Was Television Responsible for a New Generation of Smokers?

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Consumers' response to mass media can be difficult to assess because individuals choose for themselves the amount of media they consume, and that choice may be correlated with their other consumption decisions. To avoid this selection problem, this article examines the introduction of television to the US, during which some cities gained access to television years before others. This natural experiment makes it possible to estimate the causal impact of television on the decision to start smoking, a consumer behavior with important public health implications. Difference-in-differences analyses of television's introduction indicate that (1) television did cause people to start smoking, (2) 16- to 21-year-olds were particularly affected by television, and (3) much of the response to television occurred within a couple of years of its introduction. Our preferred estimates suggest that television increased the share of smokers in the population by 5–15 percentage points, generating roughly 11 million additional smokers between 1946 and 1970. More broadly, these results offer causal evidence that (1) mass media can have a large influence on consumers, potentially affecting their health, (2) media exerts an especially strong influence on teens, and (3) mass media can influence consumers more than typical changes in prices.

Keywords: smoking initiation, television, health, natural experiment, advertising, mass media

Smoking for the first time can initiate a lifetime of addiction with potentially severe health consequences. For this reason, academics have taken an interest in understanding the factors that drive smoking initiation, including the role of the media. Media influence has especially captured the interest of marketers: both media's impact on tobacco consumption in particular (Leeflang and Reuijl 1985; Pechmann and Knight 2002; Pechmann and Ratneshwar 1994; Pechmann and Shih 1999; Pollay et al. 1996) and behavioral investigations into the mechanics of media influence in general (McQuarrie and Mick 1996; Mick 1986; Mick and Buhl 1992; O'Guinn and Shrum 1997; Petty, Cacioppo, and Schumann 1983; Shrum, Burroughs, and Rindfleisch 2005; Shrum, Wyer, and O'Guinn 1998). Across disciplines, a positive association between media and smoking has been reported in studies with differing data sources and methodologies, investigating differing media types and populations (Chaloupka and Warner 2000; Wellman et al. 2006). Broadly, studies of media and smoking take one of two approaches: they either report associations in the field between smoking behavior and media consumption, or they experimentally manipulate media exposure in the laboratory and report its impact on attitudes toward smoking.

Each of these approaches presents strengths and weaknesses. Laboratory studies have motivated the importance

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of media influence on smoking decisions, especially for teens (Pechmann et al. 2005), through a variety of behavioral mechanisms. However, they do not estimate the magnitude of their effect on actual smoking behavior, making it difficult to assess whether they recommend cost-effective policy interventions. In contrast, observational studies use data from the field to investigate actual smoking decisions made by large samples of individuals, but these typically lack the experimental conditions produced in the laboratory. The absence of experimental conditions means that, despite positive associations between tobacco and media consumption, bans on advertising and other media restrictions may have no impact on smoking whatsoever. Instead, these associations may simply reflect a world in which those who like to consume more media also tend to like to smoke.

To overcome these concerns, this article takes what may be considered a hybrid approach. Ultimately, we rely on observational data: a national survey, which includes tens of thousands of individuals from across the US. But, unlike typical observational studies, we seek to mimic the experimental ideal generated in the laboratory. To this end, we examine a natural experiment that delayed television's introduction to many US cities (Gentzkow 2006; Gentzkow and Shapiro 2008). These delays allows us to compare, for example, two populations of the same age, living in similar cities, one of which had access to television and another that did not gain access until several years later. Using econometric methods, we test whether the first population's early access to television caused them to initiate smoking more readily than the second.

The historical setting examined in this study is not without its own limitations, however. Unlike laboratory studies, we are constrained by the conditions of the natural experiment and cannot generate new experiments to test deeper behavioral mechanisms. And, unlike the existing observational studies, this article estimates the impact of access to just one medium—television—and does so for a period that occurred decades in the past, which may bear limited resemblance to the modern media environment. Furthermore, the approach is not able to denominate television's impact in terms of hours watched or empirically differentiate between the possible channels of televisions influence: standard advertisements, product placement, nonsponsored smoking by stars, and so on. Such nuances require richer data environments.

Instead, this study contributes to the literature by studying actual smoking decisions by a large, nationally representative sample while also avoiding the selection concerns that arise when individuals choose for themselves the amount of media they consume. We believe this union complements both observational and laboratory research by providing evidence in support of the claims that (1) the associations found in observational studies include causal effects, and (2) the behavioral mechanisms found in the laboratory manifest in the general population's smoking decisions with enough magnitude to recommend effective policy interventions. Indeed, while considering the current media environment with its new complexities—now presenting selfies of "microinfluencers" smoking on social media (Cortese et al. 2018)—policy makers require a firm understanding of whether viewing images of smoking *causes* consumers to take up the habit themselves.

ARTICLE OVERVIEW

In the next section we provide a review of the related literature on media and smoking. We follow this with a description of the natural experiment that caused some cities to gain access to television before others (Gentzkow 2006; Gentzkow and Shapiro 2008), along with its historical context. To exploit this natural experiment, we use data from the National Health Interview Surveys (NHIS), which we discuss in detail. In short, the NHIS provides data on individuals' smoking status, age, and geographic location, which allows us to approximate when each individual first gained access to television and therefore estimate its influence on their smoking behavior.

Given this background, we present two difference-in-differences analyses of smoking initiation. First, we make use of the multiple birth cohorts represented in the 1965–66 NHIS data to test whether television access at some ages has a greater impact on smoking initiation than at other ages. Second, using a question about the age of smoking initiation included in the 1970 NHIS, we test whether the hazard of becoming a smoker increased at, or around, the time that television entered a given city. Using the estimates from each of these analyses, we then approximate the long-term effect of television on the number of smokers in the population. We finish with a general discussion of the results and a conclusion.

PRIOR RESEARCH ON MEDIA AND SMOKING

Although studies of the cigarette industry have come from a variety of disciplines with different objectives, many have focused on whether media exposure affects smoking behavior.[1] We briefly review observational studies of individual-level consumer responses to media and then review laboratory studies that shed light on the potential mechanisms of media's influence and point to teens as one of the most responsive age groups.

1 The economics literature, which has often relied on aggregate data, is reviewed by Chaloupka and Warner (2000). The medical literature, which includes many observational studies of individual-level smoking choices, is surveyed in a metastudy by Wellman et al. (2006).

Individual-Level Observational Studies

Numerous observational studies of individuals have revealed correlations between smoking behavior and individuals' media environments. For example, Lewit et al. (1981) study the effect of antismoking advertising on teen smoking and find a positive correlation between television-viewing hours and the likelihood of smoking, as reported by teens. Furthermore, the authors find this correlation weakened after 1967, and they suggest the significant increase in televised antismoking advertisements from 1967 under the Fairness Doctrine is responsible. Similarly, Pollay et al.'s (1996) findings suggest advertising may be more influential on teens than on adults. They estimate brand-specific advertising elasticities that are three times larger for teens than for adults, based on correlations between brand shares and advertising shares between 1979 and 1993. Finally, Pierce, Lee, and Gilpin (1994) use the NHIS to estimate smoking initiation across time and find it increased for women in the late 1960s and early 1970s, about the time sales and advertising of women's cigarette brands also increased. While these studies recover correlations suggestive of media's influence on smoking, selection concerns arise from their lack of robust control groups.

Laboratory Studies

In contrast to the above observational studies, laboratory studies introduce experimental variation to explore potential mechanisms of media influence. Work by Shrum and others (O'Guinn and Shrum 1997; Shrum et al. 1998; Shrum et al. 2005) suggests that television may help to form consumers' perceptions of reality by presenting a world that is different from reality that consumers come to accept as accurate. While they focus on the prevalence of affluence on television, the high prevalence of smoking may similarly influence consumers. Additional evidence suggests that consumers may be influenced by advertising, even with little involvement (Greenwald and Leavitt 1984) or conscious awareness on their part (Chartrand 2005). Petty et al. (1983) propose that advertising may influence consumers via at least two paths: a "central route" in which the consumer carefully considers the value proposition, and a "peripheral route" in which the consumer simply infers value based on positive or negative characteristics that are associated with the product (e.g., celebrity endorsement). Consumer mimicry may also play a role (Tanner et al. 2008).

Additional laboratory evidence suggests that teens respond to tobacco and alcohol advertising more than other age groups (Pechmann et al. 2005). This high sensitivity is suggested by variety of factors, including adolescents' greater impulsivity (Cauffman and Steinberg 2000; Spear 2000), lower skepticism toward advertising messages (Boush, Friestad, and Rose 1994), novelty seeking (Martin et al. 2002), and greater inclination toward social comparisons (Martin and Kennedy 1993; Richins 1991). Additional experiments suggest that (anti-)cigarette advertising may influence the way teens perceive their (non-)smoking peers and therefore enhance advertising effectiveness for this age group (Pechmann and Knight 2002; Pechmann and Ratneshwar 1994). Outside the domain of smoking, research finds the importance of sensitive age ranges in which persistent preferences are formed (Belk, Bahn, and Mayer 1982; Moore and Stephens 1975); for example, sensitivity may peak around age 24 when individuals are developing preferences for popular music (Holbrook and Schindler 1989). Together, this prior work helps to motivate our hypothesis that the influence of television is highly age-sensitive, and perhaps largest for teens.

