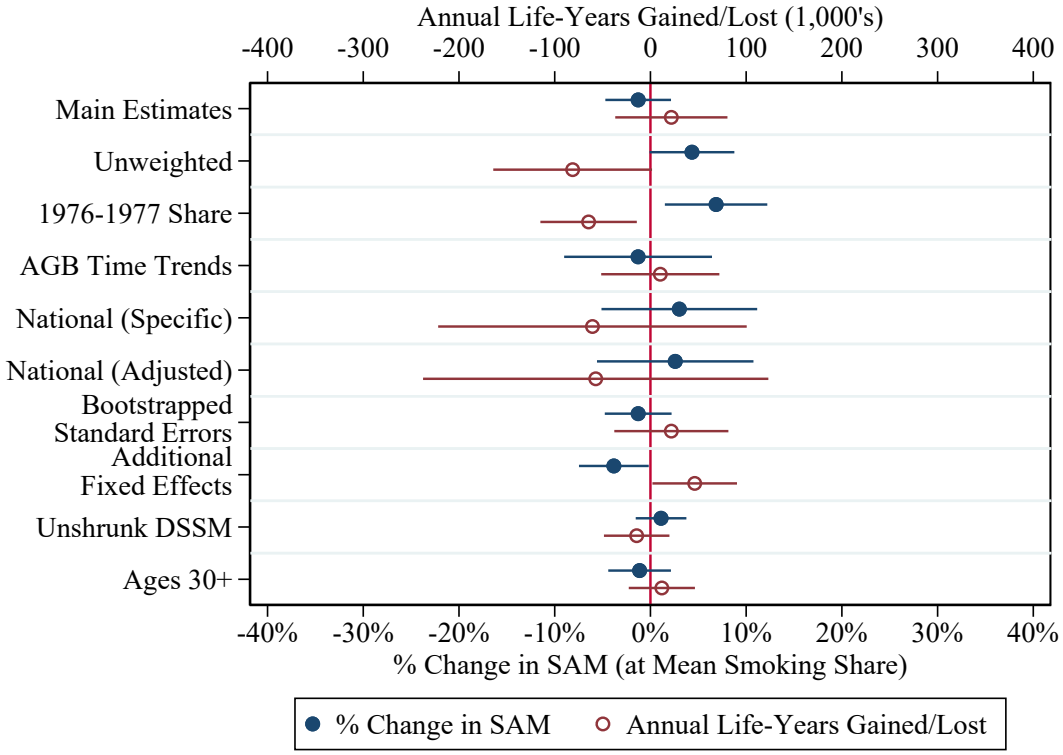


$\hat{\beta}_{Post\,2011}\,(SE) = -9.91\%^{***}\,(1.15\%)$; Δ LY $= 75,203$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.74\%^{***}\,(1.30\%)$; Δ LY $= 59,048$

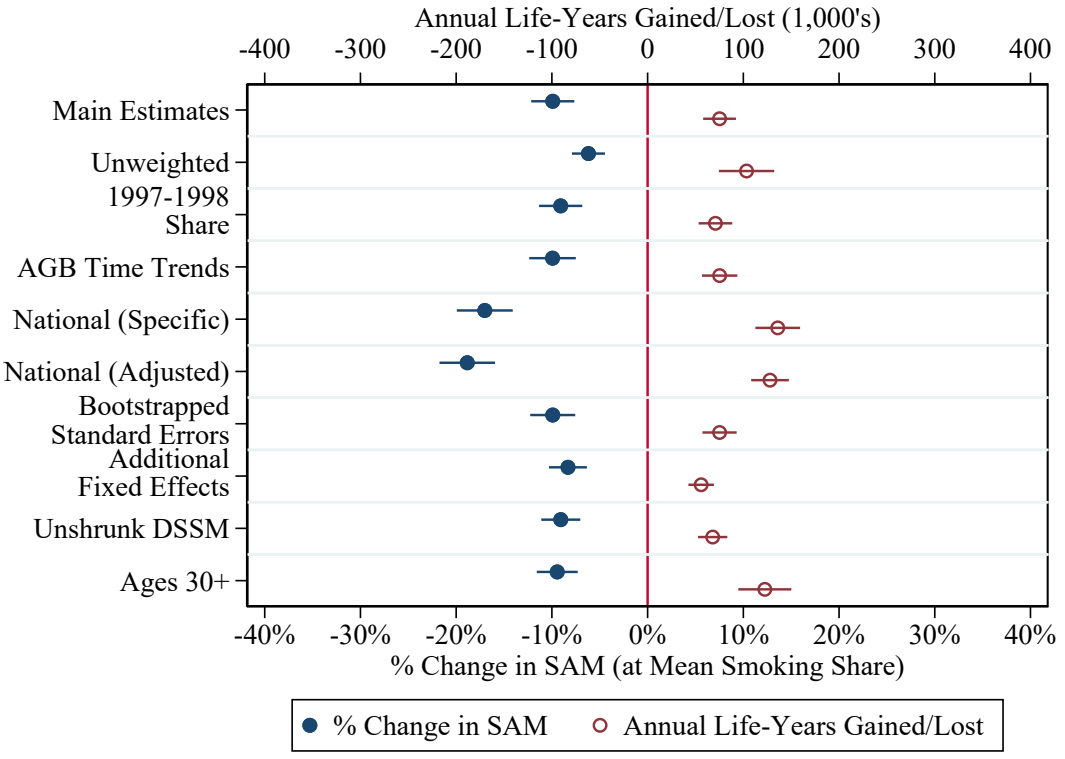
Notes: Mortality data is from the NVSS. We construct our smoking share measures using 1983 NHIS data and 2005–2006 BRFSS data. We apply Bayesian shrinkage to our smoking share measures (Morris, 1983). Outcome is SAM rate per 1,000 population for diseases established as caused by smoking. We weight causes by sex–specific SAF's (Lariscy, 2019). We apply ICD–9-10 comparability ratios following guidance from Anderson et al. (2001). Estimates along with 95% confidence intervals are shown. Event study leads and lags are interpreted as the effect of drug introduction/exemption, evaluated at the mean level of our smoking share measure, on deviations in logged mortality rate per 1,000 from the predicted linear trend, relative to reference period. Event studies are adjusted using the linear projection method from Freyaldenhoven et al. (2019). We conduct regressions using ordinary least squares, control for demographic group and year–by–age group fixed effects, and weight using reference period population. Standard errors are clustered at the demographic group level. Sample is restricted to groups ages 30–79 with available smoking share measures, yielding 287 groups for our NRT analysis and 719 groups for our e–cigarette analysis. Demographic groups are sex (male/female), 3 race categories for our NRT analysis (White, Black, other races) and 5 race/ethnicity categories for our e–cigarette analysis (White non-Hispanic, Black non-Hispanic, American Indian non–Hispanic, Asian/Pacific Islander non-Hispanic, Hispanic), census region (Northeast, Midwest, South, West), 2013 urban status (urban/rural) for our e–cigarette analysis, and 10 5–year age groups (30–34, . . . , 75–79). The weighted mean of our DSSM is 27.86% for our NRT analysis and 14.4% for our e–cigarette analysis. Weighted mean mortality (in reference period year) is 2.1 deaths per 1,000 for our NRT analysis and 1.5 deaths per 1,000 for our e–cigarette analysis. The shaded region from 2007-2010 denotes the time following e–cigarette introduction to the U.S. in August 2006 drug exemption in December 2010. Average annual Δ life–years (LY) are calculated using Equation 7 from Appendix F. *** p<0.01, ** p<0.05, * p<0.1

Figure 5: Summary of Main Estimates and Robustness Estimates

(a) NRT Drug Introduction



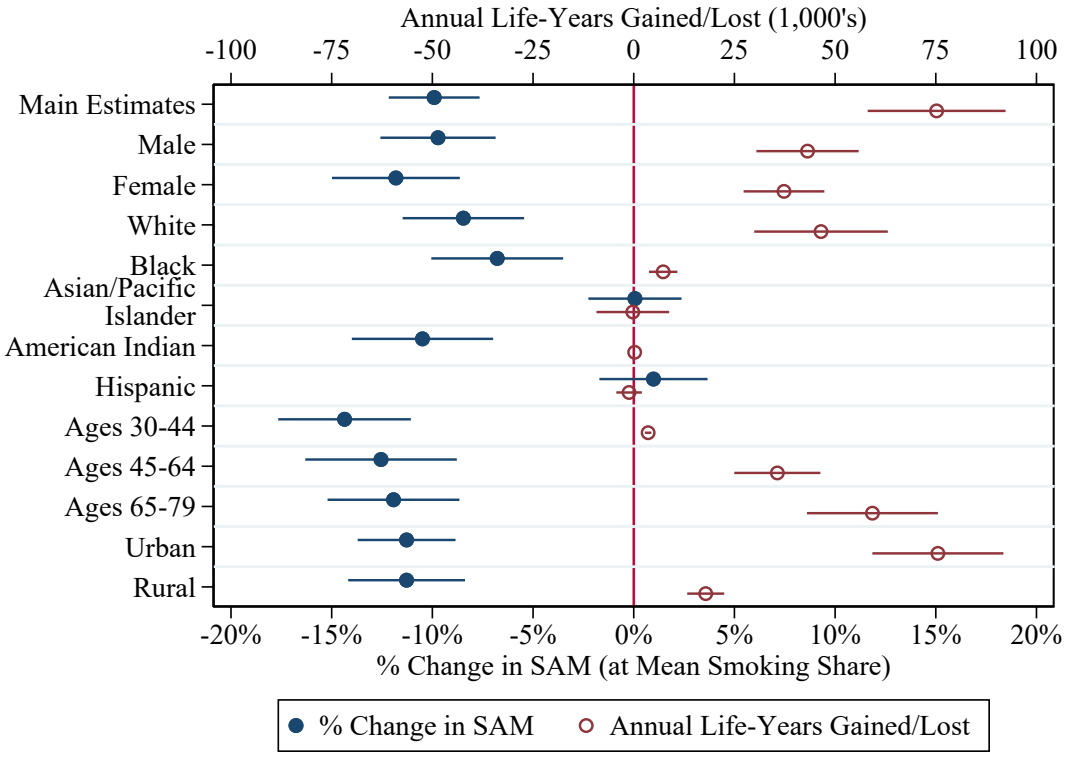
(b) E–cigarette Drug Exemption



Notes: Estimates with 95% confidence intervals are shown for our main specification. Estimates come from Figures 4, I1, I2, I3, I4, I5, I6, I7, and I8.

Figure 6: Summary of Main Estimates by Demographics & Age

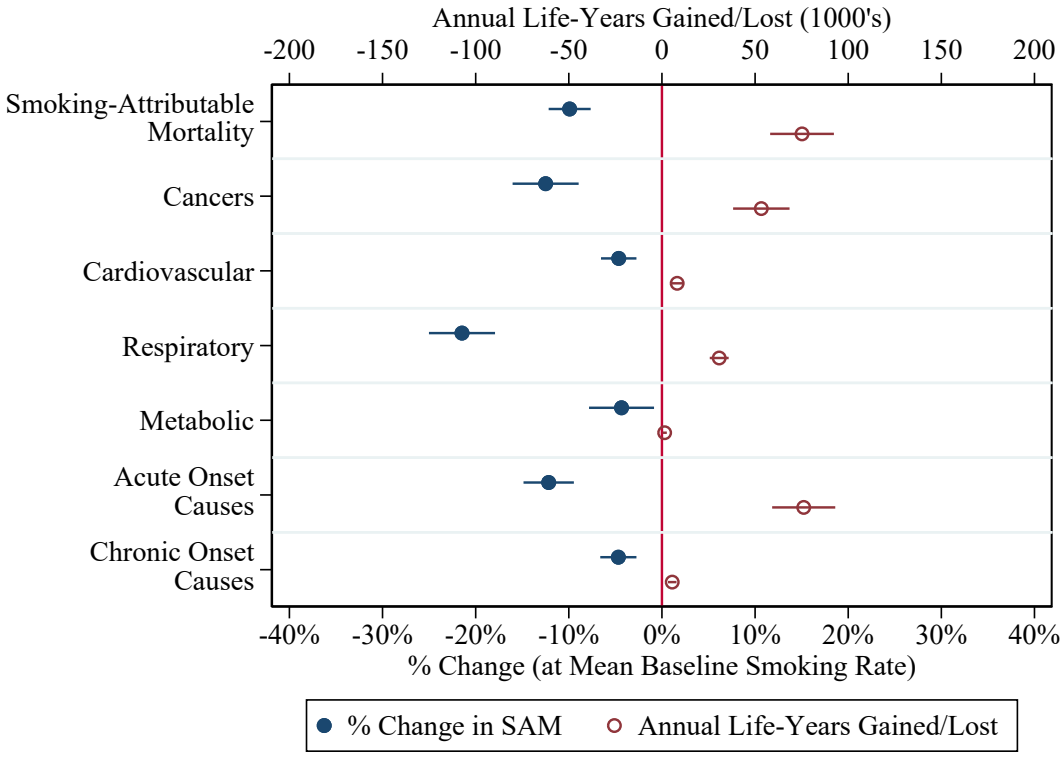
(a) E–cigarette Drug Exemption



Notes: Estimates with 95% confidence intervals are shown for our main sample. Sub–group estimates are scaled by sub–group mean daily smoking shares. Estimates come from Figures 4, J1, J2, J3, and J5.

Figure 7: Summary of Main Estimates by Cause of Death

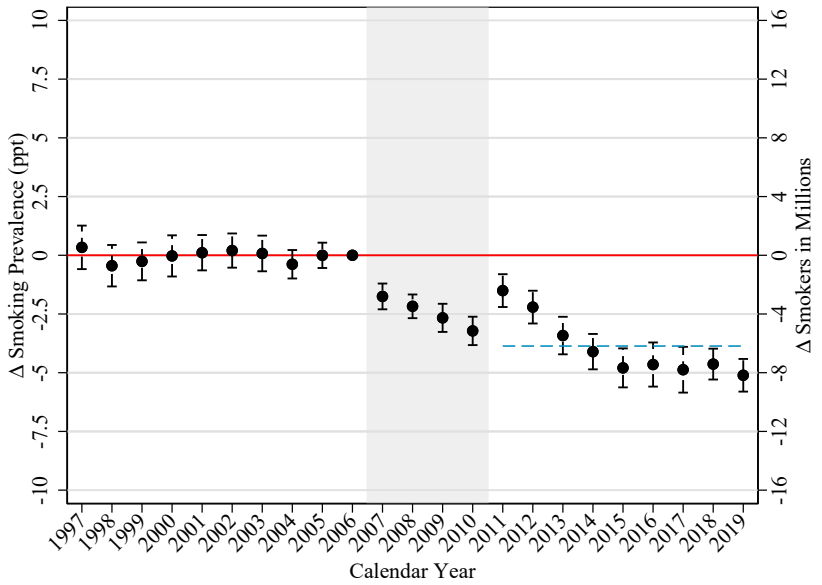
(a) E–cigarette Drug Exemption



Notes: Estimates with 95% confidence intervals are shown for our main sample. Estimates come from Figures 4, K1, and K2.

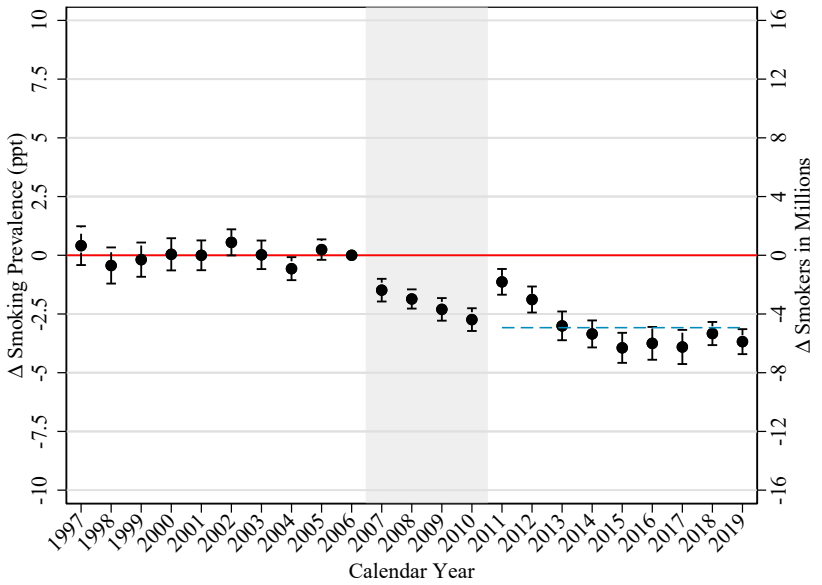
Figure 8: Event Studies for Smoking Prevalence Outcomes

(a) Current Smoking Prevalence (BRFSS)



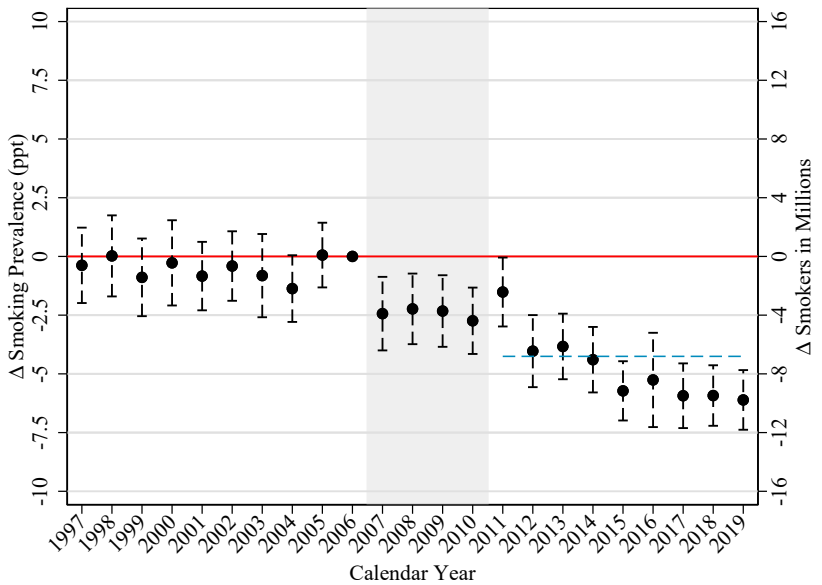
$\hat{\beta}_{Post\,2011}\,(SE) = -3.87\text{ppt}^{***}\,(0.19\text{ ppt});\ \Delta\text{SY} = -6.42\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -2.41\text{ppt}^{***}\,(0.22\text{ ppt});\ \Delta\text{SY} = -5.68\text{m}$

(b) Daily Smoking Prevalence (BRFSS)



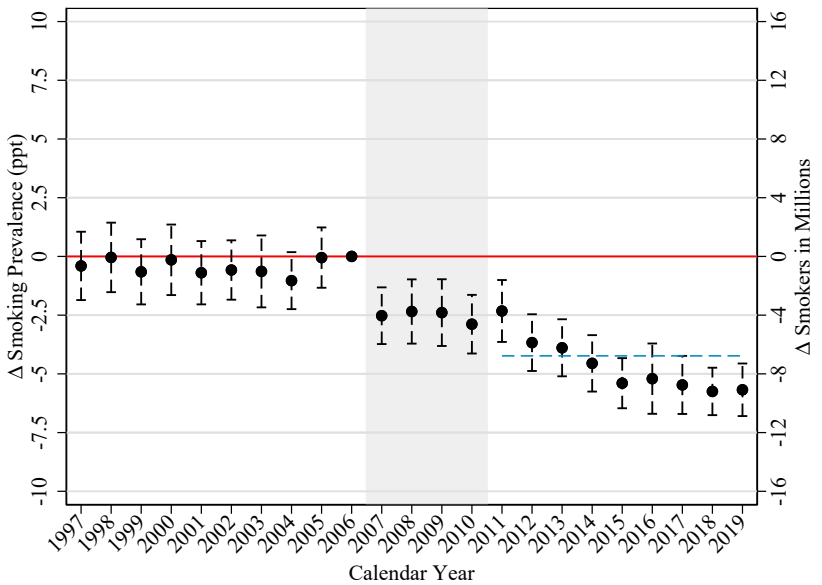
$\hat{\beta}_{Post\,2011}\,(SE) = -3.08\text{ppt}^{***}\,(0.15\text{ ppt});\ \Delta\text{SY} = -5.08\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -2.11\text{ppt}^{***}\,(0.18\text{ ppt});\ \Delta\text{SY} = -4.57\text{m}$

(c) Current Smoking Prevalence (NHIS)



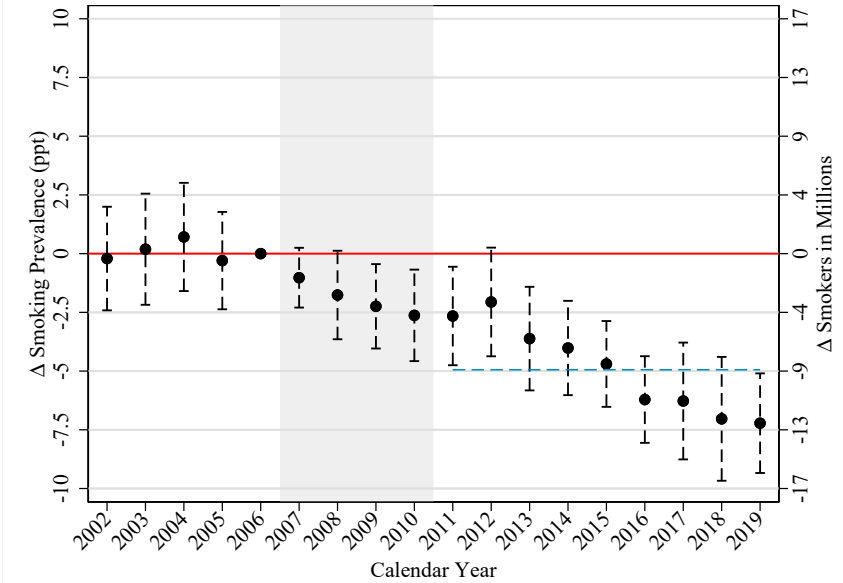
$\hat{\beta}_{Post\,2011}\,(SE) = -4.25\text{ppt}^{***}\,(0.26\text{ ppt});\ \Delta\text{SY} = -7.72\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -1.95\text{ppt}^{***}\,(0.38\text{ ppt});\ \Delta\text{SY} = -6.56\text{m}$

(d) Daily Smoking Prevalence (NHIS)



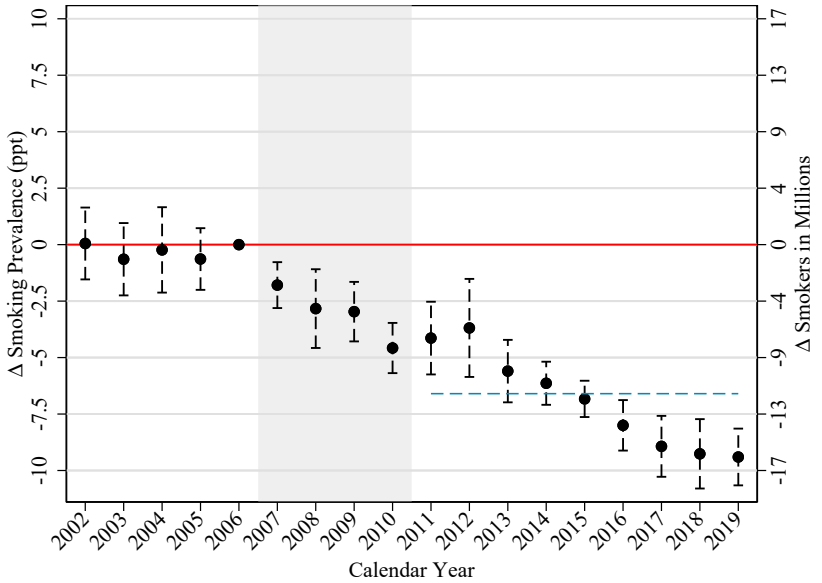
$\hat{\beta}_{Post\,2011}\,(SE) = -4.23\text{ppt}^{***}\,(0.25\text{ ppt});\ \Delta\text{SY} = -7.55\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -2.11\text{ppt}^{***}\,(0.35\text{ ppt});\ \Delta\text{SY} = -6.49\text{m}$