A BRIEF HISTORY OF TELEVISION AND TOBACCO

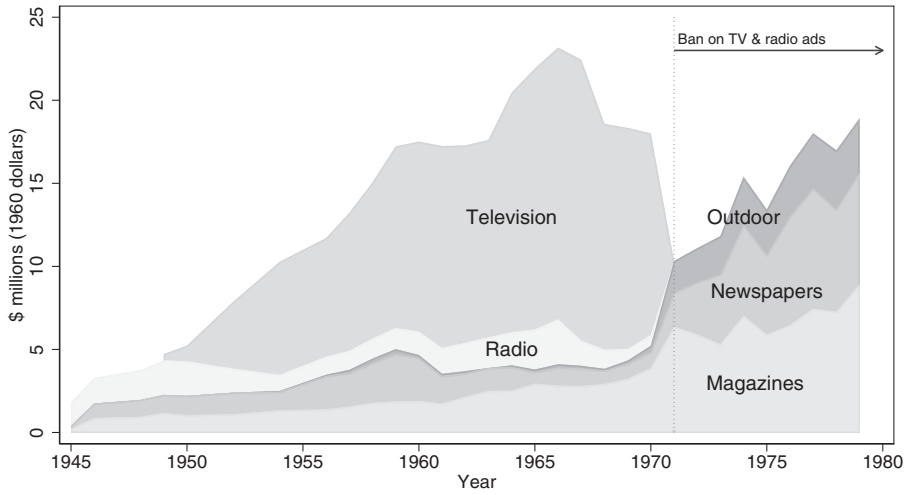
In this section, we present evidence of the significant presence tobacco had from the earliest days of television. We also detail television's introduction to the US and key events that caused delays for some cities—delays we will later use to examine television's influence on smoking behavior using analyses akin to Gentzkow (2006) and Gentzkow and Shapiro (2008).

Viewers experienced cigarettes as an integral part of television from early broadcasts in the 1940s at least until the ban on cigarette advertising in 1971 (see figure 1 for advertising expenditures over time by medium). Undoubtedly, viewers experienced cigarettes on television outside of advertising, but advertising offers some of the clearest evidence of its presence. For example, the "Dancing Pack" commercial from 1948 featured a woman dressed in a pack of cigarettes, with only her legs showing, as if it were one of the acts on the *Original Amateur Hour*. Similarly, the eponymous host of *Arthur Godfrey and Friends* from 1949 could be seen chain-smoking Chesterfield cigarettes on the air, and—during an occasional sponsor announcement—ostensibly dismiss his script to offer a personal endorsement of the brand. Whereas sponsorship of early television was typically a season-long investment, cigarette advertisers also sponsored sporting events and bought spot advertising, together creating a presence that did not go unnoticed. According to *Television* Magazine (1948–1949), cigarette brands consistently topped "sponsor identification" tables based on surveys that asked respondents to spontaneously list three advertisers on television.

These experiences did not immediately reach the entire US population, however, due to interventions by the Federal Communications Commission (FCC) (Gentzkow 2006). The FCC issued the first licenses for commercial television in 1941 for a few cities, but shortly thereafter banned further station development in order to focus resources on World War II. Following the war, those cities

FIGURE 1

TOBACCO ADVERTISING EXPENDITURE



NOTE.— Reported are the total advertising expenditures of the top six cigarette firms, which include a small amount of nontobacco advertising. Data are from *Advertising Age* (1945–1980).

with station infrastructure already in place began to rapidly adopt television. By the end of the 1940s, more cities had developed the infrastructure to offer broadcasts, but in 1948 the FCC imposed a "freeze" on any further licenses in order to address issues with spectrum allocation, leaving many cities without access to television. The freeze was not lifted until 1952, at which point nearly all of the remaining population received television within a couple of years (Gentzkow and Shapiro 2008). These three waves of television introduction are illustrated in the histogram at the bottom of figure 2 and generate the cross-sectional differences in television access that allow us to construct plausible control and treatment groups.

Such natural experiments are rare. While numerous other potentially influential events took place around the time of television's introduction (shown on the top of figure 2), they lack the critical cross-sectional differences in timing that allow us to construct meaningful control and treatment groups. Thus, we focus on the effect television access had on smoking.

DATA

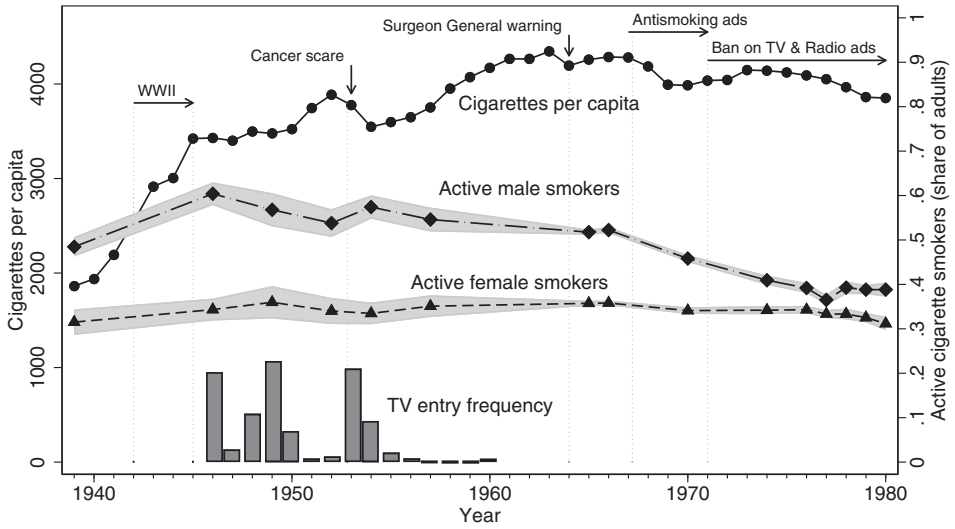
The National Health Interview Survey (NHIS) serves as our primary data source. It reports individuals' smoking status and the age at which they first smoked regularly, which provide our dependent variables. Additionally, the NHIS reports age and geographic information for individuals, which, combined with the television-entry dates from Gentzkow (2006), allow us to determine the age at which a respondent first had access to television. Together, these data allow us to estimate whether access to television influenced individuals' self-reported smoking status later in life.

Outcome variables for this study come from two NHIS questions. First, the 1965–66 surveys asked all respondents 17 and older, "Have you smoked 100 cigarettes in your entire life?" This question provides this article's definition of a smoker and functions as the dependent variable in the age-group analysis below. Second, the 1970 survey asked, "At what age did you start smoking regularly?" We use this question in the hazard analysis.

Geographic information from the NHIS allows us to determine the Designated Marketing Area (DMA) an individual lived in at the time of the survey. The NHIS contains Standard Metropolitan Statistical Area (SMSA) identifiers for respondents living in a 1960-definition SMSA. For surveys before 1973, only the Census Region is known for individuals living outside of an SMSA, so these, representing 35% of the total, are excluded from the analysis. We match the known SMSAs to DMAs, geographic regions with common television reception, which Gentzkow and Shapiro (2008) treat as having common television-entry dates. Although nearly all SMSAs lie within the DMA definitions, the few that cross DMA borders are assigned to the DMA that contains most of its population. Note that SMSA identifiers are restricted variables and not included in the public version of the NHIS. For this reason,

FIGURE 2

SMOKING TRENDS AND TELEVISION ENTRY DATES



NOTE.— From top to bottom: At the top of the plot are major events that may have affected smoking. Next, the trend line with circles represents per-capita consumption in the US as reported by "The Tobacco Situation," which is produced by the US Department of Agriculture. In the middle, the banded series report the share of active smokers in the adult population for each gender (diamonds: male, triangles: female). Gallup Polls are used for 1939–1957 and report the share of respondents selecting "cigarettes" to answer the question "Do you smoke?" NHIS data are used for 1965–1980 and report the share of respondents that report smoking one or more cigarettes per day. For each, 95% confidence intervals are shown and are adjusted for the NHIS survey design but not for the Gallup. Along the bottom (labeled "TV entry frequency") is a histogram of television entry dates, weighted by population.

the SMSA identifiers were merged with the public NHIS data by the Centers for Disease Control and Prevention, and the resulting data were analyzed at a Research Data Center.

The age at which a respondent first had access to television can be approximated using the age and location they report in the NHIS. Specifically, we set age of first television access equal to an individual's age at the time of the survey minus the number of years that had passed since his DMA first received television. This calculation assumes the respondent's DMA at the time of television entry is the same as when he was interviewed. A similar assumption regarding migration is made in Gentzkow and Shapiro (2008), which estimates television's impact on children's performance on a test given in 1965. While migration biases our respective television estimates toward zero, their analysis suggests that the impact should be limited.

Respondents were selected for the NHIS with a multistage sampling design from the civilian, noninstitutionalized US population. The first stage samples are drawn from Primary Sampling Units (PSUs) that are composed of SMSAs and other clusters of one or two contiguous counties. From the total of 1,900 PSUs, 357 were selected. Within the selected PSUs, further sampling steps were applied, eventually leading to final sampling units called "segments," which consist of six or nine nearby households. All members of these households were interviewed, although some questions could be answered by a proxy (i.e., family member). The 1965–66 surveys were conducted between July 1964 and July 1966; the 1970 survey was conducted over its calendar year. Summary statistics for individuals used for this analysis are reported in table 1.