(e) Current Smoking Prevalence (NSDUH)



$\hat{\beta}_{Post\,2011}\,(SE) = -4.94\text{ppt}^{***}\,(0.38\text{ ppt});\ \Delta\text{SY} = -8.50\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -1.99\text{ppt}^{***}\,(0.51\text{ ppt});\ \Delta\text{SY} = -6.92\text{m}$

(f) Daily Smoking Prevalence (NSDUH)

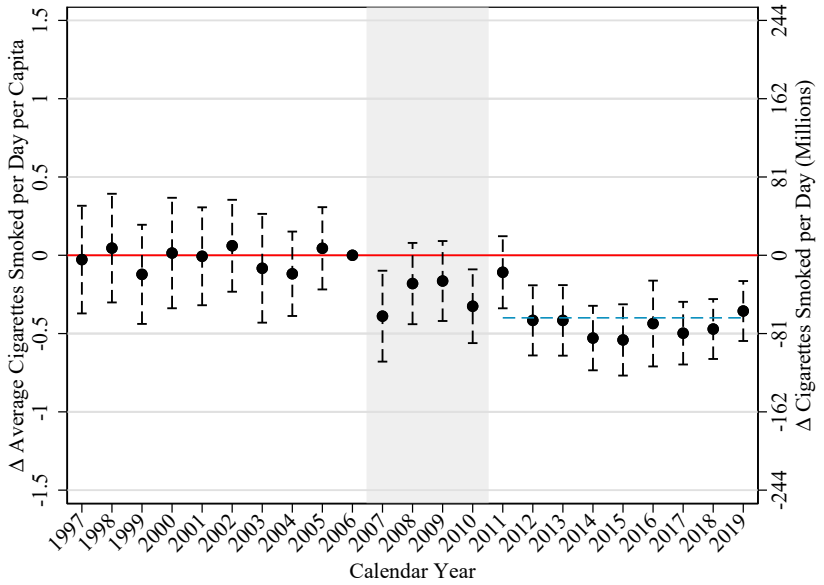


$\hat{\beta}_{Post\,2011}\,(SE) = -6.60\text{ppt}^{***}\,(0.30\text{ ppt});\ \Delta\text{SY} = -12.04\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -2.75\text{ppt}^{***}\,(0.34\text{ ppt});\ \Delta\text{SY} = -9.97\text{m}$

Notes: Survey data is from the BRFSS, NHIS, and NSDUH. Estimates along with 95% confidence intervals are shown. Outcomes are population–level current and daily smoking rates. See Table D2 for details on each data source. We construct our DSSM using 2005–2006 data, and utilize each data source's smoking share in that data source's regression. We apply Bayesian shrinkage to smoking shares and smoking outcomes (Morris, 1983). Event study leads and lags are interpreted as the effect of drug introduction/exemption, evaluated at the mean level of our smoking share measure, on deviations in our smoking–prevalence outcome from the predicted linear trend, relative to reference period. Event studies are adjusted using the linear projection method from Freyaldenhoven et al. (2019). We conduct regressions using ordinary least squares, control for demographic group and year fixed effects, and weight using reference period population. Standard errors are clustered at the demographic group level. Sample is restricted to groups ages 30–79 with available smoking share measures, except for NSDUH, which is ages 30 and older. Our estimation sample consists of 719 groups for BRFSS, 219 groups for NHIS, and 39 groups for NSDUH. The weighted mean of our DSSM is 14.36% for BRFSS, 16.9% for NHIS, and 16.2% for NSDUH. Weighted mean current smoking rates (in reference period year) is 18.6% in BRFSS, 20.5% in NHIS, and 22.6% in NSDUH. Weighted mean daily smoking rates (in reference period year) is 14.0% in BRFSS, 16.9% in NHIS, and 16.2% in NSDUH. The shaded region from 2007-2010 denotes the time following e–cigarette introduction to the U.S. in August 2006 drug exemption in December 2010. Average annual Δ smoking-years (SY) are calculated using Equation 9 from Appendix F. *** p<0.01, ** p<0.05, * p<0.1

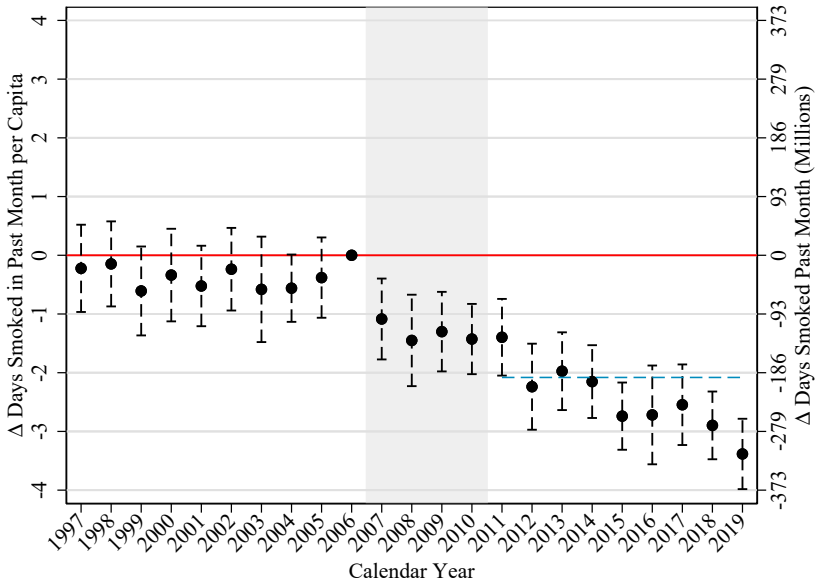
Figure 9: Event Studies for Smoking Frequency/Intensity Outcomes

(a) Average Cigarettes Smoked per Day (NHIS)



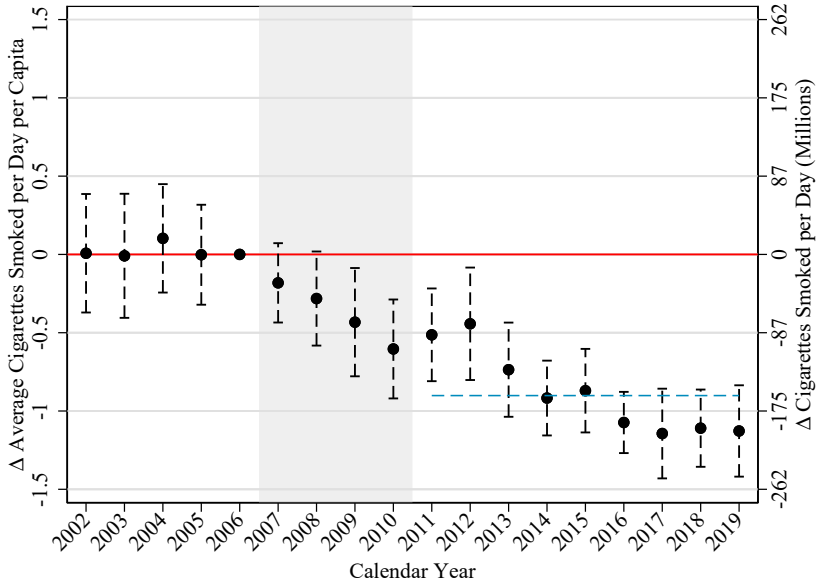
$\hat{\beta}_{Post\,2011}\,(SE) = -0.40\text{cigs}^{***}\,(0.06)$; 64.89m cigs per day
$\hat{\beta}_{Post\,2007}\,(SE) = -0.25\text{cigs}^{***}\,(0.75)$; 40.01m cigs per day

(b) Days Smoked in Past Month (NHIS)



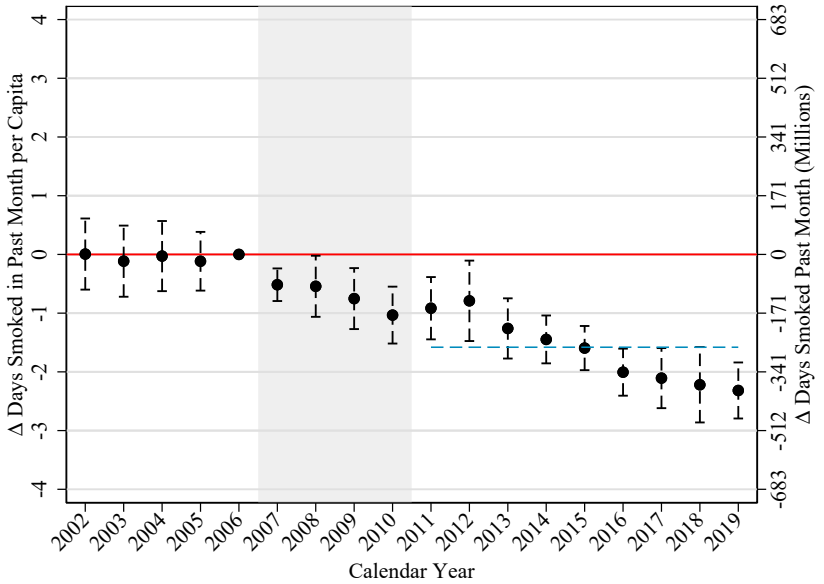
$\hat{\beta}_{Post\,2011}\,(SE) = -2.08\text{days}^{***}\,(0.12)$; −194m days per month
$\hat{\beta}_{Post\,2007}\,(SE) = -0.95\text{days}^{***}\,(0.20)$; −89m days per month

(c) Average Cigarettes Smoked per Day (NSDUH)



$\hat{\beta}_{Post\,2011}\,(SE) = -0.90\text{cigs}^{***}\,(0.09)$; 157.6m cigs per day
$\hat{\beta}_{Post\,2007}\,(SE) = -0.39\text{cigs}^{***}\,(0.10)$; 69.04m cigs per day

(d) Days Smoked in Past Month (NSDUH)



$\hat{\beta}_{Post\,2011}\,(SE) = -1.58\text{days}^{***}\,(0.11)$; −269.8m days per month
$\hat{\beta}_{Post\,2007}\,(SE) = -0.66\text{days}^{***}\,(0.13)$; −113.3m days per month

Notes: Smoking data is from NHIS and NSDUH. Estimates along with 95% confidence intervals are shown. Outcomes are per–capita past–month days smoked and average cigarettes smoked per day. Smoking share measures are constructed using 2005–2006 NHUS and NSDUH data for each respective analysis. Bayesian shrinkage estimator is applied to smoking shares (Morris, 1983). Event study leads and lags can be interpreted as the effect of drug introduction/exemption, evaluated at the mean level of our daily smoking share measure, on deviations in our per–capita outcomes from the predicted linear trend, relative to reference period. Event studies are adjusted using the linear projection method from Freyaldenhoven et al. (2019). Regressions are conducted using ordinary least squares, control for demographic group and year fixed effects, and weighted using reference period population. Standard errors are clustered at the demographic group level. Sample is restricted to groups ages 30–79 with available smoking share measures (ages 30 and older for NSDUH), yielding 219 groups in NHIS and 39 groups in NSDUH. Demographic groups are sex (male/female), 3 race categories (White, Black, other races) in NHIS and 5-race/ethnicity categories in NSDUH (White non-Hispanic, Black non-Hispanic, American Indian non-Hispanic, Asian/Pacific Islander non-Hispanic, Hispanic), census region (Northeast, Midwest, South, West) in NHIS, and 10 5-year age groups (30–34, . . . , 75–79) in NHIS and 4 age groups in NSDUH (30–34, 35–49, 50-64, 65 and older). The weighted mean of our daily smoking share measure is 16.9% in NHIS and 16.2% in NSDUH. Weighted mean per–capita cigarettes smoked per day is 3.0 in NHIS and 3.2 in NSDUH. Weighted mean per–capita days smoked in past month is 6.1 in NHIS and 5.7 in NSDUH. The shaded region from 2007-2010 denotes the time following e–cigarette introduction to the U.S. in August 2006 drug exemption in December 2010. Δ smoking-years (SY) are calculated using Equation 9. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

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Appendix A Analysis of Patent Applications

Table A1: Effect of E–cigarette Drug Exemption on Logged Patent Applications

	Pre-2011	Post-2011	Time-Difference
E–cigarettes:			
United States:	0.267	5.395	5.108***
	(0.105)	(0.248)	(0.261)
Australia:	0.070	2.486	2.416***
	(0.041)	(0.186)	(0.184)
		Differences-	2.693***
		in-Differences:	(0.319)
	Pre-2011	Post-2011	Time-Difference
Non–E–cigarette Smoking Cessation Drugs:			
United States:	1.156	2.674	1.518***
	(0.180)	(0.103)	(0.207)
Australia:	0.700	1.867	1.167***
	(0.136)	(0.135)	(0.189)
		Differences-	0.351
		in-Differences:	(0.280)
		DDD Estimator:	2.342***
			(0.425)

Notes: Difference-in-differences estimates (DD) and triple-differences (DDD) estimates with robust standard errors shown for log(patent applications + 1) from 1970-2019, with the post-period beginning after FDA drug exemption in 2011. DD estimates show before-and-after differences in patent applications between the United States and Australia by drug type. DDD estimate is the difference between the DD estimate for e–cigarettes and the DD estimate for non–e–cigarette smoking cessation drugs.

Appendix B Background Figures on E–cigarette Innovation

Figure B1: Ruyan V8 E–cigarette



Notes: This is a promotional image of the first e–cigarette imported to the United States, the Ruyan v8 e–cigarette, in August 2006. It was advertised with a nicotine concentration of 16 mg/ml (Bullen et al., 2010) and were not flavored. If e–cigarettes had been consistently regulated as drugs, they are unlikely to look much different today. Source: https://bit.ly/4a3udWE.

Figure B2: E–cigarette Vape Shop

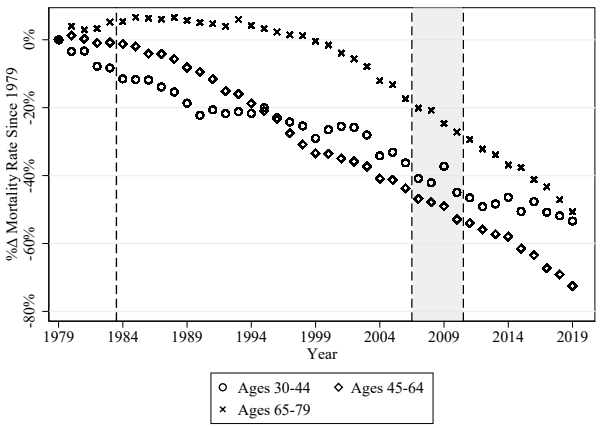


Notes: This image of a modern-day vape shop provides an illustration of the rapid innovation in the e–cigarette marketplace made possible by the judicial exemption of e–cigarettes from drug regulation. Source: https://bit.ly/3x0XH8Z.

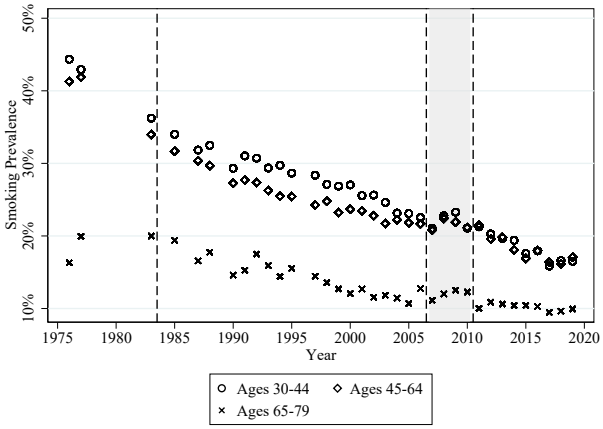
Appendix C Descriptive Figures

Figure C1: Trends in Smoking–Attributable Mortality and Smoking Rates by Demographic Category

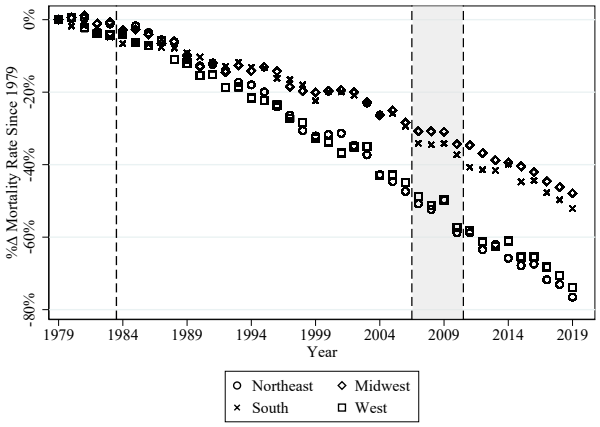
(a) Smoking–Attributable Mortality, Age



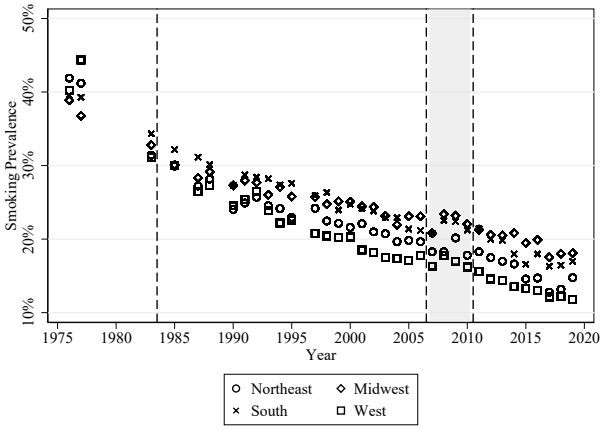
(b) Current Smoking, Age



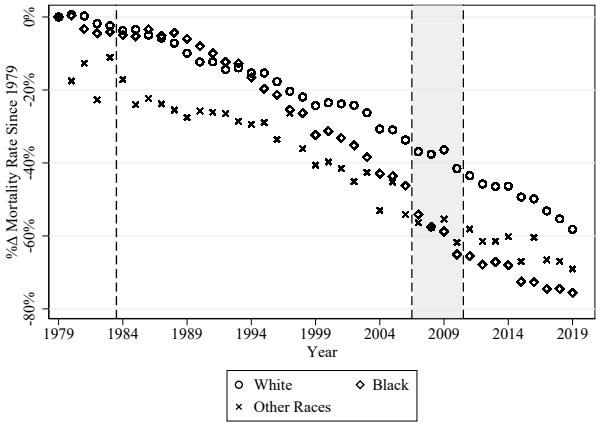
(c) Smoking–Attributable Mortality, Census Region



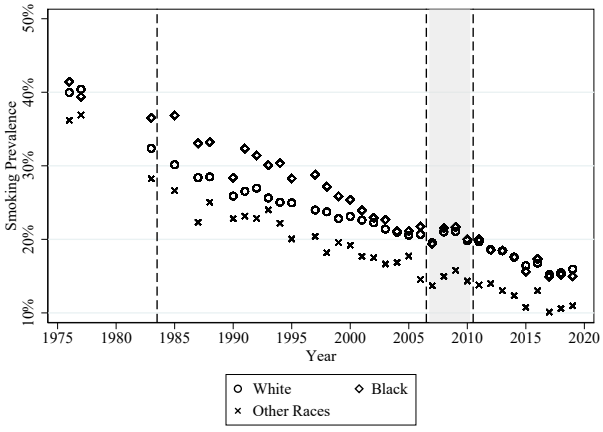
(d) Current Smoking, Census Region



(e) Smoking–Attributable Mortality, Race



(f) Current Smoking, Race



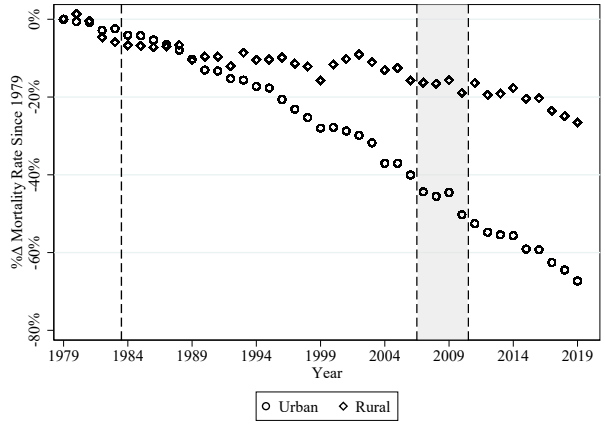
(g) Smoking–Attributable Mortality, Sex



(h) Current Smoking, Sex



(i) Smoking–Attributable Mortality, Urban Status



Mortality data is from NVSS. Smoking data is from NHIS. Population data is from SEER. Mean logged adjusted SAM rates per 1,000 population are shown for adults ages 30–79. Mean adjusted smoking prevalences are shown for adults ages 30–79. Smoking outcomes are weighted using NHIS sampling weights and shrunk using Bayesian shrinkage. Calendar year 2000 used as U.S. standard population.

Appendix D Data Appendix

D.1 Mortality Data

We provide a listing of ICD codes and SAF's for each of the 21 causes established as caused by smoking below in Table D1.

We examine four sub-categories of SAM in Appendix K: cancers, cardiovascular, respiratory, and metabolic causes.[D1] We also examine SAM with acute onset of risk reduction following smoking cessation ("acute–onset causes") and causes of death with a more delayed, or chronic, onset of risk–reduction following smoking cessation ("chronic–onset causes"). We create these two categories based primarily on evidence from Godtfredsen et al. (2002), who studies cause–specific survival rates following smoking–cessation. We include details on these sub–categories in Table D1.

[D1] We weight sub-categories of SAM using SAF's.

Table D1: Trends in Mortality and Smoking Rates by Demographic Category

Cause of Death	Male SAF	Female SAF	ICD–9	ICD–10	Acute/Chronic
Cancers:					
Lung cancer	87%	83%	162	C33-C34	Acute
Lip and oral cavity cancer	35%	14%	140-149	C00-C14	Acute
Laryngeal cancer	34%	45%	161	C32	Acute
Colorectal cancer	32%	14%	153, 154	C18-C20	Chronic
Esophageal cancer	27%	20%	150	C15	Chronic
Kidney and renal pelvis cancer	18%	10%	189	C64-C66	Chronic
Stomach cancer	18%	2%	151	C16	Chronic
Acute myeloid leukemia	16%	11%	205	C92.0	Chronic
Urinary bladder cancer	15%	18%	188	C67	Chronic
Pancreatic cancer	3%	13%	157	C25	Chronic
Cervical cancer	—	0%	180	C53	Chronic
Liver cancer	0%	0%	155	C22	Chronic
Cardiovascular:					
Aortic aneurysm	45%	26%	441	I71	Acute
Other arterial diseases	38%	22%	442-448	I72-I78	Chronic
Other heart disease	23%	25%	390-398, 415-429.1, 429.3-429.9	I00-I09, I26-I51	Chronic
Ischemic heart disease	20%	25%	410-414, 429.2	I20-I25	Acute
Atherosclerosis	9%	0%	440.1, 440.2	I70	Acute
Stroke	7%	8%	430-434, 436-438	I60-I69	Acute
Respiratory:					
Chronic obstructive pulmonary disease	43%	61%	490-492, 496	J40-J44	Acute
Pneumonia, influenza, tuberculosis	31%	33%	010-018, 480-487	A16-A19, J10-J18	Acute
Metabolic:					
Diabetes mellitus	28%	0%	250	E10-E14	Acute

Notes: Cause of death categories for diseases established as caused by smoking, as well as sex–specific SAF's, and ICD–9 and -10 codes are presented (Lariscy, 2019).