AGE GROUPS THAT RESPONDED TO TELEVISION

This analysis exploits the fact that television entered different parts of the US at different times due to exogenous policy interventions, discussed earlier. These interventions allow us to compare the smoking status of people who either did or did not have television available to them at a given age and point in time.

We begin this section by presenting model-free evidence suggestive of television's influence on smoking by age groupings, then discuss how this approach may include confounding variation. Next, we introduce a model that allows for more robust estimates of television's influence

TABLE 1

NHIS RESPONDENT SUMMARY STATISTICS BY TV ENTRY GROUP

		Early entry	Middle entry	Late entry
Smokers:	Share smokers	.60	.60	.58
Birth year:	Average birth year	1927	1927	1928
	Minimum birth year	1899	1899	1899
	Maximum birth year	1953	1953	1953
TV access:	Average year TV first available	1946	1949	1953
	Average age TV first available	19	21	25
	Share that gained access to television between ages 16 and 21	.14	.13	.12
Demographics:	Share female	.53	.53	.53
	Share educated beyond high school	.09	.08	.08
	Median annual income	$8,187	$7,479	$6,728
	Share nonwhite	.13	.11	.12
	Median DMA size	2,739,997	492,693	103,519
Veterans:	Korean War veterans, share of men	.09	.10	.09
	WWII veterans, share of men	.25	.25	.22
	"Critical age" share: WWII veterans	.26	.09	.00
	"Critical age" share: Korean War veterans	.27	.55	.48
NHIS survey:	Share of men responding by proxy	.30	.31	.31
	Share of women responding by proxy	.13	.11	.11
	Total number of respondents	49,548	68,961	25,126

NOTE.—Each statistic is reported for groups of respondents that received television at different times: "Early entry" DMAs received television in 1946; "Middle entry," between 1947 and 1952; and "Late entry," 1953 or later. Summary statistics are for respondents from the 1965, 1966, and 1970 NHIS surveys with the exception of "Median annual income," which reports the median nominal values reported in the 1965–66 surveys only. "Median DMA size" reports the median number of total households in the DMA in which the survey respondents live; estimates of total households in each DMA are from Gentzkow and Shapiro (2008). "Critical age" share of veterans reports the share of veterans that were between the ages 16 and 21 when the DMA in which they were interviewed gained access to television.

on smoking by using only variation from the natural experiment to estimate television's effect. Finally, we present and discuss estimates using this model.

Model-Free Evidence of Responses by Age Group

To start thinking about how television might have varying effects across ages on people's decision to start smoking, consider figure 3, a histogram of the ages at which people report having started smoking. For both men and women, the vast majority of smokers start in their late teens or early twenties. If these years are the critical ages at which a person decides whether to become a smoker, we may hypothesize that these are also the ages at which television is most influential on smoking initiation. This hypothesis is also suggested by Pechmann et al. (2005), who review evidence showing that adolescents respond to media influence more than other ages for tobacco.

First, consider simple evidence for this hypothesis: the smoking rate averages by the age at which people first gained access to television. Based on the evidence presented above, ages 16–21 are critical years in which many people start smoking, so we will consider smoking rates for people who gained access to television before, during, and after this age range. Further, because smoking behavior has changed dramatically over the years, we will make these comparisons within birth cohorts, which is made possible by the staggered rollout of television's introduction. These smoking rates are reported in figure 4, which shows that, within a birth cohort, individuals who received television earlier in life almost always had higher smoking rates. This does not hold for every birth cohort, but it does hold for the vast majority.

However, this simple analysis does not rely exclusively on variation from the natural experiment and, therefore, may produce confounded estimates of television's influence. The natural experiment described earlier varied only the *timing* of television's introduction across the US, but it did not vary the *order* in which it was received across DMAs. In general, the larger, wealthier DMAs received television before the smaller, poorer DMAs, and this fact may confound the analysis in figure 4. For example, if the larger DMAs happened to experience increases in teen smoking before smaller DMAs, we would misattribute this to television. Gentzkow (2006) argues that the main determinants of television's entry order are the DMA's population and wealth, and, after controlling for the logs of these values, shows that other observable DMA characteristics are not independently or jointly predictive of the

FIGURE 3

AGE STARTED SMOKING REGULARLY, REPORTED

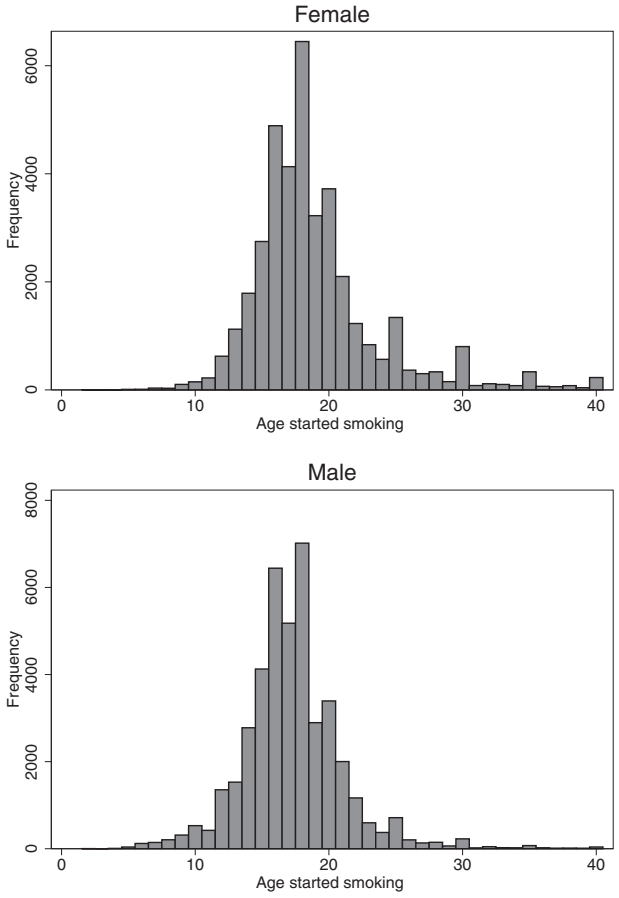
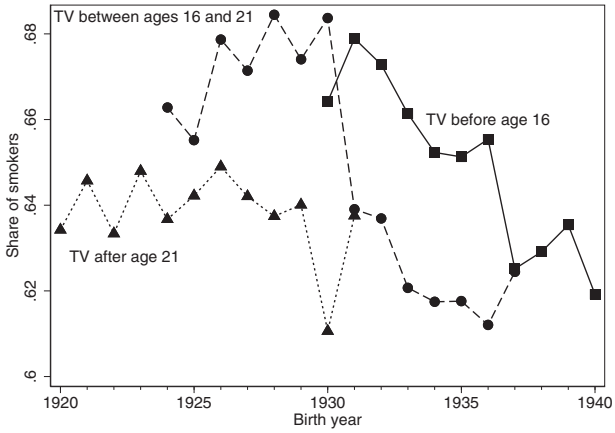


FIGURE 4

SHARE OF SMOKERS BY THE AGE THEY FIRST GAINED ACCESS TO TELEVISION



NOTE.—Share of people answering "yes" to the 1965–66 NHIS question "Have you smoked 100 cigarettes in your entire life?" grouped by birth year and the age people first received television. Triangles represent people who received television after age 21; circles represent people who received television between ages 16 and 21; squares represent people who received television before age 16.

television-entry order. Additionally, web appendix A demonstrates that even within DMAs with similar size or median wealth, there is still significant variation in the television-entry dates. Given these analyses, the models below will include controls for differential behavior in smoking across DMAs of different size and wealth for each birth cohort, plus cohort and DMA fixed effects.

A Model for Estimating Responses by Age Group

Although the simple averages in figure 4 provide a useful look at the data, we wish to control for ordering effects. The following model allows for a more robust analysis that ensures only the exogenous variation in the timing of television's introduction, and not the order of television entry across cities, estimates the effect of television. This model generalizes a difference-in-differences analysis by including an additional term to control for differential smoking behavior of cohorts across different types of DMAs, which should also control for any confounding effect of television's entry order.

For an intuitive example of the identification strategy employed by this model, consider two cities that were of similar size and wealth, but one received television in 1946 and the other received television in 1950. Also, consider two birth cohorts, one born in 1910 and the other born in 1930. As a result of these television-entry dates, the cohort born in 1930 gained access to television at age 16 if they lived in the first city, but not until age 20 if they lived in the second. Similarly, the cohort born in 1910 gained access to television at age 36 if they lived in the first city and age 40 if they lived in the second city. If access to television during the late teens was more effective at causing people to start smoking than it was during their late thirties, the difference in the smoking rates between the 1930 and the 1910 cohorts would be much greater in the first city than in the second city. Furthermore, if we are willing to assume that the effect of television on smoking initiation is zero past age 36, then this difference-in-differences approach estimates the absolute effect of television on smoking initiation between the ages of 16 and 24 rather than the effect relative to the effect on people in their late 30s. Such an analysis could be repeated for other birth cohorts to estimate the effect of television across other age groups.

To generalize such comparisons across multiple DMAs and birth cohorts, assume that the utility of becoming a smoker is given by

$$u_{icd} = \boldsymbol{\alpha}\mathbf{q}_{cd} + \delta_{gcd} + \zeta \mathrm{x}_{icd} + \epsilon_{icd}, \quad (1)$$

where δ_{gcd} represents a taste parameter specific to birth-year cohort c living in DMA d of gender g, x_{icd} represents observable characteristics for person i (i.e., marital status, income, and whether they provided their own survey response or it was by proxy), and ϵ_{icd} represents an individual-specific taste shock that is unobserved.

The remaining term, $\boldsymbol{\alpha q}_{cd}$, captures the effect that television access had on the utility of becoming a smoker. Specifically, the elements of vector $\boldsymbol{q}_{cd}$ contain counts of the number of years cohort c from DMA d was exposed to television in each of the age groups, $j \in \{9\text{–}12, 13\text{–}15, 16\text{–}18, 19\text{–}21, 22\text{–}25\}$. Hence, the elements of $\boldsymbol{\alpha}$ represent the effect of one year of television access on the utility of becoming a smoker for each of the age groups.

Note that because everyone in the 1965–66 surveys had access to television, we can estimate only the relative response across age groups, not the level response; no one in the survey can serve as the control group that has never been exposed to television. However, some cohorts in the survey may have had close to zero response to television and can serve as a reference group. Given the way $\boldsymbol{q}_{cd}$ is defined, the reference group consists of those who received television before age nine or after age 25. As a result, assuming the effect of television on the smoking behavior of ages 0–8 and 26+ is zero, each element of $\boldsymbol{\alpha}$ provides an estimate of television's impact on the corresponding age group. Alternatively, if this assumption is false and the people in the reference group experienced relatively large responses to television, our estimates understate the true impact of television on people ages 9–25.

Additionally, assume that the cohort-DMA-specific shocks have the following form:

$$\delta_{gcd} = \rho_{gd} + \tau_{gc} + \phi_{gc} W_d, \quad (2)$$

where ρ_{gd} is a DMA fixed effect for each gender and τ_{gc} is a birth cohort fixed effect for each gender.[2] These two terms resemble a standard difference-in-differences specification: we have controlled for location effects with ρ_{gd}, and, instead of time effects, we control for birth cohorts, τ_{gc}. However, controlling for the potential order effects requires a more general specification. We attain such a specification by including $\phi_{gc} W_d$, where W_d is a vector containing the log of the population and the log of median income in DMA d. W_d also includes shares of the population that are WWII veterans and the share that are Korean War veterans, because Bedard and Deschênes (2006) show that service in these wars had a significant impact on smoking behavior. ϕ_{gc} is a vector of cohort-specific coefficients. Now if a certain birth cohort happened to have higher smoking rates in larger DMAs, for example, this will not be misattributed to television.

Under the assumption that ϵ_{icd} has a logistic distribution, the probability of becoming a smoker for person i is:

$$P_{icd} = \frac{(\exp \boldsymbol{\alpha}\mathbf{q}_{cd} + \delta_{gcd} + x_{icd})}{1 + \exp(\boldsymbol{\alpha}\mathbf{q}_{cd} + \delta_{gcd} + \mathrm{x}_{icd}).} \quad (3)$$

The parameters of this logit utility model are estimated using maximum likelihood.

Potential for Biased Estimates

With the full model in place, we now consider the potential for the unobservables, ϵ_{icd}, to confound our analysis. For this purpose, the return of veterans from WWII and the Korean War provides an illustrative example because these wars had a large effect on smoking initiation (Bedard and Deschênes 2006), and veterans returned from these wars around the time of television's introduction. Imagine, as a plausible example, that WWII veterans returned around 1946 and disproportionately repatriated to the largest cities in the US. Because the model allows cohorts to vary as a function of DMA size ($\phi_{gc} W_d$), this scenario would not confound the television estimates. To confound the television estimates, the WWII veterans would have had to disproportionately return to large DMAs that also received television early. While such a scenario seems unlikely, the data shed some light on the question.

The survey data allow us to calculate the concentration of veterans across DMAs in 1965–1966, which is likely to be correlated with their repatriation rates at the end of the wars. Web appendix B presents evidence that after we control for DMA size and wealth, the share of WWII veterans in a DMA does not predict television entry order, but DMAs that received television later also tended to have higher shares of Korean War veterans. For this reason, the model includes controls for the concentration of veterans across DMAs.[3]

Another potentially confounding factor for the cross-age, television-response estimates, $\boldsymbol{\alpha}$, comes from differential migration rates across ages. We have mentioned that migration biases our estimates toward zero, but that bias is not even across age groups if migration rates differ across ages. The 1960 US census reports migration rates over the previous five years and indicates this rate reaches its peak of .26 at age 24, up from a local minimum of .08 at age 16.

2 Including separate controls for each gender is motivated by the significant differences in smoking behavior observed for each gender in figure 2.

3 The results presented in this article are robust to dropping veterans from the analysis.

However, these differences overstate the impact on our analysis. The time between television access and the 1965 survey is an average of 16 years, not five, and the longer time horizon should compress the differences across ages because more ages are covered in the longer horizon. We approximate this effect in web appendix C and find that the compression is significant and that the differential migration rates should work against the television-response estimates presented below.

Model-based Estimates of the Response by Age Group

Estimates of television's influence on different age groups, $\boldsymbol{\alpha}$, are presented in table 2. This table includes estimates from variants equation (1). Consistent with the earlier discussion of figure 4, the regression estimates in table 2 suggest a greater response to television from people in their late teens than people of other ages. The first column, which does not include controls for any DMA characteristics or fixed effects, reports relatively scattered results, although the estimates for 16- to 18-year-olds are significant. All other pooled specifications include various DMA characteristics and report significant effects for both the 16–18 and 19–21-year-old age groups but not other age groups. The importance of the DMA controls helps demonstrate the limitations of the simpler analysis presented in figure 4.

Exploration of possible differences in television responsiveness between genders is motivated by their different smoking patterns (figure 2) and differences in their television viewing habits.[4] The last two columns of table 2 look at each gender separately. While the point estimates suggest women responded to television at earlier ages than men, the differences across genders are not significant.

Unfortunately, the magnitude of the logit model's parameters are not easily interpreted. To understand the size of television's influence on smoking, we convert estimates of $\boldsymbol{\alpha}$ to estimates of the impact of one year of television access on the probability of becoming a smoker using the probability expression in equation (3):

$$\tilde{\alpha}^j \equiv \frac{\exp(\alpha^j + \delta_0)}{1 + \exp(\alpha^j + \delta_0)} - \frac{\exp(\delta_0)}{1 + \exp(\delta_0)}, \qquad \textbf{(4)}$$

where we set δ_0 equal to its average for individuals in the data set. Standard errors for $\tilde{\boldsymbol{\alpha}}$ are obtained using the delta method.

The magnitude of these estimates under the complete model is reported in figure 5 and suggests the response to television access was large. According to the values in this figure, each additional year of television access between ages 16 and 21 increased the probability of becoming a smoker by somewhere between .5 and 2.8 percentage points per year. Later, we use these values to estimate the impact of television on the number of smokers in the US. Finally, comparing the age responses in figure 5 with the ages people reported smoking in figure 3 suggests that the ages at which people respond most to television roughly correspond with the ages at which people typically start smoking.

THE HAZARD OF SMOKING INITIATION

Another method of evaluating television's impact on smoking considers the rate at which nonsmokers became smokers and tests whether this rate increased significantly upon television entry. These rates can be estimated using a hazard model, which at the individual level estimates the risk (or "hazard") that an individual will become a smoker at different points in her life. As in the analysis above, we compare individuals of the same birth cohort who either did or did not have television at a given age to estimate whether television affected their propensity to start smoking.

This section begins with an explanation of how we estimate the hazard of smoking at each age. We build on this analysis to provide model-free evidence of television's impact on smoking. Finally, we introduce a model for estimating how television changed the hazard of becoming a smoker and discuss estimates based on the model.

Hazard Estimates for Each Age

The NHIS data on starting ages lend themselves to thinking about the risk of becoming a smoker over discrete periods of time: people's ages. For this analysis, we assume everyone is born a nonsmoker but is at risk of becoming a smoker either until they are converted or their life is over. Once a person becomes a smoker, she can never return to being a nonsmoker. In this setting, the hazard is the probability that a person who is not yet a smoker becomes a smoker over the next year of her life.

We can estimate the hazard of smoking at each age by simply calculating the share of people in the sample who started smoking at each age, conditional on having survived to that age without starting smoking. In anticipation of later analysis, we perform this calculation by constructing a panel with an observation for each age over each respondent's life up until the age she becomes a smoker and, for nonsmokers, the last age she completed without becoming a smoker. By constructing the data this way, each observation conditions on the

4 Albert and Meline (1958) report women as viewing slightly more television than men. However, to our knowledge, large marketing campaigns to target women did not start until the late 1960s (Pierce et al. 1994).