The process of smoothing out discontinuities in cause-specific mortality between ICD editions is known as "harmonization". Standard practice in the literature is to apply comparability ratios when data spans more than one ICD edition (Anderson et al., 2001). Comparability ratios between ICD–9 and -10 are computed as follows:

$$\sigma_i = \frac{D_i^{ICD--10}}{D_i^{ICD--9}} \tag{5}$$

Above, σ_i is the comparability ratio for cause i, $D_i^{ICD--10}$ is the number of deaths assigned to cause i under ICD–10, and likewise D_i^{ICD--9} is deaths under ICD–9. Calculating these ratios requires assigning ICD–9 causes to their corresponding cause under ICD–10 using both automated software and manual intervention by experts.[D2] We pull final comparability ratios for 113 selected causes of death from the National Center for Health Statistics.[D3]

We comment on a few data–related details unique to the NVSS data below. First: Some states began reporting Hispanic ethnicity to the NCHS in 1990, but full participation from all states was not achieved until 1997. 5-race/ethnicity mortality counts prior to 1997 may therefore be unreliable, with changes driven by mortality as well as increased state participation. For this reason, we use our 3-race mortality panel whenever our analysis spans 1997, otherwise we use 5-race/ethnicity wherever possible.

Second: Prior to 1982, counties in the mortality files are delineated using *NCHS* codes, not FIPS codes. NCHS codes are similar to FIPS codes but are assigned to counties within each state sequentially based on alphabetical order.[D4] Prior to 1982, we use the 1970 Geographic Code Manual from the Division of Vital Statistics in conjunction with the NCHS-to-FIPS crosswalk to back-out urban status.[D5] In rare cases where we are unable to recover the FIPS code of a county from the NCHS code, we assign urban status manually based on the county's name and that present–day county's urban status. We define urban status based on metropolitan status using the 2013 rural-urban continuum codes (RUCC) codes from the Economic Research Service.[D6]

[D2] Both the Mortality Medical Indexing, Classification, and Retrieval (MICAR) program and Automated Classification of Medical Entities (ACME) are used. The MICAR is used to assign codes to each entity based on the configuration of entity codes, and the ACME then takes these codes and generates an underlying cause based on a series of decision tables and selection rules.

[D3] Final and preliminary estimates available from https://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/Comparability/icd9_icd10/Comparability_Ratio_tables.xls

[D4] Virginia also distinguishes between counties and cities, with NCHS codes for cities being distinct from counties and being listed after counties.

[D5] Available at https://data.nber.org/mortality/1991/docs/geocode.txt.

[D6] Due to historical changes in geographic definitions in the state of Alaska, we assign Alaska as rural.

Third: We begin our mortality panel in 1979 as the first year that the NVSS data uses the 9th Edition of the International Classification of Diseases (ICD–9). Harmonizing causes of death between the ICD–8 and ICD–9 is particularly challenging due to major structural changes in how diseases and ailments are classified starting in ICD–9. In addition, Centers for Disease Control (CDC) cause of death recodes, which are categories used to assign individual causes of death to larger, more logical groupings on the basis of pathology, are discontinuous between ICD–8 and –9, unlike between ICD–9 and -10, thus making ICD–8 and –9 harmonization impractical.

We comment briefly on our use of specific rates below. Define the count of deaths for group i in year t as $D_{i\,(t)}$, and similarly the population of group i in year t as $P_{i\,(t)}$. The specific mortality rate for group i in year t, or $D_{i\,(t)}$, is defined as the ratio of total deaths over total population, or $\frac{D_{i\,(t)}}{P_{i\,(t)}}$. If group i contains sub-groups, denoted by $m \in i$, then group i's specific mortality rate can also be expressed as the weighted-average of each sub-group's mortality rate (weighted by group m's share of population in group i), or $\Sigma_{m \in i} \frac{D_{m\,(t)}}{P_{m\,(t)}} \cdot \frac{P_{m\,(t)}}{P_{i\,(t)}}$. In contrast, the adjusted mortality rate holds fixed the share of population of each subgroup at an arbitrary year's population distribution, typically the standard U.S. population in 2000. The formula for group i's adjusted rate in year t, using the U.S. 2000 standard population, is $\Sigma_{m \in i} \frac{D_{m\,(t)}}{P_{m\,(t)}} \cdot \frac{P_{m\,(2000)}}{P_{i\,(2000)}}$, which is identical to the previous calculation of specific rates, except that each sub-group is weighted by their population share in year 2000.

The use of adjusted rates is generally recommended in two situations: first, when mortality rates are calculated for relatively large subsets of the population; second, when examining long-run trends in mortality rates. The use of specific rates in either of these situations introduces the possibility of changes in mortality rates over time being driven by changes in the population distribution, as opposed to changes in the underlying mortality burden.[D7] Neither of these situations appear applicable to our specification, since we define groups at extremely granular levels (5-year age, sex, race, urban status, region). The assumption we introduce, however, by our use of group–specific rates is that the composition of each demographic group remains stable over time, which appears reasonable here. In Appendix C, however, we present adjusted rates, since groups are defined more broadly (mortality by sex, by race, ...). We also show using national demographic groups and adjusted rates matters little to our estimates in Figure 5.

[D7] For example, age is strongly correlated with mortality. Changes in the distribution of age in the population will mechanically change mortality rates, even if underlying deaths are unchanged. If the composition within groups changes substantially over time, then analysis of trends can yield misleading conclusions.

D.2 Survey Data

In Table D2, we present an overview of the Behavioral Risk Factor Surveillance System (BRFSS), the National Health Interview Survey (NHIS), and the National Survey on Drug Use and Health (NSDUH). We work directly with the raw data files for BRFSS and NSDUH. For NHIS, we use the harmonized data from the Integrated Public Use Microdata Series (IPUMS).

[D8] BRFSS achieves full state-participation in 1990 but years prior to 1997 are unnecessary for our analysis. NHIS is administered annually but contains years with missing smoking outcomes. NSDUH underwent a major redesign in 2002 when it was renamed from the National Household Survey on Drug Abuse (NHSDA) to NSDUH. To ensure consistency we do not examine years prior to 2002 in NSDUH.

[D9] County FIPS codes are available through 2005. Beginning in 2005 the rate of county FIPS suppression increases until 2013, when county FIPS are dropped from BRFSS. The metropolitan status variable MSCODE is introduced in 2005, which we use to supplement missing county FIPS codes. From 2013-2017, MSCODE is exclusively used to determine urban/rural status, despite missing values. Beginning in 2018, _METSTAT is introduced and used in place of MSCODE as it contains substantially fewer missing values.

Table D2: Overview of Surveys and Outcomes

	BRFSS	NHIS	NSDUH
Purpose of Data	Monitor health risk behaviors, chronic diseases, and preventative services; monitor progress towards state and national health objectives	Collect data on health behaviors and status; monitor progress towards national health objectives	Monitor trends in substance abuse, mental health, and treatment to inform public health policy
Structure of Data	State-representative repeated cross-section	Nationally representative repeated cross-section	Nationally representative repeated cross-section
Average Annual Sample Size	279,701 adults	29,829 adults	38,095 adults
Years in Our Analysis[D8]	1997-2019	1976-2019 with gaps	2002–2019
Smoking Prevalence Outcomes	Ever, curent, & daily smoking status	Ever, curent, & daily smoking status	Ever, curent, & daily smoking status
Prevalence Variable Names	Smokeday, smokenum, smoke100, smokev, smokestatus1, smokestatus2, smokestatus3, smokestatus4, smokestatus5, smokenow	Smokestatus2, smokestatus1, smokestatus3, smokestatus4, smokestatus5, smokenow, cigsday, cigsday1, cigsday2, cigsdayes	cig100lf, cig30use, cig30av
Prevalence Variable Years	1997–2019	1976, 1977, 1983, 1985, 1987, 1988, 1990–1995, 1997-2019	2002–2019
Smoking Intensity/Other Outcomes	–	Average cigarettes smoked per day, average days in past month smoked	Average cigarettes smoked per day, average days in past month smoked
Intensity/Other Variable Names	–	Cigsday, cigsdayes, cigsday1, cigsdaymo	cig30av, cig30use
Intensity/Other Variable Years	–	1976, 1977, 1985, 1989, 1991-1995, 1997-2019 (Cigarettes per day); 1991-2019 (Days per month)	2002–2019
Weighting Variable	_finalwt (1997-2010), _llcpwt (2011–2019)	perweight (1976), sub77wt (1977), supp1wt (1983), supp2wt (1988, 1993-1995), sampweight (1985, 1987, 1990-1992, 1997-2019)	analwt_C
Weighting Notes	BRFSS begins including cell-phone respondents in 2011, plus a change made to weighting methodology (raking). BRFSS omits urban status for landline respondents from 2014-2017 due to confidentialty.	Weights valid for adult smoking sample	Weights valid for 12+ sample.
Lowest unit of Geography Available	County of Residence[D9]	Census region. Urban status is available in some years.	National
Race/Ethnicity	5-race/ethnicity	3-race	5-race/ethnicity
E–cigarette Prevalence Outcomes	Ever–, curent, & daily–e–cigarette use	–	–
E–cigarette Variable Names	ecignow, ecigaret,	–	–
E–cigarette Variable Years	2016–2018	–	–

For BRFSS and NSDUH, we work directly with the raw survey data files. For NHIS, we pull harmonized data from IPUMS.[D10] Across all 3 survey sources, we follow convention and define ever smokers as those individuals who have smoked at least 100 cigarettes in their lifetime. Current smokers are individuals who have ever smoked and who report smoking at least some days in the past month. Heavy smokers are individuals who have ever smoked and who report smoking every day in the past month. We can back out former–smokers by subtracting current smokers from ever smokers, and likewise we can back out light smokers by subtracting heavy smokers from current smokers. We express each smoking–status as a population–level prevalence, weighting by the applicable survey weights.

Each survey data source has strengths and weaknesses relative to one another. BRFSS contains by far the largest number of observations, followed by NSDUH, followed by NHIS. Larger sample sizes generally make our estimates of smoking prevalences more precise by reducing measurement error, and also ensures that a greater number of demographic groups is available in our analysis, improving statistical power.[D11] BRFSS is also the only survey data that contains urban status, unlike NHIS and NSDUH. A notable weakness of BRFSS, however, is a change made in 2011, when BRFSS begins including cell–phone respondents and begins a new weighting–method known as *raking*. This presents challenges in interpreting pre–2011 outcome levels to post–2011 outcome levels.[D12] In contrast, NSDUH and NHIS both contain consistent variables for smoking intensity/frequency outcomes, which BRFSS lacks. NSDUH contains fewer observations than NHIS, but utilizes 5-race/ethnicity categories instead of 3-race categories, while NHIS contains census region (NSDUH contains no geographic indicators). Lastly, NSDUH contains coarser age-groupings, while BRFSS and NHIS contain single–year age.[D13]

For our smoking–intensity/frequency measures (NHIS, NSDUH), we define average cigarettes smoked per day per capita and average days smoked in the past month per capita. We calculate past–month average days smoked per capita by assigning 30 days to heavy smokers and the average of the past–month average days smoked for light smokers. We apply Bayesian Shrinkage (Morris,

[D10] IPUMS provides harmonized NHIS data at https://nhis.ipums.org/nhis/. BRFSS data is available starting in 1984 but only a handful of states participated prior to 1990.

[D11] We use BRFSS to construct our smoking–share measure for this reason, and because BRFSS contains urban status, in our e–cigarette analysis.

[D12] In our results, this 2011–change appears to attenutate our results, but otherwise our BRFSS results are quite comparable to those from NHIS and NSDUH.

[D13] For BRFSS and NHIS, we use 5-year groupings starting from 30–34 through 75–79 in our estimation sample. For NSDUH, we use ages 30–34, 35–49, 50–64, and 65–99. Our NSDUH panel does not exclude groups greater than age 80.

1983) to outcomes derived from survey data to account for group–specific measurement error. We discuss shrinkage further in Section 3.3.

Our smoking outcomes are comparable across surveys both in levels and in trends over time. Starting with the first year of our NSDUH data, from 2002–2019, the population–weighted current smoking rate for adults ages 18 and older fell 8.2ppt from 22.3% in 2002 to 14.1% in 2019 in NHIS. For BRFSS, current smoking fell 7ppt from 22.0% in 2002 to 15.0% in 2019. For NSDUH, current smoking fell 7.4ppt from 24.5% in 2002 to 17.1% in 2019.

From 2002–2019, the population–weighted daily smoking rate for adults ages 18 and older fell 7.2ppt from 18.3% in 2002 to 11.1% in 2019 in NHIS. For BRFSS, daily smoking fell 6.7ppt from 17.0% in 2002 to 10.3% in 2019. For NSDUH, daily smoking fell 6.5ppt from 17.5% in 2002 to 11.0% in 2019.

From 2002–2019, the population–weighted past–month average cigarettes smoked for adults ages 18 and older fell by 1.7 cigarettes per capita per day from 3.3 cigarettes per capita per day in 2002 to 1.6 cigarettes per capita per day in 2019 in NHIS. For NSDUH, average cigarettes smoked fell by 1.9 cigarettes per capita per day from 3.5 cigarettes per capita per day in 2002 to 1.9 cigarettes per capita per day in 2019.

From 2002–2019, the population–weighted past–month average days smoked for adults ages 18 and older fell by 2.6 days per capita per month from 6.6 days per capita per month in 2002 to 4.0 days per capita per day per month in 2019 in NHIS. For NSDUH, average days smoked fell by 2.3 days per capita from 6.4 days per capita per month in 2002 to 4.1 days per capita per month in 2019.

D.3 Additional Information on Share Measure

Our daily smoing–share measure (DSSM) is the sample–weighted mean population–level daily smoking prevalence for each demographic group for 1983 (NRT analysis) or 2005–2006 (e–cigarette analysis). We weight using the applicable survey data weights (Table D2). We apply Bayesian shrinkage to our smoking (and vaping) share measures to address group–specific measurement error. Unless otherwise stated we utilize the shrunk smoking shares throughout this paper (Section 3.3 for details on our Bayesian shrinkage procedure). We also use all of the same procedures described above to construct alternative share measures, including a daily–vape share measure and smoking share measures for alternative years, for use in sensitivity analyses.

For our mortality analysis of 1984 NRT drug introduction, we construct our DSSM from 1983 NHIS data.[D14] Our estimation sample of ages 30–79 with non–missing smoking shares consists of 287 groups and 15,400 survey respondents. On average, each group's smoking share is constructed using 40 survey respondents. In our NRT estimation sample, the population–weighted mean of our 1983 DSSM is 28.2% (SE = 5.2%) before applying Bayesian shrinkage. After applying Bayesian shrinkage, the mean is 27.9% (SE = 0.5%).[D15]

For our mortality analysis of e–cigarette introduction in 2007 and drug exemption in 2011, we construct our DSSM from 2005–2006 BRFSS data. BRFSS contains significantly larger sample sizes compared to the other surveys, making it our preferred data source to construct our smoking shares. We use 2 years to improve precision, particularly for small demographic groups, in the smoking share measure.[D16] Our estimation sample of ages 30–79 with non–missing smoking shares consists of 719 groups and 399,610 survey respondents. On average, each group's smoking share is constructed using 503 survey respondents. In our estimation sample, the population–weighted mean of our 2005–2006 DSSM is 14.4% (SE = 1.6%) before applying Bayesian shrinkage. After applying Bayesian shrinkage, the mean is 14.3% (SE = 0.2%).

We study effects of 2007 e–cigarette introduction/drug exemption on smoking across three surveys individually, and for each survey we use the 2-year daily smoking share generated from within the same survey. Our e–cigarette smoking–outcome estimation sample for BRFSS, NHIS, and NSDUH consists of 719, 219, and 39 groups, respectively. The total number of survey respondents used to construct our smoking share for our estimation samples are 281,666, 17,804, and 15,978 respondents in total, respectively. Each group's smoking share consists of, on average, 392, 81, and 410 survey respondents, respectively. The population–weighted mean of our 2-year DSSM, before Bayesian shrinkage, is 14.1% (SE = 2.1), 17.0% (SE = 2.5%), and 16.2% (SE = 1.2%), respectively. After Bayesian shrinkage, the population–weighted weighted mean of our 2-year DSSM is 14.3% (SE = 0.2%), 16.9% (SE = 0.4%), and 16.2% (SE = 1.1%), respectively.

Our DSSM contains significant variation by demographic characteristics. Consider our 2005–2006 2-year DSSM from BRFSS. The age group with the highest daily smoking share is ages 45-49 at

[D14]We use just a single year since smoking data was not collected by NHIS in 1982. However, we conduct a sensitivity analysis using a smoking share measure created using the first two years of NHIS smoking data, from 1976–1977 data, in Section I.

[D15]We apply Bayesian shrinkage using adults ages 18 and older. The shrunk means in our age 30–79 estimation sample are therefore slightly different from the unshrunk means because the adult population–average smoking rate is the prior that smoking rates are shrunk towards, not the age 30–79 average.

[D16]We conduct a sensitivity analysis using a smoking share measure created using the first 2 years of our e–cigarette analysis, 1997–1998, in Section I.

18.2% on average, while the age group with the lowest daily smoking share is ages 75–79 at 5.4% on average. The smoking share–age gradient is non–linear: the smoking share increases linearly from 13.9% for ages 30–34, peaks with ages 45-49, and falls linearly with age reaching its minimum for ages 75–79. By 5-race/ethnicity, American Indian non-Hispanics have the highest daily smoking share at 19.6%, followed by White non-Hispanics at 15.5%, Black non-Hispanics at 14.9%, Hispanics at 9.8%, and Asian/Pacific Islanders have the lowest share at 6.5%. By census region, the Midwest has the highest daily smoking share at 15.9%, followed by the South at 15.7%, the Northest at 14.0%, and the West census region at 11.0%. The daily smoking share is 15.4% on average for men and 13.3% for women. The daily smoking share is higher for rural groups at 18.1%, compared to urban groups at 13.6%.

Age is the most predictive demographic characteristic of our DSSM and trends in logged SAM. A regression of 2005–2006 daily smoking shares from BRFSS on our demographic dimensions (sex, 5-race/ethnicity, region, urban, 5-year age) yields an adjusted R-squared of 71.9%. Dropping age-group as a predictor reduces the model's adjusted R-squared by 38.9ppt; dropping race by 16.4ppt; region by 5.9ppt; urban by 4ppt; sex by 2.7ppt. A regression of logged SAM on our demographic dimensions yields an adjusted R-squared of 98.85%. Dropping age-group as a predictor reduces the model's adjusted R-squared by 87.6ppt; dropping sex by 3.3ppt; race by 3ppt; region by 0.2ppt; urban by 0.1ppt.

Demographic variation in our 1983 1-year DSSM from NHIS shows similar patterns to our 2005–2006 daily smoking share from BRFSS overall. The overall mean daily smoking rate was significantly higher in the 1980's, however. By 5-year age groups, ages 45-49 have the highest mean daily smoking share at 33.1%. Ages 75–79 have the lowest mean daily smoking share at 12.3%. The pattern of daily smoking by age is similar to the 2005–2006 pattern, with smoking rates higher across all age groups. Ages 30–34 have a 30% mean daily smoking mean, which increases to its peak daily smoking share at ages 45-49, and then falls with age monotonically. By 3-race categories, Black non-Hispanics have the highest mean daily smoking share at 28.1%, followed by Whites at 27.9%, and all other races at 22.7%. The South census region has the highest mean smoking share at 29.4%, followed by the Midwest at 28.5%, the Northeast at 26.6%, and West census region at 25.9%. The mean daily smoking share for men is 31.0%, and 25.0% for women. The mean daily smoking share for urban groups is 28.1% and 26.8% for rural groups.

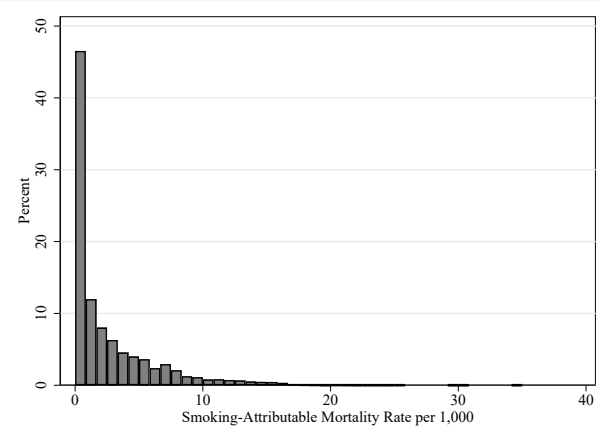
Lastly, our 2005–2006 DSSM from BRFSS is significantly correlated with 2016–2018 ever–vape share and heavy–vape share measures. The pair-wise correlation coefficient between our 2005–2006

daily smoking share and 2016–2018 ever–vape prevalence is 0.49 ($p < 0.01$). The correlation between our 2005–2006 daily smoking share and the 2016-2019 daily–vape prevalence is 0.46 ($p < 0.01$). We can further examine how ever– and heavy–vape prevalence in 2016–2018 varies by quartile of our 2005–2006 daily smoking share (p25 = 10.5%, p50 = 14.8%, p75 = 18.2%). For groups in the lowest quartile of our daily smoking share, their ever–vape use was 11.9% and heavy–vape use was 0.5% from 2016–2018. For groups in the second–lowest quartile, their ever–vape use was 15.6% and heavy–vape use was 1.0% from 2016–2018. For groups in the second–highest quartile, their ever–vape use was 23.7% and heavy–vape use was 1.2% from 2016–2018. Lastly, for groups in the highest quartile of our 2005–2006 daily smoking share measure, their ever–vape use was 24.9% and heavy–vape use was 1.8% from 2016–2018.

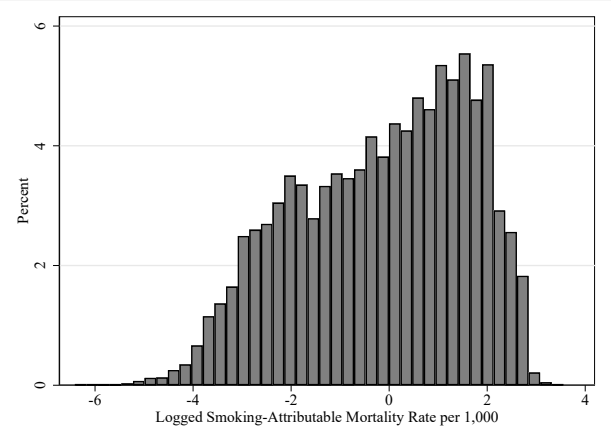
Appendix E Functional Form

Figure E1: Distribution of Smoking–Attributable Mortality and Current Smoking Prevalence

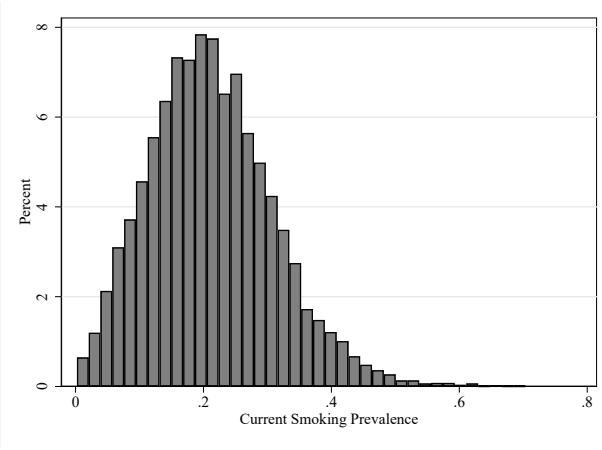
(a) SAM



(b) Logged SAM



(c) Current Smoking Prevalence



Notes: Mortality data is from NVSS. Smoking data is from NHIS. Data is shown for years 1979-2019.

Appendix F Mortality & Smoking Calculations

In our analysis, we estimate the causal impact of drug introduction/exemption (the *intervention*) on mortality rate growth. Our outcome is logged SAM rates (per 1,000 population) and our independent variable is the smoking share measure (daily smoking rate prior to intervention). After scaling our estimates by the mean smoking share in our sample, we interpret our event study leads/lags as the effect of the intervention on percent growth in the mortality rate (per 1,000 population), evaluated at the mean smoking share in our sample. Denoting event time as k, the event study lag in period k is $\hat{\beta}_k$, which is relative to the reference period, the year before the intervention ($\beta_0 = 0$).

To convert percent–changes in mortality rates to level–changes in mortality rates, we multiply β_k by the average (population-weighted) mortality rate in the reference period year.[F1] We then convert changes in the mortality rate to total changes in mortality by dividing by 1,000, so that mortality rates are in deaths per–capita, not deaths per 1,000 population.[F2] Lastly, we multiply by the total population in our estimation sample in the reference period.[F3] This final *net* change in mortality in period k we denote as D_k.

To calculate consumer surplus, we need to convert net changes in mortality to net changes in life years (LY). If the intervention increases mortality on net, then this translates to a *reduction* in life years (fewer years of life). Likewise, reductions in mortality translate to *gains* in life years (additional years of life). We can calculate the cumulative gain/reduction in life years following the intervention directly by taking the net mortality reduction/increase in each year after the intervention, multiplying by the number of years remaining until last year, and then summing these estimates starting with the year of the intervention through the final year. This calculation does not require knowledge of the the true change in life-expectancy resulting from the intervention, which is unobserved.

To illustrate, suppose that intervention A causes 150 individuals to each lose 1 year of life. 100 deaths that would have occurred in year 1 (absent the intervention) now occur in year 0, and 50 deaths that would have occurred in year 2 (absent the intervention) now occur in year 1. As the researcher, we do not observe the reduction of 1 life–year per person, but we do observe the net change in mortality in each year. The *net* change in deaths is therefore +100 deaths in year 0, −50

[F1] For example, if the mean mortality rate is 50 deaths per 1,000, and we estimate a 10% decrease in mortality rates, then after multiplying we have a reduction of 5 deaths per 1,000.

[F2] From the previous example, the reduction of 5 deaths per 1,000 population, divided by 1,000, translates to a reduction of 0.005 deaths per capita.

[F3] From before, if the total population in the reference period year is 1 million, then a reduction of 0.005 deaths per–capita, multiplied by 1 million people, yields a reduction of 5,000 deaths

deaths in year 1, and −50 deaths in year 2.[F4] The cumulative change since year 0 is 300 LY's lost.[F5] The cumulative change since year 1 is 200 LY's lost.[F6] The cumulative change since year 2 is 150 LY's lost.[F7] In conclusion, our estimate of 150 LY's lost since the intervention is equal to the true estimate of 150 LY's lost (150 deaths times 1 year of life lost per decedent).

In contrast, suppose that intervention B causes 150 individuals to each gain 1 year of life. 100 deaths that would have occurred in year 0 (absent the intervention) now occur in year 1, and 50 deaths that would have occurred in year 1 (absent the intervention) now occur in year 2. As the researcher, we do not observe the gain of 1 life–year per person, but we do observe the net change in mortality in each year. The *net* change in deaths is therefore −100 deaths in year 0, +50 deaths in year 1, and +50 deaths in year 2.[F8] The cumulative change since year 0 is 300 LY's gained.[F9] The cumulative change since year 1 is 200 LY's gained.[F10] The cumulative change since year 2 is 150 LY's gained.[F11] In conclusion, our estimate of 150 LY's gained since the intervention is equal to the true estimate of 150 LY's gained (150 deaths delayed by 1 year, or 150 LY's gained).

Extending the examples above, the *cumulative* life years gained since the intervention by the end of period m is therefore the sum from $k = 0$ to $k = m$ of the net mortality change in period k, multiplied by the number of years remaining until period $k = m$ (including period k), multiplied by -1 to convert mortality gains/losses to life–year losses/gains:

$$\text{Cumulative Life-Years Gained/Lost} = \sum_{k=0}^{m} (m - k + 1) \cdot (-D_k) \tag{6}$$

[F4] 100 individuals who would have died in year 1 absent the intervention have now died in year 0, so there is a net mortality increase of 100 deaths in year 0. In year 1, the net change in mortality is 50 additional deaths due to the intervention, minus 100 deaths that were shifted from year 1 to year 0, or −50 deaths in year 1. In year 2, there are no deaths caused by the intervention, but 50 deaths were shifted earlier in time from year 2 to year 1, so the net change in mortality is −50 deaths in year 2.

[F5] A net increase of 100 deaths in year 0, multiplied by 3 years remaining until end of year 2, yields 300 LY's lost.

[F6] A net decrease of 50 deaths in year 1, multiplied by 2 years remaining until the end of year 2, yields 100 LY's gained. Adding the cumulative change in LY's since year 0 (300 LY's lost) yields 200 LY's lost.

[F7] A net reduction of 50 deaths in year 2, multiplied by 1 year remaining until end of year 2, yields 50 LY's gained. Adding the previous change since year 1 (200 LY's lost) yields 150 LY's lost.

[F8] 100 individuals who would have died in year 0 absent the intervention have now died in year 1, so there is a net mortality reduction of 100 deaths in year 0. In year 1, the net change in mortality is 50 fewer deaths due to the intervention, plus 100 deaths that were shifted from year 0 to year 1, or +50 deaths in year 1. In year 2, there are no deaths caused by the intervention, but 50 deaths were shifted later in time from year 1 to year 2, so the net change in mortality is +50 deaths in year 2.

[F9] A net reduction of 100 deaths in year 0, multiplied by 3 years remaining until end of year 2, yields 300 LY's gained.

[F10] A net increase of 50 deaths in year 1, multiplied by 2 years remaining until the end of year 2, yields 100 LY's lost. Adding the cumulative change in LY's since year 0 (300 LY's gained) yields 200 LY's gained.

[F11] A net increase of 50 deaths in year 2, multiplied by 1 year remaining until end of year 2, yields 50 LY's lost. Adding the previous change since year 1 (200 LY's gained) yields 150 LY's gained by end of year 2.

The average change in life years per year is simply the cumulative life–years gained since the intervention by the end of period m, divided by the total number of periods, $m+1$:

$$\text{Average Annual Life-Years Gained/Lost} = \sum_{k=0}^{m} \frac{m-k+1}{m+1} \cdot (-D_k) \tag{7}$$

We now turn to our calculation of net changes in smokers and cumulative increases/reductions in smoking years. Recall that the current smoking prevalence (CS) is defined as the proportion of ever smokers who currently smoke at least 1 day in the past month.[F12] Changes in current smoking can occur through two channels: changes in smoking initiation or changes in smoking–cessation. Changes in the population–level daily smoking prevalence (DS), or current smokers who smoke everyday, can occur through three channels: changes in the frequency of smoking among current smokers, changes in smoking–cessation among daily smokers, or changes in smoking–initiation among daily smokers. We assume that the effect of mortality on current smoking prevalence is negligible.

In our survey–data analysis, we estimate the causal impact of drug introduction/exemption (the *intervention*) on the population–level current smoking prevalence. Our outcome is the current smoking prevalence and our independent variable is the smoking share measure (daily smoking rate prior to intervention). After scaling our estimates by the mean smoking share in our sample, we interpret our event study leads/lags as the *cumulative* effect of the intervention on the current smoking rate in levels, evaluated at the mean smoking share in our sample. Denoting event time as k, the event study lag in period k is $\hat{\beta}_k$, which is relative to the reference period, the year before the intervention ($\beta_0 = 0$). Since smoking–status is a status that can turn on and off, $\hat{\beta}_k$ denotes the cumulative change in the current smoking prevalence since the intervention.

To convert cumulative net changes in the current smoking prevalence to cumulative net changes in smokers, we multiply β_k by the total population in our estimation sample in the reference period.[F13] The *net* change in smokers in period k we denote as S_k.

The *cumulative* net change in the population–level number of smokers since the intervention by period m is given by the event study lag in period m, S_m. The *year–over–year* net change in smokers is equal to the difference of cumulative changes in smokers in year k, S_k, and year $k-1$,

[F12] Ever smokers are those who have smoked at least 100 cigarettes in their lifetime. Ever smoking status cannot "turn off" for individuals, but the population–level ever smoking rate can decrease over time due to changes in smoking initiation or other demographic changes.

[F13] If the total population in the reference period year is 1 million, and we estimate an average reduction in the current smoking prevalence of 5 ppt, then multiplying yields a reduction of 50,000 smokers.

S_{k-1}. The average net change in smokers in the population by period m is simply the cumulative change in smokers by period m divided by the number of periods since the intervention, or $\frac{S_m}{m}$

Smoking years (SY) are the additional years that an individual in the population is classified as a current smoker. A gain (reduction) in smoking years indicates greater (less) usage of combustible tobacco in the population. Measuring smoking–years accumulated up to a point in time does not account for future accumulation of smoking–years; e.g., somebody that quits permanently will contribute a smoking year reduction in each year from that point forward.[F14] Cumulative gains/reductions in SY's can be calculated by summing the cumulative change in smokers in each period following the intervention up to period m.

To illustrate, suppose that intervention A causes 50 individuals to start smoking permanently in year 0 and 50 individuals to start smoking permanently in year 1. Suppose that an additional 50 individuals start smoking in year 0, but later quit permanently by the end of year 1. Like mortality, we only observe the *net* change in smokers in each year. The net change in smokers is +100 in year 0, +100 smokers in year 1, and +100 smokers in year 2.[F15] The cumulative change in SY's in year 0 is 100 additional SY's.[F16] The cumulative change in SY's in year 1 is 200 additional SY's.[F17] The cumulative change in year 2 is 300 additional SY's.[F18] Our final estimate of 300 additional SY's accumulated since the intervention aligns with the true increase of SY's.[F19]

In contrast, suppose that intervention B causes 50 smokers to permanently quit smoking in year 0 and 50 smokers to permanently quit in year 1. Suppose that an additional 50 smokers quit smoking in year 0, but later relapse by the end of year 1 and return to smoking. Like mortality, we only observe the *net* change in smokers in each year. The net change in smokers is −100 in year 0, −50 smokers in year 1, and −100 smokers in year 2.[F20] The cumulative change in SY's in year 0 is

[F14] We calculate cumulative changes in SY's through year 2019, as we do with life–years, but smokers who quit permanently continue to reduce SY's in the population beyond 2019.

[F15] 100 individuals started smoking in year 0. In year 1, 50 smokers quit, 50 individuals who started smoking in year 0 continue to smoke, and 50 other individuals begin smoking. In year 2, 50 smokers who started smoking in year 0 continue to smoke and 50 individuals who started smoking in year 1 continue to smoke.

[F16] 100 additional smokers since the intervention in year 0 multiplied by 1 year yields a net increase of 100 SY's.

[F17] 100 additional smokers since the intervention in year 1 multiplied by 1 year yields 100 additional SY's. Adding the cumulative change in year 0 (100 additional SY's) yields 200 fewer SY's in year 1.

[F18] 100 additional smokers since the intervention in year 2 multiplied by 1 year yields 100 additional SY's. Adding the cumulative change in year 1 (200 additional SY's) yields 300 additional SY's accumulated by year 2.

[F19] The 50 smokers who began smoking permanently in year 0 contribute 150 additional SY's. The 50 smokers who began smoking in year 0 but quit by end of year 1 contribute 100 additional SY's. The 50 smokers who began smoking permanently starting in year 1 contribute 100 additional SY's. The cumulative change in SY's by end of year 2 is therefore $100 + 100 + 100 = 300$

[F20] 100 smokers quit in year 0. In year 1, 50 smokers continue to abstain from the prior year. Additionally, 50 additional smokers quit and 50 smokers who quit in the previous year relapse, which offset each other. In year 2, 50 smokers who quit in year 0 continue to abstain and 50 smokers who quit in year 1 continue to abstain.

100 fewer SY's.[F21] The cumulative change in SY's in year 1 is 150 fewer SY's.[F22] The cumulative change in year 2 is 350 fewer SY's.[F23] Our final estimate of 350 fewer SY's accumulated since the intervention aligns with the true reduction of SY's.[F24]

Extending the examples above, the cumulative change in smoking–years by period m is therefore the sum from $k = 0$ to $k = m$ of the cumulative net change in the number of smokers in period k, S_k:

$$\text{Cumulative Smoking-Years Gain/Lost} = \sum_{k=0}^{m} S_k \tag{8}$$

Above, the net cumulative change in smokers in each period since the intervention is multiplied by 1 year. The average annual net change in smoking–years by period m is simply the sum of cumulative changes in the count of smokers in each year following the intervention divided by the number of periods since the year before the intervention, or $m + 1$:

$$\text{Average Annual Net Change in Smoking-Years} = \sum_{k=0}^{m} \frac{S_k}{m+1} \tag{9}$$

[F21] 100 fewer smokers multiplied by 1 year yields a net reduction of 100 SY's.

[F22] 50 fewer smokers in year 1 since the intervention multiplied by 1 year yields 50 fewer SY's. Adding the cumulative change in year 0 (100 fewer SY's) yields 150 fewer SY's in year 1.

[F23] 100 fewer smokers in year 2 since the intervention multiplied by 1 year yields 200 fewer SY's. Adding the cumulative change in year 1 (150 fewer SY's) yields 350 fewer SY's accumulated by year 2.

[F24] The 50 smokers who quit permanently in year 0 contribute 150 fewer SY's. The 50 smokers who quit in year 0 but relapse by end of year 1 contribute 100 fewer SY's. The 50 smokers who quit permanently starting in year 1 contribute 100 fewer SY's. The cumulative change in SY's by end of year 2 is therefore $-150 - 100 - 100 = -350$

Appendix G Social Surplus Calculation

We denote the net change in mortality in period k after treatment as D_k ($D_{-1} = 0$ in reference period), where k is the number of periods following drug introduction/exemption.

From Equation 6, for NRT drug introduction, we calculate changes in life–years during our 9-year post-period (1984–1992); for e–cigarette drug introduction, we calculate life–years during our 13-year post-period (2007–2019); for e–cigarette drug exemption, we calculate life–years during our 9-year post-period (2011–2019).

To calculate consumer surplus, we borrow from the methodology of Philipson and Jena (2006). First, we calculate the net mortality gain/reduction in each year following drug introduction/exemption, or D_t. We then calculate the cumulative life–years gained/lost from Equation 6, denoted by ΣLY_t. The cumulative life-year estimates are calculated from the net change in mortality in year t and the number of years remaining until the final post-period. Sustained reductions in mortality following drug introduction lead to faster accumulation of life–years because of continuing life-year gains from previous deaths averted, plus additional life-year gained from contemporaneous mortality reductions in year t.

We then calculate the year–over–year change in cumulative life–years by taking the difference in cumulative life–years in year t minus cumulative life–years in year $t-1$. This allows us to estimate the additional life–years gained/lost in each year.

The additional life–years gained or lost in each year are then multiplied by $100,000 value of statistical life–year (VSLY), discounted to 2007 using a market interest rate of 3%, and then converted to $2019 using the consumer price index. The consumer surplus gained/lost in each year–the period VSLY–is then summed over time in order to calculate the cumulative VSLY since drug introduction.

Tables G1 and G2 show our consumer surplus calculations. Column 1 (D_k) shows the net mortality change in each year following drug introduction. Column 2 (ΣLY_t) shows the cumulative life–years gained/lost (positive life–years indicate mortality reductions, and vice versa). Column 3 shows the year–over–year difference in life–years accumulated in each year following drug introduction. Column 4 shows the period-VSLY, or the estimate from the previous column discounted to 2007 and expressed in $2019. Column 5 shows the cumulative VSLY in each year following drug introduction. We also include grand total VSLY, average annual VSLY, and the corresponding 95% confidence intervals.

To calculate producer surplus, we use estimates of market revenue by year from a variety of industry and government sources. We rely on Surgeon General reports, Statista Market Insights, Grand View Research, in addition to estimates from private industry. Because we are unable to find reliable estimates of market revenue from 2007-2009, we impute revenues of $0 during these years. After discounting to 2007 and adjusting for inflation, we then assume an average variable cost of 35%.[G1] Based on these calculations, we estimate a total producer surplus of $9.9 billion from 2011–2019 following e–cigarette drug exemption, or an average of $1.1 billion each year, expressed in 2019 dollars and discounted to 2007. Following e–cigarette introduction, we estimate a total producer surplus of $14.3 billion from 2007–2019, expressed in 2019 dollars and discounted to 2007.

[G1] https://philipmorrisinternational.gcs-web.com/static-files/a8eed5ab-aac5-410b-94ea-fa98bf659b0d

Table G1: Consumer Surplus Calculations: NRT Drug Introduction

Year	D_k (Deaths)	$\Sigma\,\mathrm{LY}_t$ (LY's)	$\Sigma\,\mathrm{LY}_t - \Sigma\,\mathrm{LY}_{t-1}$ (LY's)	Period–VSLY (Billions of \$2019)	Cumulative VSLY (Billions of \$2019)
1984	-3,462	31,162	31,162	\$7.44	\$7.44
1985	-800	37,564	6,403	\$1.48	\$8.92
1986	2,192	22,219	-15,345	-\$3.45	-\$5.47
1987	-2,117	34,923	12,704	\$2.77	\$8.24
1988	-1,142	40,633	5,710	\$1.21	\$9.45
1989	-6,253	65,646	25,012	\$5.13	\$14.58
1990	1,715	60,501	-5,145	-\$1.02	\$13.55
1991	-13,172	86,844	26,344	\$5.08	\$18.64
1992	-5,902	92,746	5,902	\$1.10	\$19.74
Grand Total D_k	-28,942 deaths			Grand Total VSLY	\$19.74 billion
	[-79,128, 21,245]				[-\$33.1b, \$72.6b]
Average Annual D_k	-3,216 deaths			Average Annual VSLY	\$2.19 billion
	[-8,792, 2,361]				[-\$3.7b,\$8.1b]

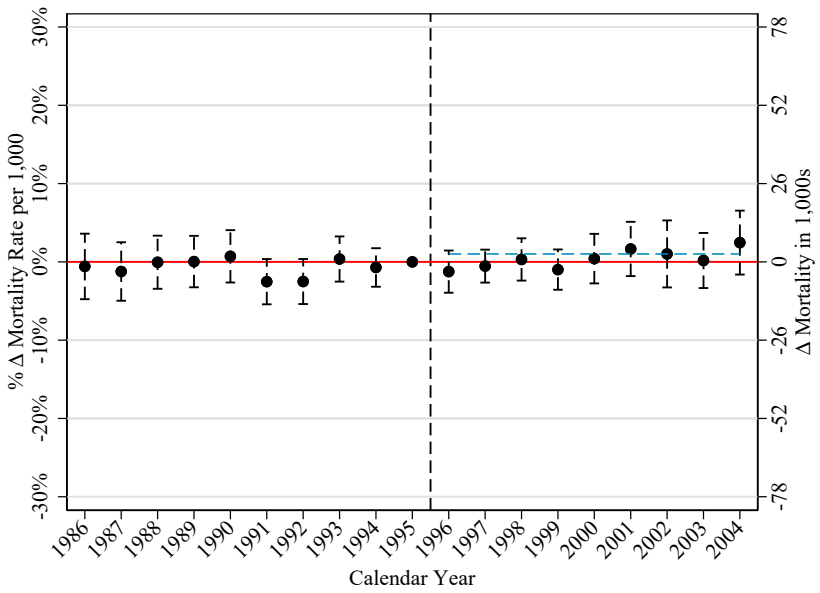
Table G2: Consumer Surplus Calculations: E–cigarette Drug Introduction/Exemption

Year	D_k (Deaths)	$\Sigma\,\mathrm{LY}_t$ (LY's)	$\Sigma\,\mathrm{LY}_t - \Sigma\,\mathrm{LY}_{t-1}$ (LY's)	Period–VSLY (Billions of \$2019)	Cumulative VSLY (Billions of \$2019)
2007	2,233	-29,028	-29,028	-\$3.47	-\$3.47
2008	-188	-26,774	2,254	-\$0.26	-\$3.21
2009	-7,126	51,609	78,383	\$8.82	\$5.61
2010	-3,918	90,793	39,184	\$4.28	\$9.89
2011	-9,268	174,202	83,408	\$8.83	\$18.73
2012	-7,849	236,996	62,794	\$6.45	\$25.18
2013	-12,018	321,119	84,123	\$8.38	\$33.56
2014	-14,977	410,980	89,861	\$8.68	\$42.25
2015	-19,152	506,738	95,759	\$8.98	\$51.23
2016	-22,359	596,174	89,435	\$8.14	\$59.36
2017	-24,897	670,865	74,692	\$6.59	\$65.95
2018	-28,412	727,690	56,824	\$4.86	\$70.82
2019	-32,931	760,621	32,931	\$2.74	\$73.55
Post E–cigarette Drug Introduction:					
Grand Total D_k	-180,862 deaths			Grand Total VSLY	\$73.55 billion
	[-229,903, -131,820]				[\$39.2, \$107.9b]
Average Annual D_k	-13,912 deaths			Average Annual VSLY	\$5.7 billion
	[-17,685, -10,140]				[\$3.0b,\$8.3b]
Post E–cigarette Drug Exemption:					
Grand Total D_k	-171,862 deaths			Grand Total VSLY	\$63.66 billion
	[-207,361, -136,363]				[\$47.0b, \$80.3b]
Average Annual D_k	-19,096 deaths			Average Annual VSLY	\$7.1 billion
	[-23,040, -15,151]				[\$5.2, \$8.9b]

Appendix H Over–the–Counter NRT Drug Introduction

Figure H1: Smoking–Attributable Mortality Event Studies, 1996 OTC NRT Introduction Date

(a) Smoking–Attributable Mortality



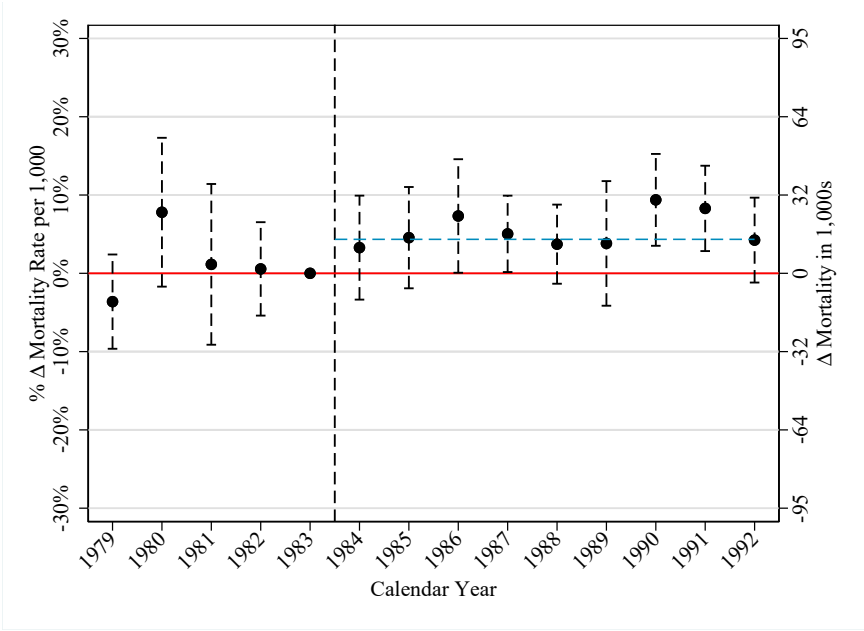
$\hat{\beta}_{Post\,1996}\,(SE) = 1.01\%\,(1.60\%)$; Δ LY $= -1,387$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. 1996 is used as the over–the–counter NRT introduction date. Our sample consists of 338 groups. The mean of our daily smoking share is 20.9%. Weighted mean mortality (in reference period year) is 1.8 deaths per 1,000 *** p<0.01, ** p<0.05, * p<0.1