TABLE 2

EFFECT OF TELEVISION ON SMOKING STATUS BY AGE OF TV ACCESS

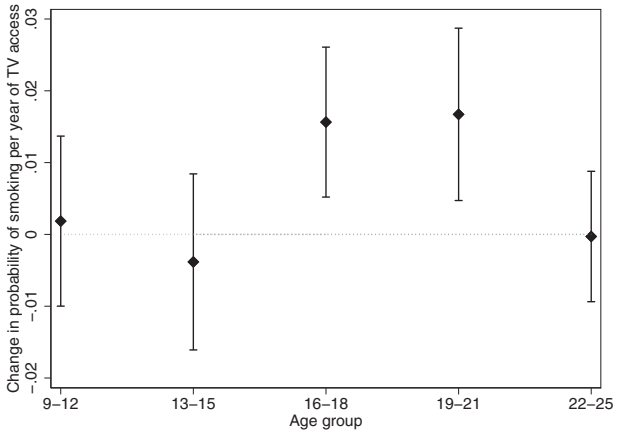
	(1)	(2)	(3)	(4)	(5)
Age group:					
9–12	.038**	−.005	.008	−.040	.019
	(.015)	(.023)	(.025)	(.032)	(.028)
13–15	−.030	−.026	−.016	−.036	−.019
	(.019)	(.025)	(.026)	(.037)	(.036)
16–18	.053***	.057***	.066***	.041	.066**
	(.018)	(.022)	(.022)	(.038)	(.031)
19–21	.025	.062***	.071***	.094***	.041
	(.022)	(.022)	(.025)	(.033)	(.028)
22–25	−.008	−.014	−.001	−.041	.003
	(.012)	(.018)	(.019)	(.032)	(.021)
Individual characteristics: x_i	X	X	X	X	X
Birth year × gender fixed effect: τ_{gc}	X	X	X	X	X
DMA characteristics × gender × year: $\phi_{gc}W_d$		X	X	X	X
DMA × gender fixed effects: ρ_{gd}			X	X	X
Gender	Pooled	Pooled	Pooled	Male	Female
Number of observations	100,416	99,549	99,549	46,838	52,711
Number of DMAs	114	112	112	112	112

NOTE.—This table presents the effect of one additional year of TV access on the utility of being a smoker for each of the listed age groups, specifically, estimates of α from equation (1). These estimates are recovered using a logit regression that controls for birth-year fixed effects plus the birth-year fixed effects interacted with DMA characteristics (log of DMA population, median income, share WWII and share Korean War veterans). Additional controls are DMA fixed effects and individual characteristics: respondent type (self or proxy), marital status, and income. Each set of controls differs by gender, but the television response is pooled across genders. The reported parameters indicate the size of TV's effect relative to that of the reference population: people who gained access to television before age nine or after age 25. Smoking status comes from responses to the 1965–66 NHIS question "Have you smoked 100 cigarettes in your entire life?" Sample weights provided by the NHIS were included in the regressions. Standard errors are clustered at the DMA level and reported in parentheses.

***indicates $p < .01$, **indicates $p < .05$, and *indicates $p < .1$ for individual hypothesis tests.

FIGURE 5

THE EFFECT OF ONE YEAR OF TELEVISION ACCESS ON THE PROBABILITY OF BECOMING A SMOKER



NOTE.—This figure presents estimates of the effect of one additional year of television access on the probability of being a smoker, broken out by age groups. The regression is the same as the third column of table 2. The model's parameter values have been converted to the marginal effects on the probability of becoming a smoker using equation (4) and the delta method. Standard errors are clustered at the DMA level, and the ranges shown represent ±1.96 * SE.

respondent not having previously initiated smoking. Then we estimate

$$y_{it} = \gamma_{gt} + \epsilon_{it}, \tag{5}$$

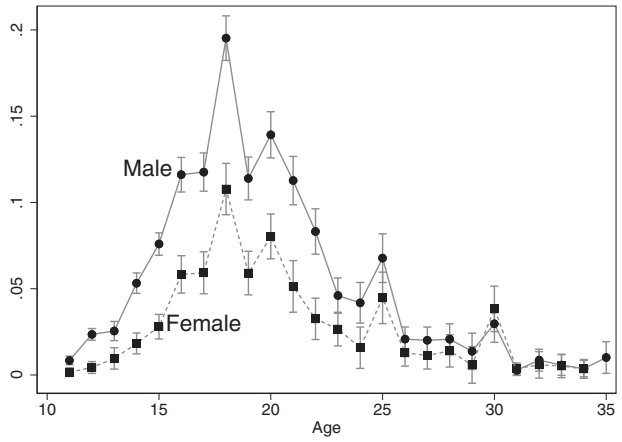
where y_{it} is an indicator that person i reported becoming a smoker at age t, and ϵ_{it} is an error term. γ_{gt} is a fixed effect for each gender-age, which provides the desired estimates of the hazard associated with each gender at each age. These estimates are presented in figure 6. For this analysis, the age distribution has been truncated to 11–36, a range that accounts for 96% of all starting ages.

Model-Free Evidence of Television's Influence: Hazard Time Trends

If television was influential on smoking initiation, we might expect that individuals who gained access to television early experienced increases to their hazards that late-television receivers did not experience until later. To test this, we make use of the three waves of television entry that are apparent in figure 2: early DMAs (received it in 1946), middle DMAs (received it from 1947 to 1952), and late DMAs (received it after the FCC ban was lifted in 1953). In principle, we could compute the average hazard rates associated with each year for each of these three

FIGURE 6

HAZARD OF SMOKING INITIATION BY AGE



NOTE.—Coefficients are the average hazard of becoming a smoker for ages 11–36 and are estimated from responses to the 1970 NHIS question "At what age did you start smoking regularly?" Standard errors are adjusted for the NHIS survey design, and the ranges shown are $\pm 1.96 * \text{SE}$.

DMA groups and inspect whether the hazard in each DMA group increased at the time it received television. However, because individuals' ages are highly predictive of smoking initiation, we obtain more precise estimates of the changes in the hazard rates by computing the averages of the residuals from equation (5) for each year and DMA group.[5] We inspect the trends in these means for evidence of responses to television.

The hazard trends for each of the television-entry groups are plotted in figure 7 and provide some graphical evidence of television's influence on the hazard rate. To see this, note how in the early 1940s the early and middle entrants follow similar trends, but in 1946, when the early entrants received television, the two trends separate. At this point, the hazard for the early entrants is much higher than the middle or late entrants until about 1948, which is what we would expect if television increased the hazard of becoming a smoker. Next, notice that 1948 and 1949 are the years in which the middle entrants primarily received television, and the hazard of the middle entrants is once again similar to the early entrants in 1949, and then overtakes early entrants in 1950–51, again suggesting the influence of television. Finally, the trend for the late entrants appears noisier than the other two, perhaps because the data contain fewer respondents from these DMAs (table 1). Nevertheless, in most years, the hazard for late entrants is lower than for middle entrants, though one of the years in which they flip is 1954, one year after most late entrants received television. Model-based analysis in the next section will help absorb more of the noise observed on this plot.[6]

A Model of Television's Impact on the Hazard

Having observed graphical evidence of television's effect on the hazard, we now turn to model-based estimates. The model specifications presented in this section allow us to account for the precise timing of television introduction to each DMA and include controls that help ensure that only variation generated by the natural experiment is used to estimate the influence of television on smoking initiation.

To illustrate how this model-based approach estimates the influence of television on smoking initiation, start by considering the model-free evidence discussed above. There, we plotted time trends of the hazard of smoking initiation and inspected whether, upon the entry of television, this hazard rose. Formalizing this approach, we could instead calculate the average hazard for each year relative to the year that television entered a given DMA; for example, year zero represents the year that television entered the average DMA, year one represents one year after television entered, and so on. This approach would allow us to average the hazard responses each DMA experienced following television's entry and test whether the jumps we saw in the model-free evidence are, together, statistically significant. In addition, this approach provides a placebo test. We know that, in the years prior to television's arrival, there should be no response to television; thus, if we detect an apparent response to television before it enters, our analysis likely failed to control for confounding factors. Alternatively, if we detect no response in the years prior to television's entry but a sudden increase in the hazard at, or shortly after, television's entry, this would provide confidence that our analysis has isolated television's influence on smoking initiation.

To implement this analysis, we specify a model with controls similar to those used in the age-group analysis. Together, these controls help isolate the quasi-experimental variation in the timing of television's entry and improve the precision of our estimates. Consider the expression:

$$\mathbf{z_i}(t)'\boldsymbol{\psi} = \theta_{\mathrm{v}\tau} + \rho_{\mathrm{d}} + \phi_{\tau} W_d + \boldsymbol{\beta}\mathbf{TV}_{it}, \qquad (6)$$

where $\mathbf{z_i}(t)'\boldsymbol{\psi}$ maps to the hazard of becoming a smoker in a manner we formalize below. The first two terms on the right-hand side are similar to a difference-in-differences specification: to control for time trends in smoking

5 Using the residuals also controls for the influence of differences in the size of birth cohorts, which may affect the average hazard rate over the years. The residuals are as estimated from equation (5). Each age, t, of a respondent's life is mapped to the calendar year in which he spent the majority of that age, τ. Each person, i, is mapped to his DMA's television-entry group. We compute the average residuals for each year and DMA group as $\widehat{\varepsilon}_{\boldsymbol{m}\tau} = \frac{1}{N_{m\tau}} \sum_{i \in \boldsymbol{m}, t \in \tau} \widehat{\varepsilon}_{it}$

6 The reader may also notice that each of these shocks appears to be temporary, lasting only a year or two. The dynamics consistent with this observation are discussed with the model-based estimates.

behavior, we include fixed effects, $\theta_{v\tau}$, for each calendar year, τ, that are allowed to differ by veteran status, v, because of the increased likelihood of initiating smoking while serving in WWII or the Korean War. Similarly, ρ_d controls for differences in the hazard across DMAs. Next, the term $\phi_\tau W_d$ controls for differential trends across DMAs that differ in their populations, wealth, or veteran concentrations. Specifically, W_d is the log of the population; the log of median income of DMA d, which i lives in; and the share of WWII and Korean War veterans, while ϕ_τ is a set of calendar-year-specific coefficients.

Ultimately, we are interested in how television may have influenced the hazard of smoking initiation in the years surrounding television's entry into a DMA. To test this influence, we include the final term in equation (6), $\mathbf{TV}_{it}$, which is a vector of dummies corresponding to each of R years before and R years after person i's DMA received television. Specifically, let t^* be the age at which person i's DMA received television; then, $\mathrm{TV}_{it}^r = \mathbf{1}_{\{r=t-t^*\}}$, where $r \in \{-R, -R+1, \ldots, 0, \ldots, R-1, R\}$. $\mathbf{TV}_{it}$ allows us to test for jumps in the hazard after television's entry and provides a placebo test for years prior to its entry.

With this specification established, we now embed it in a standard hazard model that includes controls for age effects, similar to those we used in equation (5). A common assumption in the hazard literature is the proportional hazard model suggested by Cox (1972), who expressed the continuous-time hazard as

$$\lambda_i(t) = \lambda_0(t) \exp\{\mathbf{z_i}(t)'\boldsymbol{\psi}\}, \quad \textbf{(7)}$$

where $\lambda_0(t)$ is the baseline hazard over time, and the exponential term allows for the influence of covariates on the hazard relative to this baseline.

However, the data on smoking ages are best modeled as discrete periods of time. Also, as figure 6 shows, the likelihood that a person reports becoming a smoker can change dramatically from one age to the next, which suggests that assuming a smooth, parametric distribution for the baseline hazard—as is common for many applications—would be inappropriate here. For this reason, we use the Prentice and Gloeckler (1978) model, which allows each age to have its own contribution to the hazard, estimated as a separate "fixed effect." This Prentice and Gloeckler model discretizes equation (7) by expressing the probability that i does not become a smoker between age t and $t+1$ as:

$$P[T_i \geq t+1 | T_i > t] = \int_t^{t+1} \lambda_0(u) \exp\{\mathbf{z_i}(t)'\boldsymbol{\psi}\} du = \exp[-h_{it}], \quad \textbf{(8)}$$

where $h_{it} = \exp[\mathbf{z_i}(t)'\boldsymbol{\psi} + \gamma_t]$.

T_i is the age at which i became a smoker, covariates $\mathbf{z_i}(t)$ are assumed to be constant between t and $t+1$, and γ_t is a constant that captures the effect of the baseline between t and $t+1$; specifically, $\gamma_t = \log\{\int_t^{t+1} \lambda_0(u)du\}$.

This model can be estimated using the likelihood expression:

$$L(\gamma_t, \boldsymbol{\psi}) = \prod_{i=1}^{N} [[1 - \exp\{-h_{it}\}]^{\delta_i} \prod_{t=0}^{k_i - 1} \exp\{-h_{it}\}], \quad \textbf{(9)}$$

where N is the number of people in the sample, and δ_i equals 1 if person i reported being a smoker and 0 if i reported being a nonsmoker. For smokers, k_i is the age at which i reports having become a smoker; for nonsmokers, k_i is the age at which person i was interviewed. The product on the right side from $t = 0$ to $k_1 - 1$ corresponds to the joint probability that person i did not become a smoker at any age between 0 and $k_1 - 1$. If the person was a smoker, the term on the left side raised to δ_i becomes non-trivial and accounts for the probability that i became a smoker at age k_i (Meyer 1990).

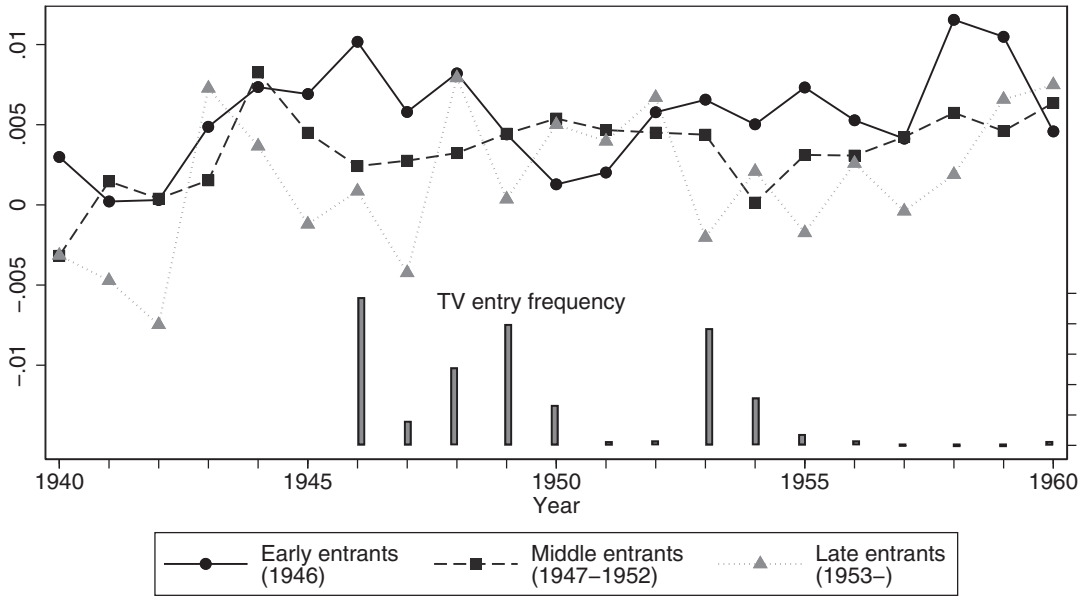
Model-based Estimates of Television's Impact on the Hazard

Estimates of how television influenced the risk of becoming a smoker, $\boldsymbol{\beta}$, are presented in table 3 for different versions of the specification in equation (6). Estimates for females are presented in the first three columns. In all three of these columns we observe significant values for the parameters associated with the year television was introduced and even stronger responses one year after its introduction. These significant coefficients contrast with those associated with years before television (years –5 to –1), which all recover null effects. These null effects are expected for a correctly specified model and indicate the placebo test did not fail. Finally, the third column presents the parameters from the full model, with a complete set of controls, which estimates the largest coefficients for years after television's introduction. Not only are years 0 and 1 large; years 3–5 are also marginally significant. Such strong estimates for this specification suggest that the heterogeneous time trends, controlled for with $\phi_\tau W_d$, mask rather than amplify the true effect of television.

Estimates for males are presented in columns 4 to 6 of table 3 and show no significant responses; the standard errors are consistently larger for men than women. Although this difference in precision and the apparent lack of a television response for men present something of a puzzle, we propose a couple of potential explanations. First, note the data contain more than twice the number of female respondents as male respondents. This difference in sample size largely follows from the decision to exclude

FIGURE 7

HAZARD OF SMOKING INITIATION BY DMA TYPE



NOTE.—The plot reports trends in the hazard rates for three DMA types: early DMAs that received television in 1946, middle DMAs that received it 1947–1952, and late DMAs that received it from 1953 onward. The hazard calculations are adjusted for the effect of age of the survey respondents; specifically, the hazard trends are derived from the residuals of the regression in equation (5). To compute the reported values for each trend, survey respondents were each mapped to their DMA type, and their ages were each mapped to the calendar year in which they spent most of that age. The values reported in the figure are the averages of the residuals for each calendar year and DMA type. The survey responses come from the 1970 NHIS question "At what age did you start smoking regularly?" Along the bottom (labeled "TV entry") is a histogram of television entry dates, weighted by population.

survey responses given by proxy due to the difficulty of reporting the precise age a family member started smoking. This step disproportionately reduces the number of men in the sample because in 1970 more women than men were at home to respond to the survey directly. A second potential explanation for the difference in precision and apparent lack of response for men comes from the psychology literature, which suggests women may generate less measurement error than men in the NHIS responses used for these estimates. Herlitz, Nilsson, and Bäckman (1997) find women possess superior episodic memory (recall of autobiographical experiences in a particular place and time), a finding that is also suggested by a number of earlier studies they review. Even if these two explanations do not fully resolve this precision puzzle, these estimates do not contradict the findings from the age-group analysis. Back in table 2, we estimated responses to television for each gender, which were indistinguishable from one another, and here in table 3 the confidence intervals for male responses are large enough that, once again, we cannot distinguish between the estimates for each gender.

The magnitudes of the parameters of the hazard model can be difficult to interpret. For this reason, we convert the estimates to values that estimate how television changed the probability of surviving an additional year without smoking. From equation (8), we see that the probability of surviving a given year is given by $\exp[-h_i(t)]$. Therefore, the change in the probability of surviving the rth year after television's introduction is

$$\Delta h^r = \exp\{-\exp(x_0)\} - \exp\{-\exp(x_0 + \beta^r)\}, \quad \textbf{(10)}$$

where $x_0 = \gamma_0 + \theta_0 + \rho_0 + \phi_0 W_0$ corresponds to the base-level hazard of becoming a smoker without television. We set x_0 equal to the average estimated value in the data set for 19-year-olds without television because earlier estimates suggest that this age is the center of the television-responsive ages.