Appendix I Robustness & Sensitivity

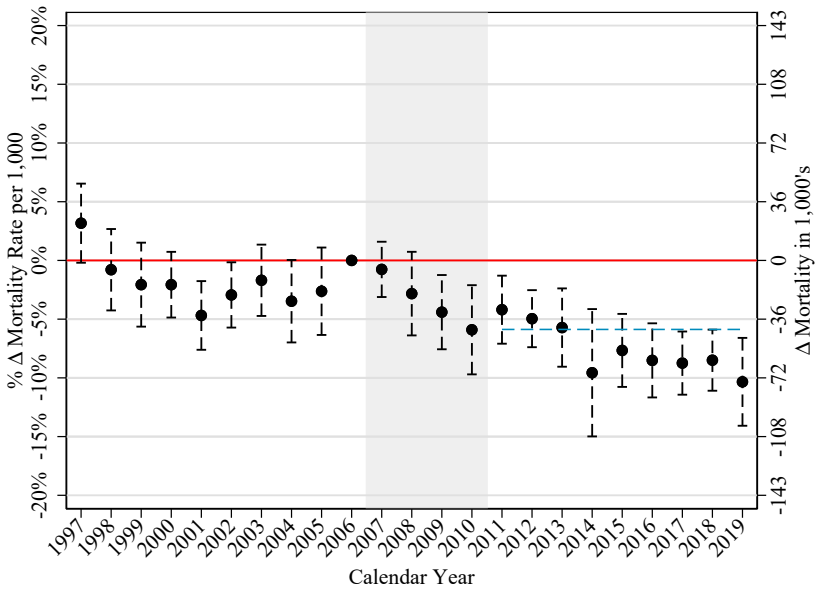
Figure I1: Event Studies for Smoking–Attributable Mortality, Unweighted

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = 4.34\%^{*}\,(2.26\%)$; Δ LY $= -81,247$

(b) E–cigarette Drug Exemption

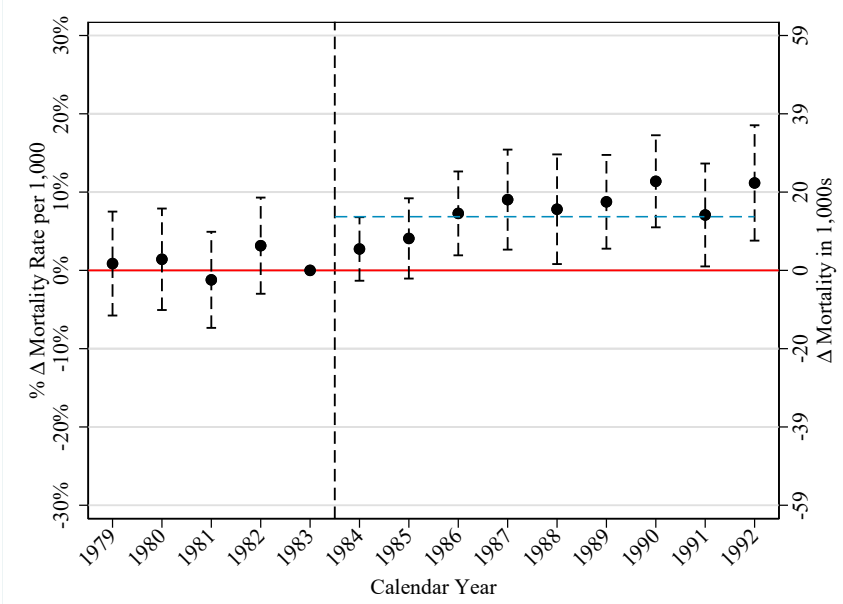


$\hat{\beta}_{Post\,2011}\,(SE) = -6.18\%^{***}\,(0.88\%)$; Δ LY $= 103,385$
$\hat{\beta}_{Post\,2007}\,(SE) = -5.03\%^{***}\,(1.00\%)$; Δ LY $= 82,945$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Regressions are unweighted. The unweighted mean of our DSSM is 26.4% for our NRT analysis and 14.0% for our e–cigarette analysis. Unweighted mean mortality (in reference period year) is 2.8 deaths per 1,000 for our NRT analysis and 2.1 deaths per 1,000 for our e–cigarette analysis. *** p<0.01, ** p<0.05, * p<0.1

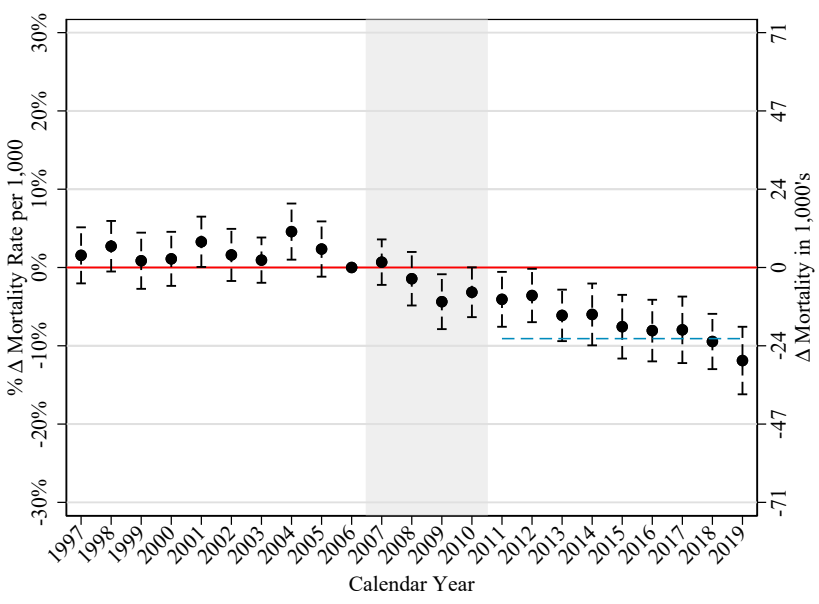
Figure I2: Event Studies for Smoking–Attributable Mortality, Alternative Smoking Shares

(a) NRT Drug Introduction, 1976–1977 DSSM



$\hat{\beta}_{Post\,1984}\,(SE) = 6.86\%^{**}\,(2.73\%)$; Δ LY $= -64,585$

(b) E–cigarette Drug Exemption, 1997–1998 DSSM

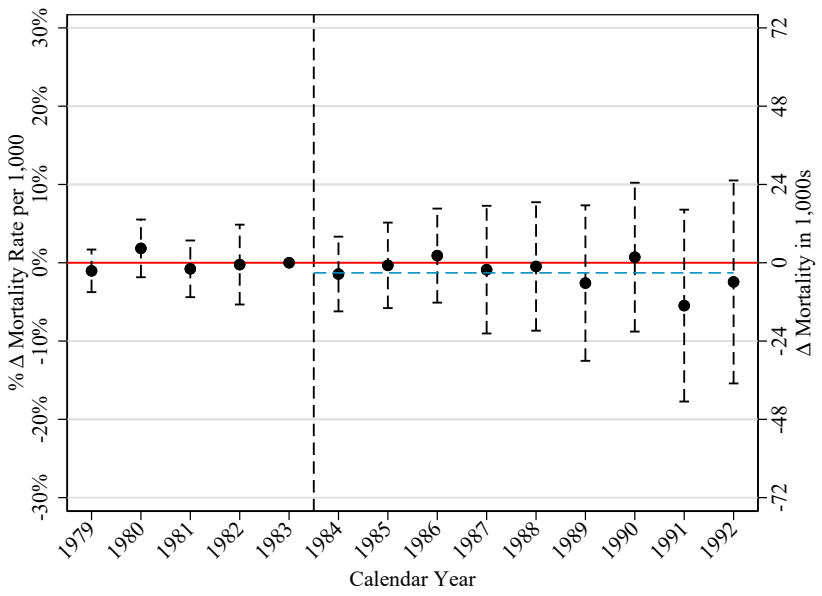


$\hat{\beta}_{Post\,2011}\,(SE) = -9.08\%^{***}\,(1.15\%)$; Δ LY $= 70,901$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.51\%^{***}\,(1.33\%)$; Δ LY $= 65,163$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Our DSSM spans 1976–1977 in our NRT analysis and 2002-2006 in our e–cigarette analysis. The weighted mean of our DSSM is 33.3% in our NRT analysis and 17.6% in our e–cigarette analysis. Weighted mean mortality (in reference period year) is 2.0 deaths per 1,000 in 1983 and 1.5 deaths per 1,000 in 2006.

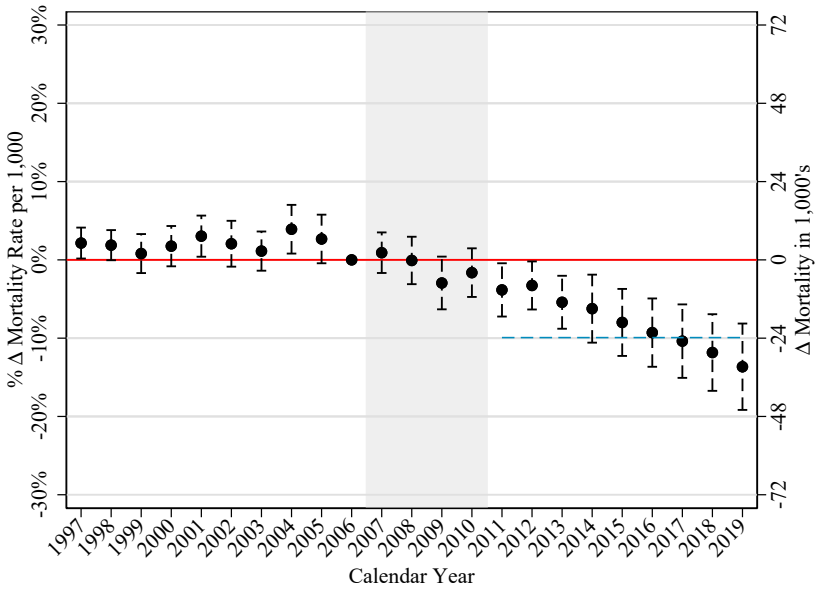
Figure I3: Event Studies for Smoking–Attributable Mortality, Alternative Specifications

(a) NRT Drug Introduction, Goodman-Bacon (2021) Time Trend



$\hat{\beta}_{Post\,1984}\,(SE) = -1.29\%\,(3.94\%);\ \Delta\ \text{LY} = 10,309$

(b) E–cigarette Drug Exemption, Goodman-Bacon (2021) Time Trend

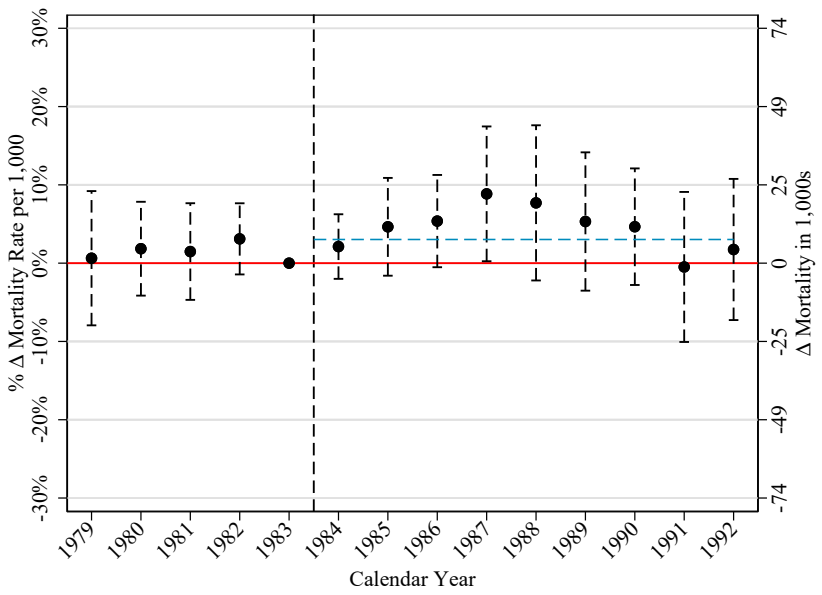


$\hat{\beta}_{Post\,2011}\,(SE) = -9.93\%^{***}\,(1.24\%);\ \Delta\ \text{LY} = 75,262$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.76\%^{***}\,(1.55\%);\ \Delta\ \text{LY} = 59,097$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. The Goodman-Bacon (2021) time trend specification uses a unit-specific linear trend estimated using pre-period years only, as opposed to a linear trend in event study leads.

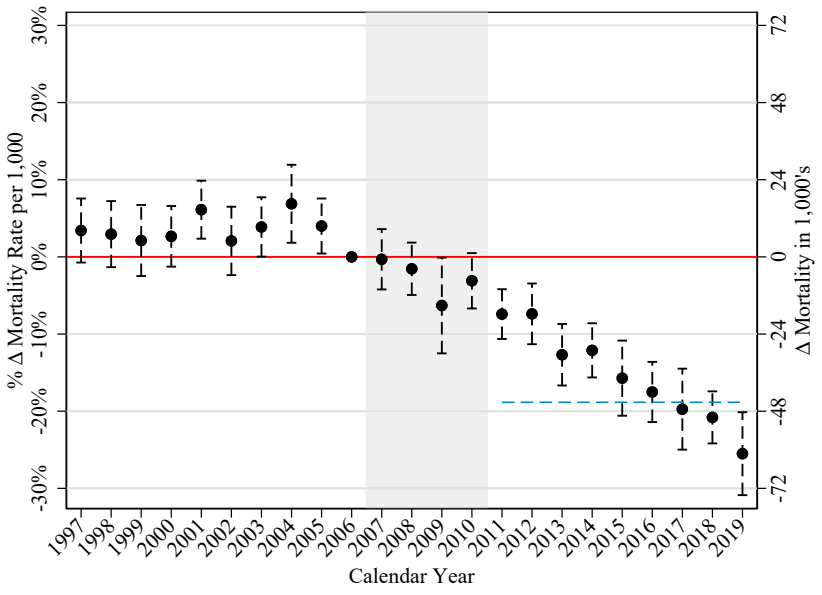
Figure I4: Event Studies for Smoking–Attributable Mortality, National

(a) Specific Rate, NRT Drug Introduction



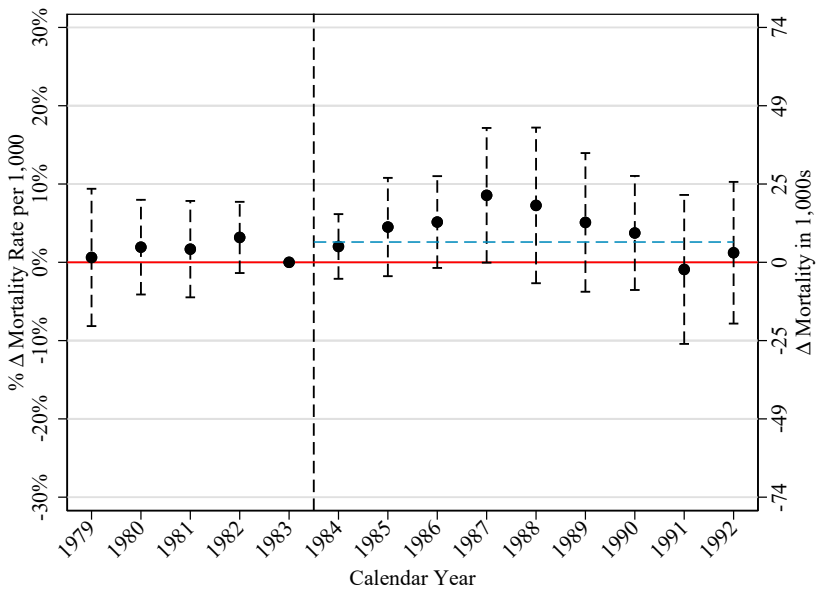
$\hat{\beta}_{Post\,1984}\,(SE) = 3.02\%\,(4.15\%)$; Δ LY $= -60,571$

(b) Specific Rate, E–cigarette Drug Exemption



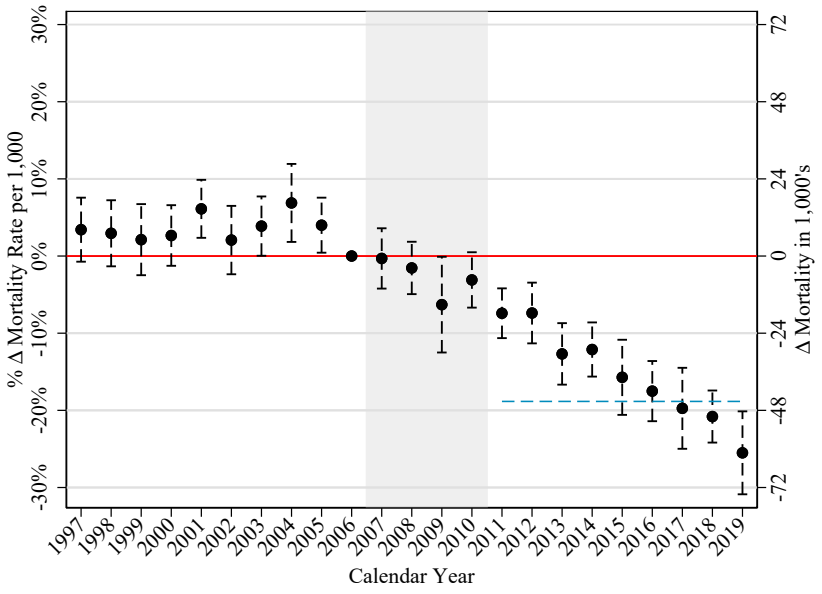
$\hat{\beta}_{Post\,2011}\,(SE) = -17.01\%^{***}\,(1.49\%)$; Δ LY $= 135,968$
$\hat{\beta}_{Post\,2007}\,(SE) = -13.39\%^{***}\,(1.61\%)$; Δ LY $= 112,885$

(c) Adjusted Rate, NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = 2.59\%\,(4.17\%)$; Δ LY $= -57,153$

(d) Adjusted Rate, E–cigarette Drug Exemption

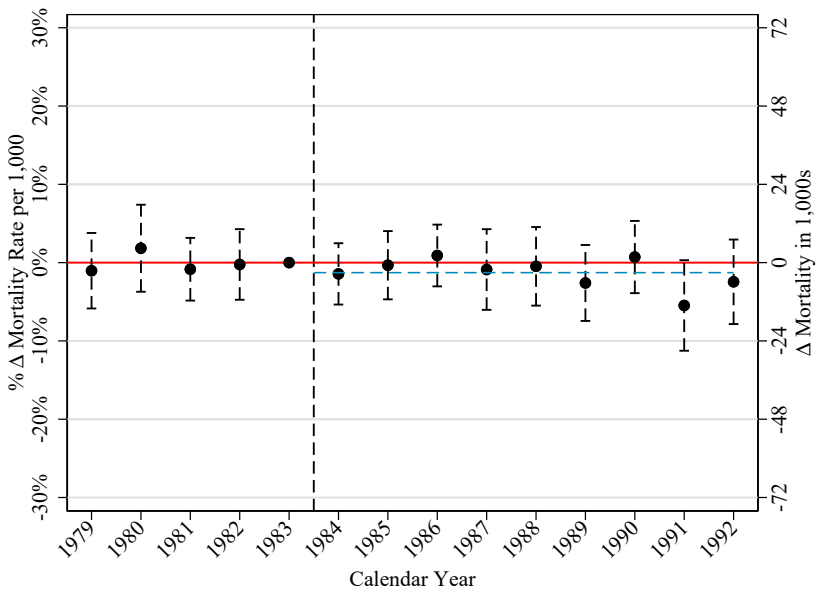


$\hat{\beta}_{Post\,2011}\,(SE) = -18.84\%^{***}\,(1.48\%)$; Δ LY $= 127,910$
$\hat{\beta}_{Post\,2007}\,(SE) = -14.96\%^{***}\,(1.60\%)$; Δ LY $= 127,080$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Demographic groups exclude census region and urban status. We show one set of results where mortality rates are adjusted, and a second set of results using specific rates. Sample consists of 55 groups for our NRT analysis and 100 groups for our e–cigarette analysis. The weighted mean of our DSSM is 27.8% for our NRT analysis and 14.3% for our e–cigarette analysis. Weighted mean mortality (in reference period year) is 2.2 deaths per 1,000 for our NRT analysis and 1.5 deaths per 1,000 for our e–cigarette analysis. When calculating adjusted rates, we use 1983 U.S. population and 2006 population in our NRT and e–cigarette analyses, respectively.

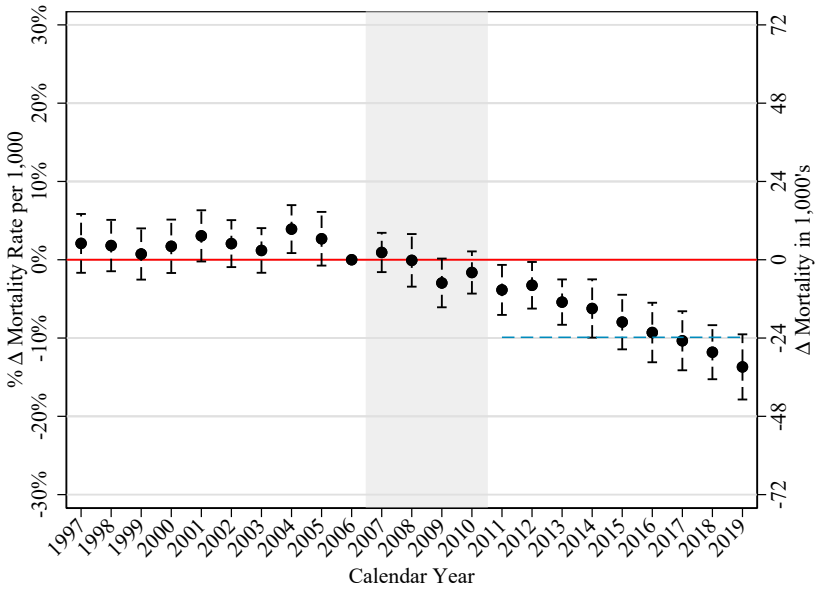
Figure I5: Event Studies for Smoking–Attributable Mortality, Bootstrapped Standard Errors

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = -1.28\%\,(1.78\%);\ \Delta\ \text{LY} = 21,852$

(b) E–cigarette Drug Exemption

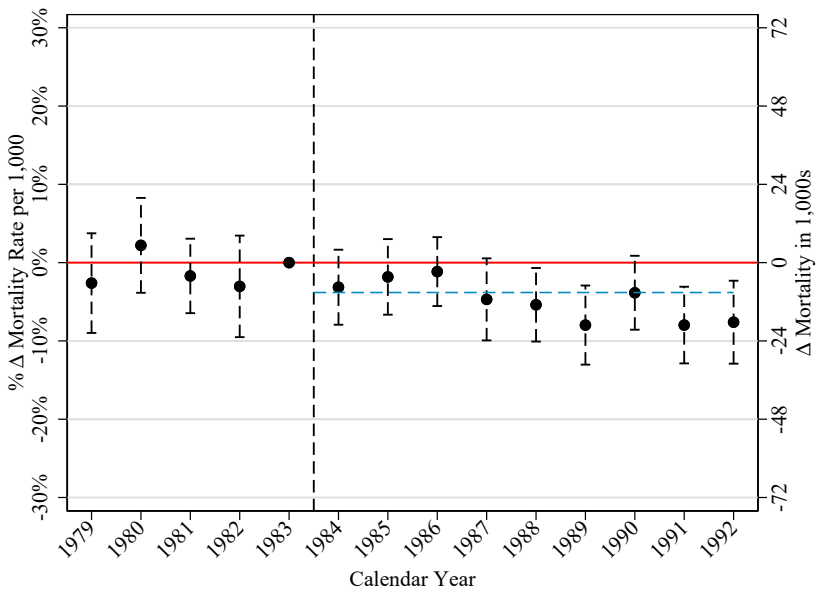


$\hat{\beta}_{Post\,2011}\,(SE) = -9.91\%^{***}\,(1.20\%);\ \Delta\ \text{LY} = 75,203$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.74\%^{***}\,(1.30\%);\ \Delta\ \text{LY} = 59,048$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Standard errors are bootstrapped using 100 replications.

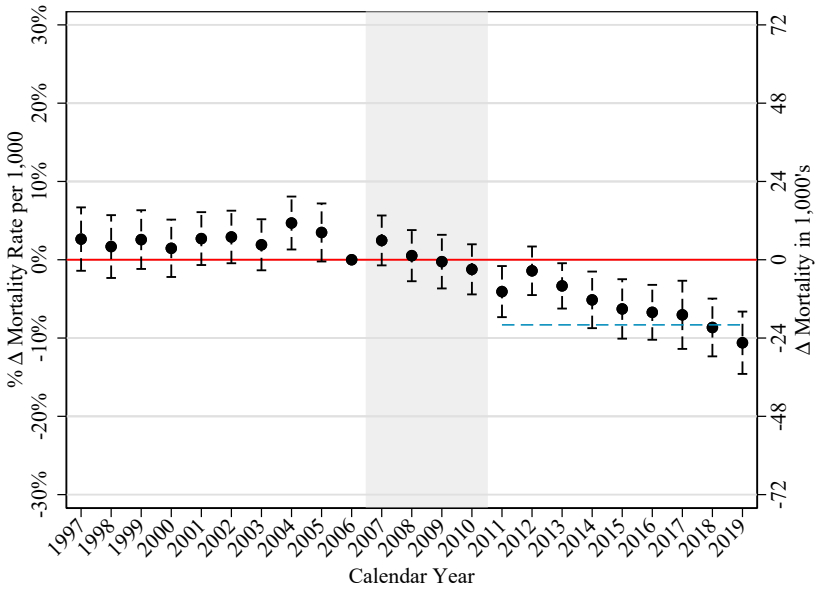
Figure I6: Event Studies for Smoking–Attributable Mortality, Additional Fixed Effects

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = -3.82\%^{**}\,(1.86\%);\ \Delta\ \text{LY} = 46,261$

(b) E–cigarette Drug Exemption

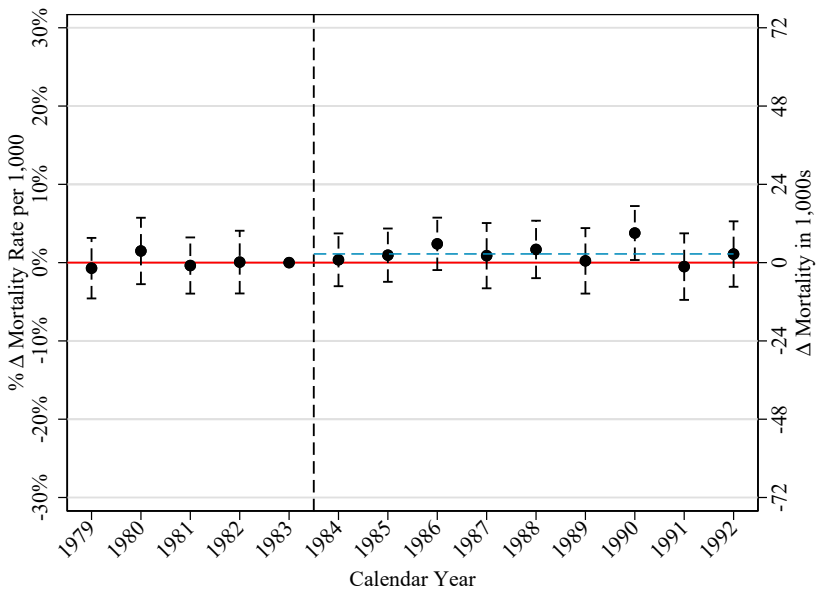


$\hat{\beta}_{Post\,2011}\,(SE) = -8.32\%^{***}\,(1.01\%);\ \Delta\ \text{LY} = 55,959$
$\hat{\beta}_{Post\,2007}\,(SE) = -6.38\%^{***}\,(1.17\%);\ \Delta\ \text{LY} = 34,422$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Regression controls for demographic group and additionally for race-by-year, sex-by-year, census region-by-year, and urban status-by-year fixed effects.

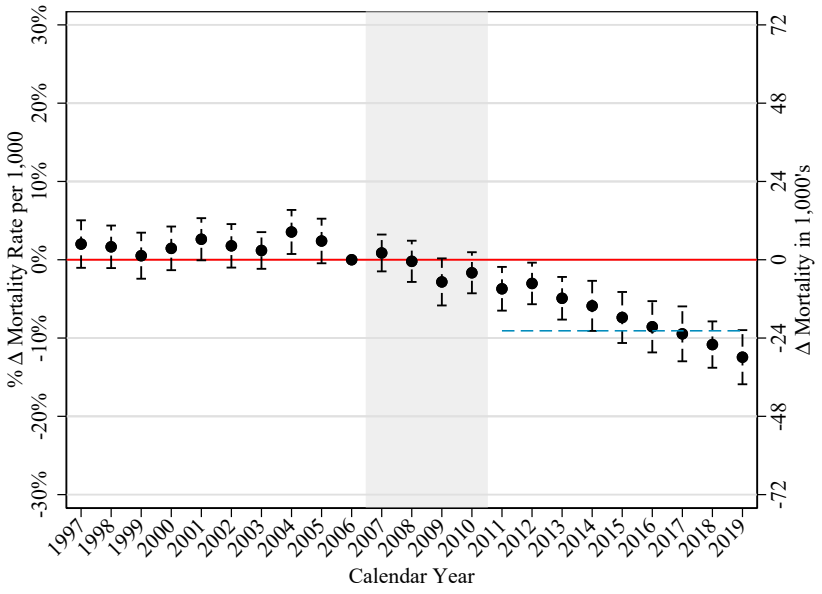
Figure I7: Event Studies for Smoking–Attributable Mortality, Unshrunk DSSM

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = 1.11\%\,(1.35\%);\ \Delta\ \text{LY} = -14,351$

(b) E–cigarette Drug Exemption

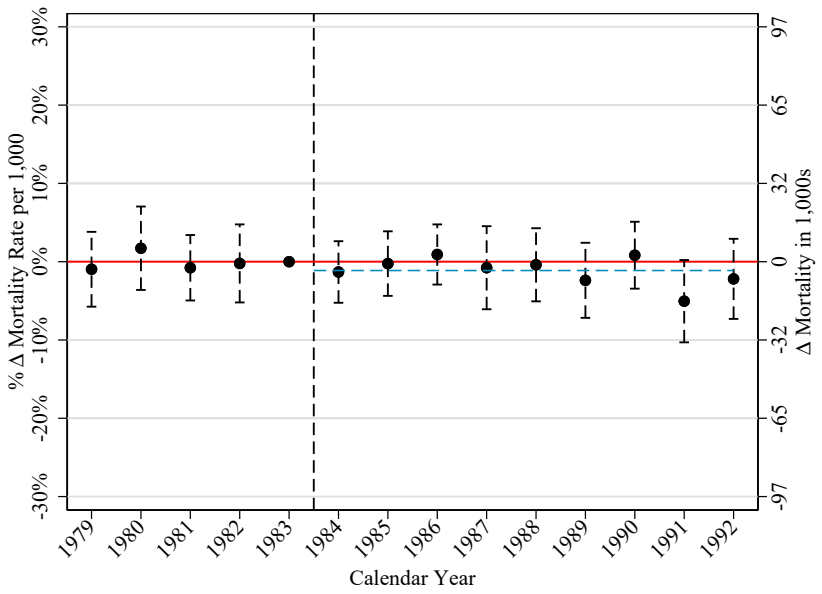


$\hat{\beta}_{Post\,2011}\,(SE) = -9.07\%^{***}\,(1.04\%);\ \Delta\ \text{LY} = 67,998$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.10\%^{***}\,(1.17\%);\ \Delta\ \text{LY} = 55,515$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. We do not apply Bayesian Shrinkage to our DSSM. The weighted mean of our DSSM is 28.1% for our NRT analysis and 14.4% for our e–cigarette analysis.

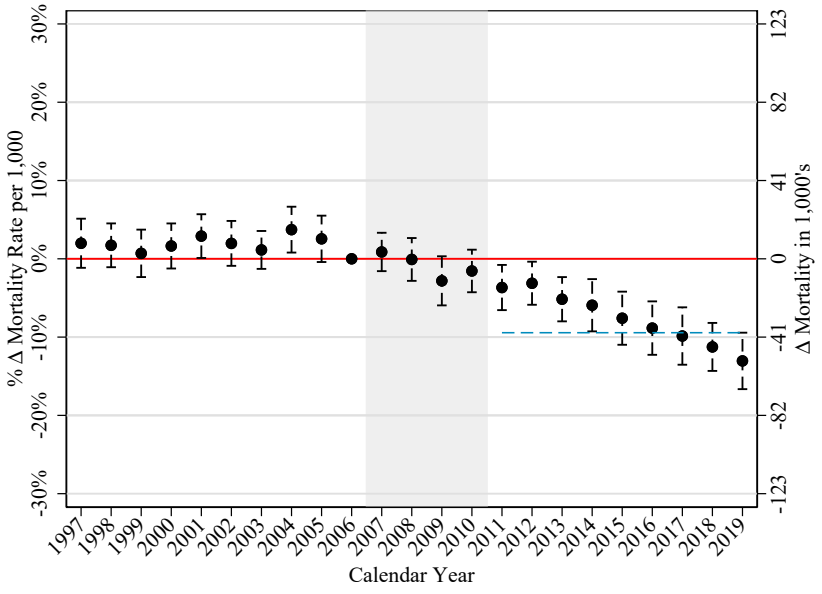
Figure I8: Event Studies for Smoking–Attributable Mortality, Ages 30 and Older

(a) NRT Drug Introduction



$\hat{\beta}_{Post\,1984}\,(SE) = -1.13\%\,(1.67\%);\ \Delta\ \text{LY} = 11,932$

(b) E–cigarette Drug Exemption



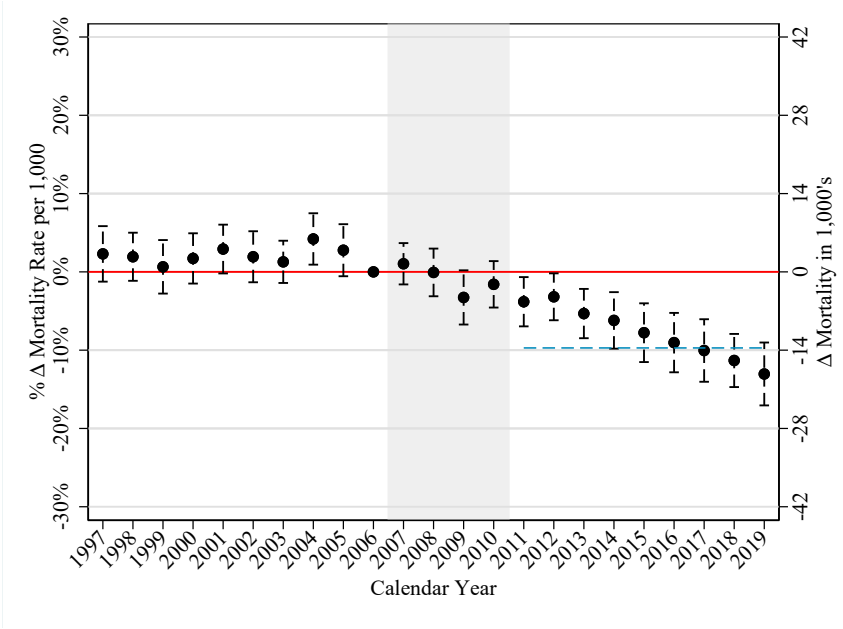
$\hat{\beta}_{Post\,2011}\,(SE) = -9.44\%^{***}\,(1.09\%);\ \Delta\ \text{LY} = 122,446$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.37\%^{***}\,(1.23\%);\ \Delta\ \text{LY} = 96,185$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Demographic groups include ages 30 and older. Sample consists of 299 groups for our NRT analysis and 772 groups for our e–cigarette analysis. The weighted mean of our DSSM is 27.1% for our NRT analysis and 13.7% for our e–cigarette analysis.

Appendix J Heterogeneity By Demographics & Age

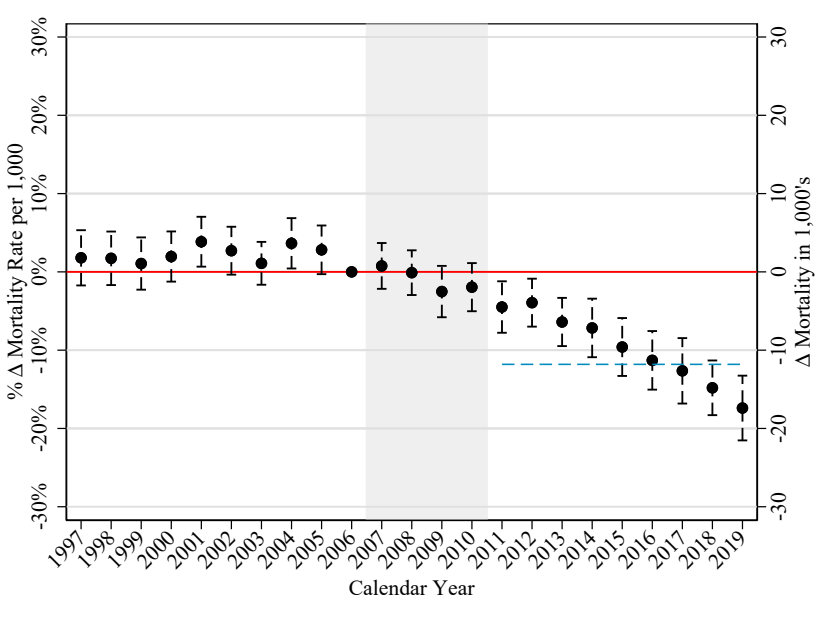
Figure J1: Event Studies for Smoking–Attributable Mortality by Sex

(a) E–cigarette Drug Exemption, Male



$\hat{\beta}_{Post\,2011}\,(SE) = -9.72\%^{***}\,(1.46\%)$; Δ LY $= 43,150$
$\hat{\beta}_{Post\,2007}\,(SE) = -7.63\%^{***}\,(1.36\%)$; Δ LY $= 34,137$

(b) E–cigarette Drug Exemption, Female

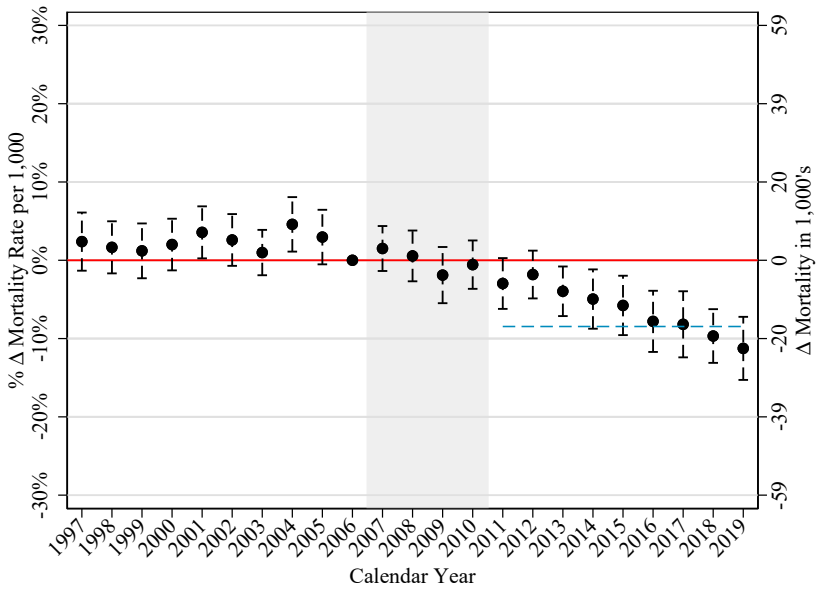


$\hat{\beta}_{Post\,2011}\,(SE) = -11.81\%^{***}\,(1.62\%)$; Δ LY $= 37,305$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.11\%^{***}\,(1.45\%)$; Δ LY $= 28,777$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Sub-groups are female and male. Our male sample contains 362 groups; the female sample contains 357 groups. The weighted mean of our DSSM for the male sample is 15.4%; the female sample is 13.3%. Weighted mean mortality (in reference period year) for the male sample is 1.8 deaths per 1,000; the female sample is 1.2 deaths per 1,000. *** p<0.01, ** p<0.05, * p<0.1

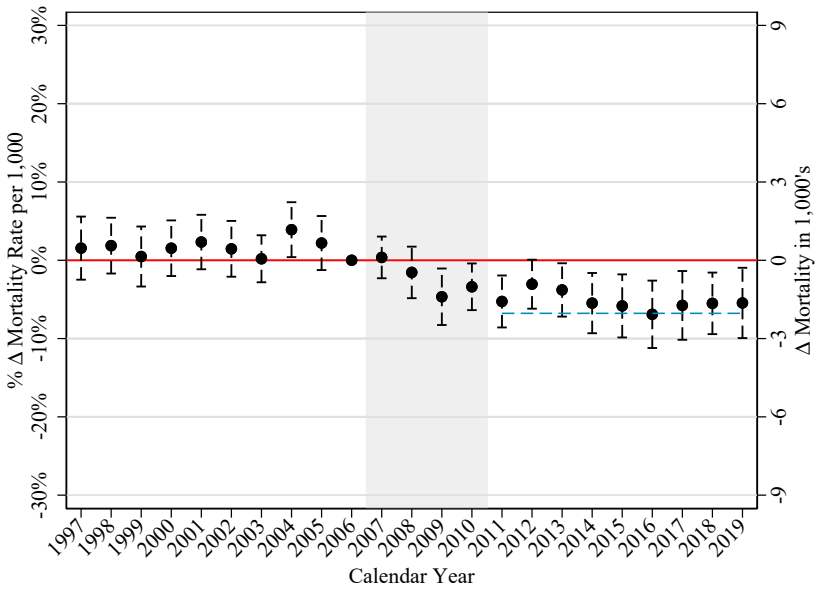
Figure J2: Event Studies for Smoking–Attributable Mortality by Race/Ethnicity

(a) E–cigarette Drug Exemption, White (5 Race)



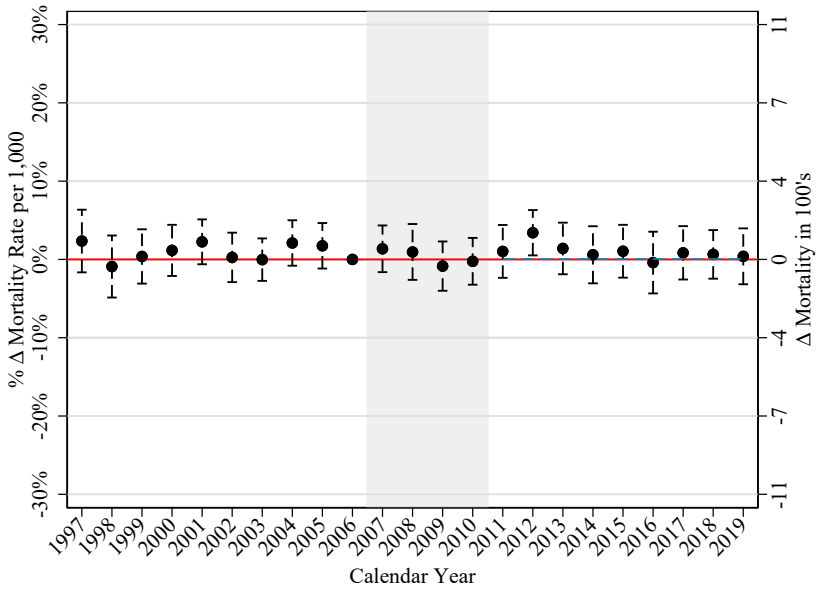
$\hat{\beta}_{Post\,2011}\,(SE) = -8.46\%^{***}\,(1.54\%)$; Δ LY $= 46,498$
$\hat{\beta}_{Post\,2007}\,(SE) = -6.56\%^{***}\,(1.38\%)$; Δ LY $= 32,228$

(b) E–cigarette Drug Exemption, Black (5 Race)



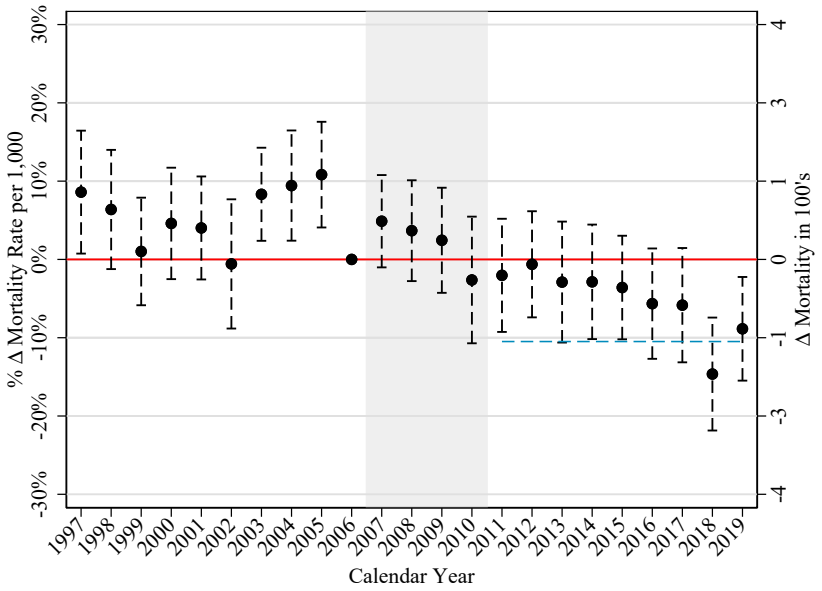
$\hat{\beta}_{Post\,2011}\,(SE) = -6.78\%^{***}\,(1.67\%)$; Δ LY $= 7,307$
$\hat{\beta}_{Post\,2007}\,(SE) = -5.88\%^{***}\,(1.47\%)$; Δ LY $= 7,319$

(c) E–cigarette Drug Exemption, Asian/Pacific Islander (5 Race)



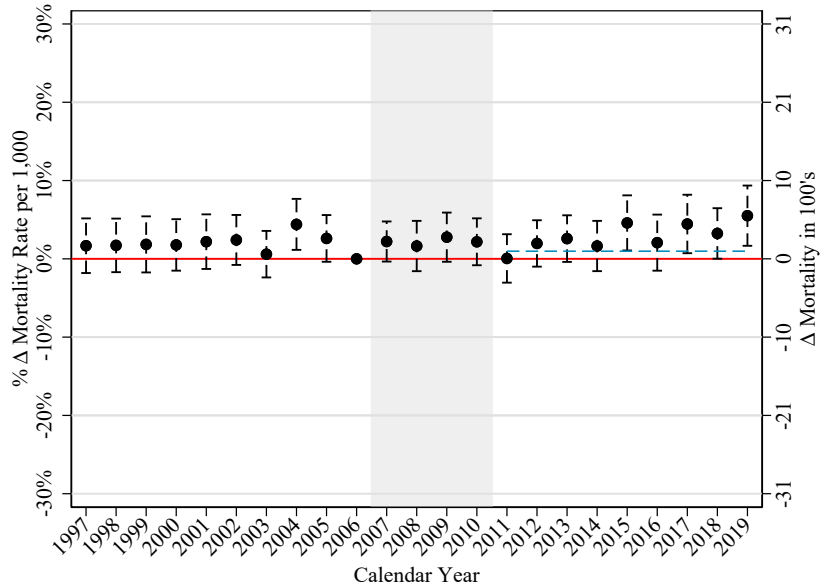
$\hat{\beta}_{Post\,2011}\,(SE) = 0.06\%\,(1.18\%)$; Δ LY $= -234$
$\hat{\beta}_{Post\,2007}\,(SE) = -0.15\%^{***}\,(1.08\%)$; Δ LY $= 210$

(d) E–cigarette Drug Exemption, American Indian (5 Race)



$\hat{\beta}_{Post\,2011}\,(SE) = -10.49\%^{***}\,(1.79\%)$; Δ LY $= 225$
$\hat{\beta}_{Post\,2007}\,(SE) = -8.25\%^{***}\,(1.61\%)$; Δ LY $= 48$

(e) E–cigarette Drug Exemption, Hispanic (5 Race)