A graphical representation of the magnitude estimates for women is presented in figure 8, which suggests that the hazard rates rose during the six years after television was introduced to a DMA. The response to television appears the year it is introduced and reaches a peak the following year. At this peak, the hazard is about .018 higher than prior to introduction, which corresponds to an average 20% increase for 19-year-olds (figure 6 shows that the average

TABLE 3

HAZARD OF SMOKING INITIATION WHEN TELEVISION IS INTRODUCED

	(1)	(2)	(3)	(4)	(5)	(6)
Year relative to TV introduction: β						
–5	.02	.03	.01	–.04	.03	–.08
	(.07)	(.07)	(.09)	(.10)	(.10)	(.11)
–4	.05	.05	.08	–.11	–.10	–.13
	(.09)	(.09)	(.11)	(.12)	(.12)	(.14)
–3	–.08	–.08	–.06	.02	.04	–.04
	(.09)	(.09)	(.10)	(.10)	(.10)	(.12)
–2	–.01	–.01	.04	.00	.02	.00
	(.09)	(.09)	(.10)	(.10)	(.10)	(.14)
–1	–.09	–.09	–.02	.13	.14	.13
	(.08)	(.08)	(.10)	(.11)	(.11)	(.15)
0	.15*	.15*	.22**	–.12	–.11	–.18
	(.08)	(.09)	(.10)	(.12)	(.12)	(.17)
1	.21***	.21***	.28***	–.05	–.04	–.14
	(.07)	(.08)	(.10)	(.12)	(.12)	(.16)
2	.05	.05	.13	–.05	–.05	–.07
	(.09)	(.09)	(.12)	(.11)	(.11)	(.14)
3	.09	.09	.15	–.06	–.06	–.10
	(.09)	(.09)	(.10)	(.12)	(.12)	(.15)
4	.08	.08	.17*	–.19	–.19	–.23
	(.07)	(.07)	(.09)	(.13)	(.13)	(.15)
5	.04	.05	.15*	–.21	–.21	–.23
	(.09)	(.09)	(.09)	(.15)	(.15)	(.16)
Age fixed-effects baseline: γ_t	X	X	X	X	X	X
Calendar-year fixed effects: $\theta_{v\tau}$		X	X		X	X
DMA characteristics × year: $\phi_\tau W_d$			X			X
Gender	Female	Female	Female	Male	Male	Male
Number of observations	301,437	301,437	301,437	96,781	96,781	96,100
Number of individuals	17,127	17,127	17,127	7,544	7,544	7,544
Number of DMAs	112	112	112	112	112	112

NOTE.—Estimates of hazard-model parameters for years relative to the introduction of television. The specification is a discrete-time hazard model in age that includes age fixed effects, calendar-year fixed effects, DMA fixed effects, and interactions of DMA characteristics (log population, log median income, and veteran concentrations) with the year fixed effects as shown in equation (6). Estimates are from responses to the 1970 NHIS question "At what age did you start smoking regularly?" Regressions use sample weights provided by the NHIS. Standard errors are clustered at the DMA level and reported in parentheses.

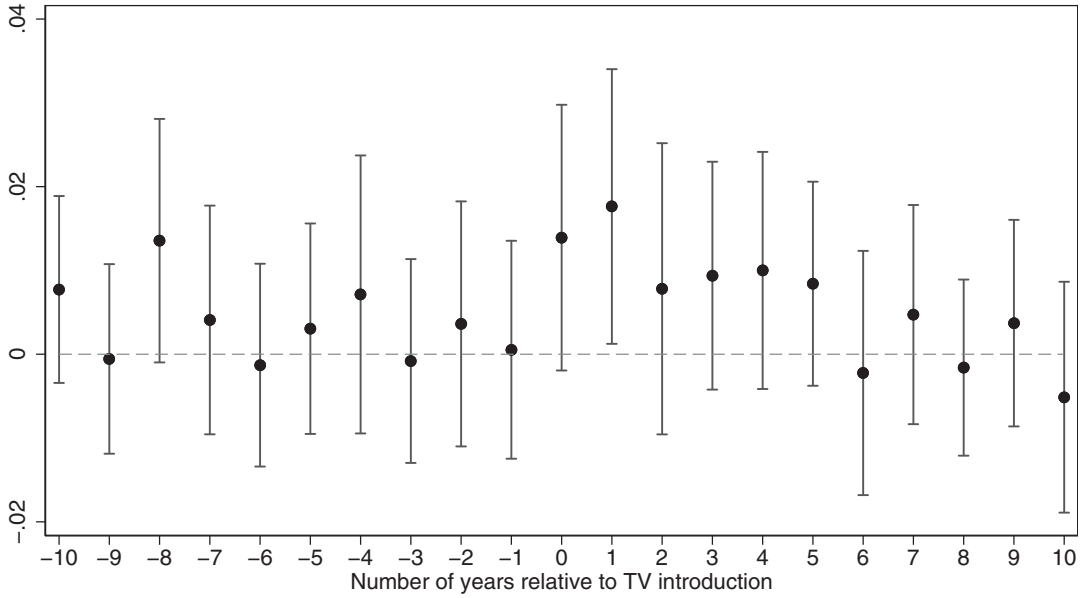
***indicates $p < .001$, **indicates $p < .05$ and *indicates $p < .1$ for individual hypothesis tests.

baseline hazard at age 19 is around .086). The quick response to television may be surprising given that broadcast availability did not mean all homes suddenly had television. However, according to surveys at the time, people who did not own televisions were frequently exposed to the new medium. A 1949 survey in metropolitan New York conducted by the National Broadcasting Company (NBC) reports that 63% of nontelevision families were exposed to television at least occasionally, with 41% seeing it once a week or more, typically at the homes of friends or family (Beville 1949). Similarly, a nationwide survey of women in 1954 reports that 47% of women in nontelevision homes had watched television in the last month (Simmons & Associates Research, Inc. 1954). The quick response to television may also be explained by network effects: a few people with televisions were converted to smokers as a result of their television exposure, while others without television converted to smokers because they saw the television owners smoking.

After the initial response to television in years 0 and 1, figure 8 shows marginally significant estimates in years 2–5, beyond which the coefficients remain close to zero. This return to zero, however, does not necessarily mean television became less effective in the years after it was introduced. To understand why, remember that (1) the hazard estimates the *rate* of smoker conversion in the population, and (2) the stock of people who might be responsive to television but have not yet been exposed is never higher than when television is first introduced. To illustrate these dynamics with a simple example, imagine only a portion of 16- to 21-year-olds respond to television while everyone else is completely unaffected by it. When television first enters a DMA, a large stock of 16- to 21-year-olds exist who have never seen television but are inclined to start smoking once they see it. As a result, when television enters, the rate of smoking initiation jumps upward as this group suddenly starts smoking. Then, with this television-responsive population converted, the rate will decrease, but

FIGURE 8

THE HAZARD OF BECOMING A SMOKER WHEN TELEVISION IS INTRODUCED, WOMEN.



NOTE.—Coefficients estimate the increase in the hazard of starting smoking in the years relative to television introduction. The regression is identical to the third column of table 3, except estimated coefficients include years up to 10 years away from the introduction of television. The model parameter estimates have been converted to the marginal effect on the hazard of becoming a smoker using equation (10), along with the delta method for the standard errors. Standard errors are clustered at the DMA level, and the ranges shown represent ±1.96 ∗ SE.

not to its initial level: new cohorts are continuously turning 16, and under our assumption, some of them are becoming responsive to television. As a result, the long-run hazard rate is higher than it was before television but lower than it was when television was first introduced. These dynamics, which are consistent with the evidence in figure 8, play out even if all cohorts were equally responsive to television.

THE NUMBER OF SMOKERS GENERATED BY TELEVISION

In the last two sections, we applied different analyses to different data, but in each case found evidence that television caused people to start smoking. However, each analysis was specific to the data we have available and therefore generated very specific measures of television's impact. Here we convert each set of estimates to capture how television changed the share of smokers in the population, which, in turn, allows us to approximate the number of new smokers that were generated as a result of television.

First, consider the age-group analysis. This analysis estimated the impact of one year of television access on the probability of becoming a smoker, and we allowed these estimates to differ by age group. By summing up the influence of television over each year of a person's life, we can estimate how the probability of an individual becoming a smoker changed as a result of television. Furthermore, if the probability of becoming a smoker changed for the average person, then we would expect the share of smokers in the population to change by the same amount. To estimate the change in the share of smokers, we make the conservative assumption that television had zero impact on people outside the ages 16–21. Therefore, we estimate

$$\Delta S^{a}_{\mathrm{TV}} = \sum_{j\in\{16-18,\ 19-21\}} n_j \tilde{\alpha}^j, \tag{11}$$

where ΔS^{a}_{TV} is the change in the share of smokers as a result of television, $\tilde{\alpha}^j$ is the coefficient estimated for age group j using equation (4), and n_j is the number of years covered by age group j.