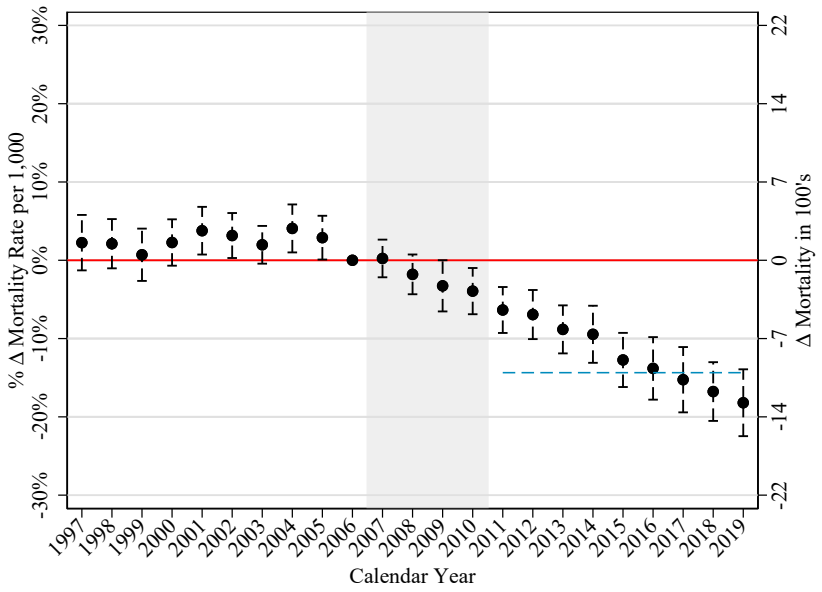


$\hat{\beta}_{Post\,2011}\,(SE) = 0.98\%\,(1.37\%)$; Δ LY $= -1,158$
$\hat{\beta}_{Post\,2007}\,(SE) = 0.76\%\,(0.87\%)$; Δ LY $= -1,603$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Sub-groups are 5 Race/Ethnicity categories (White non-Hispanic, Black non-Hispanic, Asian/Pacific Islander non-Hispanic, American Indian non-Hispanic, Hispanic). Our White sample contains 160 groups; the Black sample contains 143 groups; the Asian/Pacific Islander sample is 105 groups; the American Indian sample is 156 groups; the Hispanic sample is 155 groups. The weighted mean of our DSSM for the White sample is 15.5%; the Black sample is 14.9%; the Asian/Pacific Islander sample is 6.5%; the American Indian sample is 19.6%; the Hispanic sample is 9.8%. Weighted mean mortality (in reference period year) for the White sample is 1.7 deaths per 1,000; the Black sample is 1.6 deaths per 1,000; the Asian/Pacific Islander sample is 4.9 deaths per 10,000; the American Indian sample is 1.1 deaths per 1,000; the Hispanic sample is 5.4 deaths per 10,000. *** p<0.01, ** p<0.05, * p<0.1

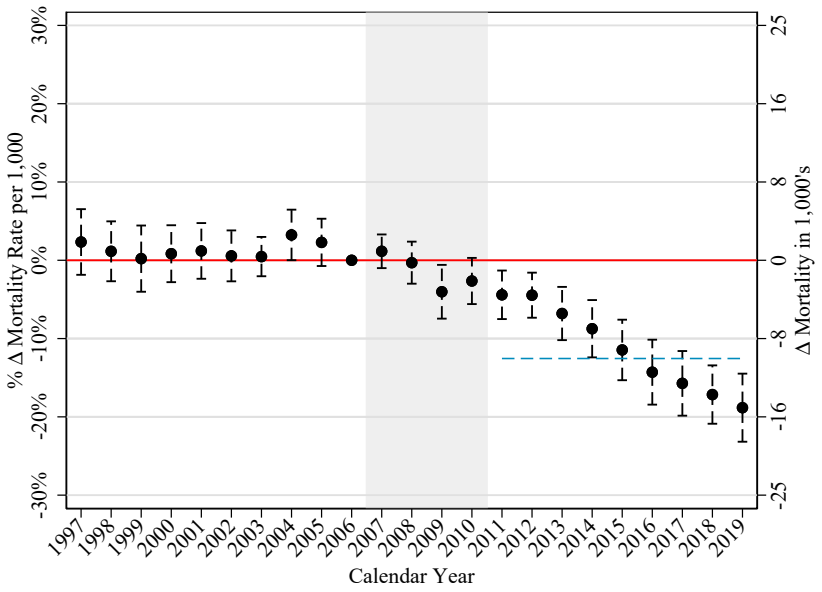
Figure J3: Event Studies for Smoking–Attributable Mortality by Age Group

(a) E–cigarette Drug Exemption, Ages 30-44



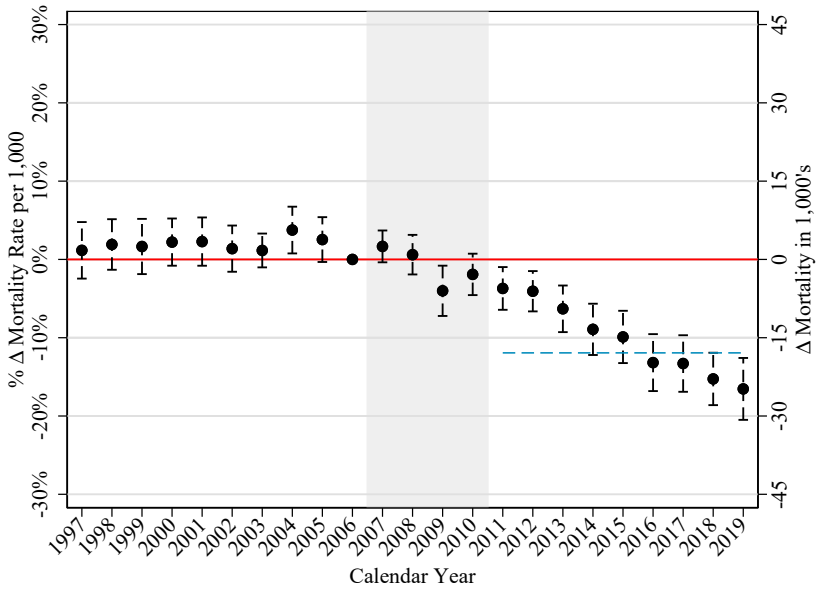
$\hat{\beta}_{Post\,2011}\,(SE) = -14.36\%^{***}\,(1.68\%)$; Δ LY $= 3,570$
$\hat{\beta}_{Post\,2007}\,(SE) = -11.33\%^{***}\,(1.51\%)$; Δ LY $= 2,990$

(b) E–cigarette Drug Exemption, Ages 45-64



$\hat{\beta}_{Post\,2011}\,(SE) = -12.55\%^{***}\,(1.92\%)$; Δ LY $= 35,649$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.51\%^{***}\,(1.77\%)$; Δ LY $= 28,443$

(c) E–cigarette Drug Exemption, Ages 65-79

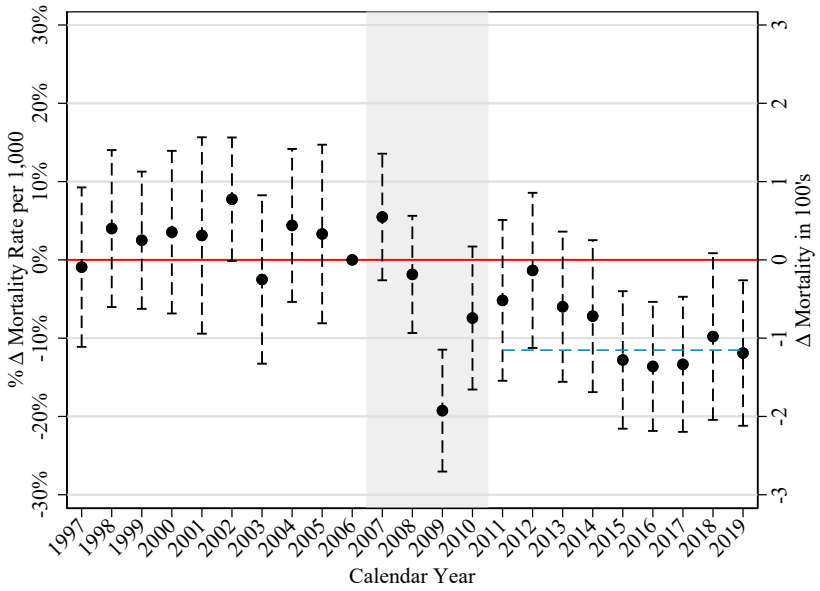


$\hat{\beta}_{Post\,2011}\,(SE) = -11.93\%^{***}\,(1.67\%)$; Δ LY $= 59,260$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.10\%^{***}\,(1.53\%)$; Δ LY $= 45,020$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Regressions are stratified by ages 30-44, 45-64, and 65-79. Our analysis for ages 30-44 contains 229 groups; ages 45-64 contains 298 groups; ages 65-79 contains 192 groups. The weighted mean of our DSSM for ages 30-44 is 15.4%; ages 45-64 15.7%; ages 65-79 is 7.9%. Weighted mean mortality (in reference period year) for ages 30-44 is 1.2 deaths per 10,000; ages 45-64 is 1.1 deaths per 1,000; ages 65-79 is 5.7 deaths per 1,000. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Figure J4: Event Studies for Smoking–Attributable Mortality, Youth

(a) E–cigarette Drug Exemption, Ages 18-29

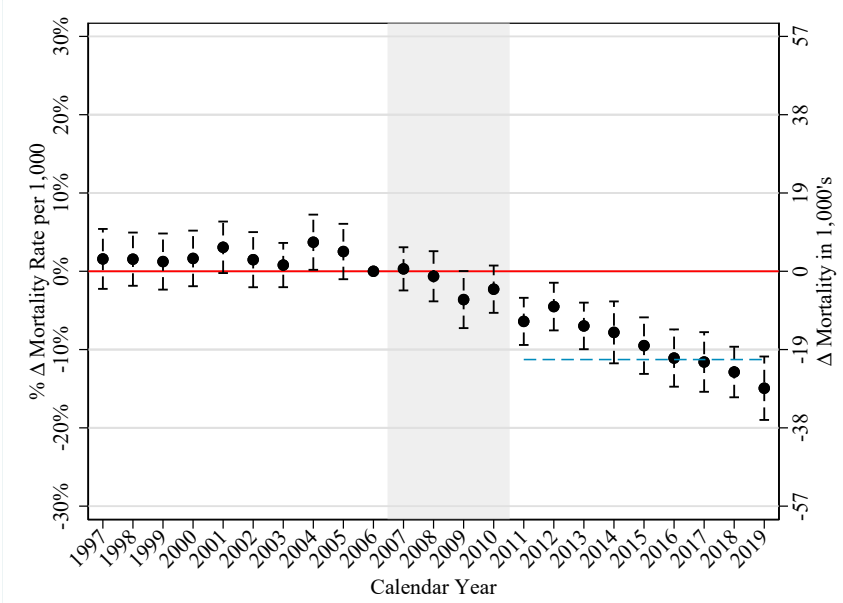


$\hat{\beta}_{Post\,2011}\,(SE) = -10.54\%^{***}\,(2.18\%)$; Δ LY $= 363$
$\hat{\beta}_{Post\,2007}\,(SE) = -10.54\%^{***}\,(2.18\%)$; Δ LY $= 420$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Youth sample consists of age groups 18-24 and 25-29. Our sample consists of 151 groups. The weighted mean of our DSSM is 17.0%. Weighted mean mortality (in reference period year) is 2.0 deaths per 100,000. *** p<0.01, ** p<0.05, * p<0.1

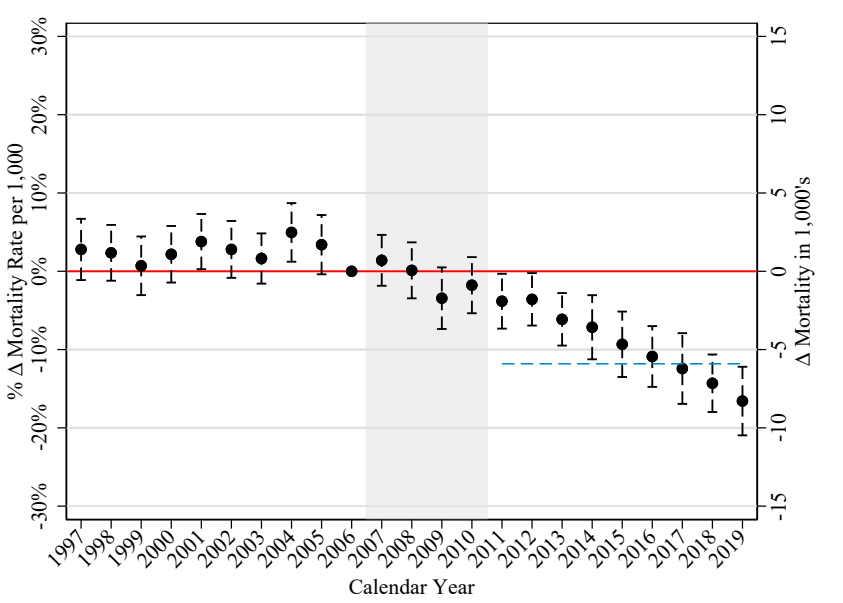
Figure J5: Event Studies for Smoking–Attributable Mortality by Urban Status

(a) E–cigarette Drug Exemption, Urban



$\hat{\beta}_{Post\,2011}\,(SE) = -11.28\%^{***}\,(1.24\%)$; Δ LY $= 75,514$
$\hat{\beta}_{Post\,2007}\,(SE) = -8.83\%^{***}\,(1.15\%)$; Δ LY $= 62,008$

(b) E–cigarette Drug Exemption, Rural



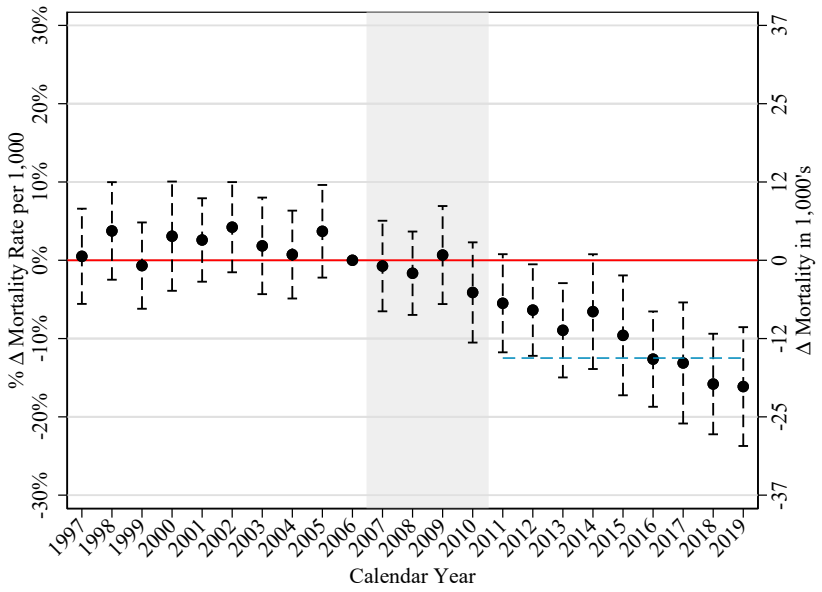
$\hat{\beta}_{Post\,2011}\,(SE) = -11.82\%^{***}\,(1.48\%)$; Δ LY $= 17,872$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.22\%^{***}\,(1.37\%)$; Δ LY $= 13,760$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Sub-groups are urban status and rural status. Our urban sample contains 386 groups; the rural sample contains 333 groups. The weighted mean of our DSSM is 18.2%; the urban sample is 13.6%. Weighted mean mortality (in reference period year) for the urban sample is 1.4 deaths per 1,000; the rural sample is 1.9 deaths per 1,000. *** p<0.01, ** p<0.05, * p<0.1

Appendix K Heterogeneity By Causes of Death

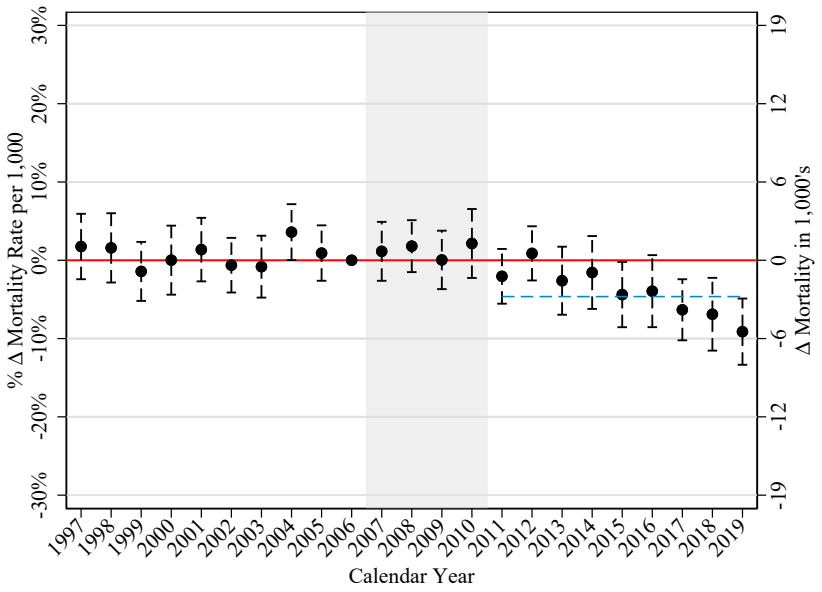
Figure K1: Event Studies, Categories of SAM

(a) E–cigarette Drug Exemption, Cancers



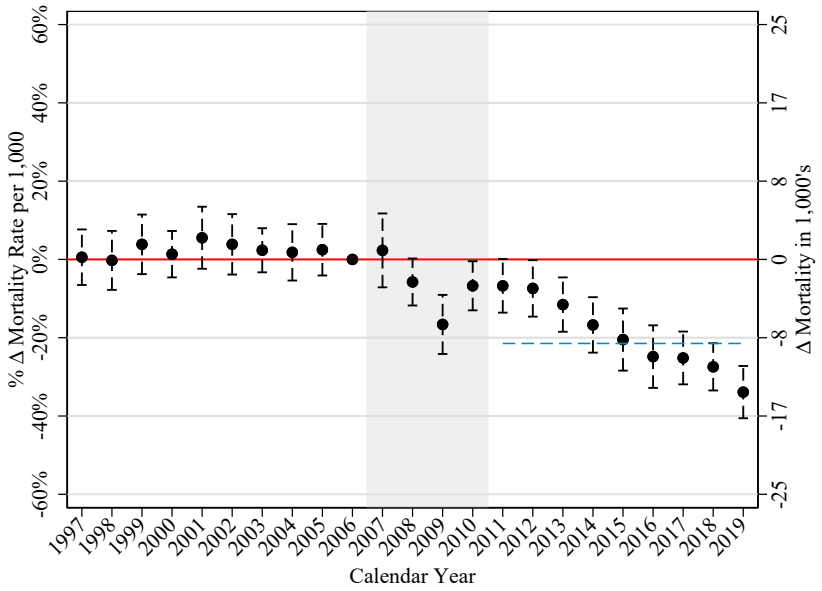
$\hat{\beta}_{Post\,2011}\,(SE) = -12.49\%^{***}\,(1.81\%)$; Δ LY $= 53,348$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.70\%^{***}\,(2.04\%)$; Δ LY $= 42,957$

(b) E–cigarette Drug Exemption, Cardiovascular



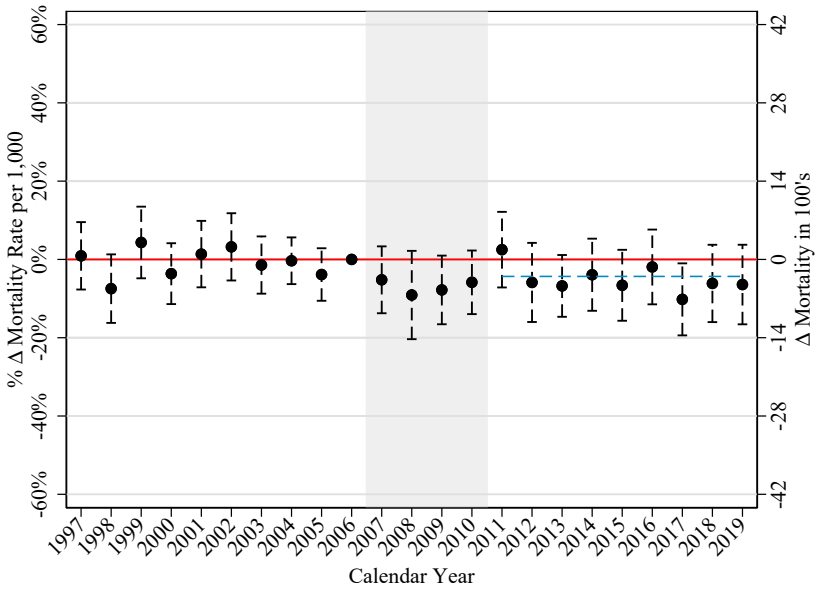
$\hat{\beta}_{Post\,2011}\,(SE) = -4.64\%^{***}\,(0.97\%)$; Δ LY $= 8,168$
$\hat{\beta}_{Post\,2007}\,(SE) = -3.02\%^{**}\,(1.19\%)$; Δ LY $= 2,872$

(c) E–cigarette Drug Exemption, Respiratory



$\hat{\beta}_{Post\,2011}\,(SE) = -21.47\%^{***}\,(1.81\%)$; Δ LY $= 30,738$
$\hat{\beta}_{Post\,2007}\,(SE) = -17.57\%^{***}\,(2.12\%)$; Δ LY $= 30,506$

(d) E–cigarette Drug Exemption, Metabolic

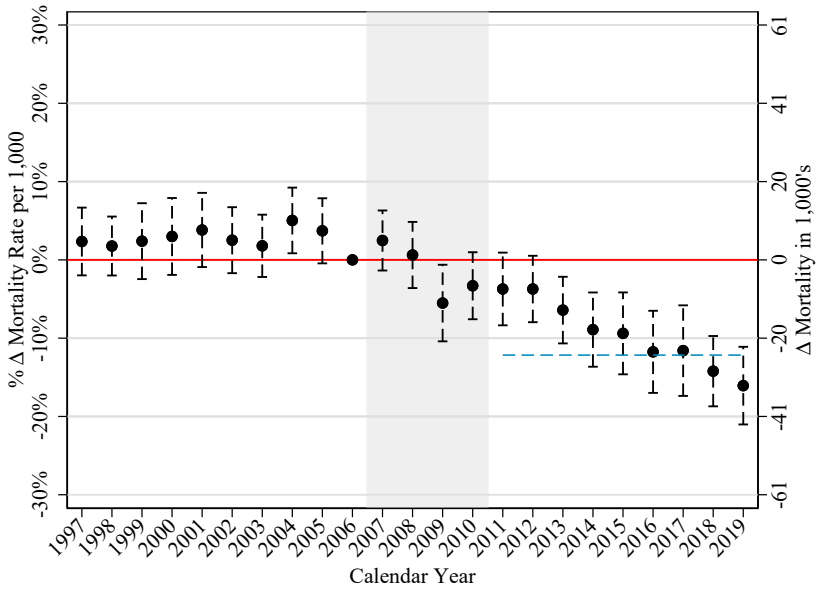


$\hat{\beta}_{Post\,2011}\,(SE) = -4.34\%\,(1.78\%)$; Δ LY $= 1,454$
$\hat{\beta}_{Post\,2007}\,(SE) = -4.93\%^{*}\,(2.77\%)$; Δ LY $= 2,749$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Regressions are stratified by categories of SAM, weighted by sex–specific SAF's: cancers, cardiovascular, respiratory, and metabolic causes. Weighted mean mortality (in reference period year) for cancers is 7.6 deaths per 10,000; cardiovascular is 3.8 deaths per 10,000; respiratory is 2.6 deaths per 10,000; metabolic is 8.9 deaths per 100,000. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

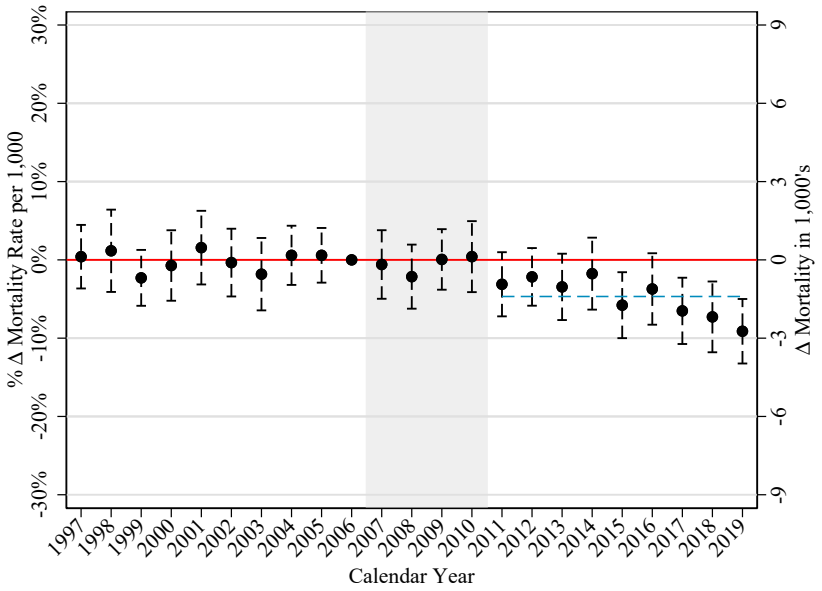
Figure K2: Event Studies, Categories of SAM by Onset of Risk Reduction

(a) E–cigarette Drug Exemption, Acute Onset of Risk Reduction



$\hat{\beta}_{Post\,2011}\,(SE) = -12.16\%^{***}\,(1.38\%)$; Δ LY $= 76,115$
$\hat{\beta}_{Post\,2007}\,(SE) = -9.68\%^{***}\,(1.60\%)$; Δ LY $= 61,201$

(b) E–cigarette Drug Exemption, Chronic Onset of Risk Reduction



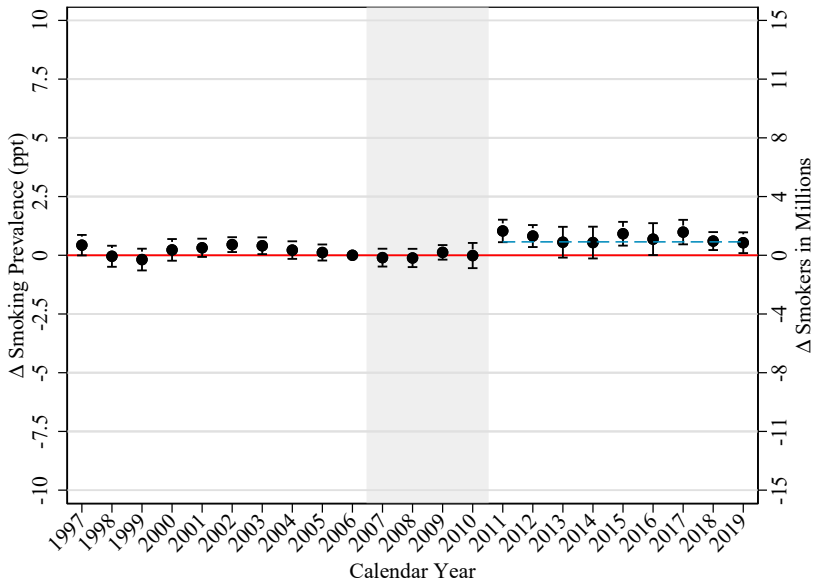
$\hat{\beta}_{Post\,2011}\,(SE) = -4.68\%^{***}\,(0.99\%)$; Δ LY $= 5,509$
$\hat{\beta}_{Post\,2007}\,(SE) = -3.39\%^{***}\,(1.10\%)$; Δ LY $= 4,467$

Notes: Refer to Figure 4 footnotes. The following changes are made relative to our main specification in Figure 4. Regressions are stratified by causes of SAM with acute onset of risk reduction from smoking-cessation and causes with chronic onset of risk reduction from smoking cessation. Causes of death are weighted by sex–specific SAF's. Weighted mean mortality (in reference period year) for acute onset causes is 1.2 deaths per 1,000; chronic onset causes is 1.8 deaths per 10,000.
*** p<0.01, ** p<0.05, * p<0.1

Appendix L Alternative Share Measure

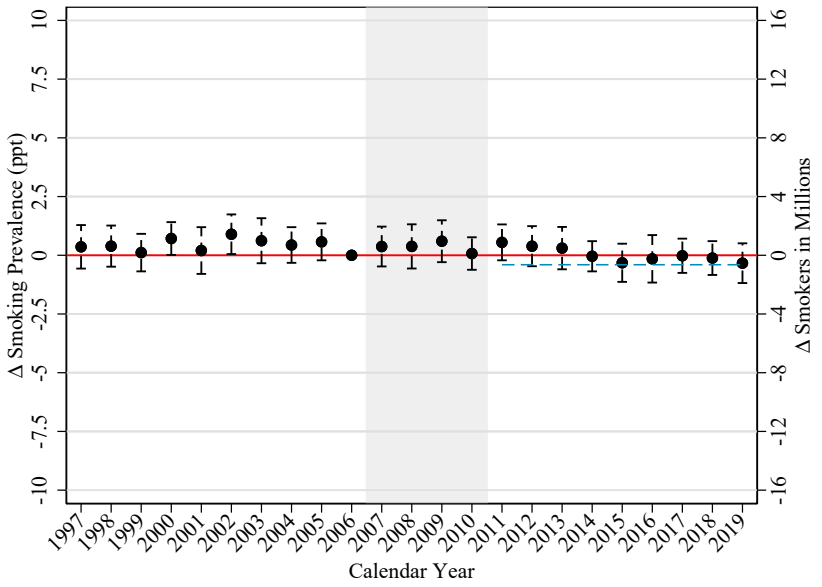
Figure L1: Event Studies Using 2016–2018 Daily–Vaping Share Measure

(a) Current Smoking (BRFSS), Daily Vaping Share



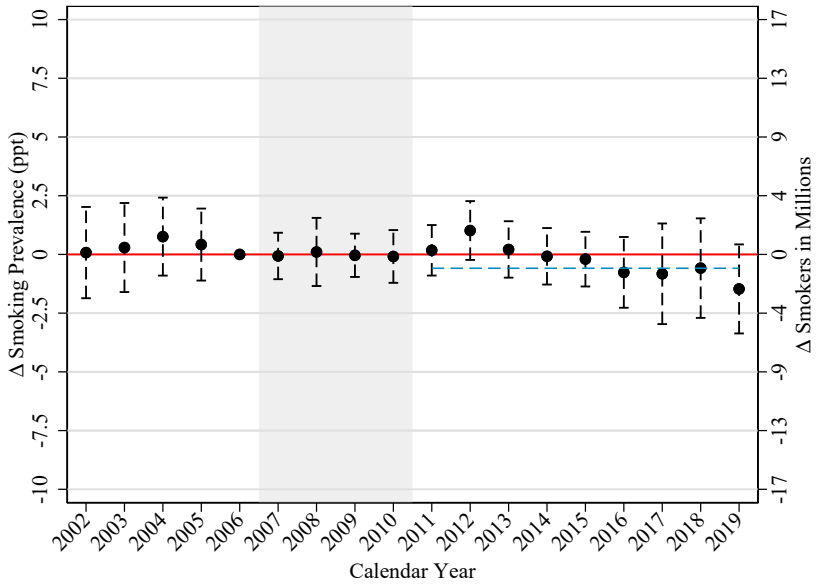
$\hat{\beta}_{Post\,2011}\,(SE) = 0.57\text{ppt}^{***}\,(0.15\text{ ppt})$; $\Delta\text{SY} = -1.14\text{m}$
$\hat{\beta}_{Post\,2007}\,(SE) = -0.22\text{ppt}\,(0.15\text{ ppt})$; $\Delta\text{SY} = -0.74\text{m}$

(b) Current Smoking (NHIS), Daily Vaping Share Measure



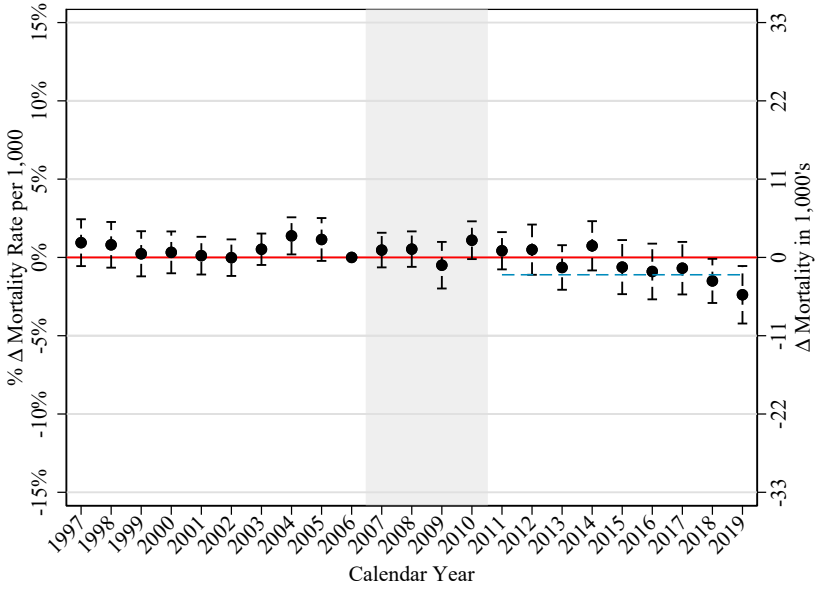
$\hat{\beta}_{Post\,2011}\,(SE) = -0.40\text{ppt}^{*}\,(0.16\text{ ppt})$; $\Delta\text{SY} = -43\text{k}$
$\hat{\beta}_{Post\,2007}\,(SE) = -0.07\text{ppt}\,(0.22\text{ ppt})$; $\Delta\text{SY} = -201\text{k}$

(c) Current Smoking (NSDUH), Daily Vaping Share Measure



$\hat{\beta}_{Post\,2011}\,(SE) = -0.59\text{ppt}\,(0.32\text{ ppt})$; $\Delta\text{SY} = -490\text{k}$
$\hat{\beta}_{Post\,2007}\,(SE) = -0.33\text{ppt}\,(0.36\text{ ppt})$; $\Delta\text{SY} = -351\text{k}$

(d) Smoking–Attributable Mortality, Daily Vaping Share Measure



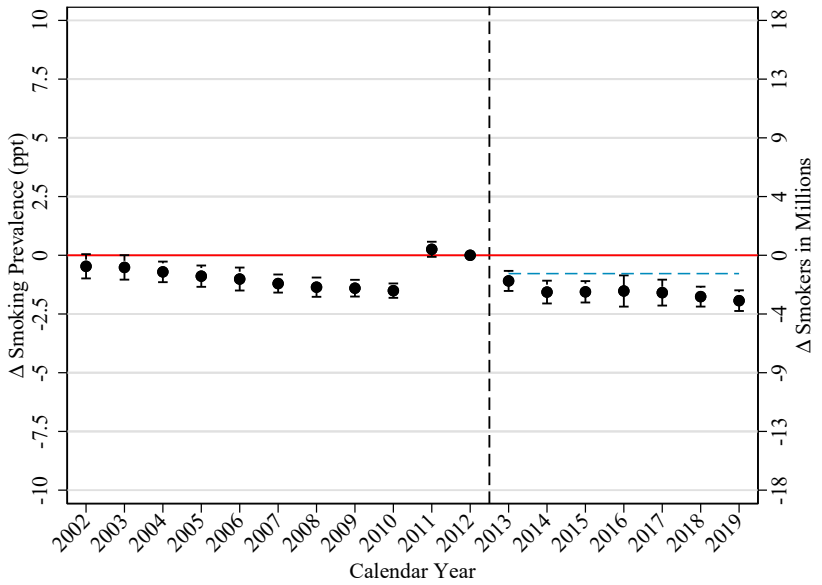
$\hat{\beta}_{Post\,2011}\,(SE) = -1.11\%\,(0.59\%)$; $\Delta\text{ LY} = 1,587$
$\hat{\beta}_{Post\,2007}\,(SE) = -0.81\%\,(0.65\%)$; $\Delta\text{ LY} = 1,986$

Notes: Refer to Figure 4 and 8 footnotes. The following changes are made relative to our main specification: We replace our 2005–2006 daily smoking share measure with a 2016–2018 daily vaping share measure (BRFSS). Outcomes are current smoking prevalence in BRFSS, NHIS, NSDUH, and logged SAM. Our sample consists of 432 groups for BRFSS, 166 groups for NHIS, 40 groups for NSDUH, and 432 groups in our mortality analysis. The weighted-mean of our daily–vape share measure is 1.2% in our BRFSS analysis, 1.2% in our NHIS analysis, 1.0% in our NSDUH analysis, and 1.2% in our mortality analysis. Weighted mean current smoking prevalence (in reference period year) is 18.8% in BRFSS, 20.6% in NHIS, 22.6% in NSDUH, and weighted mean SAM mortality is 15 deaths per 10,000. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Appendix M Alternative E–cigarette Introduction Date

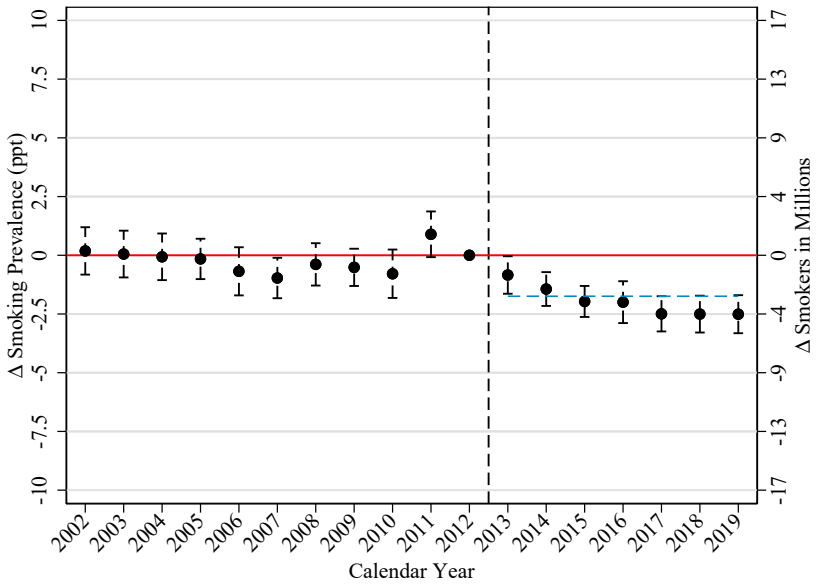
Figure M1: Event Studies for 2013 Alternative Introduction Date

(a) Current Smoking (BRFSS)



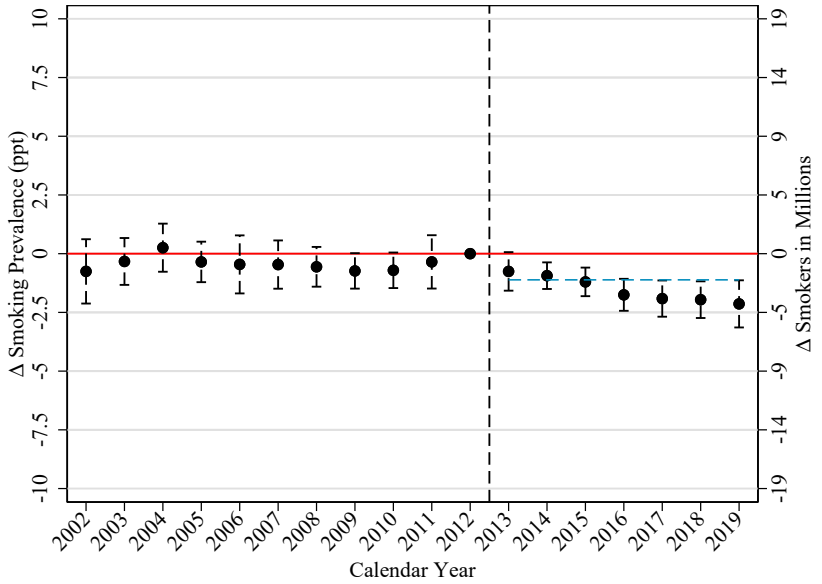
$\hat{\beta}_{Post\,2013}\,(SE) = -0.78\text{ppt}^{***}\,(0.16\text{ ppt});\ \Delta\text{SY} = -2.76\text{m}$

(b) Current Smoking (NHIS)



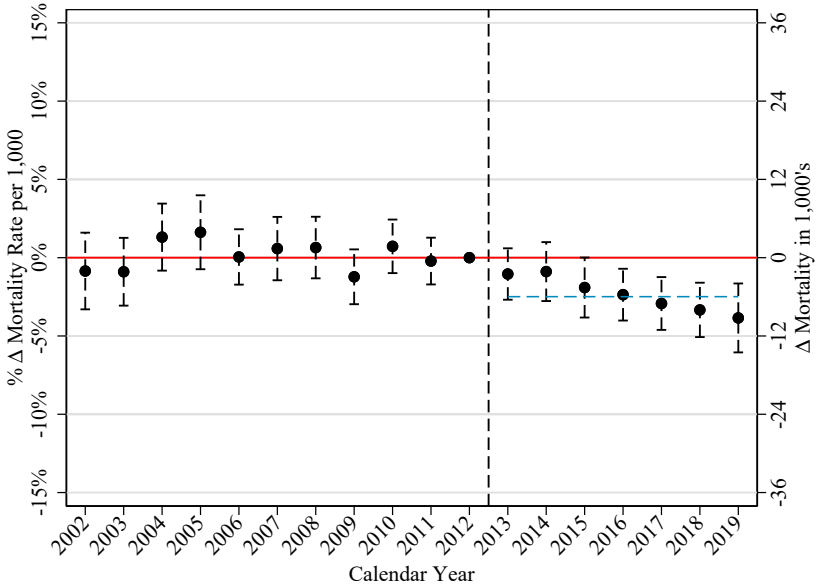
$\hat{\beta}_{Post\,2013}\,(SE) = -1.74\text{ppt}^{***}\,(0.27\text{ ppt});\ \Delta\text{SY} = -3.43\text{m}$

(c) Current Smoking (NSDUH)



$\hat{\beta}_{Post\,2013}\,(SE) = -1.11\text{ppt}^{***}\,(0.25\text{ ppt});\ \Delta\text{SY} = -2.84\text{m}$

(d) Smoking–Attributable Mortality

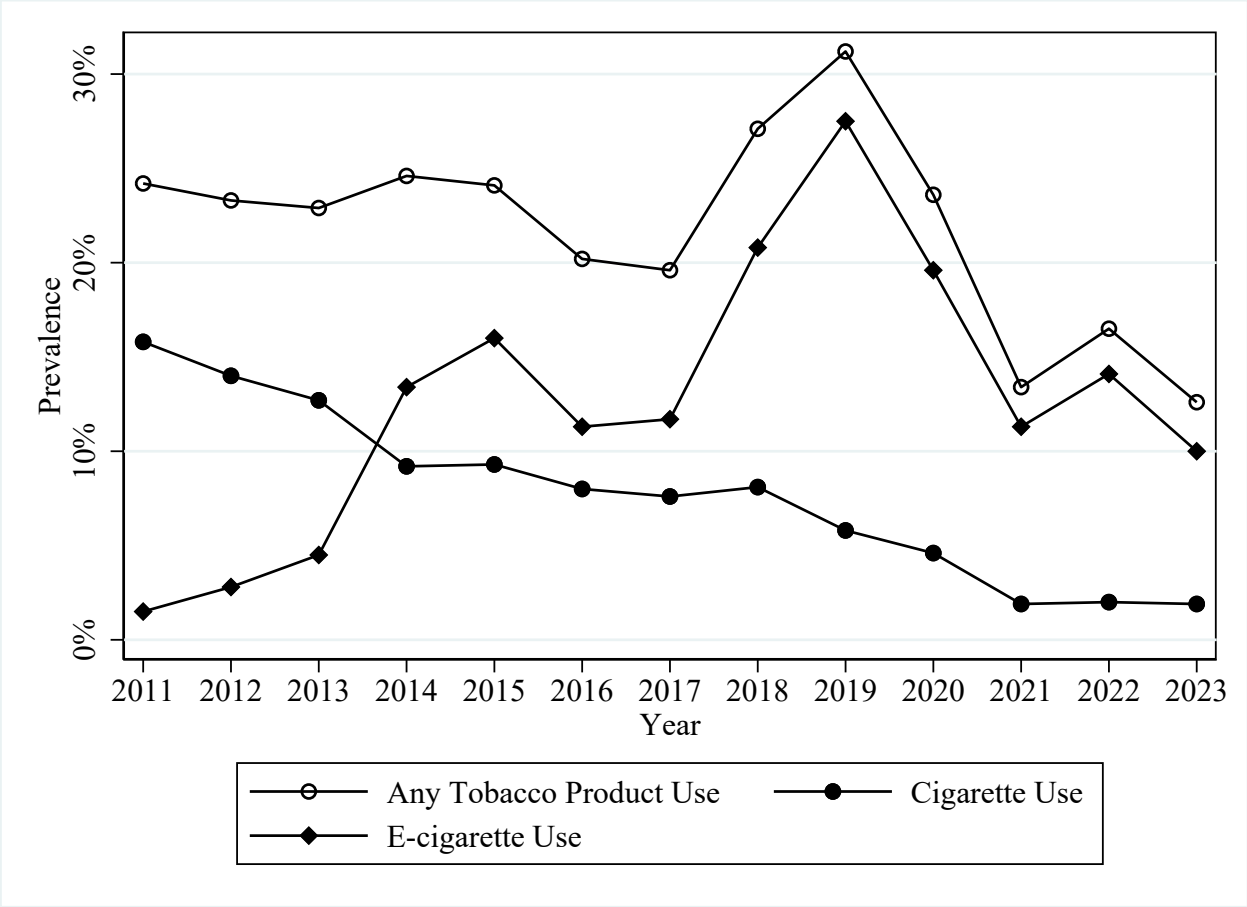


$\hat{\beta}_{Post\,2013}\,(SE) = -2.49\%^{***}\,(0.61\%);\ \Delta\text{ LY} = 9,323$

Notes: Refer to Figure 4 and 8 footnotes. The following changes are made relative to our main specification: We use an alternative e–cigarette introduction date of 2013. We replace our 2005–2006 DSSM with a 2011–2012 DSSM. Outcomes are current smoking prevalence in BRFSS, NHIS, NSDUH, and logged SAM. Our sample consists of 733 groups for BRFSS, 230 groups for NHIS, 40 groups for NSDUH, and 734 groups in our mortality analysis. The weighted-mean of our DSSM is 13.9% in our BRFSS analysis, 14.9% in our NHIS analysis, 14.1% in our NSDUH analysis, and 13.8% in our mortality analysis. Weighted mean current smoking prevalence (in reference period year) is 18.8% in BRFSS, 18.1% in NHIS, 20.4% in NSDUH, and weighted mean SAM mortality is 13.6 deaths per 10,000. *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Appendix N Youth Trends in Nicotine Consumption

Figure N1: Trends in High School Student Current (Past 30-Day) Tobacco Use



Notes: Data from the National Youth Tobacco Survey, as described in a variety of reports (Gentzke, 2019, 2020, 2022; Park-Lee, 2022; Birdsey, 2023).

Appendix References

Birdsey, J. (2023). Tobacco product use among us middle and high school students—national youth tobacco survey, 2023. *MMWR. Morbidity and Mortality Weekly Report*, 72.

Gentzke, A. S. (2019). Vital signs: tobacco product use among middle and high school students—united states, 2011–2018. *MMWR. Morbidity and mortality weekly report*, 68.

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