Similarly, the change in the share of smokers can be estimated from the hazard analysis, but this requires stronger assumptions. In particular, we must make an assumption about whether the increased hazard rate comes from people who would not have otherwise smoked or from people who were going to smoke anyway but started sooner as a result of television. For this analysis, we assume the former, which, if incorrect, would artificially raise our estimate of

the change in the share of smokers in the population. On the other hand, the estimates from the hazard analysis are prone to attenuation bias because they are based on a question posed to respondents about an event that occurred two decades in the past and are therefore likely to contain significant reporting errors. Thus, for this hazard-based calculation, we have two potential sources of bias pushing our estimates in opposite directions. Although we can only speculate as to the net direction and magnitude of these opposing effects, the calculation is still useful for comparison purposes. Web appendix D shows that the change in the share of smokers from television can be estimated as

$$\Delta S^{h}_{\mathrm{TV}} = \exp\{-H_0\} - \exp\{-H_0 - \sum_{k=0}^{5} \Delta h^{r}\}, \qquad (12)$$

where $\Delta S^{h}_{\mathrm{TV}}$ is the estimated change in the share of smokers as a result of television, H_0 is the cumulative hazard of becoming a smoker in the absence of television, and Δh^{k} is estimated using equation (10). We perform this computation only for women because the hazard analysis produced only null effects for men.

The estimates from these exercises suggest that television was highly influential on the number of smokers in the US. In particular, the age analysis suggests that the share of smokers in the population was increased by 5–15 percentage points (i.e., $\Delta S^{a}_{\mathrm{TV}} = .10$, $\mathrm{SE}_{\Delta S^{a}_{\mathrm{TV}}} = .26$; SE estimated with the delta method). To calculate the number of new smokers television generated by 1970, the last year that cigarette advertising was allowed, we account for the fact that only about half of the 1970 population had been exposed to television during the critical ages of 16–21. This leads us to estimate that television generated about 11 million new smokers by 1970. Alternatively, our estimates from the hazard analysis suggest that the share of smokers was increased by 1–7 percentage points (i.e., $\Delta S^{h}_{\mathrm{TV}} = .037$, $\mathrm{SE}_{\Delta S^{h}_{\mathrm{TV}}} = .015$), implying that television generated around four million new smokers by 1970. While the confidence intervals for ΔS_{TV} from each analysis contain some overlap, they represent statistics that are significantly different from one another. This difference is not unexpected given the strong assumptions required to convert the hazard estimate. For this reason, we treat the age-group results as our preferred estimates.

GENERAL DISCUSSION

The media environment has undoubtedly changed since the 1950s, and data sources have become increasingly rich, as reflected in many of the studies referenced in this article. These and other studies are able to look at different types of media separately (e.g., advertising, television, movies, and other promotional activity) in ways that are simply not possible using data from the mid-20th century. On the other hand, the ubiquity of modern media has made establishing a causal link between smoking and media difficult due to selection concerns. By avoiding these selection concerns, the historical events presented in this article offer new clarity on the impact of media on smoking.

Previous works reveal a strong association between media exposure and smoking, but these studies critically lack random assignment of media exposure. The micro-based studies must assume that, after we control for available demographic characteristics, the remaining *self-selected* level of media consumption does not correlate with smoking preferences. If this assumption is incorrect, their estimates contain a selection bias, the size of which is unknowable but is possibly responsible for all of the correlation they report. In other words, the existing literature leaves open the possibility that media exposure does *not* cause people to smoke, but those who choose more media consumption also tend to smoke. By exploiting the quasi-random assignment of television access, this study attempts to address this issue and provide more robust evidence that media caused teens to start smoking than was previously available.

Although the natural experiment we examine does not identify which features of television are responsible for the estimated response, a number of factors suggest advertising was responsible for at least some of it. Earlier, we saw evidence that the cigarette industry quickly adopted television as its primary advertising medium, and consumers were quite aware of the cigarette brands promoted on television. Indeed, firms had strong incentives to support any aspect of television that encouraged smoking. Although unsponsored features of television might also have encouraged smoking—for example, seeing famous people smoke on television—firms would have likely found ways to promote such features, whether through standard advertising, product placement, or by other means. Under the assumption that advertising was responsible for some of the effect we measure, this article provides new support for the effectiveness of tobacco advertising bans. Some readers may find this assumption more palatable than the assumption required by the existing literature that associations between smoking and advertising reveal a causal effect.

While our evidence lends support to the decades-old bans on television and radio advertising of tobacco products, it may also shed light on the renewed question of the media's influence on smoking, now in the digital environment. Today, much media content is consumed online, especially on social media platforms, and a new type of celebrity has emerged, the "microinfluencer." Evidence provided in a recent petition to the Federal Trade Commission (Myers, Muggli, and Henigan 2018) suggests that tobacco companies have hired microinfluencers to promote their brands and smoking in general. The borderless nature of social media allows companies to endorse microinfluencers in countries where it is legal while gaining an

audience worldwide. Tobacco companies contend that their efforts do not target new smokers and are intended only to recruit existing smokers to their brands (Kaplan 2018). In theory, their advertising may play such a role, but ultimately this is an empirical question. Our estimates suggest that media promotion can actually generate a large number of new smokers.

In particular, we find causal evidence for the frequently cited concern that teens' smoking behavior is most responsive to media content. Extensive laboratory research has offered explanations for teens' greater sensitivity to tobacco advertising (Pechmann et al. 2005), and our results are consistent with the external validity of these explanations. Furthermore, the correspondence between "high-risk" ages and "high-response" ages provides support for targeting at-risk groups that may be particularly influenced by external factors such as the media. This offers new support for the US Surgeon General's focus on tobacco use by young people (US Department of Health and Human Services 1994).

Encouragingly, our evidence for television's influence on smoking does not hinge on a single data source, which helps demonstrate robustness. Using a separate survey question to measure the response to television, our hazard analysis reveals a sudden rise in smoking initiation that accompanied television's entry into a city. That this analysis detects any effect at all may be surprising given that (1) these estimates are likely to be biased toward zero due to errors from respondents reporting on an event that occurred decades in the past, and (2) similar analyses of this NHIS question reveal no response to prices (Douglas 1998; Douglas and Hariharan 1994).

Indeed, this study estimates a response to television that is large relative to that typically found for prices. Bader et al. (2011) review the literature estimating how prices influence smoking initiation and find that of 22 studies, nine found no effect of prices, six found effects in some cases, and seven found significant effects. The study with a time span closest to this article, Douglas and Hariharan (1994), finds an insignificant influence of prices from 1954 to 1978. Alternatively, some of the largest estimates in this literature come from Sen and Wirjanto (2010), who estimate a price elasticity of smoking initiation of –.2 to –.5. Together, our respective point estimates suggest a 29% drop in prices would be required to produce the same increase in smoking initiation as television. Because prices are known to influence a wide variety of decisions, we believe this comparison helps to underscore the importance of mass media on consumer behavior.

In contrast to the price literature, the media effects estimated in this article are smaller than those found in the medical literature. For example, the metastudy by Wellman et al. (2006) reports an average odds ratio of 2.23 for being "treated" by media exposure, compared to our point estimate of 1.50 (web appendix E). While it is possible that media's influence on smoking initiation has increased dramatically between the time of television's introduction and the 1980s, the period of the earliest studies in the metastudy, the selection concerns our study has attempted to avoid may explain the difference. For example, in the metastudy "treatments" correspond to events such as the respondent recognizing a brand name, seeing advertisements, seeing a movie, or moving from the lowest media exposure quartile to a higher one, each of which deviates significantly from the experimental ideal.

Finally, a number of factors suggest that the estimates in this article underestimate the influence of media. This article has already discussed the potential for attenuation bias due to reporting errors and migration. Additionally, the estimates presented here measure the response to television availability, not exposure. Not all people who gained access to television purchased one immediately. Quantifying this distinction, figure 1 from Gentzkow and Shapiro (2008) shows that in 1950 television penetration from DMAs that gained access to television in 1948 was about .17, leaving most of the population without television two years after its introduction. While those without televisions may still have been influenced by their television-owning neighbors, this limited penetration offers an additional reason to interpret this article's estimates of television's influence as a lower bound.

CONCLUSION

This study makes use of a natural experiment to investigate the relationship between media and smoking while avoiding the selection concerns that trouble existing investigations. These analyses provide consistent evidence that television did affect smoking behavior, particularly for 16- to 21-year-olds, an age group that is already at high risk of smoking initiation.

By offering causal evidence of television's impact on smoking initiation, this study offers robust support for the view that mass media can influence the behavior of individuals in ways that are detrimental to their health. Comparisons of this study's estimates to those in the literature suggests television was more influential than prices on the decision to initiate smoking.

DATA COLLECTION INFORMATION

This study relies on preexisting data that were not collected by the author. The primary data source is the National Health Interview Survey (years 1965, 1966, and 1970), which is administered by the Centers for Disease Control and Prevention. Other preexisting data used in the article include TV entry dates from Gentzkow and Shapiro (2008); 1960 Census data on migration sourced from IPUMS; advertising expenditures for tobacco companies

reported by *Advertising Age*; per-capita consumption in the United States as reported by as reported by "The Tobacco Situation," which is produced by the US Department of Agriculture; and Gallup Polls from 1939 to 1957 on "active smokers."

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